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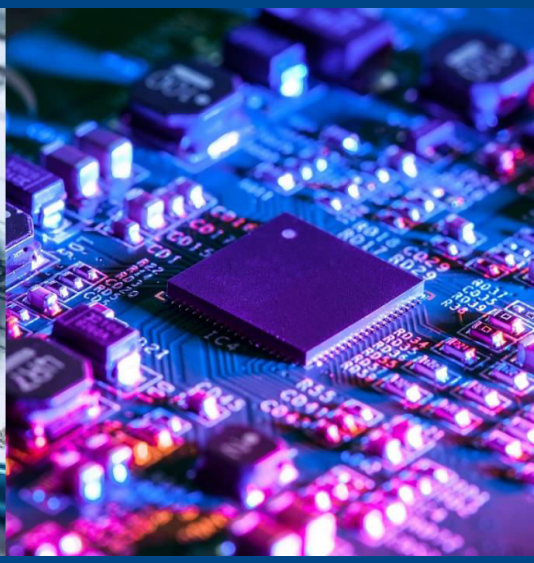

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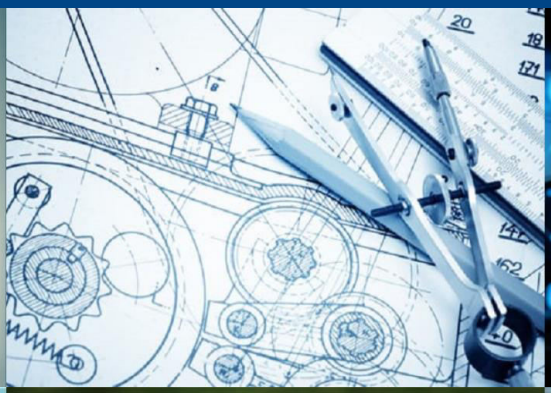

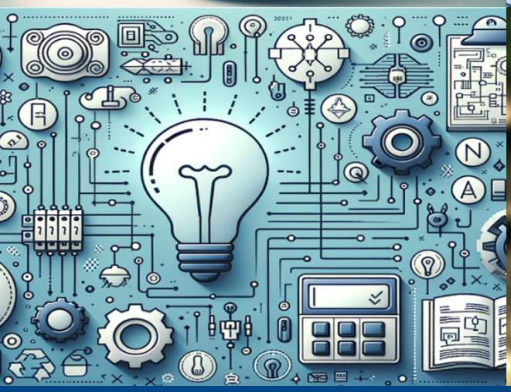

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'Pathways of Pain' from the Face and Trunk- A Narrative Review

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ABSTRACT: Understanding the complex and distinct neuroanatomical circuits that process pain from the face and trunk is essential to understanding how pain is experienced and treated. Both neuroplastic and nociceptive (pain-signaling) mechanisms that contribute to pain perception are involved in the various cranial and spinal channels that innervate the face and trunk. The primary source of facial discomfort is the trigeminal nerve (CN V), which is made up of the ocular, maxillary, and mandibular branches. Nociceptive information is sent by the sensory fibres of these branches to the trigeminal nerve ganglia, which then relays pain signals to the trigeminal nucleus caudalis in the brainstem, which then relays them to the thalamus and somatosensory cortex. The pain pathways in the face are closely related to the corticospinal and cortical networks that control pain perception.

