Athletes with Disability

Handbook

ATHLETES WITH DISABILITY COMMITTEE
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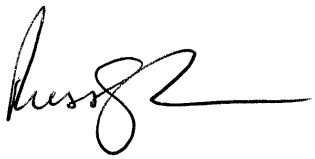
I would also like to thank the many authors who contributed their time and expertise to write the chapters in this manual. Without your help this project would not have been a success.

Sincerely,



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1. RED FLAGS

TOPIC: CHANGE IN MOTOR OR SENSORY FUNCTION

Definition: Deteriorating strength, sensation, a change in bowel or bladder function, or a significant change in spasticity for an athlete with a spinal cord injury are RED FLAGS.

Clinical Concern: For the vast majority of athletes with a disability the underlying disease and resultant neurological function should be considered to be stable. A change in the neurological function is not considered normal. A search for an underlying cause should be performed. Central neurologic problems include: **Spinal cord-** syrinx or syringomyelia, compressive myelopathy (neck or thorax), **Brain-** stroke, tumor, hemorrhage (subdural etc), infection. Peripheral neurologic problems include: **Nerve root** – disc herniation, **Brachial plexus** – trauma or traction, **Peripheral nerve** – compression, fracture.

Pertinent History: The most important thing is to recognize the "new weakness or numbness" and delve further. The team doctor or physio will be a big help.

What was the athlete's **last normal level of motor function**? (See SCI chapter)
Did they have motor function below this level to some extent? How do they feel weaker?
What was the athlete's **last normal level of sensation** and did they have incomplete or altered sensation below this? (See SCI chapter) What areas have they lost feeling in?
What was the normal pattern for the athlete's bowel and bladder? What did they have to do to empty? How has this changed?
Onset? History or trauma – spine, head, plexus? Progression? Does anything make it worse- cough flexion – disc?
Associated symptoms- Fevers? Night sweats? New or progressive spinal pain? Weight loss?
Do they have any bulbar symptoms? – Dysphagia, dysarthria, diplopia, dysmetria (reduced coordination) – as these symptoms could signify a problem in the brainstem. Have they ever had a syrinx? When was the last spinal MRI done if any?

Physical: Document muscle strength in arms and legs.
Arms- C5 – Biceps, C6 - Wrist Extension, C7 – Triceps, C8 – Finger flexion (DIP), T1 - Finger abduction
Legs- L2 - Hip Flexion, L3 – Quadriceps, L4 – Dorsi Flexion, L5 - Toe extension, S1 - Plantar flexion
Check sensation in dermatomes.
Check cranial nerve exam.
Check rectal tone, sensation perianally, ability to bear down (volitional anal contraction), and reflex anal contraction (either anal wink or bulbocavernosus reflex).
Look for a change in muscle stiffness.

Investigations: MRI spinal cord if concerned re change in neurological level. If change in spasticity or muscle tone then look for noxious stimulus (see change in spasticity chapter)
Head imaging (CT) if clinically warranted (trauma/ headache/ decreased LOC)
Labs – infectious workup depending on symptoms, urine, blood etc

Treatment:
Syrinx – refer to a neurosurgeon for opinion. These are often observed with serial imaging. Surgical decompression can be done for individuals losing neurological function in an attempt to stop further progression. If there is an underlying trigger- treat it e.g. infection making spasticity worse (see change in spasticity chapter).

References:

- Consortium for Spinal Cord Medicine Clinical Practice Guidelines, July 2001. Supported by Paralyzed Veterans of America. Copies available at www.pva.org
- Greitz D. Unraveling the riddle of syringomyelia. Neurosurgical Review. 29(4):251-63; discussion 264, 2006 Oct.
- Bergman SB. Yarkony GM. Stiens SA. Spinal cord injury rehabilitation. 2. Medical complications. Archives of Physical Medicine & Rehabilitation. 78(3 Suppl):S53-8, 1997 Mar.

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TOPIC: NEW OR SIGNIFICANT CHANGE IN SPASTICITY

Definition: Spasticity is the increased muscle stiffness often seen in patients with an upper motor neuron insult. It is defined as a velocity dependent increase in muscle tone when the muscle is stretched. It is associated with abnormally brisk reflexes due to the exaggerated muscle stretch reflex, weakness, and often pathological reflexes such as a Babinski reflex. It is due to loss of inhibition of central descending signals from the brain that normally dampen the muscle stretch reflex arch or response. A significant change in spasticity is a RED FLAG and requires the clinician to look for a reason why.

Clinical Concern: Any noxious or painful stimulus from the skin, abdominal organs, bones, joints etc can amplify the muscle stretch response and worsen spasticity. An increase in spasticity can be a sign of infection or injury in athletes with a spinal cord injury or other conditions of the central nervous system. There is often day to day variability in spasticity for athletes often worse first thing in the morning or when lying supine. A persistent history of increased muscle spasms should be investigated. Triggers can be any noxious or painful signal to the spinal cord. However, the athlete may not feel pain if they lack sensation. The clinician's job is to search out and find the offending cause.

Pertinent History: The most important thing is to recognize the "Change in spasticity" and delve further. Go through systems approach. Onset? History or trauma – spine, head? Progression? Associated symptoms- Fevers? New or progressive spinal pain? Weight loss?Any recent changes in medication?

Neurological- has there been any change in the strength or sensation? - suggests a new problem with the spinal cord- syrinx, compression (see Change in motor or sensory function chapter) Do you have a Baclofen pump?
Genitourinary - change in bladder pattern? New incontinence, frequency of catheterizations, blockage of catheter, change in color of urine (pyuria or hematuria)
Gastrointestinal - Change in bowel function (constipated, diarrhea, nausea, vomiting, blood in stool), intraabdominal injury or infection?
Skin – any new sores, ingrown toe nails
Musculoskeletal – any trauma or swelling of extremities
Vascular – change in color or swelling of a leg or arm?
Infectious- look for infection! Fever, chills, rigors, malaise etc

Physical: Look for a noxious stimulus!
- Check vitals – BP/HR/ Temp
- Check for change in strength and sensation if suspicious for problem with spinal cord.
- Check each system Genitourinary – urine, check genitals for infection or injury, Gastrointestinal- bowel for constipation, abdominal exam (may not have pain so need to have high index of suspicion for problem),
- Skin – look for new sores; check nails, MSK- check for new injuries, fractures, joint injuries, or infection. Need to look at legs, and spine particularly carefully, Vascular - any signs of ischemia or deep vein thrombosis, Infection- look for signs

Investigations: Labs – infectious workup depending on symptoms, urine, blood, stool, CSF if warranted
Imaging – to rule out abdominal (impaction, ileus), extremities (occult fracture) as clinically indicated. If suspicious of a new spinal cord problems like a syrinx MRI is the imaging of choice.

Treatment: Treat underlying cause or trigger. Then if spasticity is functionally limiting treat the spasticity with stretching, oral medications usually Baclofen as first line intervention.

References:

- Satkunam LE. Rehabilitation medicine: 3. Management of adult spasticity. CMAJ Canadian Medical Association Journal. 169(11):1173-9, 2003 Nov 25.
- http://www.emedicine.com/pmr/topic177.htm Elizabeth A Moberg-Wolff, MD
- http://www.wemove.org/spa/default.htm

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TOPIC: AUTONOMIC DYSREFLEXIA (AD)

Definition: AD, together with its sudden and severe rise in blood pressure, is a potentially life-threatening condition that affects individuals with a spinal cord injury (SCI) at or above the T6 level. AD results from various noxious stimuli, which in turn trigger dysregulated sympathetic hyperactivity.

Clinical Concern: If unrecognized, and untreated, the severe rise in blood pressure associated with AD can potentially lead to stroke and death.

Pertinent History: An individual with AD presents with a sudden, significant increase in both systolic and diastolic blood pressure *above their usual levels*. Note that individuals with SCI above T6 often have a usual systolic blood pressure of 80-110 mm Hg. A rise of 20 to 40 mm Hg above baseline may be a sign of AD. Other common symptoms include a pounding headache, bradycardia (may be a *relative* slowing of the heart), profuse sweating and/or flushing above the level of the lesion and especially at the face, neck and shoulders, piloerection (goose bumps) above the level of the lesion, blurred vision, nasal congestion, appearance of spots in the visual field and feelings of apprehension or anxiety over an impending physical problem.
**Be aware that these other symptoms may be minimal, or even absent, despite a significantly elevated blood pressure.

Physical: Observe for signs & symptoms mentioned above. Evaluate blood pressure and heart rate.

Investigations: Find the cause/trigger of the AD if possible.

Treatment: Monitor blood pressure and if elevated, sit the individual up if they are supine. Loosen clothing or constricting devices. Quickly survey for an instigating cause starting with the urinary system. If no in-dwelling catheter is in place, insert a catheter. Instill 2% lidocaine jelly (if immediately available) into urethra and wait 2 minutes prior to catheterization if possible. If a catheter is in situ, check for kinks, blockages and correct placement. Remedy any abnormalities. Continue to monitor BP during bladder drainage, as the individual may then go hypotensive.

If AD symptoms persist, suspect fecal impaction. If systolic BP >150 mm Hg, consider pharmacologic management before checking for impaction. Prior to disimpaction, use 2% lidocaine jelly (if immediately available), apply to rectum and wait 2 minutes prior to checking for the presence of stool. Monitor BP during rectal evaluation.

If pharmacologic management required, use an agent with a rapid onset and short duration while other causes of AD are being investigated. Try Nifedipine 5 mg bite and swallow, 2% nitroglycerin ointment (2.5 cm strip) above the level of injury or Captopril 25 mg. Monitor BP frequently.
If no cause for AD identified, consider admission to hospital for pharmacologic management, and further investigations regarding the instigating event.

If AD trigger resolves, monitor for 2 hours to ensure that the BP and heart rate have stabilized. Make sure the individual is able to recognize the signs of AD so they can seek appropriate help if a recurrence develops. Educate individual, family, teammates etc. about AD prevention.

References:

- Consortium for Spinal Cord Medicine Clinical Practice Guidelines, July 2001.
- Supported by Paralyzed Veterans of America. Copies available at www.pva.org
- Blackmer J. Rehabilitation medicine: 1. Autonomic dysreflexia. *CMAJ* 2003;169(9):931-5.

Author: Rhonda Willms, MD, FRCPC – Physical Medicine and Rehabilitation

TOPIC: FRACTURES IN A PARALYZED ATHLETE

Definition: Fractures in paralyzed athletes represent stress fractures (due to repetitive strain) or acute fractures (due to trauma often seemingly minimal trauma). These injuries may occur at a higher incidence in the paralyzed athlete population, as these athletes often have reduced bone density and less protective musculature in the affected limbs.

Clinical Concern: The following complications can arise due to a fracture:
- neurovascular compromise
- autonomic dysreflexia (see AD chapter)
- fat emboli with large long bone fractures / increased DVT risk post-injury
- pressure sores from immobility or attempts at casting (should be avoided)
- further patient disability: loss of independence with mobility / transfers
- Inquire about the functional implications of the fracture (just because they don't walk on it doesn't mean they don't use it)

Pertinent History:
- review the athlete's medical issues prior to competition
- remain attentive during the competition to traumatic events
- Signs of Autonomic dysreflexia: High BP, relative bradycardia, piloerection, flushing, headache, nasal congestion above the level of injury.
- CNS: headache, visual symptoms, confusion, worsened spasticity, altered pain or sensation in the affected areas
- Cardiorespiratory: dyspnea, unexpected diaphoresis, chest pain, (pre)syncope
- MSK: 'sound of bone breaking', pain in the affected limb, altered movement

Physical:
- ABC's, attend to general neurologic and cardiorespiratory status (see above)
- carefully examine below the level of paralysis, as affected areas may have complete anesthesia. The patient could be pain free and unaware of injury!
- inspect limbs on both sides for symmetry, deformity, swelling, erythema, ROM
- assess neurovascular status of the affected limb

Investigations:
- Imaging – be suspicious and image area if concerned with x-ray or if need CT scans at physician's discretion
- bone scans have limited utility in the acute setting

Treatment:
- ACLS guidelines and acute fracture approach: similar for 'able-bodied' athletes
- manage autonomic dysreflexia early; judiciously employ DVT prophylaxis
- considerations prompting urgent investigations / treatment (if answered 'yes'):
- Could this injury affect a patient's mobility, transfers or general function?
- Would a return to play be unsafe?
- involve a Physical Medicine and Rehab specialist early
- important to ask how patient transfers etc. Even though they might not walk they may well weight bear on extremity during transfers.
- if fracture is unstable or going to affect function referral to orthopedic surgeon with experience with SCI or insensate patients is important. Surgical fixation either ORIF or ex fix may be required. Casting usually is discouraged given the high rate of pressure sores in the insensate limb. Consider alternate means of immobilization.

