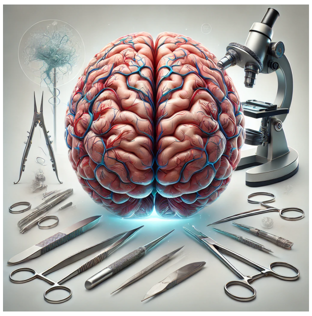
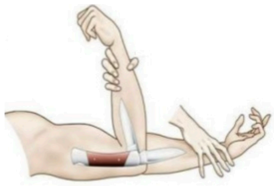


CNS—Anatomy~5
Written by: Dr.Ali Abujammil



Clasp-Knife Reaction: This is a neurological response observed in muscle spasticity, which occurs due to overactivity of the pontine excitatory system.

Phases of the Reaction:

1. Initial Resistance: When attempting to move the affected muscle, there is a strong initial resistance due to an exaggerated stretch reflex, This reflex is hyperactive because of the loss of normal inhibitory control from the central nervous system (CNS); The resistance resembles the difficulty in opening a clasp-knife, which requires a significant effort initially.

2. Sudden Release: As sustained pressure is applied, muscle tension increases to the point where the Golgi Tendon Organs (GTOs) are activated; GTOs serve as protective sensors in the tendons; once they detect excessive tension, they send inhibitory signals to motor neurons, leading to sudden muscle relaxation, This sudden release is comparable to how a clasp-knife quickly snaps open after overcoming the initial resistance.

Key Points:

 # Spasticity results from overactivity in both the pyramidal and extrapyramidal systems, leading to hyperactive reflexes and muscle stiffness.

 # Golgi Tendon Organs (GTOs) are specialized sensory receptors located in tendons that detect excessive tension and send inhibitory signals to prevent overcontraction.

 # This phenomenon is commonly seen in central nervous system (CNS) injuries, such as stroke-related spastic paralysis or spinal cord injuries.

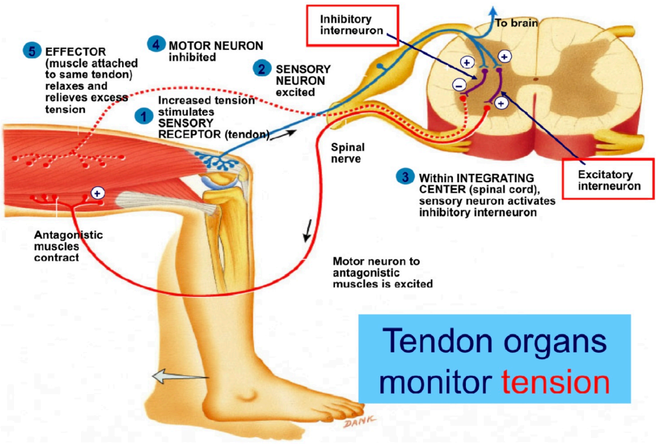
The tendon reflex is a polysynaptic reflex arc, meaning it involves multiple synapses between neurons in its pathway. This is different from monosynaptic reflexes (such as the stretch reflex), which have only one synapse and therefore generate a faster response.

1. Polysynaptic Reflex Arc: It consists of a sensory neuron, an interneuron, and a motor neuron in the reflex pathway, Because it involves interneurons, the response is slightly slower than monosynaptic reflexes but allows for more precise control of muscle activity; A key example is the Golgi Tendon Reflex, where excessive tension in the tendon activates Golgi Tendon Organs (GTOs), sending inhibitory signals to motor neurons, leading to muscle relaxation.

2. Law of Reciprocal Innervation: This law states that when one muscle group contracts, the opposing (antagonistic) muscles are inhibited to ensure smooth movement; For example: When the biceps brachii contracts, inhibitory signals are sent to the triceps brachii,

preventing it from contracting simultaneously, allowing coordinated movement, This is mediated by inhibitory interneurons in the spinal cord.

Significance of These Concepts: Protects muscles from injury by preventing excessive contraction, Facilitates coordinated movements such as walking, jumping, and writing and Regulates muscle response based on applied load, which is crucial in exercise and strenuous activities.



The image depicts the tendon reflex, an involuntary response to stretching a muscle tendon. This reflex is a polysynaptic reflex arc, meaning it involves more than one synapse. The diagram illustrates the mechanism:

1. Sensory Receptors: Located in the tendons, these detect excessive tension in the muscle.

2. Sensory Neuron: Transmits the nerve impulse from the sensory receptors to the spinal cord.

3. Integrating Center: In the spinal cord, the nerve impulse is processed. Here, a motor neuron to the stretched muscle is activated, and a motor neuron to the antagonistic muscle is inhibited. This is known as the law of reciprocal innervation.