I. INTRODUCTION

Pain is transmitted from the trunk through the spinal nerves by sensory neurons in the dorsal root ganglion and nociceptive fibres that enter the spinal cord. These impulses ascend to the spinothalamic tract, then go to specific regions of the somatosensory cortex via the brainstem before arriving at the thalamus. Both intense, acute pain and diffuse, chronic pain from the trunk are transmitted via both A-delta and C fibres. Furthermore, by exercising inhibitory control over nociceptive transmission, descending systems from the periaqueductal grey (PAG) and rostral ventromedial medulla (RVM) affect the trunk's pain pathways. [1,2]

Understanding the various but connected pathways for pain from the face and trunk is necessary to develop customized treatment for a range of pain disorders, such as trigeminal neuralgia, postherpetic neuralgia, and chronic back pain. Advances in neuroimaging, neurostimulation, and pharmaceutical therapies continue to enhance the treatment of a variety of pain conditions by providing insight into the anatomical and neurophysiological mechanisms that underline the sensory experience of pain. [3]

Pain pathways from face and trunk, its pathways

Pain from the face and trunk is transmitted through distinct but connected pathways involving the peripheral and central nervous systems. These channels follow specific routes through the cranial and spinal nerves before being processed in the brainstem, thalamus, and cerebral cortex. Understanding these pathways is necessary for the diagnosis and treatment of pain-related conditions such trigeminal neuralgia, postherpetic neuralgia, and neuropathic pain syndromes.

1. Pain Pathways from the Face

Pain sensation from the face is primarily mediated by the trigeminal nerve (cranial nerve V), which carries nociceptive signals to the brainstem and higher centers.

A. Peripheral Pathway (Trigeminal System) [4]

Receptors: Nociceptors in the skin, mucosa, and deep structures detect painful stimuli.
Primary Neurons: Located in the trigeminal ganglion, transmitting signals from:
Ophthalmic (V1) nerve – forehead, eyes, upper nose.
Maxillary (V2) nerve – midface, upper teeth, palate.
Mandibular (V3) nerve – lower face, tongue, lower teeth.

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B. Central Pathway (Brainstem & Thalamus) [4]
Trigeminal Sensory Nuclei (in the Brainstem)
Spinal Trigeminal Nucleus (Pain & Temperature processing)
Located in the medulla and upper cervical spinal cord.
Receives nociceptive input and transmits it to the thalamus.
Principal Sensory Nucleus (Touch & Pressure processing)
Trigemino-Thalamic Tract (Ascending Pathway)
Second-order neurons decussate (cross) to the opposite side and ascend via the trigemino-thalamic tract.
Synapse in the ventral posterior medial (VPM) nucleus of the thalamus.
Cortical Processing
From the thalamus, pain signals are relayed to the somatosensory cortex (S1, S2) and limbic system for pain perception and emotional response.

2. Pain Pathways from the Trunk
The spinal cord and ascending pain pathways provide a unique conduit for pain from the trunk, limbs, and internal organs.
A. Peripheral Pathway (Spinal Nerves)
Primary Afferents (Nociceptors): Pain receptors in the skin, muscles, joints, and viscera detect stimuli.
Dorsal Root Ganglia (DRG): First-order neurons carry signals from nociceptors to the spinal cord via Aδ (sharp pain) and C fibers (dull pain).
B. Central Pathway (Spinal Cord & Brainstem)
Spinal Cord Processing (Dorsal Horn & Laminae I-V)
Pain signals enter the dorsal horn of the spinal cord and synapse onto second-order neurons in the substantia gelatinosa (Lamina II).
Interneurons modulate pain through inhibitory mechanisms (e.g., opioid-mediated inhibition).

3.Ascending Pathways
Spinothalamic Tract (STT) – Primary Pain Pathway
Second-order neurons cross to the contralateral spinal cord and ascend to the ventral posterior lateral (VPL) nucleus of the thalamus.
Transmits sharp, localized pain and temperature information.
Spinoreticular & Spinomesencephalic Tracts – Emotional & Reflexive Pain Processing
Project to the reticular formation and periaqueductal gray (PAG) for modulation.
Associated with autonomic and emotional pain responses.
Cortical Processing
Pain signals reach the somatosensory cortex, insular cortex, and anterior cingulate cortex for conscious perception and affective pain responses. **[5]**

4. Modulation of Pain Pathways (Descending Control)
The brainstem's descending inhibitory mechanisms, which control spinal and trigeminal nociceptive input, modify pain perception.
A. Periaqueductal Gray (PAG) & Brainstem Modulation
PAG (Midbrain) activates serotonergic & noradrenergic pathways to inhibit pain transmission in the spinal cord and brainstem.
Opioid receptors in the PAG, nucleus raphe magnus (NRM), and dorsal horn mediate pain suppression. **[6]**
targeted pain management strategies. Future research into pain modulation mechanisms and neuroplasticity will further advance pain treatment approaches.