References:

- Braddom's Physical Medicine and Rehabilitation.
- Meiners T. Keil M. Flieger R. Abel R. Use of the ring fixator in the treatment of fractures of the lower extremity in long-term paraplegic and tetraplegic patients. Spinal Cord. 41(3):172-7, 2003 Mar.

Author: Jon Hawkeswood MD

TOPIC: SWOLLEN LIMB IN AN ATHLETE WITH A NEUROLOGICAL IMPAIRMENT

Definition:
The transient abnormal enlargement of a single limb that lacks normal sensation and/or motor function.

Clinical Concern:
A person with a sensory and/or motor impairment of a limb will not present with the usual level of pain that conditions which result in unilateral limb swelling will cause. The differential diagnosis of unilateral limb swelling in this setting includes: fracture, deep venous thrombosis (DVT), heterotopic ossification (HO), infection and impending pressure sore. Fractures may occur due to relatively minimal trauma given the underlying osteopenia present in a limb that has been non-weight-bearing. Patients with a spinal cord injury (SCI) are at highest risk for a DVT in the months immediately following the injury. Heterotopic ossification is the formation of bone in ectopic sites. It has been reported to occur in 16-35% of persons with SCI. The bone formation can lead to severe joint restriction.

Pertinent History:
- Type of neurological impairment (i.e. SCI, traumatic brain injury, spina bifida, etc)
- Degree of sensory impairment
- Degree of motor impairment
- Onset
- Associated features (i.e. fever, increase in spasticity, etc)
- History of trauma

Physical:
- Vital signs including temperature
- Presence of swelling, warmth, erythema, obvious bony deformity, skin breakdown
- Sensory exam
- Motor exam
- Joint ROM

Investigations:
- X-ray
- Doppler ultrasound
- Triple phase bone scan
- CBC, alkaline phosphatase

Treatment:
Management will depend on the diagnosis. Many fractures in paralyzed limbs do not require surgical fixation. DVT is treated with low molecular weight heparin. HO is treated with Etidronate. Occasionally, radiation therapy and surgical removal of extra bone is considered. Maintenance of joint ROM with physiotherapy is important.

References:
- Bryce TN, Ragnarsson KT, Stein AB: Spinal cord injury. In Braddom RL, editor *Physical medicine and rehabilitation,* ed 3, China, 2007, Elsevier Inc, pp. 1285-1349.

Author: Nancy Dudek MD, MEd, FRCPC – Physical Medicine & Rehabilitation

TOPIC: BALCOFEN WITHDRAWAL SYNDROME (BWS)

Definition: BWS is a potentially life-threatening syndrome that appears to be reversible if sufficient dosages of GABAergic drugs are given in time. It presents as an abrupt change in mental status, together with other symptoms such as increased spasticity, pruritis, hyperthermia, myoclonus or seizure, and is instigated by a sudden cessation of Baclofen.

Clinical Concern: The withdrawal syndrome may be misidentified as sepsis, intoxication, psychiatric illness, malignant hyperthermia (MH), neuroleptic malignant syndrome (NMS), autonomic dysreflexia (AD) or a CNS event. If untreated, BWS can lead to multisystem organ failure, DIC, cardiac arrest, coma or even death.

Pertinent History: Individuals with an impairment that involves spasticity (such as spinal cord injury, multiple sclerosis, organic brain dysfunction) may be taking Baclofen (Lioresol) to manage the negative effects of increased tone. This can be in the form of either oral or intrathecal Baclofen. A sudden cessation of the oral routine, or a Baclofen pump system failure/failure to refill pump will initiate the clinical syndrome. All patients will experience "loss of drug effect" such as the reappearance of baseline (or higher) level of spasticity associated with pruritis, anxiety and or disorientation. The more severe symptoms have been well reported and represent the full-blown potentially life threatening BWS.

Physical: Increased spasticity may present in conjunction with fever, tachycardia, hallucinations, diminished level of consciousness, autonomic dysfunction (low or labile BP), myoclonus and/or seizures.

Investigations: Septic work-up. Evaluate CK and renal function to monitor rhabdomyolysis. Can see elevated transaminase levels, DIC and/or evidence of renal and hepatic failure. If the Baclofen is infused intrathecally, evaluation of pump function will be necessary.

Treatment: Initiate life-support measures (ABC's) as needed. Benzodiazepines are helpful in controlling both spasticity and seizures during the BWS. Titrate the dosage until the desired therapeutic effect is achieved. Re-initiation of Baclofen therapy is necessary. If possible, determine the previous dosing regime, and time since cessation of the Baclofen. For those who have been on intrathecal Baclofen, the oral replacement doses may not be adequate, and administration of a CSF bolus has been reported. If possible, reinitiation of the intrathecal Baclofen dosing is best. Supportive measures may be required to manage the other symptoms of the associated syndrome.

References:

- Coffey et al. Arch Phys Med Rehabil Vol 832, June 2002, Abrupt Withdrawal From Intrathecal Baclofen: Recognition and Management of a Potentially Life-Threatening Syndrome

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TOPIC: FEVER

Definition: Fever (also known as pyrexia, from the Greek pyretos meaning fire, or a febrile response, from the Latin word febris, meaning fever) describes an increase in internal body temperature to levels above normal. Fever is most accurately characterized as a temporary elevation in the body's thermoregulatory set-point, usually by about 1–2 °C. The common oral measurement of normal human body temperature is 36.8±0.7 °C (98.2±1.3 °F). This means that any oral temperature between 36.1 and 37.5 °C (96.9 and 99.5 °F) is likely to be normal.

Clinical Concern:
In spinal cord injured patients fever may be due to impaired thermoregulation. For example, below the level of injury there is impaired reflex vasodilation when the set point temperature increases. Due to this, SCI athletes cannot dissipate excess heat predictably. The occurrence of fever is quite high in acute spinal cord injury, however, despite impaired thermoregulation; fever not attributable to infectious and inflammatory etiologies is still not common. The most common reason for fever in SCI is urinary tract infections. Also, respiratory tract infections, soft tissue infections and infections associated with pressure ulcers are common reasons for fever. Thromboembolism and heterotopic ossification are less common reasons.

Pertinent History:
Any history associated with the aforementioned conditions. A history of urinary tract infection, presence of indwelling catheter, pressure sores, skin infections, respiratory difficulty, and thromboembolic risk factors. Also, decreased ROM and bony prominences consistent with heterotopic ossification.

Physical:
Elevated temperature, increased spasticity, fatigue, malaise, swelling, erythema, dyspnea, diaphoresis, decreased ROM.

Investigations:
Diagnostic workup that includes lab work, blood cultures, Urinalysis, Doppler, VQ scan, radiographs.

Treatment:
Dictated by diagnosis. Antibiotics for infectious causes (UTI, Respiratory, pressure ulcers). NSAIDs for HO, Anticoagulation for thromboembolism.

References:

- Incidence, Etiology, and Risk Factors for Fever Following Acute SCI. McKinley W, McNamee, et al. J Spinal Cord Med. 2006; 29(5): 501-506
- Occurrence of fever associated with thermoregulatory dysfunction after acute traumatic spinal cord injury. Colachis, S.C., Otis S. M. American Journal of Physical Medicine and Rehabilitation. 1995

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2. TYPES OF DISABILITY

TOPIC: AMPUTATION

Definitions:
Amputation – removal of a limb or other body appendage
Congenital limb deficiency – complete or partial absence of a limb at birth

Clinical Patterns:
- Lower extremity much more common than upper
- Transtibial and transfemoral levels are most common
- Many congenital limb deficiencies are surgically revised to create a limb with the optimal potential for prosthetic use
- Causes of amputation: diabetes, peripheral vascular disease, trauma, tumour, infection
- Most common causes of amputation seen in athletes participating in Paralympic sport are trauma and congenital limb deficiencies
- Wide variety of prostheses are used in sport
- Athletes may choose to use or not use a prosthesis for sport

Common Clinical problems unique to condition:

1. Residual limb pain
- important to identify the cause of the residual limb pain
- common causes: neuroma, bone overgrowth (including pediatric bony overgrowth, heterotopic ossification & bone spurs), poor prosthetic fit, skin lesions
- treatment often involves prosthetic adjustment
- other treatment depends on the cause but may include: medication, local injections and surgery

2. Phantom pain
- pain felt in the limb that is no longer present
- distinguished from phantom limb sensations which are not painful
- for many people this pain diminishes with prosthetic use and time
- some people will have chronic phantom pain
- non-pharmacologic treatments focus on desensitizing the limb
- neuropathic pain medications and narcotics can be considered
- residual limb pain may exacerbate phantom limb pain and sensations

3. Residual limb skin problems
- wide variety usually associated with friction or pressure problems
- grafted skin is particularly vulnerable to the forces applied in a prosthesis
- often associated with prosthetic fit issues
- include: abrasions, ulcers, epidermoid cysts
- need to consider the environment in prosthesis when choosing a dressing
- dressings should be thin to prevent causing extra pressure inside prosthetic socket
- prosthetic liner material choice is often key to decreasing skin issues

Reference:
- Smith DG, Michael JW, Bowker JH (eds): Atlas of Amputations & Limb Deficiencies – Surgical, Prosthetic and Rehabilitation Principles, 3rd ed. Rosemont, IL, AAOS, 2004.

Author: Nancy Dudek MD, MEd, FRCPC – Physical Medicine & Rehabilitation

TOPIC: SPINAL CORD INJURY

Spinal cord injured athletes have impaired motor, sensory and autonomic function below their level of injury to varying degrees. Common medical issues include spasticity, mechanical and neuropathic pain, pressure sores, autonomic instability (autonomic dysreflexia, orthostatic hypotension, impaired thermoregulation), urinary tract infections, insufficiency fractures and contractures. They may not localize pain upon injury, and careful inspection is needed! Headache, flushing and hypertension may signal a medical issue in an athlete with T6 or higher injury.

Cervical (neck) injuries usually result in **tetraplegia** (formerly quadriplegia):

* **C3 and above**: Typically lose diaphragm function and require a ventilator to breathe.
* **C4:** May have some use of biceps and shoulders, but weaker.
* **C5:** May retain the use of shoulders and biceps, but not of the wrists or hands.
* **C6:** Generally retain some wrist control, but no hand function.
* **C7/8:** Can usually straighten their arms but still may have dexterity problems with hands.

Injuries at the thoracic level and below result in **paraplegia:**

* **T1 to T8:** Lack control of the intercostals, trunk and abdominals. Sitting balance impaired, reduced cough and respiratory capacity. Effects are less severe with lower level of injury. Injuries T6 and higher may be at risk of autonomic dysreflexia.
* **T9 to T12:** Allows trunk and abdominal muscle control, and sitting balance is good.
* **L2 to S5:** Varying degrees of leg weakness, impaired bowel and bladder function.

The **American Spinal Injury Association (ASIA) Impairment Scale**[1] assigns a neurological level and degree of distal preservation of sensory and motor function, based on bilateral assessment of 10 index muscles, 28 sensory points (dermatomes), rectal sensation and voluntary anal contraction. Typically expressed as C, T or L and numeric assignment for neurologic level followed by impairment severity rating A, B, C, D or E. i.e. "C5 ASIA A spinal injury"

Neurologic Level: most caudal spinal segment with entirely normal sensory and motor function.