4. Motor Neuron: Transmits the nerve impulse from the spinal cord to the muscle.

5. Effector: The muscle that responds to the nerve impulse by contracting or relaxing. In this case, the stretched muscle relaxes to relieve excessive tension.

In short, when tendon tension increases, sensory receptors send a signal to the spinal cord, causing the stretched muscle to relax and the antagonistic muscle to contract, maintaining balance. Tendon organs monitor muscle tension.

Corticospinal Tract Dysfunction and Withdrawal Reflex in the Foot

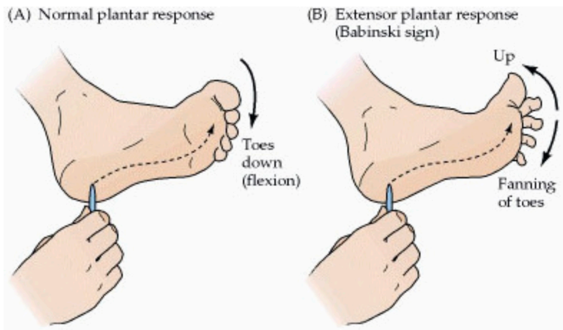
When the corticospinal tracts become nonfunctional, the influence of other descending pathways on toe movement becomes evident. This results in a withdrawal reflex upon stimulation of the sole, manifesting as the dorsiflexion of the great toe and fanning out of the other toes, a response known as the Babinski Sign.

Neural Explanation:

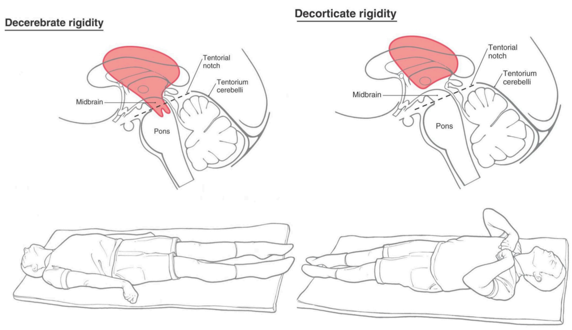
1. Under Normal Conditions: The corticospinal tracts inhibit primitive reflexes, including the Babinski reflex, Upon sole stimulation, the normal response is plantar flexion of all toes (downward movement).

2. When the Corticospinal Tracts Are Damaged: This inhibitory control is lost, allowing other descending tracts, such as extrapyramidal pathways, to take over, When the sole is stimulated with a blunt object, it triggers a Positive Babinski Sign, characterized by: Dorsiflexion of the great toe and Fanning out of the other toes, This response resembles a withdrawal reflex, as if the body is trying to pull the foot away from the stimulus.

Clinical Significance of Babinski Sign: Normal in infants (up to 1-2 years old) because the corticospinal tracts are still developing but Abnormal in adults, indicating central nervous system (CNS) dysfunction, commonly seen in: Spinal cord injuries, Stroke, Amyotrophic lateral sclerosis (ALS) and Multiple sclerosis (MS).



The image shows two drawings illustrating the response of the sole of the foot to stimulation. Part (A) shows the normal response, where the toes curl downwards in response to touch or stimulation. Part (B) shows the abnormal response, known as the Babinski sign, where the big toe extends upwards and the other toes fan out. The Babinski sign occurs when the corticospinal tracts (the nerve pathways connecting the cerebral cortex to the spinal cord) are nonfunctional. In this case, the influence of other descending tracts becomes apparent, resulting in the withdrawal reflex shown in image (B).



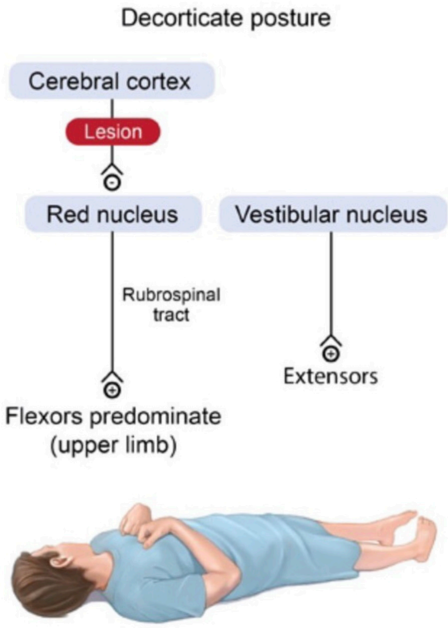
The image illustrates two types of muscle rigidity resulting from damage to the central nervous system: decerebrate rigidity and decorticate rigidity. Both represent characteristic body postures resulting from damage to different areas of the brain.

Decerebrate Rigidity: The anatomical drawing shows the location of the brain injury. It depicts damage to the brainstem area (between the cerebellum and pons). This damage leads to loss of muscle control, It is characterized by extension (straightening) of the limbs with the hands flexed at the wrists. Both upper and lower limbs are rigidly extended.