Anatomical and Neurophysiological pathways for pain from the face and trunk
Anatomical and Neurophysiological Mechanisms of Face and Trunk Pain
The complex network that senses pain from the face and trunk includes thalamic relay centres, spinal and brainstem pathways, peripheral nociceptors, primary sensory neurones, and cortical processing areas. These pathways are essential for nociceptive signal recognition, processing, and modulation. Here is a detailed analysis of the anatomical and neurophysiological pathways via which pain is transmitted from both regions:

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1. Pain Pathway from the Face (Trigeminal Nociceptive System)

A. Peripheral Component

Pain from the face, mouth, and anterior head is primarily carried by the trigeminal nerve (cranial nerve V) and minor contributions from other cranial nerves.

Nociceptors & Primary Afferents:

Free nerve endings of Aδ (sharp pain) and C fibers (dull pain) detect nociceptive stimuli (mechanical, thermal, or chemical).

Located in the skin, mucosa, muscles, periosteum, and teeth.

Trigeminal Ganglion (TG):

Contains the cell bodies of first-order neurons transmitting pain from the face.

Divided into three branches:

Ophthalmic (V1): Forehead, eyes, upper nose.

Maxillary (V2): Midface, upper jaw, palate.

Mandibular (V3): Lower face, lower jaw, anterior tongue. **[7]**

B. Central Pathway

Trigeminal Nuclei in the Brainstem:

Spinal Trigeminal Nucleus (STN):

Main relay for pain and temperature.

Divided into subnucleus caudalis (important for pain) and subnucleus interpolaris & oralis.

Located in the medulla and upper cervical spinal cord (C1-C2).

Principal Sensory Nucleus: Processes touch and proprioception (not pain).

Trigemino-Thalamic Tract (Ascending Pathway):

Second-order neurons cross to the contralateral side of the brainstem and ascend via the trigemino-thalamic tract.

Synapse in the ventral posterior medial (VPM) nucleus of the thalamus.

Cortical Processing:

Third-order neurons project from the thalamus to the somatosensory cortex (S1, S2), insular cortex, and anterior cingulate cortex for pain perception and emotional response. **[8]**

2. Pain Pathway from the Trunk (Spinal Nociceptive System)

A. Peripheral Component

Pain from the trunk, limbs, and viscera is transmitted via spinal nerves and follows the spinothalamic and spinoreticular pathways.

Nociceptors & Primary Afferents:

Free nerve endings detect pain stimuli.

Aδ fibers (sharp, localized pain) and C fibers (diffuse, burning pain).

Dorsal Root Ganglia (DRG):

Cell bodies of first-order neurons lie in the DRG of spinal nerves.

Axons enter the dorsal horn of the spinal cord for synapsing with second-order neurons. **[9]**

B. Central Pathway

Spinal Cord Processing (Dorsal Horn & Laminae I-V):

Pain signals enter the dorsal horn and synapse in Lamina I (marginal layer) and Lamina II (substantia gelatinosa).

Modulation of pain occurs via interneurons, inhibitory neurotransmitters (GABA, opioids), and descending inhibition from the brainstem.

Ascending Pathways:

Spinothalamic Tract (STT) – Primary Pain Pathway

Second-order neurons cross the spinal midline (anterior white commissure) and ascend to the ventral posterior lateral (VPL) nucleus of the thalamus.

Transmits sharp, localized pain and temperature.