ASIA Impairment Rating:

A - Complete: no sensory or motor function is preserved in the sacral segments S4/5
B - Sensory Incomplete: sensory but no motor function is preserved below the neurologic level and includes the sacral segments S4/5
C - Motor Incomplete: motor preservation below the neurologic level, and more than half of key muscles below last normal level have a muscle grade less than or equal to 3. Must have some spared sensation or motor S4/5.
D - Motor Incomplete: motor function preserved below the neurologic level, and at least half of key muscles below last normal level have muscle grade > or equal to 3. Must have some spared sensation or motor S4/5.
E - Normal: sensory and motor function are normal. May have reflex abnormalities

Central Cord Syndrome: cervical injury with greater loss of upper limb function compared to lower limbs.
Brown-Séquard Syndrome: cord hemisection; weakness and proprioceptive loss on the side of injury and loss of pain and thermal sensation of the other side.
Anterior Spinal Syndrome: weakness and loss of pain and thermal sensation but preservation of proprioception.
Conus Medullaris Syndrome: injury to the tip of the spinal cord, located at L1 vertebra.
Cauda Equina Syndrome: not really spinal cord injury but injury to the spinal roots below the L1 vertebra. Results in lower motor neuron weakness, bowel and bladder impairments.

References:

- Marino RJ, Barros T, Biering-Sorensen F, Burns SP, Donovan WH, Graves DE, Haak M, Hudson LM, Priebe MM. International standards for neurological classification of spinal cord injury. J Spinal Cord Med 2003; 26(suppl.1):S50-56.

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TOPIC: VISUAL IMPAIRMENTS

DEFINITION: is vision loss(of a person) having reduced vision as to constitute a handicap that constitutes a significant limitation of visual capability resulting from disease, trauma, or a congenital or degenerative condition that cannot be corrected by conventional means, including refractive correction, medication, or surgery. This functional loss of vision is typically defined to manifest with

1. best corrected visual acuity of less than 20/60, or significant central field defect,
2. significant peripheral field defect including homonymous or heteronymous bilateral visual, field defect or generalized contraction or constriction of field, or
3. reduced peak contrast sensitivity either of the above conditions.

"**Legal blindness**" describes arbitrary criteria that are established by national governments (within most developed countries) to determine a citizen's eligibility for government-sponsored benefits and services for people with disabilities. Legal blindness describes neither low vision nor functional blindness, but may include individuals from either category; it does not necessarily indicate a person's *ability* to function. Legal blindness criteria often are described in related legislation, such as The Social Security Act of 1935 (U.S.) or The Blind Persons Act (Canada). The definitions and requirements are common to both countries, and are as follows:

> "Central visual acuity is 20/200 or less in the better eye with corrective glasses or central visual acuity of more than 20/200 if there is a visual field defect in which the peripheral field is contracted to such an extent that the widest diameter of the visual field subtends an angular distance no greater than 20 degrees in the better eye."

VISUAL CLASSIFICATION: Classification is simply a structure for competition in disabled sports - a structure to organize the framework for competition. In abled sports like wrestling, boxing or weightlifting athletes are categorized by weight classes. In disabled sports athletes are grouped in classes defined by the degree of function presented by the disability. Classification is an ongoing process. When an athlete with disability starts competition he is allocated a class that maybe review and changed throughout the athlete's career. The visual classification has two important roles: First to determine the eligibility to compete based on the athlete's visual impairment, second to group visually impaired athletes for competition i.e. to ensure that the athlete competes equitably with other athletes according to the classification system. The visual classification system is based on the official IBSA (International Blind Sports Federation) three class system and refers to visual acuity and / or visual field. The three classes are:

B 1: From no light perception in either eye to light perception but inability to recognize the shape of a hand at any distance or in any direction
B 2: From ability to recognize the shape of a hand up to visual acuity of 2 / 60 and or a visual field of less than 5 degrees
B 3: From visual acuity above 2 / 60 to visual acuity of 6 / 60 and / or a visual field of more than 5 degrees and less than 20 degrees

The attached slide gives an impression about the visual abnormities in blind sports.
Experience shows that in winter sports around 50 % of visually impaired athletes compete in class B 3, 40% in B 2 and 10 % in B 1. This in contrast to summer sports with an almost consistent distribution of athletes in all three classes.

CLINICAL PATTERNS: INCIDENCE OF EYE DESEASES
According to our stocktaking in the Salt Lake City 2002 Winter Paralympics the most frequent eye disease was macular degeneration followed by high myopia, retinitis pigmentosa and optic atrophy. In the Torino 2006 Winter Paralympics we differentiated our results and noticed that the causes of visual impairment of Nordic visually impaired skiers differed from alpine visually impaired skiers:

Alpine Skiing:		Nordic Skiing:	
Macula degeneration	20.0%	Retinitis pigmentosa	30.8%
Trauma	15.5%	High myopia	17.6%

Optic atrophy	13.3%	Macular degeneration	11.7%
Congenital glaucoma	11.1%	Trauma	7.3%
Congenital cataract	8.8%	Congenital cataract	7.3%
Retinitis pigmentosa	6.6%	Optic atrophy	5.8%

Accordingly, in alpine skiing macular degeneration was the most frequent eye disease - a disorder with a normal peripheral visual field. However in Nordic skiing the most frequent eye disease was retinitis pigmentosa - a disorder with a tube visual field. The conclusion is that the nature of interference of the visual impairment has an influence on the discipline of sports visually impaired athletes prefer to go in. In the high speed event alpine skiing the ability to detect motion, peripheral vision and dynamic acuity are more important. Whereas in Nordic skiing athletes are less dependent on the peripheral visual field.

EYE PROBLEMS:
Common eye problems visually impaired skiers seek medical attention for are quite rare and relate mostly to ultraviolet keratoconjunctivitis, infectious conjunctivitis and corneal erosion. Injury from ultraviolet radiation can occur from exposure to high-altitude sunlight with the eyes open without eye protection or due to the ultraviolet sunlight reflected off snow when skiing at high altitudes. Intensive ultraviolet sunlight can lead to ultraviolet keratoconjunctivitis within a short time. Ultraviolet radiation penetrates slightly and causes superficial necrosis in the ocular corneal epithelium. The exposed areas of the cornea and conjunctiva become edematous, disintegrate, and are finally cast off. Symptoms typically manifest themselves after a latency period of 6 - 10 hours. This causes patients to seek ophthalmologic aid to night complaining of "acute blindness" accompanied by pain, photophobia, epiphora and foreign-body sensation. Often severe blepharospasm will be present ("snow blindness"). The symptoms of the "blinded patient" will resolve completely under treatment within 24 - 48 hours. The best prevention are sufficient sun glasses or ski goggles with side protection which totally absorb the damaging ultraviolet wavelengths of the light.

The infectious conjunctivitis usually occurs as a result of bacterial or viral infection due to a direct contact with pathogens. Typical symptoms include severe reddening, swelling of the conjunctiva, purulent or viscous discharge. Bacterial conjunctivitis usually responds well to antibiotic treatment and remits within a few days. In viral conjunctivitis the hygiene prophylaxis is very important. Because the disease is spread by contact patients should avoid direct contact with other peoples.
Corneal erosion follows initial trauma to the surface cornea such as fingernail, branch or contact lens. Immediately after the injury, the patient experiences a severe foreign-body sensation associated with tearing. Symptoms are eyelid swelling, conjunctival injection and blepharospasm accompanied by severe pain. Properly treated, the epithelial defect usually heals within 1 - 2 days.

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- BOLSINGER, A. (2004). Early Education: A Precondition for High Performance in Visually Impaired Athletes. THE PARALYMPIAN, Issue 2 / 2004, p. 11, International Paralympic Committee (Ed.), Bonn.
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- For further references see Bibliography.

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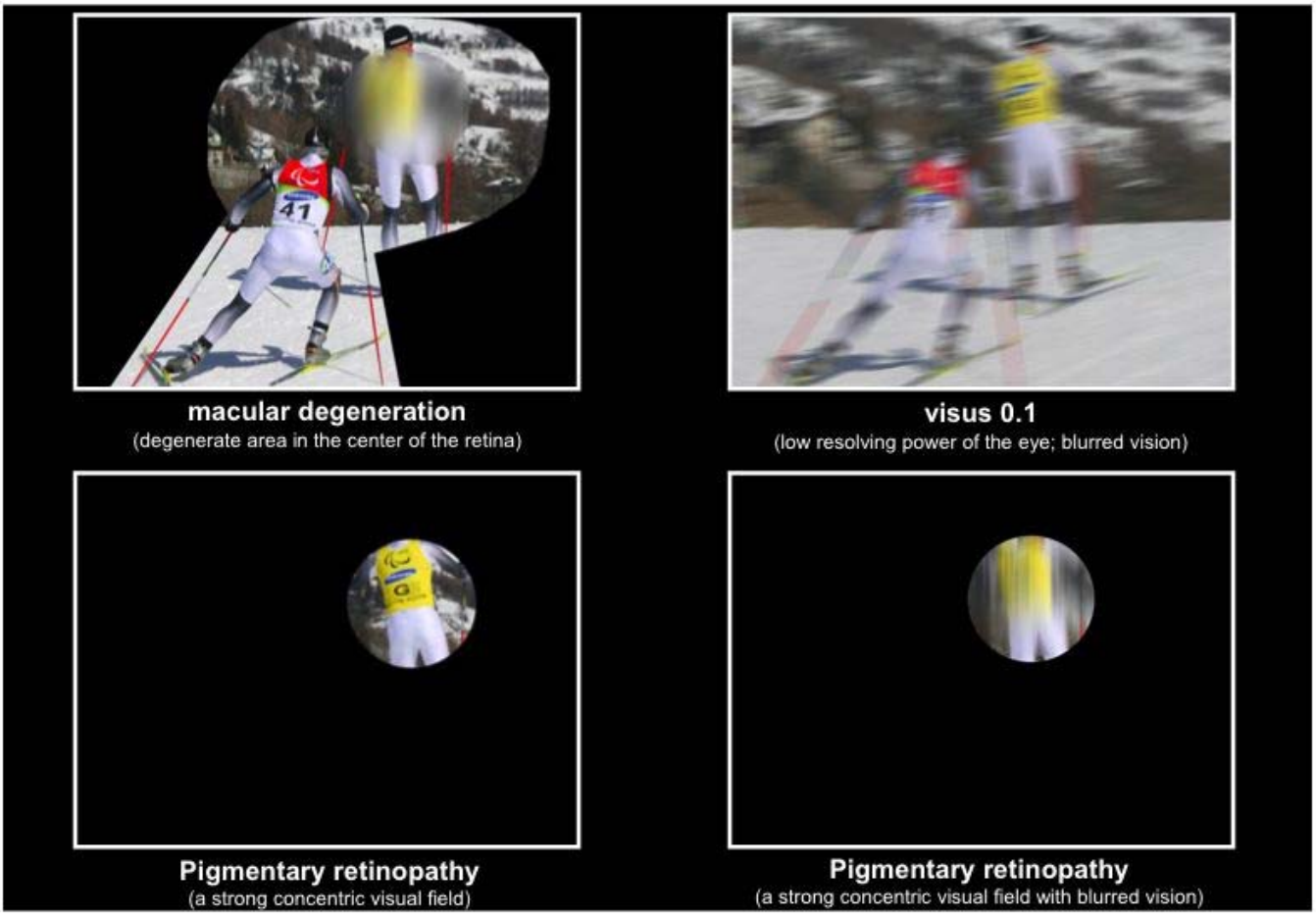
ATHLETES WITH DISABILITIES VISUAL IMPAIRMENT DEMONSTRATION



TOPIC: CEREBRAL PALSY

Definition: Cerebral palsy (CP) describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain.

Clinical Patterns: Traditional classification is according to the nature of the movement disorder (spasticity, ataxia, dystonia, and athetosis) and the anatomic or topographic distribution of the motor abnormalities (hemiplegia, diplegia, triplegia, and quadriplegia). Spastic cerebral palsy is found most commonly in prematurity, and is usually classified topographically with spastic diplegia signifying bilateral leg involvement greater than arm involvement; spastic hemiplegia signifying unilateral arm and leg involvement; and quadriplegia signifying bilateral involvement with arm involvement equal to or greater than leg involvement.