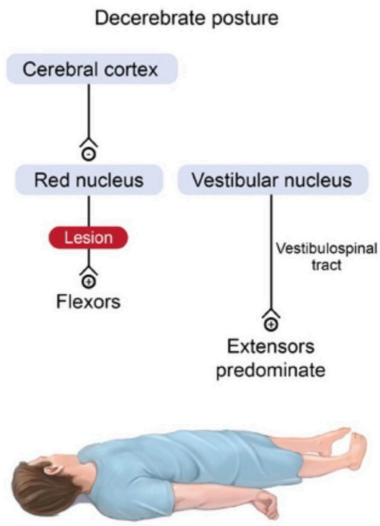
Decorticate Rigidity: The anatomical drawing shows the location of the brain injury. It depicts damage higher in the brainstem, in the cerebral cortex or corticospinal

tracts, It is characterized by flexion (bending) of the upper limbs towards the chest with clenched fists, while the lower limbs remain extended.

The main difference between the two is the posture of the upper limbs. In decerebrate rigidity, the upper limbs are extended, while in decorticate rigidity, the upper limbs are flexed towards the chest. Both conditions indicate serious neurological damage and require immediate medical attention.



The image depicts a "Decorticate posture." This is a characteristic body posture that occurs due to damage to the central nervous system, specifically in areas above the brainstem, such as the cerebral cortex or corticospinal tracts.The diagram shows the location of the injury in the brain. It shows a lesion in the cerebral cortex. This lesion affects the red nucleus and the vestibular nucleus.The red nucleus sends nerve signals via the rubrospinal tract to the flexor muscles in the upper limbs. Damage to the cerebral cortex leads to increased activity of this tract, causing flexor muscle dominance.The vestibular nucleus sends signals to the extensor muscles in the lower limbs. In decorticate rigidity, the influence of the vestibular nucleus on the lower limbs remains intact, causing extension of the lower limbs.The anatomical drawing shows the patient's posture. It is characterized by flexion of the upper limbs towards the chest with clenched fists, while the lower limbs remain extended. This reflects the dominance of flexor muscles in the upper limbs and extensor muscles in the lower limbs, In short: Decorticate rigidity results from damage to the cerebral cortex or corticospinal tracts, leading to increased activity of the rubrospinal tract and dominance of flexor muscles in the upper limbs, while the lower limbs remain extended due to the activity of the vestibular nucleus. It is a sign of serious neurological damage and requires immediate medical attention.



The image illustrates a "Decerebrate posture." This is a characteristic body posture that occurs due to damage to the central nervous system, specifically in the brainstem area, below the hypothalamus.The diagram shows the location of the injury in the brain. It shows a lesion above the red nucleus and in the neural pathways connecting the cerebral cortex to the brainstem. This damage disrupts the function of the red nucleus.The red nucleus sends nerve signals via the rubrospinal tract to the flexor muscles in the upper limbs. Damage to the red nucleus or the pathways leading to it leads

to decreased activity of this tract, causing weakness in the flexor muscles.The vestibular nucleus sends signals to the extensor muscles in the upper and lower limbs. In decerebrate rigidity, the influence of the vestibular nucleus becomes dominant, causing extension of the limbs. It is characterized by extension (straightening) of the limbs with the hands flexed at the wrists. Both upper and lower limbs are rigidly extended. The face is usually turned downwards.In short: Decerebrate rigidity results from damage to the brainstem, leading to decreased activity of the rubrospinal tract and dominance of extensor muscles in the upper and lower limbs. It is a sign of very serious neurological damage and requires immediate medical attention.

Clonus refers to rhythmic, involuntary contractions and relaxations of a muscle when it is subjected to a sudden and sustained stretch. It occurs due to the inability of the central nervous system (CNS) to properly regulate muscle reflexes, leading to repetitive activation of the stretch reflex.

Neural Mechanism:

1. Sudden Muscle Stretch:When a muscle is rapidly and continuously stretched, it activates muscle spindles, which are sensory receptors that detect changes in muscle length.

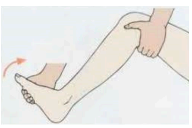
2. Exaggerated Stretch Reflex: The muscle spindles send excitatory signals to the spinal cord, triggering a muscle contraction in response to the stretch; Normally, this reflex is regulated by the corticospinal tracts, but when these pathways are damaged, the reflex becomes overactive.

3. Repetitive Contractions and Relaxations: Due to the lack of inhibitory control from the CNS, motor neurons continue to fire rhythmically, causing alternating contractions and relaxations of the muscle.

