BIOS252 Week 3 Case Study: Spinal Cord Injury

Introduction recap

The spinal cord is a cylindrical bundle of nerve fibers extending from the base of the brain through the vertebral canal. It plays a vital role in transmitting neural signals between the brain and peripheral nervous system, facilitating sensory perception, motor control, and autonomic regulation. The spinal cord consists of gray matter, containing cell bodies of neurons, and white matter, composed of myelinated axons organized into tracts. Motor neurons located in the anterior horn of gray matter transmit signals for muscle contraction, while sensory neurons in the dorsal root ganglia convey sensory information to the brain.

Discussion of Spinal Cord Injury

Spinal cord injury (SCI) is a devastating condition resulting from trauma or disease affecting the spinal cord, leading to partial or complete loss of sensory, motor, and autonomic function below the level of injury. Understanding the anatomy and physiology of the spinal cord is crucial for comprehending the consequences of SCI.

Consequences of Spinal Cord Injury:

1. **Motor Function Impairment:** SCI often results in varying degrees of motor dysfunction, depending on the level and severity of the injury. Damage to upper motor neurons (UMNs) leads to spastic paralysis characterized by increased muscle tone, exaggerated reflexes (hyperreflexia), and impaired voluntary movements below the level of injury. Conversely, damage to lower motor neurons (LMNs) results in flaccid paralysis, decreased muscle tone, diminished reflexes (hyporeflexia), and muscle atrophy. Paralysis of both lower limbs is known as (Paraplegia)
2. **Sensory Loss:** SCI can cause sensory deficits, including loss of pain, temperature, touch, and proprioception sensation below the level of injury. The extent of sensory impairment depends on the specific sensory pathways affected by the injury. For example, damage to the spinothalamic tract may result in loss of pain and temperature sensation, while damage to the dorsal columns may lead to loss of touch and proprioception sensation.
3. **Reflex Abnormalities:** SCI often disrupts normal reflex pathways, leading to alterations in reflex responses. Upper motor neuron lesions commonly result in hyperactive reflexes, such as the Babinski sign (extension of the big toe and fanning of other toes) in response to plantar stimulation, clonus (repetitive rhythmic contractions) upon muscle stretch, and exaggerated deep tendon reflexes (e.g., knee jerk reflex). Conversely, lower motor neuron lesions may cause diminished or absent reflexes.