Due to the complexity and poor inter-rater reliability of traditional methods of classification the Gross Motor Functional Classification System (GMFCS) has become increasingly important. It stratifies children based on gross motor ability in different age categories. The GFMCS is not only excellent for initial classification, but also for prognosis. GMFCS levels follow predictable development curves as a child ages.

Common Clinical problems:
Neurological: Most neurological findings occur early. These include hypotonia, poor feeding and motor delay. Persistence of primitive reflexes is also a hallmark of CP and is considered abnormal between 4-7 months depending on the reflex. Commonly, persistence of the asymmetric tonic neck reflex (fencer pose) and Moro (startle) reflex beyond 6 months is an indication to suspect the diagnosis of CP.

With athletes, increasing spasticity may signify pain or high stress. Also, seizures may require further investigation.

Musculoskeletal: The long term affects of disordered muscle tone, control and balance have extensive musculoskeletal sequelae in CP. The most obvious are abnormal gait patterns, poor walking efficiency and speed, kyphosis scoliosis, rotational deformities, hip subluxation and contractures. This leads to long term issues with pain, abnormal bone growth and early OA.

Most children with CP who can sit by the age of 2 years or crawl by 30 months will eventually ambulate independently or with a walking aid. As an ambulatory child with CP grows and gains weight and muscle mass they are often no longer able to tolerate the high biomechanical stresses of ambulation and prefer wheelchair mobility. Once non-ambulatory, scoliosis can progress more quickly and respiratory function can become compromised. Surgical intervention is often necessary in curves greater than 60 degrees.

Systemic and Associated Medical Conditions: Forty percent or more of patients with CP will have associated visual problems. Retinopathy of prematurity, strabismus, esotropism, exotropism, and visual field deficits are common. Hearing impairments, especially in CP associated with TORCH infections should be assessed early. Malnutrition, aspiration and hypoxemia caused by oral motor, gastroesophageal dysfunction and reduced bowel motility can have long term effects on growth, overall health and, ultimately, mortality. Urinary symptoms including frequency, incontinence, and difficulty urinating are common in CP. Seizure disorders occur in up to 33% of patients and are associated with higher severity of CP involvement. Osteoporosis and increased risk of pathological fracture is universal in non-ambulatory CP patients and supplementation with Calcium, Vitamin D and early bone density monitoring should be implemented.

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TOPIC: SPINA BIFIDA

Definition: Neural tube defects resulting from failure of the neural tube closure; defects occur between 25 and 28 days after conception.

Clinical Pattern: Three categories: Spina bifida occulta, meningocele and spina bifida cystica (myelomeningocele). The most common location of the malformations is the lumbar and sacral areas. Myelomeningocele (MMC) is the most significant form and leads to disability in most affected individuals. Spina bifida and myelomeningocele are usually used interchangeably.
The majority of patients with myelomeningocele also have hydrocephalus and Chiari II malformations.

Etiology: The cause of NTDs is unknown. It is thought to be mutlifactorial; including chromosomal disorders, environmental exposure to valproic acid, carbamazepine, maternal diabetes mellitus, and folic acid deficiency.

Common Clinical Problems unique to this condition:

1-*Neurological -* Neurologic deficits depend upon the level of the lesion. In most cases, the entire spinal cord distal to the lesion is nonfunctional. The deficits usually are severe, resulting in complete paralysis and absence of sensation. The bladder and bowel are affected in nearly all patients, resulting in urinary and fecal incontinence (neurogenic bladder and bowel). The majority of patients with MMC have hydrocephalus (obstruction of fourth ventricular outflow or flow of CSF through the posterior fossa due to Chiari malformation or aqueductal stenosis. Shunting is needed immediately in most patients. Brain stem dysfunction due to the Chiari malformation occurs in some patients with MMC. This may result in swallowing difficulties, vocal cord paresis and has a high mortality rate. Strabismus and facial weakness can also occur. Hydromyelia (dilatation of the central canal of the spinal cord) and tethered cord syndrome that results from surgical adhesions which can tether the cord to the low lumbar or sacral region may also occur. Scoliosis occurs in most cases with MMC who have lesions above L2 and is uncommon when the lesion is below S1.

2-*Musculoskeletal -* Deformities result from congenital skeletal anomalies, unbalanced muscle action around joints and fractures. Hip dislocation and feet deformities occur in 24 to 50%. Management techniques include casting and corrective appliances, surgical procedures on soft tissue and bone, and the use of orthoses. Fractures of the lower extremities occur in 30%. They may develop without traumatic injury or during forceful physical therapy. Factors that increase the risk of fracture include sensory loss of the legs, osteopenia, nonambulation, foot arthrodesis, and higher level of paralysis. A fracture should be strongly suspected when a patient presents with a red, warm, and swollen limb which confused with cellulitis or osteomyelitis.

3-Systemic and Associated Conditions

Pressure sores: develop on the sacrum, buttocks, back, and feet. Children with MMC are susceptible to burns because they lack sensation in their lower extremities. Deep ulceration especially over bony prominences and beneath orthotic devices should be evaluated for evidence of osteomyelitis. Factors contributing to skin breakdown include; increased pressure, limited mobility, overweight, infection, trauma, poor circulation, lack of sensation and fecal and urinary incontinence.

Latex allergy: Many MMC children have allergic reactions to latex. Latex products should be avoided.

Endocrine disorders: Children with MMC have reduced development of the lower limbs and spine, complex central nervous system abnormalities and hypothalamic-pituitary dysfunctions, including central precocious puberty and growth hormone deficiency. Obesity is also very common among children with MMC.

Psycho social: children with MMC have lower IQ scores. Verbal IQ is usually higher than performance IQ (cocktail party personality). Verbose but irrelevant conversation is often described. These children also have difficulty in math and visual perceptual tasks than their able-bodied peers do. They are less adaptable, more withdrawn, more distractible, less attentive, and less predictable. Stimulant medications (Ritalin) are often prescribed for these children.

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TOPIC: TRAUMATIC BRAIN INJURY

Definition: Traumatic brain injury is an insult to the brain, not of a degenerative or congenital nature but caused by an external physical force, that may produce a diminished or altered state of consciousness, which results in an impairment of cognitive abilities or physical functioning. It can also result in the disturbance of behavioral or emotional functioning.

Clinical Patterns:
Traumatic brain injury may be initially categorized as mild, moderate or severe based on prognostic indicators including the Glasgow Coma Scale Score (Severe ≤8) or the duration of Post Traumatic Amnesia (PTA). TBI can manifest clinically as impairment of physical, cognitive or behavioural functioning. Outcome following TBI is rated using the Glasgow Outcome Scale which indicates the level of independent functioning that can be influenced by physical, cognitive or behavioural factors. Patients may present with marked physical deficits due to the central nervous system injury which may result in spasticity, weakness, ataxia or balance impairment. Impaired cognition and behaviours may affect personal interactions where there may be more of a tendency for impulsivity or emotional volatility.

Common Clinical problems unique to condition:

1. Neurological

Post traumatic seizure disorder and need for anti-seizure medications
Hydrocephalus – may have ventriculo-peritoneal shunt
Spasticity
Ataxia
Aphasia
Dysarthria
Visual Field Deficits
Cognitive impairments
Behavioral impairments

Cognitive impairments due to traumatic brain injury may or may not be apparent. They can include problems with memory, attention, information processing speed and visuospatial abilities. Executive functions such as planning, problem solving and multi-tasking may also be impaired.

Behavioral impairments may also exist post brain injury including impulsivity, poor self-monitoring, irritability and a decreased ability to control anger.

2. Musculoskeletal

Often may have residual musculoskeletal impairments from multiple traumas such as pain and deformity post fracture.

References:

- Boake C, Francisco GE, Ivanhoe CB, Kothari S. Brain Injury Rehabilitation. In Physical Medicine and Rehabilitation, Second Edition. Ed: Braddom RL. WB Saunders, Toronto. 2000.

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TOPIC: MULTIPLE SCLEROSIS

Definition: MS is a central nervous system autoimmune demyelinating disease likely triggered by infectious agents in patients who are genetically susceptible

Clinical Patterns: The clinical patterns generally identified include:

1. Relapsing-remitting (60%)
   Most relapses (discrete episodes of neurological deterioration) occur in the first 5-10 years and the relapse rate decreases as the disease progresses
   Most will become secondarily progressive eventually
2. Primary progressive (20%)
   Disease is progressive from the outset
3. Benign (20%)
   Occasional relapses without real disease progression
4. Some also include a 4th type, Malignant
   5% of patients die within 5 years

Common Clinical problems unique to condition:

1. Neurological

- weakness
- sensory loss
- spasticity
- ataxia/incoordination
- subcortical dementia

2. Musculoskeletal

- limb contractures secondary to weakness and spasticity
- musculoskeletal (and neuropathic) pain
- gait deviations with resultant back and hip pain syndromes

3. Systemic

- severe fatigue
- heat intolerance

References:

- Fox et al. Multiple sclerosis: Advances in understanding, diagnosing and treating the underlying disease. Clev Cli J Med 2006: 73 (1): 91-102.
- Kraft GH. Rehabilitation principles for patients with multiple sclerosis. J Spinal Cord Med. 1998; 21(2): 117-120.

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3. COMMON MEDICAL PROBLEMS

TOPIC: SPASTICITY

Definition: As part of the upper motor neuron (UMN) syndrome, spasticity is a velocity dependent resistance to passive movement, and is associated with weakness and increased reflexes. “Spasms” are the involuntary movements that may be triggered by touch or movement.

Clinical Concern: An increase in spasticity may reflect the presence of a noxious stimulus. This may be traumatic in nature (focal injury), systemic (underlying infection such as a UTI) or pharmacologic (missing a dose of an antispastic agent). Some spasticity may be beneficial in maintaining muscle bulk, in providing functional resistance during activities such as transfers or weight bearing and in improving venous return. Spasticity in excess however, may increase risk for falls or skin breakdown (shear, friction or direct trauma), may increase pain and can reduce stability/balance. Increases in spasticity may even affect respiratory function (increasing the restrictive pattern of air flow) or sphincter tone. A sudden decrease in spasticity may indicate a medication error (too much of oral or intrathecal Baclofen, excess benzodiazepine, tizanidine etc.) or if focal, may reflect a peripheral nerve injury. Drugs such as cocaine or amphetamines may increase tone.

Pertinent History:
By definition, an individual with spasticity has an UMN (at or proximal to the level of the alpha motor neuron) impairment. If a change in spasticity is identified, inquire about medications or drugs. **Note that sudden cessation of Baclofen (Lioresal) may lead to a potentially life-threatening syndrome. Ask about any trauma history, infection symptoms, skin breakdown, symptoms of autonomic dysreflexia (AD) or other systemic illness. Inquire about any change to bowel or bladder function.

Physical:
Check vital signs to rule out autonomic dysreflexia (hypertension with relative bradycardia) in individuals with a spinal cord injury at or above T6. Observe if the spasticity increase/decrease is focal or generalized. Evaluate skin for any breakdown and look for any areas of trauma such as swelling, bruising, redness or deformity (in sensory impaired individuals, the increase in spasticity may be the only sx they “feel”). CNS: level of consciousness. H&N: signs of AD, lymph nodes. RESP: air entry/respiratory excursion, atelectasis, consolidation, effusion. CVS: signs of DVT, murmur, vascular sufficiency. ABD/GU: bowel sounds, rigidity, masses, catheter function, scrotal swelling, prostate inflammation, rectal fissures or hemorrhoids.

Investigations:
As guided by history and physical exam, lab work and cultures to evaluate for sepsis, imaging for trauma or systemic illness. Evaluate Baclofen pump function if indicated. Tox/drug screen.

Treatment: If possible, remove the underlying noxious stimulus and perpetuator of the change in tone. Identify and manage the trigger. Treat the symptoms of spasticity as required, changing doses gradually if possible. If respiratory function is impaired because of unmanageable trunk/chest wall tone, benzodiazepines with respiratory function monitoring may be required. If concerns regarding Baclofen pump function arise, seek assistance as needed.