Possible Causes are Central nervous system (CNS) disorders, including: Stroke, Multiple sclerosis (MS), Spinal cord injuries, Amyotrophic lateral sclerosis (ALS) and Hypertonia (Increased muscle tone) associated with neurological conditions.

Common Sites for Clonus Testing; The most frequent locations to test for clonus include: Ankle Clonus Rapid dorsiflexion (upward movement) of the foot and Patellar Clonus Sudden pressure on the kneecap (patella).

Clinical Significance: The presence of clonus is a key pathological sign indicating upper motor neuron (UMN) lesions, It is used in neurological assessments to evaluate motor function impairments.



The image depicts a test for ankle clonus. The diagram shows a hand holding someone's knee while the other person's foot is sharply dorsiflexed. This is known as the clonus test, which checks for involuntary rhythmic muscle contractions and relaxations in response to a sudden sustained stretch. If the muscles respond in this way, it may indicate a neurological problem.

Hypertonia and Hyperreflexia Due to Increased Gamma Motor Neuron Activity.

Hypertonia is an abnormal increase in muscle stiffness, making movement difficult and resistant to passive stretching. It is often accompanied by Hyperreflexia, which refers to exaggerated reflex responses when testing muscle reflexes, such as the knee-jerk reflex.

Role of Gamma Motor Neurons: regulate muscle tone by controlling muscle spindle sensitivity, which detects changes in muscle length, These neurons stimulate intrafusal muscle fibers within the spindles, making them more sensitive to stretch and Increased gamma motor neuron activity leads to hypersensitivity of muscle spindles, resulting in Hyperreflexia and Hypertonia.

How Increased Gamma Motor Neuron Activity Causes Hypertonia and Hyperreflexia:

1. Gamma motor neurons become overactive, making muscle spindles highly sensitive to stretch.

2. This leads to excessive sensory input to the spinal cord, exciting alpha motor neurons.

3. The result is continuous muscle contraction, causing Hypertonia (muscle stiffness).

4. Additionally, muscle reflexes become exaggerated, leading to Hyperreflexia.

Possible Causes of Increased Gamma Motor Neuron Activity:

Central Nervous System (CNS) Lesions, such as: Stroke, Spinal cord injuries, Multiple sclerosis (MS), Cerebral palsy.

Loss of Inhibitory Control from the CNS: Normally, the corticospinal tracts and extrapyramidal inhibitory pathways regulate gamma motor neuron activity; Damage to these pathways leads to excessive gamma motor neuron activity, resulting in Hypertonia and Hyperreflexia.

Clinical Impact: Severe Hypertonia can cause muscle rigidity and movement difficulties; In some cases, it can develop into Spasticity, a condition where muscle stiffness is associated with exaggerated reflexes; Treatment may involve physical therapy, muscle relaxants like Baclofen, or even surgical interventions in severe cases.

The lamination (layered arrangement) of nerve fibers within the ascending tracts, particularly the spinothalamic tract, is clinically significant because it determines how sensory loss progresses when the spinal cord is subjected to external pressure or tumors.

1. Effects of External Pressure on the Spinothalamic Tract: If external pressure is applied to the spinal cord in the region of the spinothalamic tract, the first sensory loss occurs in the sacral dermatome (pain and temperature sensations); As the pressure increases, the sensory loss progresses upward, affecting higher segmental dermatomes (lumbar, thoracic, and cervical regions).

2. Anatomical Arrangement of Fibers in the Spinothalamic Tract: Cervical fibers are located medially (toward the center) while Sacral fibers are located laterally (toward the outer edges);This lamination determines which fibers are affected first in spinal cord compression or injury.

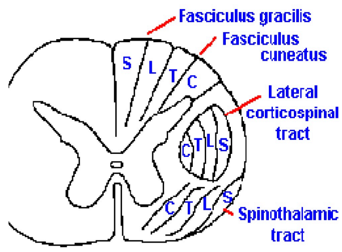
3. Effects of Intramedullary vs. Extramedullary Tumors

Intramedullary Tumors (inside the spinal cord): Affect medial fibers first (cervical region), This causes sensory loss in the upper body before the lower limbs are affected.

Extramedullary Tumors (outside the spinal cord): Affect lateral fibers first (sacral and lower limb regions),This leads to sensory loss in the legs and feet before affecting the upper body.

4. Sacral Sparing in Intramedullary Tumors: In intramedullary tumors, the sacral fibers (located laterally) may remain intact for a longer period, This results in preserved sacral sensation (Sacral Sparing), even in cases of advanced spinal cord involvement.

This understanding is crucial for diagnosing spinal cord lesions and predicting the pattern of sensory loss based on the location of the pathology.



