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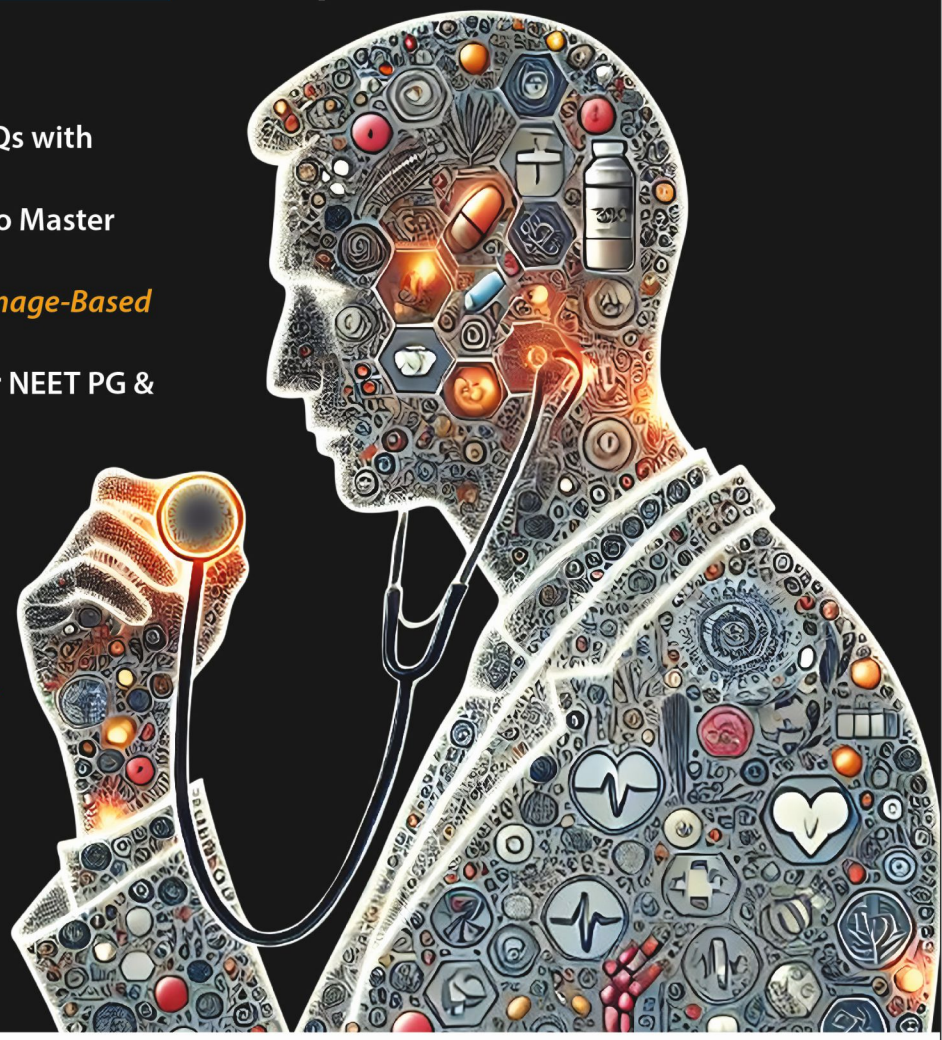
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SECTION 1

General Medicine

1. **Which of the following fibers are primarily responsible for transmitting sharp, well-localized pain?**

a. A-beta fibers
b. A-delta fibers
c. C fibers
d. A-alpha fibers

EXPLANATION

Answer: b. A-delta fibers

A-delta fibers are myelinated fibers that transmit sharp, well-localized pain. They carry fast pain signals and are activated by mechanical or thermal stimuli, producing an immediate pain sensation. ***A-beta fibers*** are large, myelinated fibers involved in the transmission of touch, pressure, and vibration sensations and are not primarily involved in pain transmission. ***C fibers*** are unmyelinated fibers that transmit dull, aching, or throbbing pain. The pain transmitted by C fibers is slow and poorly localized. ***A-alpha fibers*** are responsible for proprioception and motor function. They do not play a role in pain transmission.