References:

- Satkunam LE. Rehabilitation medicine: 3. Management of adult spasticity. CMAJ Canadian Medical Association Journal. 169(11):1173-9, 2003 Nov 25.

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TOPIC: NEUROPATHIC PAIN

Definition:
Neuropathic pain is pain caused by a lesion of the nervous system. It can be central (examples are spinal cord injury, multiple sclerosis, head injury) or peripheral (examples are diabetic neuropathy, amputation, nerve injury). Neuropathic pain can be spontaneous (continuous or intermittent) or stimulus-evoked. Stimulus-evoked pain can be allodynia (pain evoked from a nonpainful stimulus) or hyperalgesia (increased pain from a nonpainful stimulus).

Diagnosis:
The diagnosis of neuropathic pain is based primarily on history and physical examination. History should include obtaining sensory description, temporal variation, functional impact and previously attempted treatments. Common sensory descriptors for neuropathic pain include hot, burning, sharp, stabbing, tingling, prickling and pins and needles. Temporal variation often involves worsening of symptoms at the end of the day. Functionally, neuropathic pain can affect athletic performance, sleep, mobility, self care and mood. Physical examination should include any evidence of allodynia or hyperalgesia. Other causes of pain should also be assessed on physical examination.

Investigations may include electromyography/nerve conduction studies (example is nerve compression), CT/MRI (example change in ASIA score in spinal cord injury patients), three phase bone scan (complex regional pain syndrome) or lab tests (B12, thyroid, glucose).

Treatment:
Treatment for neuropathic pain can be divided into non-pharmacological and pharmacological. Non-pharmacological treatments may include exercise, transcutaneous electrical nerve stimulation, percutaneous electrical nerve stimulation, graded motor imagery, cognitive behavioral therapy, psychotherapy or tactile stimulation. Pharmacological agents can be topical (Capsaicin or Lidocaine) or oral (anti-depressant, anti-convulsant, opioids or cannabinoids). Anti-depressants used to treat neuropathic pain include Tricyclic (Amitriptyline, Nortriptyline) and less effective but also used selective serotonin reuptake inhibitors and serotonin noradrenalin reuptake inhibitors. Anti-convulsant treatments include Carbamazepine, Phenytoin, Gabapentin and Pregabalin. Adverse side effects should be considered when prescribing anti-convulsants. Opioid analgesic and opioid/mixed serotonin-noradrenalin reuptake inhibitors are also used to treat neuropathic pain. Cannabinoids have been found to play a role in the treatment of neuropathic pain. When treating neuropathic pain for athletic events, consideration of substances considered banned or pre-approval should be considered.

References:

- Gilron, Ian. Peter Watson, Catherine Cahill, Dwight Moulin. Neuropathic pain: practical guide for the clinician. CMAJ. August 1, 2006, 175(3), p. 265-275.
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TOPIC: ORTHOSTATIC HYPOTENSION

Definition: A decrease in systolic blood pressure (BP) of 20mmHg or more, or a reduction in diastolic blood pressure of 10mmHg or more, upon the change in body position from a supine position to an upright posture, regardless of the presence of symptoms. There are a number of changes that occur after SCI that can mitigate the severity of OH, including the level and the severity of SCI (higher lesions usually worse), recovery of spinal sympathetic reflexes, development of spasticity and increased muscle tone, and changes in the renin-angiotensin system.

Clinical Concern: The pooling of blood in the lower extremities and decrease in blood pressure results in a reduction in cerebral flow which presents as a number of signs and symptoms. OH can be asymptomatic up in 40% of individuals with SCI. Orthostatic hypotension can significantly impair the quality of life of individuals with SCI. In athletes this condition could also interfere with physical activity and alter the athletic performance. Therefore, early recognition and timely management of cardiovascular dysfunctions in this population are crucial.

Pertinent History: Signs and symptoms of OH are similar to those in able-bodied individuals. Presence of the following symptoms and signs could suggest the presence of OH in spinal cord-injured individuals: light-headedness, dizziness, blurred vision, dyspnea, restlessness, fatigue, weakness and episodes of syncope.

Other non neurogenic causes of OH should be considered. These include low plasma volume/dehydration, hyponatremia, infection, new medications and cardiovascular deconditioning due to bed-rest. Medications that could contribute to development of OH: beta-blockers, diuretics, and tricyclic antidepressants.

Physical examination/Investigations:Vitals Resting blood pressure and heart rate in lying and sitting or standing Blood & urine samples + pregnancy test. Consider an ECG. Remember pain may be absent.Look for any cause of low blood pressure – dehydration, infection, trauma (long bone fractures)

Treatment: There are both pharmacological and non-pharmacological measures that are recommended. But first rule out an underlying cause is important especially if this is a new symptom. Medications and high volume, high salt diets can cause supine hypertension and this needs to be watch for.

Non Pharmacologic Treatments - can be tried immediately include: crossing the athlete's legs and bending forward, leg compression stockings, abdominal binder. Other non-pharmacological treatment options cited in the literature includes the regulation of fluids and salt intake, as well as electrical muscle stimulation. The studies used salt and fluid regulation in combination with other pharmacological interventions and thus, the effects of salt and fluid regulation cannot be determined. Recent evaluation of Evidence Based Practice for management of OH in spinal cord jury (SCIRE 2008) reported following: No evidence exists on the effect of salt or fluid regulation alone for OH management in SCI. There is conflicting evidence that elastic stockings/abdominal binders have any effect on cardiovascular responses in individuals with SCI however this is common practice. There is level 2 evidence from randomized controlled trials that FES is an important treatment adjunct to minimize cardiovascular changes during postural orthostatic stress in individuals with SCI and this can be used by the athlete long term. There is level 2 evidence that simultaneous upper extremity exercises does not improve orthostatic tolerance during a progressive tilt exercise.

Pharmacologic treatments- Midodrine hydrochloride 2.5mg administered orally 2 to 3 times daily (Level 2 evidence (one randomized clinical trail) for use of short acting alpha adreno-mimetic – not on banned list). There are only limited evidence that fludrocortisone (mineralocorticoid), ergotamine (alpha adrenergic receptors agonist), and ephedrine (non-selective, alpha and beta receptor agonist – which is banned) are effective for the management of OH in SCI.

References:

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TOPIC: NEUROGENIC BOWEL

Definition:
Dysfunction of voluntary defecation caused by impairment of the sympathetic, parasympathetic or somatic control of the bowel and anorectal mechanisms.

Clinical Concern:
Fecal incontinence
Constipation
Perforated viscus

Pertinent History:
Past History of gastrointestinal difficulties
Frequency, duration and urgency of bowel movements
Bowel routine - Medications, positioning, time of day, trigger foods, urinary function
Pain - Abdominal or referred pain – NOTE: may not have any pain if are insensate
Spasticity - Increase in total body or abdominal wall
Blood in stool - Melena or Bright red blood
Travel history
Odor of stool

Physical: Vary depending on origin of neurogenic bowel
Vital signs
    Presence of autonomic dysreflexia (see Autonomic Dysreflexia Chapter)
Abdominal exam
    Presence/Absence of bowel sounds
    Palpate for presence of feces
    Assess for acute abdomen
    Hemorrhoids

Investigations:
CBC, electrolytes, BUN, Creatinine, Stool for Culture
    Infectious diarrhea or electrolyte depletion due to diarrhea
Plain abdominal film
    Presence of stool in bowels/obstruction

Treatment:
Constipation
    Manual disimpaction with use of anesthetic jelly
    Adjustment of bowel medications
Diarrhea
    Adjustment of bowel medications
    Replete electrolytes and dehydration
    Referral to surgical specialist if needed
Infectious diarrhea
    Appropriate antibiotics

References:

- Stiens SA; King JC: Neurogenic Bowel: Dysfunction and Rehabilitation. In Braddom RL, editor *Physical medicine and rehabilitation,* ed 2, Philadelphia, 2000, WB Saunders, pp. 637-667.
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TOPIC: NEUROGENIC BLADDER

Definition: Malfunctioning urinary bladder due to neurologic dysfunction or insult emanating from internal or external trauma, disease, or injury.

Clinical Concern:

Urosepsis
Urinary Tract Infection (UTI)
Autonomic Dysreflexia (see Autonomic Dysreflexia Chapter)
Urinary retention
Incontinence
Bladder stones/Renal stones

Pertinent History:

Voiding complaints
- Frequency, urgency, hesitancy, dysuria, incontinence, hematuria

Change in spasticity/neuropathic pain
Method of bladder management
- Indwelling/condom catheter, intermittent catheterization, valsalva voiding

Constitutional symptoms
Fluid intake and output
Autonomic dysreflexia (see Autonomic Dysreflexia Chapter)
Past history of urologic issues- UTI, kidney/bladder stones, BPH, stricture or urethral false passage

Physical: Vary depending on origin of neurogenic bladder- may not have pain if lack sensation

Vital signs
- Blood pressure and signs of Autonomic dysreflexia

Abdominal exam
- Distended bladder
- Abdominal pain
- Flank pain

Spasticity
- Increase in total body or abdominal wall

If indwelling/condom catheter – check if bag appears to be draining well

Investigations:

CBC, Electrolytes, BUN, Cr
Urinalysis, Urine culture and sensitivity
Plain film
- Radioopaque stones

Treatment: Bacteria in the urine does not necessarily require treatment

Symptomatic UTI
- Treat with 10 – 14 day course of antibiotic.

Kidney/Bladder stones
- Hydration
- Pain management
- Urologic referral

Urinary retention/Catheter failure to drain
- In and out catheterization
- Replace indwelling or condom catheter

References:

- Cardenas DD, Mayo ME: Management of bladder dysfunction. In Braddom RL, editor *Physical medicine and rehabilitation,* ed 2, Philadelphia, 2000, WB Saunders, pp. 561-578.
- Linsenmeyer TA, Stone JM, Stiens SA: Neurogenic bladder and bowel dysfunction. In DeLisa JA, Gans BM, Walsh NE, *et al*., editors: *Physical medicine and rehabilitation: principles and practice*, ed 4, Philadelphia 2005 LIppincott, Williams & Wilkins, pp. 1619-1654.

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TOPIC: PRESSURE SORES

Definition: an area of unrelieved pressure over a defined area, usually a bony prominence, resulting in ischemia, cell death and tissue necrosis. Also known as bedsores or decubitus ulcers. Pressure sores can result in autonomic dysreflexia, worsening spasticity, cellulitis, sepsis, osteomyelitis, pyarthroses, anemia, fistula, gangrene and very rarely malignant transformation.

Athlete factors: anemia, infection, fever, ischemia, malnutrition, spinal cord injury, spasticity, impaired sensation and movement, decreased lean body mass, smoking, diabetes, new equipment.
Extrinsic factors: unrelieved pressure, maceration, shear, friction

National Pressure Ulcer Advisory Panel Classification:[1]

Suspected Deep Tissue Injury: Purple/maroon discolored intact skin or blood-filled blister due to underlying soft tissue damage from pressure and/or shear. Area may be indurated, painful, and warmer or cooler than surrounding tissue. May evolve to skin ulceration despite intervention, as this represents early response to damage already incurred.

Stage I: non-blanchable erythema of intact skin. Stage I ulcers can be distinguished from reactive hyperemia in two ways: a) reactive hyperemia resolves itself within 3/4 of the time pressure was applied, and b) reactive hyperemia blanches when pressure applied.

Stage II: partial-thickness loss of skin, including the epidermis and possibly dermis; includes blisters, abrasions, shallow erosions.

Stage III: involves full skin thickness (epidermis and dermis) through to subcutaneous tissue and fat, but not fascia. Bone, tendon and joint are not exposed.

Stage IV: full-thickness skin loss, destruction of underlying fascia, exposing muscle, bone or joint.

Stage X: base of ulceration necrotic (black/yellow) and full depth of ulceration indeterminate

Exam: Assess width, length and depth of wound, associated maceration or induration of adjacent tissue. Quantify odor, drainage, tissue color. Probe for undermining and fistula presence. Rule out sepsis, fever, advancing cellulitis.