The image showing a cross-section of the spinal cord with an explanation of the clinical significance of the lamination of ascending tracts. The diagram illustrates the location of different ascending tracts, including the spinothalamic tract, the lateral corticospinal tract, the fasciculus gracilis, and the fasciculus cuneatus. Look how external pressure on the spinal cord affects these tracts, and how pressure will first affect the sacral dermatome, then extend to higher areas if the pressure increases. And look the difference between intramedullary and extramedullary tumors and how they affect these tracts, as well as the concept of sacral sparing which occurs in intramedullary tumors. In short, illustrates the relationship between the anatomy of the spinal cord and its function, and how lesions or tumors can affect spinal cord function.

The Lateral Spinothalamic Tract (LSTT) is one of the ascending sensory pathways in the central nervous system, responsible for transmitting pain and temperature sensations from the body to the brain. When this tract is damaged, it results in a characteristic pattern of sensory loss.

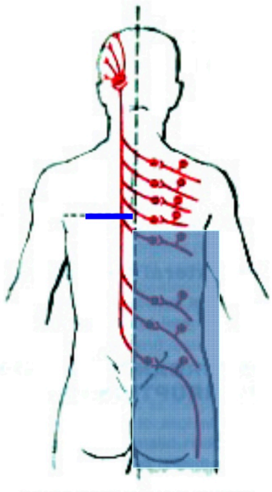
1. Symptoms of LSTT Destruction: Loss of pain (Analgesia) and temperature sensation (Thermoanesthesia), Sensory loss occurs on the contralateral (opposite) side of the body; The sensory loss occurs below the level of the lesion, because the fibers in LSTT cross to the opposite side within 1-2 spinal segments after entering the spinal cord.

2. Why Does the Damage Affect the Opposite Side? Sensory fibers for pain and temperature enter the spinal cord through the dorsal roots and synapse in the dorsal horn; Then, these

fibers cross to the opposite side (decussate) within the spinal cord before ascending in the LSTT to the thalamus in the brain Therefore, any damage after the crossing point results in sensory loss on the opposite side of the body.

3. Clinical Effects on the Patient: The patient will not recognize pain or differentiate between hot and cold sensations on the contralateral side below the lesion. This increases the risk of unnoticed injuries, such as burns or cuts, which could lead to infections or complications; The sensory loss can be partial or complete, depending on the extent of LSTT damage.

4. Clinical Conditions That Can Damage the LSTT: Spinal cord injuries, Brainstem strokes affecting sensory pathways, Spinal cord tumors, Multiple sclerosis (MS), where demyelinating plaques may damage the LSTT and Brown-Squard Syndrome: Occurs with hemisection of the spinal cord and Causes contralateral loss of pain and temperature sensation and ipsilateral loss of motor function and deep touch sensation.



LATERAL SPINOTHALAMIC TRACT

The image is a presentation slide illustrating the clinical correlation between damage to the Lateral Spinothalamic Tract (LSTT) and the loss of pain and temperature sensation.The slide shows a diagram of the human body with the Lateral Spinothalamic Tract (LSTT) highlighted in blue. This tract is responsible for transmitting pain and temperature signals from the body to the brain.what happens when this tract is damaged: Loss of pain and temperature sensation: If the LSTT is damaged, the patient will lose the ability to feel pain and temperature. On the contralateral side: Importantly, the loss of sensation will be on the *opposite* side of the body from where the damage occurred. For example, if the LSTT is damaged on the right side of the spinal cord, the patient will lose pain and temperature sensation on the left side of the body. Below the level of the lesion: The loss of sensation will be *below* the level of the injury in the spinal cord. If the damage occurs in the thoracic region, for example, the loss of sensation will be in the lower parts of the body. Inability to discriminate between hot and cold: As a result of LSTT damage, the patient will not be able to distinguish between different temperatures (hot and cold).

The Fasciculus Gracilis and Fasciculus Cuneatus are part of the Dorsal Column-Medial Lemniscus Pathway, which is responsible for transmitting fine touch, vibration, proprioception (joint position sense), and tactile discrimination.

1. Symptoms of Fasciculus Gracilis and Fasciculus Cuneatus Damage: Loss of proprioception, vibration sensation, and fine touch discrimination; Sensory loss occurs on the same side (Ipsilateral) of the body; Sensory loss occurs below the level of the lesion, because these fibers do not cross over until they reach the Medulla Oblongata.

2. Difference Between Fasciculus Gracilis and Fasciculus Cuneatus:

Fasciculus Gracilis: Carries sensory information from the lower limbs and lower trunk; Located medially in the spinal cord.

Fasciculus Cuneatus: Carries sensory information from the upper limbs and upper trunk, Located laterally in the spinal cord.

 # Damage to the Fasciculus Gracilis affects the legs, while damage to the Fasciculus Cuneatus affects the arms.

3. Why Does the Sensory Loss Occur on the Same Side? These fibers ascend in the spinal cord without crossing (decussating); They cross only at the Medulla Oblongata, where they synapse in the Nucleus Gracilis and Nucleus Cuneatus Therefore, a lesion within the spinal cord affects sensation on the same side below the lesion.