2. **A 35-year-old male presents with severe, unilateral headache around the eye, associated with tearing and redness of the eye, lasting for 30 minutes and multiple attacks over a few weeks. What is the most likely diagnosis?**

a. Migraine
b. Tension headache
c. Cluster headache
d. Trigeminal neuralgia

EXPLANATION

Answer: c. Cluster headache

Cluster headache are severe, unilateral, and often localized around the eye. They occur in clusters (multiple attacks over a few weeks), and autonomic symptoms like tearing, nasal congestion, and ptosis are common. ***Migraines*** are often unilateral and associated with throbbing pain, nausea, photophobia, and phonophobia. However, they last for hours to days, not in short clusters like cluster headaches. ***Tension headache*** are usually bilateral, with a band-like pressure around the head. They are not associated with autonomic symptoms such as tearing and eye redness. ***Trigeminal neuralgia*** causes sharp, stabbing facial pain in the distribution of the trigeminal nerve but lacks the autonomic features seen in cluster headaches.

3. **The emotional component of pain is primarily processed in which of the following areas of the brain?**

a. Somatosensory cortex
b. Insular cortex
c. Amygdala
d. Thalamus

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EXPLANATION

Answer: c. Amygdala

Amygdala is crucial for the emotional processing of pain, including the generation of fear, anxiety, and emotional distress in response to pain stimuli. ***Somatosensory cortex*** processes the sensory aspects of pain, such as the location, intensity, and quality of the pain stimulus, but not the emotional response to it. ***Insular cortex*** integrates sensory, emotional, and cognitive information related to pain, but the amygdala is more directly involved in emotional processing. ***Thalamus*** acts as a relay center for sensory signals, including pain, but it does not directly process the emotional aspect of pain.

4. A 45-year-old woman presents with burning pain in her feet that worsens at night. She also has diminished ankle reflexes and reduced sensation in a stocking-glove pattern. What is the most likely underlying cause of her pain?

a. Diabetic neuropathy
b. Complex regional pain syndrome
c. Multiple sclerosis
d. Trigeminal neuralgia

EXPLANATION

Answer: a. Diabetic neuropathy

Diabetic neuropathy is common in patients with diabetes and presents with burning, tingling pain in a stocking-glove distribution. Diminished reflexes and sensory loss are common due to peripheral nerve damage from chronic hyperglycemia. ***Complex regional pain syndrome*** typically occurs after trauma and is characterized by severe pain, swelling, and changes in skin appearance, often affecting a single limb. ***Multiple sclerosis*** can cause neuropathic pain, but it typically presents with neurological deficits like muscle weakness, vision problems, and coordination issues, not a stocking-glove pattern of sensory loss. ***Trigeminal neuralgia*** causes facial pain in the distribution of the trigeminal nerve and does not involve the feet or a stocking-glove distribution.

5. Which of the following is the primary neurotransmitter involved in the transmission of pain signals in the spinal cord?

a. Acetylcholine
b. Glutamate
c. Dopamine
d. Serotonin

EXPLANATION

Answer: b. Glutamate

Glutamate is the main excitatory neurotransmitter responsible for transmitting nociceptive (pain) signals in the spinal cord. It plays a key role in fast synaptic transmission of pain. ***Acetylcholine*** is involved in neuromuscular junction transmission and parasympathetic nervous system functions, not in pain transmission. ***Dopamine*** is involved in the reward and motivation pathways, as well as motor control, but is not the primary neurotransmitter for pain transmission. ***Serotonin*** is involved in mood regulation and can modulate pain, but it is not the primary neurotransmitter responsible for pain signal transmission.