Spinoreticular & Spinomesencephalic Tracts – Emotional & Reflexive Pain

Project to the reticular formation and periaqueductal gray (PAG) for modulation.

Associated with autonomic and emotional pain responses.

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Cortical Processing:
Pain signals from the thalamus project to the somatosensory cortex, insular cortex, and cingulate cortex for sensory, emotional, and autonomic responses. **[10]**

3. Descending Pain Modulation System
Pain perception is regulated by descending inhibitory pathways from the brainstem to the spinal cord and trigeminal nuclei.
Periaqueductal Gray (PAG, Midbrain):
Releases endogenous opioids (enkephalins, endorphins) to inhibit pain transmission.
Activates the nucleus raphe magnus (NRM, medulla).
Descending Pathways:
Serotonergic and noradrenergic projections from the NRM and locus coeruleus inhibit dorsal horn neurons.
Suppression of pain via GABAergic and opioid mechanisms in the dorsal horn and spinal trigeminal nucleus. **[11]**

Referred Pain and Its Physiological Basis
"Referred pain" is the phenomenon where pain is experienced somewhere other than its source. This pain is often associated with visceral diseases, but it can also be brought on by neuropathic pain or musculoskeletal disorders. Interactions between brain processing, nociceptive pathways, and somatic afferents are some of the complex physiological mechanisms behind transferred pain.
1. Mechanisms of Referred Pain
Referred pain is thought to be caused by nociceptive fibres from different tissues converging into the same neurons at the brainstem or spinal cord level. This convergence causes the brain to get confused and misinterpret the source of the pain impulses.
A. Convergence-Projection Theory
In the dorsal horn of the spinal cord, pain fibres from the somatic (skin, muscles) and visceral (internal organs) regions converge on the same second-order neurons. The brain struggles to distinguish between the two sources of pain because somatic pain pathways are more developed than visceral pathways. As a result, pain may be felt in areas of the body that are distant from the underlying problem. For example, after a myocardial infarction, heart pain is often felt in the left arm or jaw. This is because nociceptive signals from the heart and the arm/jaw are received by the same spinothalamic tract neurons in the spinal cord. **[12]**

B. Central Sensitization
Central sensitization is a syndrome in which the brain and spinal cord become hyperresponsive to nociceptive input due to chronic pain or persistently unpleasant stimuli. This may lead to allodynia (pain from normally non-painful stimuli) or hyperalgesia (increased sensitivity to pain). When visceral discomfort sensitizes somatic sensory pathways, the brain may erroneously assume that pain sensations from both somatic and visceral regions come from the same source. **[13]**

C. Embryological and Anatomical Basis
Referred pain may occur because somatic tissues and visceral organs share embryological segments, according to the theory of embryological development. For instance, heart pain is often directed to the left shoulder and arm since the nerves innervating the heart and the arm come from the same spinal region (T1–T4). **[14]**

D. Somatic and Visceral Pain Pathways Interaction
Visceral pain is usually extensive and poorly localized, but somatic pain is often localized and intense. When two distinct forms of pain converge at the same spinal segment, the brain misinterprets the signal, giving the impression that the pain is coming from a separate location. Visceral nociceptive fibres travel via the sympathetic nervous system before entering the spinal cord at the dorsal horn levels. Somatic sense fibres and neurons often share circuits there. This contact may lead to referred discomfort in faraway areas. **[16]**

2. Examples of Referred Pain
Cardiac Pain (Angina or Myocardial Infarction)
Heart pain is often referred to the left arm, neck, jaw, or upper back. This phenomenon occurs due to the convergence of nociceptive fibers from the heart and the left arm or neck at the same spinal cord segments (C8-T4).

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Gallbladder Pain (Cholecystitis)

Pain from the gallbladder is typically referred to the right upper quadrant of the abdomen or the right shoulder. This is due to the C3–C5 spinal segments being shared by both the gallbladder and diaphragm.