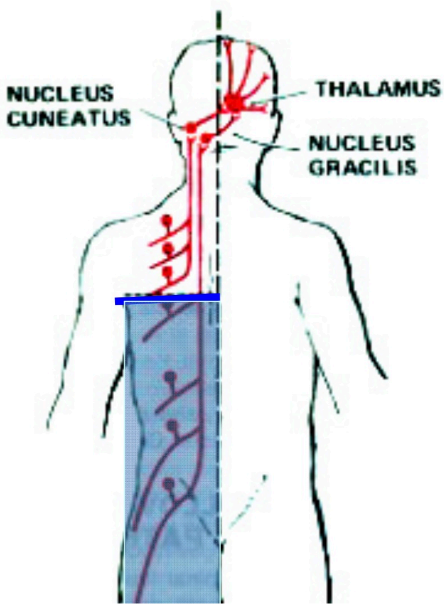
4. Clinical Effects on the Patient: The patient cannot sense limb position without looking, leading to balance problems and sensory ataxia (difficulty walking in the dark or with closed eyes), Loss of vibration sense prevents detection of tuning fork vibrations, Loss of tactile discrimination (e.g., inability to distinguish between a coin and a key by touch alone); Gradual sensory loss can be seen in degenerative conditions such as Tabes Dorsalis (a late-stage syphilitic infection affecting the posterior columns).

5. Clinical Conditions That Can Damage These Tracts: Amyotrophic Lateral Sclerosis (ALS) with dorsal column involvement, Spinal cord trauma, Tabes Dorsalis (Neurosyphilis), which affects the dorsal columns, Vitamin B12 Deficiency (Subacute Combined Degeneration), Tumors or Multiple Sclerosis affecting the posterior columns.

Note:It is extremely rare for a spinal cord lesion to affect only one sensory tract, as most injuries impact multiple pathways.

The image shows a slide from a medical presentation illustrating the relationship between damage to the fasciculus gracilis and fasciculus cuneatus tracts in the spinal cord and the appearance of specific clinical symptoms.

Fasciculus gracilis and fasciculus cuneatus: These two tracts are part of the posterior columns of the spinal cord, forming the posterior column-medial lemniscus pathway. This is a sensory pathway that transmits fine touch information from the body to the brain, including: Position sense Awareness of the location of body parts in space, Vibration sense Sensation of vibrations, Tactile discrimination Ability to distinguish between two separate touches, Muscle joint sense Sensation of joint position and movement.



Damage: The diagram illustrates the pathway of the two tracts from the spinal cord to the thalamus in the brain. Damage to one or both of these tracts will result in a loss of the sensations mentioned above.

Clinical symptoms: indicates that damage to these tracts results in a loss of position sense, vibration sense, tactile discrimination, and muscle joint sense on the same side of the body (ipsilateral) below the level of the lesion in the spinal cord. That is, symptoms will appear on the same side of the body as the damage, and below the level of the injury in the spinal cord.

Rarity of single tract involvement: it is extremely rare for spinal cord damage to be localized to only one sensory tract. Usually, more than one sensory pathway is affected.

Syringomyelia is a neurological disorder characterized by the formation of a fluid-filled cavity (syrinx) within the central regions of the spinal cord. Over time, this cavity expands, causing progressive damage to the neural pathways, particularly in the central spinal cord.

1. How Does Syringomyelia Affect the Nervous System? Since the syrinx forms in the central spinal cord, it damages the fibers that cross through the anterior white commissure, These fibers are responsible for transmitting pain and temperature sensations; As a result, patients develop: Bilateral loss of pain and temperature sensation and Touch, vibration, and proprioception remain intact because the dorsal column pathways (Fasciculus Gracilis & Fasciculus Cuneatus) are unaffected.

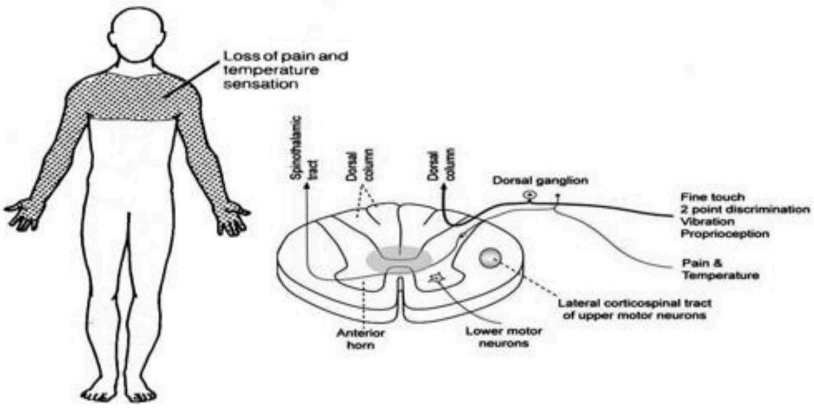
2. Classic Cape-Like Sensory Loss Pattern in Syringomyelia; When the syrinx is located at C4 - C5 spinal cord levels, it produces a distinct sensory loss pattern - Cape-Like Distribution of sensory loss: Involves the shoulders, upper arms, and extends to the nipple level (T4); This distribution resembles a cape draped over the shoulders, It is a classic diagnostic feature of Syringomyelia.