Investigations: CBC, albumin, pre-albumin, total protein, iron studies. Wound cultures of questionable value. X-Ray, CT scan, bone scan and bone biopsy if stage IV and osteomyelitis suspected. Blood cultures if septic.

Treatment: Prevent new or further tissue injury by removing cause of pressure. Minimize risk factors, weight shift when seated q15 min, reposition in bed q2 hours, pad bony prominences, check proper cushion inflation. Upgrade degree of pressure reducing cushion and/or mattresses. Keep head of bed <45 degrees. Ensure adequate protein (1-2 g/kg/d) and caloric intake (35-40 kcal/kg/d), consider multivitamin, zinc and vitamin C supplementation.

Non-operative management for Stage I and II includes appropriate wound care, debridement of necrotic tissue, optimize nutrition, relief of pressure, minimized muscle spasticity and smoking cessation. Goal is a moist, clean, pink, granulating wound base and secondary intention healing.

Antibiotics are not routinely indicated. Pressure ulcer infection is suggested by the presence of necrotic tissue, wound edge erythema, purulent discharge, and foul odor. Wound cultures are of questionable value, as they may reflect colonization. Debridement, topical antibiotics and silver-impregnated dressings can help reduce bacterial load and excess drainage. Consider systemic antibiotics only if local wound measures ineffective.

Hospitalization and IV antibiotics for sepsis, cellulitis or osteomyelitis. Consider surgical consult for Stage III and IV ulcers, although some can heal with conservative treatment.

References:

- http://www.npuap.org
- Grey JE. Harding KG. Enoch S. Pressure ulcers. BMJ. 332(7539):472-5, 2006 Feb 25.

Author: David Koo MD, FRCPC – Physical Medicine and Rehabilitation

TOPIC: OSTEOPOROSIS

Definition: Osteoporosis is a disease of bone that leads to an increased risk of fracture. In osteoporosis the bone mineral density (BMD) is reduced, bone micro architecture is disrupted, and the amount and variety of non-collagenous proteins in bone is altered.

Clinical Concern: Osteoporosis is a known consequence of spinal cord injury (SCI) and occurs in almost every patient with a SCI. It manifests itself as an increase in the incidence of lower extremity fractures. The pattern of bone loss seen in patients with a SCI is different from that usually encountered with endocrine disorders and disuse osteoporosis. In general, there is no demineralization in supralesional areas following SCI. Several factors appear to have a major influence on bone mass in individuals with a SCI, such as the degree of the injury, muscle spasticity, age, gender and duration after injury. At the lumbar spine, bone demineralization remains relatively low compared to that of the long bones in the sublesional area. Rapid loss of bone mineral occurs during the first 4 months following SCI. A new steady state level between bone resorption and formation is reestablished about 2 years after SCI. The net effect is a 10-21% bone loss by 10 years. SCI may not only cause bone loss, but also alter bone structure and microstructure. Trabecular bone is more affected than cortical bone in the SCI population.

Pertinent History: Osteoporosis by itself is a sub clinical condition. There are no associated clinical signs or symptoms. Osteoporosis is usually diagnosed incidentally in patients with a SCI when radiographs are taken and reveal the significant bone loss. Osteoporotic fractures are those that occur in situations where healthy people would not normally break a bone; they are therefore regarded as *fragility fractures*. For example, a person with a SCI may fracture their femur during a transfer. Therefore, the focus of history taking should be on determining the person's risk of osteoporosis based on the history of the neurological impairment and a high index of suspicion for fracture even in minor trauma.

Physical: Examination of a person with a SCI who is suspected to have a fragility fracture should focus on looking for the typical signs of fracture such as swelling, erythema and deformity. Pain will be absent in patients with complete SCI but may be there for incomplete SCI. Spasticity often increases in the presence of a painful stimulus and this should also be addressed in the examination.

Investigations: Dual energy X-ray absorptiometry (DXA, formerly DEXA) is considered the gold standard for the diagnosis of osteoporosis. If there is clinical suspicion of a fracture, an x-ray must be obtained.

Treatment:
- Education regarding nutrition, early mobilization, transfer techniques & wheelchair sport safety --Weight-bearing exercises are effective when started within 6 weeks of injury to prevent osteoporosis
- Weight-bearing exercises are ineffective in preventing osteoporosis or restoring bone mineral density past the original time of injury
- Conservative treatment for non-displaced fractures
- Care to prevent skin irritation with splints/casts in insensate limbs
- Surgical intervention may be required for displaced fractures
- Pharmacologic intervention for osteoporosis after SCI includes calcium, phosphate, vitamin D, calcitonin and biphosphonates. However, the evidence for the effectiveness of these medications in the prevention and treatment of osteoporosis remains low

References:

- Osteoporosis after spinal cord injury. : Jiang SD, Dai LY, Jiang LS. Osteoporos Int. 2006 Feb;17(2):180-92. Epub 2005 Oct 11. Erratum in: Osteoporos Int. 2006;17(8):1278-81.
- Osteoporosis and risk of fracture in men with spinal cord injury. M G Lazo et al. *Spinal Cord* April 2001, Volume 39, Number 4, Pages 208-214
- Osteoporosis after spinal cord injury. Gülçin Demirela, et al. *Spinal Cord* Dec 1998, Volume 36, Number 12, Pages 822-825.

Author: Doug Dittmer MD, FRCPC – Physical Medicine & Rehabilitation

TOPIC: SHOULDER PAIN IN THE WHEELCHAIR ATHLETE

Definition: Pain involving the shoulder and/or arm with active shoulder motion resulting in reduced sport performance and challenged activities of daily living such transfers and mobility.

Clinical Concern: Upper extremity injuries are very common amongst wheelchair athletes.(6) The stresses across the shoulder with the demands of wheelchair propulsion make the shoulders the most common upper extremity injury.(9) Wheelchair athletes have a higher frequency of shoulder complaints compared to able-bodied athletes secondary to a higher ratio of shoulder abduction to adduction muscular strength. The position of the shoulder in flexion and internal rotation and muscular imbalances places the rotator cuff at risk for impingement under a reduced subacromial space.(2) Peak shoulder joint loading has been noted to occur when the arm is extended and internally rotated and with higher wheelchair propulsion velocity putting the shoulder joint at risk.(3)

Pertinent History: Most common subjective complaints of athletes with rotator cuff and impingement problems are: pain, difficult overhead activities, nocturnal pain, loss of endurance during activities, deterioration in sporting performance, catching and grinding (crepitus), weakness and stiffness.(5) Associated symptoms such as shoulder clicking, instability, numbness, radiating sharp pains and/or paresthesias in the neck or down the arm into hand would suggest an associated diagnosis. Additional diagnoses to consider during the assessment of these athletes are as follows:

- Bicipital Tendonopathy
- Glenohumeral Instability+/- 2* impingement
- Osteolysis of the distal clavicle
- Labral injuries
- Osteoarthritis of the Glenohumeral Joint
- Osteoarthritis of the Acromioclavicular joint
- Myofascial Pain Syndrome
- Suprascapular Neuropathy
- Cervical Pathology (Facet Joint, Cervical Root, Deg Disc Disease, Syringomyelia/Syrinx)

Physical: An organized assessment with inspection, range of motion, palpation, special tests and neurovascular screen is essential in establishing an accurate diagnosis. A discrepancy between active and passive range of motions, weakness and pain specific motion of specific rotator cuff muscles are suggestive of rotator cuff pathology. Special tests specific for impingement syndrome: forced forward flexion (Neer's) and abduction internal rotation (Hawkin's/Kennedy).(5)

Investigations: The impingement test involves injection of local anesthetic into the subacromial region after a positive impingement signs. A positive response suggestive of an impingement problem is noted with subjective relief of symptoms and reduction of previously demonstrated impingement signs.(5) Standard radiographic x-rays of the shoulder(s) may reveal a high riding humeral head suggestive of rotator cuff disease. Osteolysis of the distal clavicle can be seen on plain x-ray. MRI may reveal a rotator cuff tear.(1) A shoulder arteriogram may increase the sensitivity of MRI alone in establishing potential rotator cuff or labral pathology.

Treatment: In constructing a treatment plan for these athletes, it is important to consider the following factors: 1) cause of the condition, 2) the athlete's level of performance and 3) severity of the problem. The causes of the condition are often related to muscular imbalances and joint forces as a result of the act of wheelchair propulsion and seating position.(2)(7) The athletes' level of performance should reflect some decision making regarding enforced therapeutic rest, however the wheelchair athlete will require good shoulder function for a number of additional basic day to day skills such as weight transfers, transfers and mobility which should be considered with high regard. The severity of the problem would than reflect a decision on a treatment plan in the following into categories: 1) Prevention, 2) Non-operative and 3) Operative. Wheelchair position has been noted to have significant impact on shoulder joint forces and risk for impingement. As a result, seating position should be considered as a preventive strategy for future shoulder health with consideration too sport performance. Well-balanced muscles are vital to prevention and maintenance of these athletes' performance

levels. (2) A 6-month home exercise protocol was effective in decreasing the intensity of shoulder pain that was interfering with functional activity of wheelchair users. (4)(8)

References:

- See Bibliography

Author: Jamie Irvine MD, FRCPC – Physical Medicine and Rehabilitation

4. WINTER SPORTS INJURY PATTERNS

TOPIC: PARALYMPIC ALPINE SKIING

Sport Description: Paralympic alpine skiing consists of 4 disciplines: slalom, giant slalom, super giant slalom (Super-G) and downhill.

Slalom and giant slalom are "technical" events and consist of 2 runs each down different courses. The times from the 2 courses are combined for an overall time. The turns in slalom are tighter and the courses are shorter than in giant slalom.

Super-G and downhill are "speed" events and consist of a single run down a course. Super-G is longer and has bigger turns (at least 25 metres apart) than slalom and giant slalom, but is held on a shorter course than downhill. Downhill has the fewest turns and the highest speeds. It is the only alpine discipline where athletes have training runs to familiarize themselves with the course.

General FIS (Federation Internationale de Ski, ski racing's governing body) rules are followed with a few modifications. Skiers with visual impairments are led through the course by guides using voice or radio signals. For B1 athletes (totally blind), the guide may be in front or behind the athlete. For B2 and B3 athletes (partially blind), the guide must be in front. For the technical events, the guide and athlete must be within 2 gates from each other. For the speed events, they must be within 1 gate.

Classification: The Athlete's race in order depending on their classification: visually impaired, sitting or standing with the visually impaired skiers competing first down the freshest course. Visually impaired athletes are classified according to the severity of their impairment. There are many complex and varied categories for the classification of sitting and standing skiers depending on the nature of their impairment and the equipment that they use.

Special Equipment: Skiers use adapted ski equipment depending on their impairment. Many skiers use modified ski poles with outriggers. These poles have short skis attached to them and help with balance. Athletes with amputations may also use upper or lower limb prostheses.

The sitting skiers use sit-skis and outriggers. Sit-skis have specially designed chairs (sometimes called "buckets") that are attached to the ski with a suspension system.

Type of Injuries: The alpine venues at the Winter Olympics and Paralympics typically record the highest numbers of injuries and the most severe injuries. Some athletes may have chronic injuries from training and overuse. However, some injuries are relatively unique to alpine skiing due to the equipment and speeds involved.

Even with the use of helmets, there is a risk of concussion and traumatic brain injury from impact during a crash. Long bone fractures and spinal fractures with or without spinal cord injuries may also occur. Ligamentous injuries such as torn anterior cruciate ligament injuries are possible. Finally, the use of outriggers may result in injuries to the upper extremities due to falling heavily on outriggers.

Sport Event and Medical Venue Organization: Venue medical organization follows FIS rules and standards. Ski patrollers provide first response to injured athletes with physicians available on course if needed. The chief of patrol and the chief of medical coordinate the medical response to an injured athlete.

References:

- Alpine Canada Alpin website **www.canski.org**

Author: Andrea F. Townson, MD, FRCPC- Physical Medicine and Rehabilitation

TOPIC: ICE SLEDGE HOCKEY

Sport Description: Sledge Hockey is basically the same sport as standard ice hockey as defined by the International Ice Hockey Federation (IIHF). The only difference is that the athlete competes on/in a sledge. A sledge is the European term for sleigh or sled. The same rules, rink size, offside lines, goal size etc. as conventional ice hockey apply.