Renal Pain

Kidney pain is often referred to the lower back, flank, or groin, due to the common T10–T12 spinal segments that receive sensory input from both the kidneys and the lower abdomen.

Somatic Musculoskeletal Pain and Visceral Pain

Pain in the abdominal muscles due to gastrointestinal issues (e.g., gastritis or appendicitis) can be perceived as pain in the shoulder, particularly in acute cholecystitis due to diaphragmatic irritation. **[11]**

3. Clinical Relevance of Referred Pain

Referred pain is essential for diagnosing visceral disorders, especially in emergency conditions. For instance, the commonly referred pain associated with heart attacks might be confused with musculoskeletal or gastrointestinal pain, which delays diagnosis and treatment. By better understanding the patterns of transferred pain, clinicians can better tailor treatment approaches and identify the root source of pain. **[16]**

Endogenous analgesic system and its significance

"Endogenous analgesic system" refers to the way the body automatically inhibits or lessens pain. The central nervous system (CNS), which consists of the brain, spinal cord, and peripheral nervous system, can reduce pain by regulating neurochemicals. When someone is hurt, sick, or under stress, this system is crucial for maintaining homeostasis and helping them manage their pain.

1. Components of the Endogenous Analgesic System

The endogenous analgesic system includes various neurotransmitters, neuromodulators, and brain structures that work together to reduce the perception of pain. Key components of this system include:

A. Opioid System

The opioid system is essential for endogenous analgesia because it releases endogenous opioid peptides such as endorphins, enkephalins, and dynorphins that attach to opioid receptors (mu, delta, and kappa). Endogenous opioids block the transmission of pain at several points along the pain pathway, including the spinal cord, brainstem, and cortex. The main endogenous ligands for mu-opioid receptors, which are particularly linked to euphoria and analgesia, are endorphins. **[17]**

B. Descending Inhibitory Pathways

Descending channels from the brainstem influence the pain signal at the spinal cord level. Important brain regions involved in these pathways include the rostral ventromedial medulla (RVM), periaqueductal grey (PAG), and locus coeruleus (LC). PAG is a crucial area for pain management. It interacts with the RVM, which generates neurotransmitters that block pain signals from getting to the spinal cord and is set off by stressful situations. Norepinephrine and serotonin (5-HT) are neurotransmitters that operate on spinal interneurons to prevent nociceptive transmission in these routes. **[18]**

C. Endocannabinoid System

The endocannabinoid system also helps regulate endogenous pain by generating endocannabinoids such anandamide and 2-arachidonoylglycerol (2-AG). These substances bind to CB1 and CB2 receptors, which are present in peripheral tissues as well as the central nervous system. Activation of CB1 receptors (mostly in the brain and spinal cord) reduce pain signals and has analgesic benefits, especially in chronic pain conditions. The peripheral nervous system and immune cells are the main locations for CB2 receptors, which have analgesic and anti-inflammatory qualities. **[19]**

D. GABA and Glycinergic Systems

The brainstem and spinal cord use inhibitory neurotransmitters like glycine and GABA (gamma-aminobutyric acid) to control pain. By acting on GABA-A receptors, GABA reduces the transmission of pain and suppresses the synthesis of excitatory neurotransmitters. Glycine works similarly in the spinal cord by obstructing nociceptive signaling pathways. **[20]**

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E. Nitric Oxide (NO) and Substance P

Nitric oxide (NO), a signaling molecule, plays a part in the activation of endogenous analgesia, particularly in spinal cord pain pathways. Substance P is a neuropeptide that plays a role in the transmission of pain signals. In certain situations, endogenous opioids and nitric oxide can inhibit substance P release, which lessens the experience of pain. **[21]**