3. Involvement of the Anterior Horn of the Spinal Cord, If the syrinx expands into the anterior horn, it causes: Weakness in the upper extremities due to lower motor neuron (LMN) damage, Muscle atrophy and flaccid paralysis; If both anterior horns are affected Bilateral weakness; If only one anterior horn is affected â†’ Ipsilateral weakness (same-side muscle weakness).

4. Clinical Impact on the Patient: The patient loses pain and temperature sensation in the affected areas, Due to sensory loss, the patient may suffer burns or injuries without noticing them, If the anterior horn is involved, muscle weakness and atrophy occur, leading to difficulty in arm movement.

5. Conditions Associated with Syringomyelia: Arnold-Chiari Malformation (a congenital brain abnormality where the cerebellum herniates into the spinal canal), Spinal cord trauma and Spinal cord tumors or infections.

This condition is progressive and requires neurological evaluation and often surgical intervention if the syrinx continues to expand.



The image is a diagram illustrating the mechanism of syringomyelia. Syringomyelia is a rare medical condition characterized by the formation of a fluid-filled cyst (syrinx) within the spinal cord. This cyst compresses the surrounding spinal cord tissue, leading to a range of neurological symptoms.The diagram shows a region of loss of pain and temperature sensation in the upper body (shoulders and arms). This is due to the pressure exerted by the syrinx on the sensory tracts in the spinal cord responsible for transmitting pain and temperature signals. These tracts are located in the anterior spinal cord. In contrast, other sensory functions such as fine touch, vibration, and proprioception remain intact because the nerve tracts responsible for them (posterior tracts) are unaffected.

Additional Information:

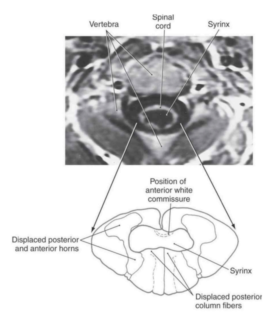
Syrinx Location: The location of the syrinx within the spinal cord varies from person to person, leading to variations in symptoms.

Symptoms: In addition to loss of pain and temperature sensation, symptoms of syringomyelia may include muscle weakness, stiffness, numbness, and dysfunction of internal organs.

Causes: The exact cause of syringomyelia is not always known, but it is often associated with congenital defects in the spinal cord, injuries, or tumors.

Diagnosis: Syringomyelia is diagnosed through clinical examination and magnetic resonance imaging (MRI) of the spinal cord.

Treatment: Treatment of syringomyelia aims to relieve pressure on the spinal cord and may include surgery or medication.



Simply put, syringomyelia is a condition where a fluid-filled cyst (syrinx) forms within the spinal cord. This cyst puts pressure on parts of the spinal cord, causing problems with sensation and movement. The diagram shows how the syrinx presses on specific parts of the spinal cord, leading to loss of pain and temperature sensation in certain body parts, while other sensations remain intact. This is because the syrinx doesn't press on all parts of the spinal cord equally.

Brown-squard Syndrome is a neurological condition caused by a hemisection or partial injury to the spinal cord, resulting in a characteristic pattern of motor and sensory deficits on either side of the body.

1. Sensory and Motor Effects in Brown-Squard Syndrome: In this condition, damage to one side of the spinal cord affects both sensory and motor pathways on that side. Specifically, this can involve: Lateral corticospinal tract damage, which controls voluntary muscle movements, Damage to the anterolateral system (ALS), responsible for transmitting pain and temperature sensations, Damage to the posterior columns, responsible for discriminative touch, proprioception, and vibration sense.

2. Symptoms of Brown-Squard Syndrome: If the lesion is on the right side of the spinal cord at C4 to C5: Right side of the body will exhibit muscle weakness or paralysis (Hemiparesis/ Hemiplegia) and Left side of the body will experience Loss of pain and temperature sensation and Right side of the body will experience Loss of discriminative touch, proprioception, and vibration sense.

3. Other Effects of Brown-Squard Syndrome:

Contralateral loss of nociceptive and thermal sensations: Loss of pain and temperature sensations will occur on the opposite side of the lesion below the level of injury.