The athletes are seated in the sledge "bucket" and use unique hockey sticks. These sticks are specially made for sledge hockey and are about 1 metre in length and consist of a curved blade at one end and an "ice pick" at the other. The hockey puck is regulation size. The blade is used to propel the puck towards the goal and the pick is used to propel the sledge. The hockey shot is usually taken using one arm.

Classification: To participate in IPC Official Competitions and IPC Sanctioned events, each player of a participating team must have a permanent disability that would normally preclude that athlete from playing regular competitive ice hockey. Determination of minimum disability and appropriate classification shall be based on accredited medical conclusion and observations by a tripartite Tournament Classification Committee composed of an IPC recognized physician, an athlete/player representative from the same team as the player being classified, and an IPC Sports Assembly appointed Technical Representative, all of whom should be familiar with the sport.

Special Equipment: Standard ice hockey equipment as approved by the IIHF. Special equipment includes a sledge mounted on two blades of various widths depending on athlete's preference and skill. The sledge must allow the puck to pass underneath.

Type of Injuries: Although a very aggressive sport, the athlete is well protected from most injuries. The commonest injuries seen are related to trauma from the ice pick on the end of the hockey stick. This usually involves lacerations. Due to the high energy contact between sledges, head injuries and spinal cord injuries must be anticipated, although very rare seen. Long bone fractures can occur, usually following sledge collisions. Provided the equipment worn by the athlete doesn't fall off or fail, injuries in sledge hockey are relatively rare and when they occur are usually minor. Exceptions do occur as in any sport.

Sport Event and Medical Venue Organization: Venue medical organization follows IIHF rules and standards. Team trainer and medical staff are first response to injured athletes with physicians available in the venue if needed. The venue medical manager or representative coordinates medical evacuation if required in cooperation with team's medical staff.

Reference:

- www.paralymic.org

Author: Richard Beauchamp MD, FRCPC - Pediatric Orthopedic Surgeon

TOPIC: PARALYMPIC NORDIC/BIATHALON SKIING

Sport Description: Paralympics Nordic consists of cross-country and biathlon. The cross-country skiing event is split into two separate races of classical and free technique. Cross-country skiers in men's and women's categories compete in a short, middle, and long distances, ranging from 2.5 – 20 kilometers.

Biathlon consists of cross-country skiing and rifle shooting. Paralympic athletes shoot in the prone position only. Short-distance biathlon skiers ski a 2.5 km loop three times with two stops for shooting. Each episode of shooting includes five shots at a metal target approximately ten meters away. The target consists of a bull's eye 15 mm in diameter. If a bull's eye is missed, the competitor is required to skate a 150-metre penalty loop for each missed shot. Long distance biathlon competitors ski a loop five times and stop four times for shooting. A one-minute time penalty is received for each miss on the shooting range. This is added to the overall skiing time. In the blind category, the poles of the athlete are taken by the guide and the guide waits in a designated area while the athlete is shooting.

Both cross-country and biathlon races have start formats every 30 seconds. IPC utilizes a Nordic percentage system in order to equalize skiers within each category as a disability time handicap. This percentage is applied to each skier's final time and the skier with the lowest calculated time is the winner. In the relay event in cross-country, each team member skis one loop. Teams consist of skiers from different categories, but the overall total percentage for each team is equal; therefore, no time calculation is required and the first team to cross the finish line wins.

Classification: There are three medal categories, which include visually impaired, sit-skiing, and standing. In the visually-impaired category athletes are classified based on light perception, ability to see shapes, and visual field. Individuals in the standing category are classified based on degree of disability. Sit skiers are classified based on sitting balance and/or disabilities in the lower limbs.

Special Equipment: Skis consist of both classical and skate. Sit-skis vary greatly, depending upon the patient's ability to sit. Sit-skis can be fully enclosed with a shell and/or open utilizing a strapping system. Skis are attached to the undersurface of the sit-ski. All individuals in the standing category use two skis, but variance in the use of two poles to no poles can occur Most visually impaired individuals are required to use a guide (exception B3 may use a guide).

For biathlon event, an acoustic system for shooting is utilized by visually-impaired skiers. Differing tones, as the rifle is aimed at the bull's eye occur, with increasing frequency of pitch occurring when a bull's eye is achieved. During the biathlon, the use of a shooting sling is permitted. Rifle support is allowed in some classes.

Type of Injuries: There are minimal statistics available with regards to injuries related to IPC Nordic and Biathlon events. The Monashee Accident Research Centre reports the most common cross-country skiing injuries include sprains, twists, fractures, and bruises. The most frequently injured body region includes knees, arms, hands, and ankles. Altered cross-country skiing technique and overuse is also reported as a cause for injury. Due to the altered equipment in sit-skiing, increased frequency of upper extremity injuries (For example rotator cuff injuries) is likely. Increased lordosis of the low back during poling in some individual's, may lead to increased back pain. Increased frequency of falls is likely due to altered technique and visual impairment.

Sport Event and Medical Venue Organization: Venue medical organization follows IPC rules and standards. Ski patrollers provide first response to injured athletes with a physician available on course or in a medical room. Chief of medical coordinates the medical response to the injured athlete.

References:

- Monashee University Accident Research Centre; IPC Nordic Skiing Biathalon and Cross-Country Skiing Rules and Regulations

Author: Heather A. Underwood, MD, FRCPC, MSc

TOPIC: WHEELCHAIR CURLING

Sport Description: The sport of wheelchair curling began in Europe in 1998. The first World Championships were held in 2002 in Sursee, Switzerland. Wheelchair curling had its first Paralympic Games debut at the 2006 Torino Paralympic Games. The sport of wheelchair curling is quickly gaining popularity and is currently being practiced by athletes in over 20 countries. [1]

- It is governed and played according to the rules of the World Curling Federation (WCF) in which all the same rules apply as able-bodied curling with only minimal modifications. The same sheets of ice, stones (weighing no greater than 44 lbs and no less than 38 lbs) and scoring system used by able-bodied athletes are used in wheelchair curling. [1]
- Each wheelchair curling team must be comprised of mixed gender. Each team is composed of four players, each player playing two stones and playing each stone alternatively with their opponent.
- All players must deliver the stone from a stationary wheelchair which is placed so that the stone is delivered from the centre line. Each player releases his or her stone before the hog line (line located 21 feet from center point of the house (round scoring area, 12 feet in diameter, with concentric circles 1, 4, 8 feet in diameter)). Delivery of the stone may be undertaken by the conventional arm/hand release or by the use on an extender cue/delivery stick. [1]
- No sweeping is permitted in wheelchair curling.[1]
- Scoring is simple. The team receives one point for each of their stones that are within the house and are closer to the center than any of the opposition's stones. The team that scores throws first in the next end. Each game is played over 8 ends and each team has 68 minutes plus one-minute time out for each game. When extra ends are required the clocks will be reset and each team shall receive 10 minutes of playing time for each extra end.[1]

Classification: Wheelchair curling is restricted to individuals with significant impairments in lower leg/gait function (i.e. Spinal cord injury, cerebral palsy, multiple sclerosis, double leg amputation), who usually require a wheelchair for daily mobility. More specifically those who are non ambulant or can walk only very short distances. Authorized sports classifiers shall make determination of minimum disability and appropriate classification. [1, 2]

Special Equipment: Any wheelchair is acceptable. The athlete's feet cannot touch the sheet of ice and need to be on the wheelchair footrest. The extender cue/delivery stick uses a lightweight plastic bracket that threads onto a standard adjustable broom handle. The hinged bracket fits over the top of the rock handle allowing the rock to be pushed forward and released smoothly with rotation. [1]

Type of Injuries: The IPC injury survey in Torino 2006 revealed no reported injuries with wheelchair curling athletes. [3] Wheelchair athletes are at particular risk for overuse injuries of the shoulder complex due to repetitive use of the upper extremities for propulsion. In wheelchair curling extension and quick forward flexion of the neck and upper back and extension of the arm for delivery of the stone using an extender cue/ delivery stick can possibly lead to musculoskeletal injuries (tendinopathy, sprains, and strains). Issues with worsening spasticity secondary to noxious stimuli such as distended bowel or bladder in upper motor neuron conditions or autonomic dysreflexia in T6 and above complete spinal cord injuries may be observed.

Sport event and medical venue organization: Venue medical organization follows IPC rules and standards. The athlete medical team comprises of 1 physician, 1 physiotherapist and 2 first aid responders responsible for athlete care on field of play and in the medical room. The venue medical officer (VMO) is responsible for the coordination of the medical team, communication of medical issues with venue management, ambulance services and reporting to the chief medical officer (CMO) or representative.

References:

- www.paralympic.ca: Canadian Paralympic Committee: Wheelchair Curling
- World Curling Federation (WCF) rules for wheelchair curling. www.worldcurling.org
- Webborn et al. IPC Injury Survey Torino 2006. The Paralympian. Issue 2: 11; 2007.

Author: Dr. Rajiv Reebye BSc.Pharm BMedSci BMBS FRCPC

5. PARALYMPIC MEDICAL BAG

SUGGESTED ADDITIONAL SUPPLIES
MEDICAL BAG

Airway Kit:

- Disposable Oral Screw
- Disposable Laryngoscope Blade
- 7.0 mm Endotracheal Tube
- #3 Oral Airway
- Reusable Ambu Bag
- Laryngoscope Light
- #14 I.V. Catheter
- 6 ml Syringe

Bandage Kit:

NON STERILE:

- 3" & 6" Tensors
- Safety Pins
- Soft Skin Protection
- Large Zip-Style Baggies
- Bandage Scissors
- 1" Plain Tape
- 2" Waterproof Tape
- 1" Micropore Tape

STERILE:

- Anchor Band-Aids
- Kling Bandage
- 4" x 4" Gauze
- SPLINT:
- Reusable SAM Splint
- Cotton Sling
- Malleable Finger Splint
- Foil Blanket

Wound Kit:

- Disposable Laceration Kit
- 5-0 & 3-0 Non Absorbable
- 6 & 12 cc Syringes
- Steristrips
- Betadine Prep
- #15 Scalpel
- 4-0 Absorbable Suture
- 7.5 Sterile Gloves
- #25 x 1.5 Needles
- Betadine Scrub Brush

Eye Kit:

- Fox Aluminum Shield
- Fluorescein Strips
- Sterile Q-tips
- Soft Patch Gauze
- Micropore Tape

I.V. Kit:

- Plasma Expander
- Standard Alcohol Swabs
- 3 & 12 cc Syringes
- #18 x 1.5 & #25 x 1.5 Needles
- Primary Line I.V. Tubing
- #18 Intercath
- 1 x 12 Penrose Tubing

Examination Instruments:

- Oto/Opthalmo Scope
- Rectal & Oral Thermometers
- Non Sterile Gloves
- Stethoscope
- B.P. Cuff
- Tongue Depressors

PARALYMPIC MEDICAL SUPPLIES

EQUIPEMENT REPAIR KIT:

Amputee/Brace
- Loctite
- Allen keys: metric up to 12 mm and standard 1/16 to 3/8 inch
- Duct tape
- Shear ban
- Multi bit screwdriver
- Double sided Velcro strips (adhesive hook)
- Fiberglass tape
- Carbon foot plate (Otto Bock) as splint for fractured foot plate

Wheelchair
- Ratchet wrench

DRUG TESTING

- Foley catheter size 14
- B. P. Cuff

WOUND CARE:

In addition to above:
Thin Form dressing eg Allevyn wound dressing (several)
Film dressing eg Tegaderm

BOWEL AND BLADDER KIT:

Dulcolax supps
Senekot
Foley
Lidocaine jelly 1% for cath or bowel check
Glycerine supps
Ditropan
Intermittent catheter (2or 3)
Leg bag

PAIN AND SPASTICITY MEDS:

Gabapentin 300 mg tablets
Baclofen 10 mg tablets

PARA EMERGENCY KIT:

Captopril 25 mg sublingual (don't chew) if BP> 150 after bladder and bowel check. Repeat in 30 min if BP still > 150.
Lidocaine 1 or 2% jelly for catheter or bowel check if patients is dysreflexic
Baclofen 10 mg for withdrawal

6. IMPAIRMENT DISABILITY HANDICAP AND INTERNATIONAL CLASSIFICATION OF FUNCTION

TOPIC: IMPAIRMENT, DISABILITY, HANDICAP & ICF

In 1980, the World Health Organization (WHO) published a document titled, “International Classification of Impairments, Disabilities and Handicaps (ICIDH). This document made the following definitions.

