**Autonomic Dysreflexia**

|  |  |
| --- | --- |
| **Pathophysiology** | * **Uninhibited** **sympathetic** **reflex** caused by a noxious stimulus below the level of SCI * **Most commonly occurs in SCI at level T6 or above**; complete more common than incomplete * Lesions below T6 have improved sympathetic inhibitory outflow to the splanchnic nerve * Clinical emergency in SCI |
| **Etiologies** | * Most commonly from **bowel, bladder, and skin irritation** * Other causes: ingrown nails, medications, systemic reactions, abdominal conditions (appendicitis, cholecystitis, or kidney stones), unhealed surgical site, orgasm, menstrual cycle and labor in female patients, etc. |
| **Presentations** | * **Hypertension** (**SBP>20mm over post-SCI baseline**), **bradycardia**/tachycardia, headache, sweating, flushing, dilated pupils, nasal congestion, piloerection, etc. (Some patients have no symptoms at all) |
| **Management** | Find and address the underlying causes:   * GU - drain bladder; flush Foley catheter * GI - evaluation of bowel program; check for rectal impaction (use lidocaine before evaluation as a rectal exam could worsen AD) * Skin - assess for pressure injury (especially **sacral** **dermatomes**) * If nothing else works: Topical nitro paste to prevent cerebral injury (contraindicated in patients on vasodilators) until cause is identified and treated |
| **Complications** | * Stroke, MI, seizures, and intracerebral hemorrhage |

**Pressure Injuries**

|  |  |
| --- | --- |
| **Common locations** | * First two years: **Sacrum**, **calcaneus**, and **ischium** (occiput and scapula in cervical level injuries) * Greater than 2 years post injury: **Ischium** and **trochanters** in chronic SCI patients * 25% of SCI patients experience at least one pressure injury in the acute hospital or rehab setting |
| **Stages of pressure injury** | * Stage I – intact skin with an area of **non-blanchable** erythema; no skin breakdown/drainage * Stage II – **partial thickness skin loss** with exposure of dermis, pink or red base * Stage III – **full-thickness skin loss with exposed fat**, granulation tissue, and rolled wound edges; no muscle, tendon, or bone present * Stage IV – **exposed muscle, tendon, ligament, or bone**; commonly have undermined edges and tunneling * Unstageable – presence of eschar or slough in the wound bed * Deep tissue injuries – **deep, maroon, non-blanchable, no skin breakdown**; possible underlying stage III or IV injury |
| **Prevention** | * Pressure relief   + **Offloading areas for 2 minutes every 15-30 minutes**   + Forward lean, side-to-side lean, push-up, tilting of chair   + If needed in an acute rehab setting, can be turned every 2 hours or be placed prone if tolerable * Close examination of wheelchair and cushion * Obtaining a pressure map |
| **Treatment** | * Debridement of slough or eschar * Alginate for substantial drainage * Cover the area with a large, foam-type dressing * Consider oral antibiotics if there is frank pus or the surrounding skin is erythematous |

**Sexual Dysfunction**

**Conversation set up – quiet space, adequate time to discuss sexuality and fertility**

|  |  |
| --- | --- |
| **Key anatomic areas for sexual function** – erection and vaginal vaso-congestion | * **Erection/vaginal vasocongestion – parasympathetic nervous system**   + Psychogenic component – centrally mediated causing modulation of the sacral reflex arc   + Reflexogenic component – parasympathetic nervous system (sacral reflex arc - S2-S4)   + Sacral reflex arc     - Afferent: genitals → pudendal nerve → spinal cord     - Efferent: parasympathetic fibers from S2-S4; also referred to as pelvic splanchnic nerve * **Ejaculation – sympathetic nervous system**, specifically the hypogastric nerve (T11-L2) |
| **Erectile dysfunction in SCI patients** | * Most SCI patients only achieve erections through the **reflexogenic mechanism** (sacral reflex)   + Reflexogenic erections occur in >90% of complete and incomplete UMN injuries, and 25% of complete LMN lesions * Psychogenic erections   + Occur in about 50% of incomplete UMN injuries, and 25% of complete LMN lesions   + Better prognosis with incomplete lesions * Treatment   + Pharmacotherapy     - Phosphodiesterase inhibitors; caution if patients are already on nitrates, have low baseline blood pressure, or are at risk for AD     - Intracorporeal injection with prostaglandin E1, alpha-blockers, or vasodilators   + Implants, vacuum devices, and ring devices |
| **Ejaculation** | * 10-20% of men can ejaculate normally after SCI injury   + Higher chance of ejaculation in incomplete lesions * Higher success rate with manual or partner masturbation, penile vibratory stimulation (home use, caution in AD patients), and electroejaculation (medical supervision required, caution in AD patients) |
| **Fertility** | * **Male fertility – decreased after SCI**   + Quality of semen decreases after SCI     - Occurs primarily due to **decreased sperm motility** related to prostatic fluid stasis     - Also impacted by atrophy of the seminiferous tubules * **Female fertility – maintained after SCI**   + Menstrual cycle often stops initially, but returns to normal by 6-12 months after the injury   + Gynecologist assessment needed if amenorrhea lasts longer than a year   + Important considerations in SCI patients who are pregnant     - Often have increased spasticity, decreased pulmonary function, recurrent UTIs     - AD may be the only clinical manifestation of labor       * Managed with epidural anesthesia |