2. Brain Regions Involved in Endogenous Analgesia

A. Periaqueductal Gray (PAG)

The PAG in the midbrain serves as the focal point of the body's natural pain control mechanism. It has a high concentration of opioid receptors and is activated by both stress and pain signals. When the PAG is active, it sends descending signals to the rostral ventromedial medulla (RVM), which controls spinal nociceptive transmission. Furthermore, stress-induced analgesia—a state in which the body's stress response inhibits the perception of pain—is influenced by the PAG. **[22]**

B. Rostral Ventromedial Medulla (RVM)

The RVM is a crucial relay station that modifies the way pain signals are transmitted in the spinal cord and trigeminal pathways.The RVM releases a number of neurochemicals, including serotonin and enkephalins, to inhibit pain signals via descending pathways. The PAG triggers the RVM, which releases opioids that block pain transmission. **[23]**

3. Clinical Significance of the Endogenous Analgesic System

A. Pain Management

Knowledge of the endogenous analgesic system has a major influence on pain treatment. The opioid system's role in natural pain suppression is a significant component of opioid analgesic therapy and its disadvantages, including tolerance and dependency. Interventions that target the PAG, RVM, and endocannabinoid system are being researched to enhance natural pain control without only relying on pharmaceutical opioids.

B. Stress and Pain

The endogenous analgesic system is closely linked to stress-induced analgesia, a condition in which the perception of pain is altered in stressful or injury-related circumstances. Understanding these pathways can help treat chronic pain conditions like fibromyalgia or chronic back pain that involve dysregulation of the endogenous analgesic system.

C. Chronic Pain and Dysfunction

Chronic pain syndromes such as neuropathy, fibromyalgia, and irritable bowel syndrome are thought to be associated with disruptions in the endogenous analgesic system. For example, a breakdown in the descending inhibitory circuits or a reduction in the release of opioid peptides may cause exaggerated pain responses. **[24]**

Specific Treatments Based on the Endogenous Analgesic System

The endogenous analgesia system, which includes neural circuits, neurotransmitters, and modulators that naturally reduce pain, offers many potential pathways for therapeutic intervention. Treatments based on these systems aim to duplicate or enhance the body's natural ability to control pain in order to reduce reliance on external drugs such as opioids. The body's natural analgesia system is the focus of the specific therapies listed below:

1. Opioid-based Treatments and Modulation

A. Opioid Agonists

Opioids (like morphine and fentanyl) directly mimic endogenous opioid peptides, such endorphins and enkephalins, which bind to opioid receptors (mu, delta, and kappa) in the central nervous system and stop pain from being transmitted. These drugs imitate the effects of endogenous opioids by activating the opioid receptors, which results in analgesia and euphoria. Although opioids are very effective in reducing pain, they have risks, including tolerance, addiction, and dependency. Research has focused on biassed agonists or drugs that selectively activate pain-relieving pathways (like mu-receptor activation) while avoiding adverse side effects (like addiction). **[25]**

B. Endogenous Opioid Enhancement

Some treatments aim to increase the body's own production of endorphins and enkephalins to boost the endogenous analgesic response. Exercise and acupuncture are examples of therapies that activate the release of endogenous opioids, thus contributing to pain relief. [**26]**

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2. Targeting Descending Inhibitory Pathways

A. Transcranial Magnetic Stimulation (TMS)

TMS is a non-invasive treatment that uses magnetic fields to stimulate certain parts of the brain, including the periaqueductal grey (PAG), which is important for descending pain modulation. TMS has been researched in relation to neuropathic pain, migraines, and fibromyalgia, among other chronic pain conditions. Research has shown that it triggers descending circuits that reduce pain signals. Studies show that TMS can provide long-lasting pain relief without the need for prescription drugs by enhancing the body's natural analgesic response. **[27]**

B. Spinal Cord Stimulation (SCS)

In order to transmit electrical impulses that activate descending inhibitory circuits from the brainstem to the spinal cord, an electrode is inserted into the spinal cord during spinal cord stimulation. It is believed that the stimulation improves pain inhibition by modifying the release of endorphins, serotonin, and norepinephrine. Chronic neuropathic pain, such as complex regional pain syndrome (CRPS) and failed back surgery syndrome is frequently treated using this technique. **[28]**