Ipsilateral loss of discriminative tactile, vibratory, and position sense: Loss of fine touch, proprioception, and vibration sense will occur on the same side of the lesion below the level of injury.

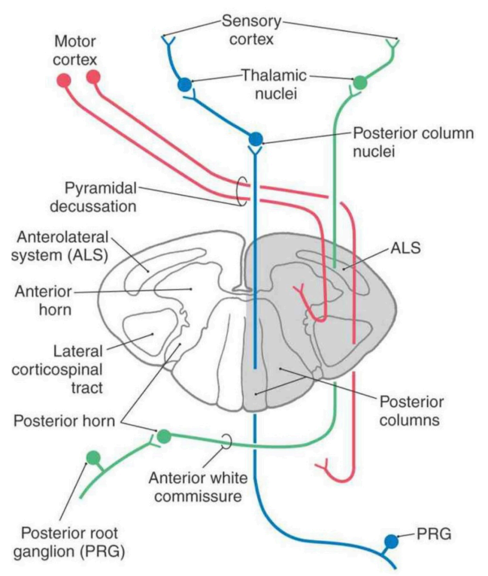
Ipsilateral paralysis of the leg or leg and arm, depending on the level of the hemisection: Weakness or paralysis will affect the same side of the body below the level of the lesion, depending on the spinal level.

4. Clinical Pattern: Contralateral loss refers to pain and temperature loss on the opposite side of the lesion; Ipsilateral loss refers to discriminative touch, proprioception, and vibration sense on the same side of the lesion; Ipsilateral paralysis affects the same side of the body below the injury.

The image is a diagram illustrating the nerve pathways in the spinal cord, specifically showing the effects of Brown-Squard Syndrome This is a rare medical condition that occurs as a result of partial damage to one side of the spinal cord. This damage leads to a characteristic set of symptoms on one side of the body, different from the other side.The diagram shows damage to one side of the spinal cord. As a result of this damage, we observe the following:

On the same side of the injury (ipsilateral):

Paralysis: Loss of movement in muscles due to



damage to the corticospinal tracts responsible for voluntary motor control. This is shown in the diagram as damage to the red pathway on the same side of the injury.

Loss of proprioception (vibration and position sense) and fine touch: This is due to damage to the posterior columns responsible for these sensations. This is shown in the diagram as damage to the green pathway on the same side of the injury.

On the opposite side of the injury (contralateral):

Loss of pain and temperature sensation: This is due to damage to the spinothalamic tracts that carry these sensations. However, note that this damage appears on the *opposite* side of the injury because these tracts cross (decussate) in the spinal cord before ascending to the brain. This is shown in the diagram as damage to the red pathway on the opposite side of the injury.

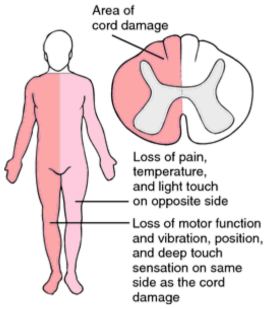
Additional Information:

Causes of the syndrome: Brown-Squard syndrome usually occurs as a result of spinal cord injuries, such as stab wounds, gunshot wounds, or car accidents. It can also be caused by tumors, infections, or vascular diseases.

Symptoms: The severity of symptoms varies depending on the size and extent of spinal cord damage.

Diagnosis: The syndrome is diagnosed through clinical examination and magnetic resonance imaging (MRI) of the spinal cord.

Treatment: Treatment aims to address the underlying cause of the injury and may include surgery, medication, or physical therapy.



The image shows a diagram illustrating Brown-Squard Syndrome. This syndrome occurs when only one half of the spinal cord is damaged. Because of the way nerve pathways are organized in the spinal cord, symptoms appear differently on the two sides of the body.Imagine the spinal cord is divided into two halves. If one half is damaged, you'll notice the following symptoms:

On the same side of the injury: You will lose the ability to move your muscles (partial paralysis) and lose the sense of fine touch, vibration, and proprioception (knowing where your limbs are in space).

On the opposite side of the body: You will lose the sensation of pain and temperature.

This is because the nerve pathways responsible for pain and temperature cross over (change sides) in the spinal cord before reaching the brain, while the pathways responsible for movement and fine touch do not cross until they reach the brainstem.

When facing the significant challenges that come with treating diseases or injuries of the nervous system, always remember that science and technology are constantly evolving. Never give up in the face of difficult cases; instead, view each case as an opportunity to learn and grow. Rely on your medical team, and continue seeking the latest methods and techniques for patient care. The nervous system may be complex, but it not beyond our capabilities. Persistence and teamwork can lead to remarkable results, so never lose hope,Working in neurosurgery requires critical thinking, adaptability, and continuous focus.