Impairment
- A loss or abnormality of physical bodily structure or function, of logic-psychic, physiologic or anatomic origin

Disability
- Any limitation or function loss deriving from impairment that prevents the performance of an activity in the time-lapse considered normal for a human being

Handicap
- A disadvantaged condition deriving from impairment or disability limiting a person performing a role considered normal in respect of age, gender, social and cultural factors

The new document titled, “International Classification of Functioning, Disability and Health” focuses on functionality as opposed to impairments. It offers new terms to deal with the above concepts.

Bodily Functions
- Physiologic functions of bodily systems

Bodily Structures
- Anatomical parts of the body such as organs and limbs

Activities
- Execution of an action on behalf of an individual

Participation
- Involvement of the individual in a life situation

Environmental factors
- Characteristics of the physical and social world and the attitudes that can have an impact in the individual’s life

Note that the word handicap has been abandoned and therefore the meaning of the word disability has been expanded to cover the restriction of activity as well as the limitation to participation.

References:
- http://www.who.ch/icidh

Author: Nancy Dudek, MD, MEd, FRCPC – Physical Medicine & Rehabilitation

7. CLASSIFICATION

TOPIC: CLASSIFICATION

Classification is simply a structure for competition. Not unlike wrestling, boxing and weightlifting, where athletes are categorized by weight classes, athletes with disabilities are grouped in classes defined by the degree of function presented by the disability.

Traditionally there are athletes who belong to six different disability groups in the Paralympic Movement: amputee, cerebral palsy, visual impairment, spinal cord injuries, intellectual disability and a group which includes all those that do not fit into the aforementioned groups (les autres).

Classes are determined by a variety of processes that may include a physical and technical assessment and observation in and out of competition. The classes are defined by each sport and form part of the sport rules.

Classification is an ongoing process. When an athlete starts competing, they are allocated a class that may be reviewed throughout the athlete's career. Sports certify individuals to conduct the process of classification and these officials are known as classifiers.

Since the 1960's, the development of sport for athletes with a disability has produced the development of classification systems; and this continues to evolve to the present day.

In 2003, the International Paralympic Committee (IPC) developed a Classification Strategy with the overall objective to support and co-ordinate the ongoing development of accurate, reliable, consistent and credible sport focused classification systems and their implementation. The IPC Classification Code is a direct result of recommendations made in this Strategy.

The trend in classification is to move towards sport specific classes rather than being disability specific. This allows for fewer events and ultimately leads to more competitive events at elite levels. Some argue that it is difficult for an athlete with a neurological illness (i.e. cerebral palsy) to compete against more physical illnesses (i.e. amputee). It is felt that there is more variation with neurological signs, such as spasticity, in sport situations.

It is likely that classification will continue to evolve over time.

Author: Dhiren Naidu MD, FRCPC Physical Medicine and Rehabilitation - Senior International Classifier CP-ISRA

8. PRE PARTICPATION EXAM QUESTIONAIRE FOR AN ATHLETE WITH A DISABILITY

PRE-PARTICIPITION EVALUATION FORM FOR AN ATHLETE WITH A DISABILITY

Date of Examination: _____ Reviewing Physician: _____

Identifying Information

Athlete name / identifying code: _____

Gender: _____ Age: _____ Date of Birth: _____

Personal Physician: _____

Address: _____

Phone (H): _____ (W): _____

Emergency Contact Person: _____

Relationship: _____ Phone: _____

Team name / identifying code: _____

Athletic group classification:

1. List Competitive Sports / Disciplines

Sporting discipline	Years involved
a.	
b.	

In the past 12 months, have you been either *sedentary* or *restricted by a physician to limit participation* in sports (including bedrest or modifed activity)? Please explain.
If needed, use the final sheet (page 6) to provide further details for the questions below.

2. List current <u>active</u> medical conditions (full details of disabled sport eligibilty are later on)

Diagnosis/Injury	Date	Treatment	Physician

3. List all previous surgical procedures:

Diagnosis/Injury	Date	Treatment	Physician

4. List all prescription medications you are currently taking:

1.	**4.**
2.	**5.**
3.	**6.**

Please respond to each question with 'Yes' or 'No'. Circle questions you do not know the answers to. Please use space provided on the final sheet (page 6) for explanations of 'yes' responses.

5. General Health Review	**Yes**	**No**
Are you currently taking non-prescription (over-the-counter) medicines, supplements or pills?		
Do you have any medication allergies?		
Have you ever applied for therapeutic use exemption for any medication / supplement?		
Do you have any allergies to pollens, foods or stinging insects?		
Do you have any dietary restrictions (e.g. vegetarian or vegan)?		
Do you smoke?		
Do you drink alcohol beverages?		
Are you happy with your weight?		
Are you trying to gain or lose weight?		
Do you limit or carefully control what you eat?		

6. Cardiovascular Review	**Yes**	**No**
Have you had any episodes of passing out DURING exercise?		
Have you had any episodes of passing out AFTER exercise?		
Have you ever experienced discomfort, pain, or pressure in you chest during exercise?		
Have you ever told you have high blood pressure, high cholesterol, heart murmur, previous heart infection? If yes, please indicate:		
Does your heart race or skip beats during exercise?		
Is there a history of early, unexplained death in your family?		
Has a doctor ever ordered a test for you heart? (for example, ECG, echocardiogram)		
Does anyone in your family have Marfan syndrome?		
Do you have a condition called 'Autonomic Dysreflexia?'		
Has any family member or relative died of heart problems or suddenly before age 50?		

7. Respiratory History	**Yes**	**No**
Do you cough, wheeze or have difficulty breathing during or after exercise?		
Is there anyone in your family who has asthmas?		
Have you ever used an inhaler or taken asthma medicine?		
Have you had pulmonary function tests (if so, please specify the reason)?		

8. Further General Medical Review	**Yes**	**No**
Do you have any issues with prolonged bleeding?		
Have you ever suffered from a deep vein thrombosis or pulmonary embolism?		
Do you have anemia?		
Have you had infectious mononucleosis (mono) within the past month?		

Do have problems with recurrent infections?		
When exercising in the heat, do you have severe muscle cramps or become ill?		
Has a doctor told you that you someone in your family has sickle cell trait or disease?		
Do you have a history of recurrent skin wounds or pressure sores?		
Do you have any rashes or other skin problems?		
Have you been hospitalized for issues surrounding skin breakdown?		
Have you had a herpes skin infection?		
Do you have diabetes?		
Do you have problems with your thyroid gland?		
Were you born without or are you missing a kidney, a testicle, or any other organ?		
Have you had problems with recurrent urinary tract infections?		
Have you ever had abnormal bowel or bladder control?		
Are you concerned about recent bladder disturbances, including pain or bleeding?		
Are you concerned about recent bowel disturbances, including blood or dark black stools?		
Have you ever had a stomach ulcer?		
Have you ever had any type of hernia?		

9. Visual History	**Yes**	**No**
Are you considered blind in one eye or are you missing one eye?		
Do you wear glasses or contact lenses?		
Do you wear protective eyewear, such as goggles or a face shield?		
Do you have any other visual disorders, including colour blindness, reduced vision in one eye, or difficulty with visual perception?		

10. Neurologic and Psychiatric History	**Yes**	**No**
After suffering a blow to the head, have you ever been confused, unconscious or had difficulty with memory?		
Have you ever had a seizure?		
Do you have headaches with exercise?		
Have you ever had numbness, tingling or weakness in your arms or legs after being hit and falling?		
During activity, have you ever experienced headaches, migraines, dizziness, nausea, irritability or unusual fatigue?		
Do you have concerns with your current mood, changes in mood, or level of energy?		
Are you currently experiencing sleep disturbances (including the need for sleeping pills)?		

11. *Female Athletes Only*	**Yes**	**No**
Are have been suffering from irregular periods or menstrual irregularities?		
Is there a possibility that you could be pregnant?		

12. Musculoskeletal History

List previous injuries. Please include sprains, muscle or ligament tears or tendonitis that caused you to miss a game or practice. Give information on Xrays, CT scan or MRI, surgery, injections, rehabilitation, physical therapy, a brace, a cast or crutches. Should you require more room for writing, please use page 6.

	Injury	**Date**	**Imaging**	**Treatment**

1.				
2.				
3.				
4.				
5.				

Musculoskeletal History Continued	**Yes**	**No**
Have you ever been told you have osteoporosis / osteopenia or low bone density?		
Have you ever had a stress fracture or a broken bone from a low impact trauma?		
Have you ever taken prednisone for greater than a one week period?		
Do you currently have any metal in your body as a result of a bone surgery?		

13. Disability Description: Please provide the following details of your disability:

Date of diagnosis: _____

Specific diagnosis (for amputees and spinal cord please include the 'levels' affected):

_____

Would you consider your condition to be progressively worsening, and if so, why?

_____

14. Please circle major categories of limitation or difficulty due to the diagnosis:

Mobility:	independent	cane/walker	wheelchair
Transfers:	independent	sliding board	assist
Hygiene:	independent	independent with modifications	assist
Dressing upper body:	independent	independent with modifications	assist
Dressing lower body:	independent	independent with modifications	assist
Eating/drinking:	independent	independent with modifications	assist
Bowel care:	normal function	modifications (specify):	assist
Bladder care:	normal function	modifications (specify):	assist

Should the treating doctor be aware of any other information in the case of an emergency?

Are there any other issues that you would like to discuss with the doctor?

Do you have any further questions for the doctor (regarding health, safety or other matters of concern)?

15. Orthoses / prosthesis / modified equipment / braces / adaptive devices / wheelchairs
If you use any of the above items (designed specifically for you), then answer the questions listed, *applying each list of questions to each piece of equipment individually.*

Item 1:	Date athletic equipment obtained (approx.):
Is this exact piece of equipment used during your routine day to day activities?	
Would you consider this athletic equipment to be optimally modified for your body?	
Is there room for further improvement in fit? Why?	
Is there room for further improvement in performance? Why?	
Is there room for further improvement in safety? Why?	

Have there been any health issues related to this product? If so, then please specify:

Injuries:	
Skin lesions:	
Pain:	
Other:	

Item 2:	Date athletic equipment obtained (approx.):
Is this exact piece of equipment used during your routine day to day activities?	
Would you consider this athletic equipment to be optimally modified for your body?	
Is there room for further improvement in fit? Why?	
Is there room for further improvement in performance? Why?	
Is there room for further improvement in safety? Why?	

Have there been any health issues related to this product? If so, then please specify:

Injuries:	
Skin lesions:	
Pain:	
Other:	

Additional space for discussing 'yes' answers (if possible, please indicate section number):

- End of Form -

PHYSICAL EXAM

Name: _____

HR: _____ BP: _____/_____

	Normal	Abnormal	Comments
Eyes			
Head			
Neck			
Ears			
Nose			
Throat			
Heart			
Lungs			
Abdomen			
Skin			
GU			

MSK:

	Normal	Abnormal
Neck		
Upper Back		
Elbows		
Shoulders		
Wrists/hands		
Low back		
Hips		
Thigh/leg		
Knees		
Ankles		
Feet		
Neuro-		C5 6 7 8 T1 L2 3 4 5 S1 LNSL R L Spasticity -

Assessment/Plan:

1.

2.

3.

_____
_____
_____
_____
_____
_____
_____
_____
_____
_____
_____
_____
_____

Cleared to play - _____

Conditional cleared to play – Follow required for -

_____
_____

Unfit to play – reason _____

Name of Physician:_____

Signature:_____

Authors: Dr. Jon Hawkswood, Dr. Heather Finlayson and Dr. Russell O'Connor

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