3. Endocannabinoid System Modulation

A. Cannabinoids and Cannabinoid Receptor Agonists

The endocannabinoid system aids in pain modulation through its interactions with CB1 and CB2 receptors. Activating CB1 receptors in the brain and spinal cord (CNS) and CB2 receptors in the immune system can reduce pain and inflammation. Cannabis-based medications, such the synthetic cannabinoid Nabilone and the THC and CBD combo Sativex, have been shown to have analgesic effects in conditions like multiple sclerosis pain, cancer pain, and neuropathic pain. The analgesic properties of cannabidiol (CBD), a non-psychoactive compound derived from cannabis that lacks THC's addictive tendency, are also being studied**.[29]**

B. Endocannabinoid Enhancement

Anandamide is one of the endocannabinoids that the body naturally makes and can be increased to reduce pain. Drugs that inhibit fatty acid amide hydrolase (FAAH), an enzyme that degrades anandamide, are being researched to enhance endocannabinoid tone and reduce pain perception. This method is now being researched for two chronic pain conditions: osteoarthritis and neuropathic pain. **[30]**

4. Modulation of GABAergic and Glycinergic Systems

A. GABA Receptor Modulators

Gamma-aminobutyric acid, or GABA, is a significant inhibitory neurotransmitter that helps reduce pain. Pain perception can be modulated by medications that improve GABAergic signaling. By boosting the activation of GABA receptors in the brain and spinal cord, benzodiazepines (like diazepam) and Gaba pentinoids (like gabapentin, pregabalin) are frequently used to treat neuropathic pain. **[31]**

B. Glycinergic Pathways

The glycinergic pathway also plays a role in pain inhibition at the spinal cord level. Through its action on glycine receptors, particularly in the dorsal horn, glycine prevents the transmission of pain. Research is being done on the potential of glycine receptor agonists as analgesics for conditions like neuropathic and inflammatory pain. **[32]**

5. Stress-Induced Analgesia and Psychological Interventions

A. Mindfulness and Cognitive Behavioral Therapy (CBT)

Psychological therapies such as mindfulness meditation and cognitive behavioral therapy have been shown to activate the body's natural pain inhibitory systems by fortifying the descending pain control pathways. By promoting the release of endogenous opioids and neurotransmitters such as serotonin and endorphins, these medicines can reduce chronic pain**. [33]**

II. CONCLUSION

The fact that the paths of pain from the face and trunk are mediated by distinct neuroanatomical systems, even though both are crucial to the overall pain experience, demonstrates the complex nature of pain processing in the human body.

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The trigeminal nerve controls facial pain through a well-defined circuit that consists of the brainstem, thalamus, and trigeminal ganglion. Cortical components of this circuit modulate the experience of pain. Acute and chronic pain sensations are associated with A-delta and C fibres, respectively, and pain from the trunk is mostly transmitted by spinal pathways, including the spinothalamic tract. Both systems are affected by the periaqueductal grey (PAG) and the rostral ventromedial medulla (RVM), two descending modulatory processes that, depending on the situation of the body, can either amplify or diminish pain signals.

Understanding these intricate pain pathways is essential for improving pain management and developing more targeted, effective treatments for a range of pain disorders, such as trigeminal neuralgia, chronic musculoskeletal pain, and neuropathic pain. Advances in neurostimulation, neuropharmacology, and neuroplasticity research are continuously revealing new therapeutic opportunities to minimize side effects and minimize pain. Given the unique characteristics of pain in different body areas, the importance of customized pain management strategies is further highlighted by the distinct and overlapping nature of these pain pathways. Future research on neuroplastic changes, pain modulation, and targeted therapeutics will be crucial to enhancing chronic pain patients' quality of life and pain management.

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