6. In chronic pain conditions, pain persists beyond the expected period of tissue healing. Which of the following mechanisms is most commonly implicated in the development of chronic pain?

a. Peripheral sensitization
b. Central sensitization
c. Referred pain
d. Neuropathic pain

EXPLANATION

Answer: b. Central sensitization

Central sensitization refers to increased sensitivity within the central nervous system, leading to persistent pain and amplification of pain signals even after tissue healing. It is a key mechanism in chronic pain syndromes. ***Peripheral sensitization*** occurs when nociceptors become more sensitive to stimuli after tissue damage. It contributes to acute pain but is not the primary mechanism in chronic pain. ***Referred pain*** is felt in a location distant from the actual source of pain, often due to shared neural pathways, but it is not a mechanism of chronic pain. ***Neuropathic pain*** arises from damage to the nervous system and can be chronic, but it is a specific type of chronic pain rather than a mechanism.

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7. **A 50-year-old male develops pain and stiffness in his neck, shoulders, and hips, which is worse in the morning and improves throughout the day. ESR is elevated. What is the most likely diagnosis?**

a. Rheumatoid arthritis
b. Polymyalgia rheumatica
c. Osteoarthritis
d. Fibromyalgia

EXPLANATION

Answer: b. Polymyalgia rheumatica

Polymyalgia rheumatica causes pain and stiffness in the neck, shoulders, and hips, particularly in older adults. Elevated ESR is a hallmark of this condition, and morning stiffness improves with activity. ***Rheumatoid arthritis*** causes joint pain and stiffness, especially in the small joints of the hands and feet, and may also have elevated ESR. However, it typically presents with joint swelling and deformities, which are not seen in polymyalgia rheumatica. ***Osteoarthritis*** causes joint pain due to wear and tear, but it is more common in weight-bearing joints like the knees and hips and does not cause elevated ESR. ***Fibromyalgia*** involves widespread musculoskeletal pain, but it does not typically present with elevated ESR or morning stiffness localized to the shoulders and hips.

8. **Which of the following is the most appropriate first-line treatment for neuropathic pain?**

a. NSAIDs
b. Acetaminophen
c. Gabapentin
d. Opioids

EXPLANATION

Answer: c. Gabapentin

Gabapentin, an anticonvulsant, is a first-line treatment for neuropathic pain. It works by modulating calcium channels and reducing neuronal excitability. ***NSAIDs*** are effective for nociceptive pain but have limited efficacy in neuropathic pain. ***Acetaminophen*** is useful for mild to moderate nociceptive pain but is not effective for neuropathic pain. While ***Opioids*** can be used for severe neuropathic pain, they are generally not first-line due to their risk of addiction and side effects.

9. **A patient presents with intense, lancinating pain in the face, triggered by light touch. The pain is located in the distribution of the maxillary branch of the trigeminal nerve. What is the most appropriate first-line treatment?**

a. Carbamazepine
b. NSAIDs
c. Amitriptyline
d. Gabapentin

EXPLANATION

Answer: a. Carbamazepine

Carbamazepine is the drug of choice for trigeminal neuralgia. It is an anticonvulsant that stabilizes inactivated sodium channels, reducing nerve excitability. ***NSAIDs*** are ineffective in treating trigeminal neuralgia, as the condition is not inflammatory in nature. ***Amitriptyline*** is a tricyclic antidepressant used for neuropathic pain but is less effective than carbamazepine for trigeminal neuralgia. ***Gabapentin*** may be used in neuropathic pain, but carbamazepine is preferred for trigeminal neuralgia due to its superior efficacy.

10. **Which of the following fibers is primarily responsible for transmitting pain signals from intense mechanical or thermal stimuli?**

a. A-beta fibers
b. A-delta fibers
c. C fibers
d. A-alpha fibers

EXPLANATION

Answer: b. A-delta fibers

A-beta fibers are involved in transmitting nonpainful touch, pressure, and vibration. They are large-diameter, myelinated fibers and do not transmit pain signals. ***A-delta fibers*** are small-diameter, myelinated fibers that transmit sharp, well-localized pain from intense mechanical and thermal stimuli. They are involved in the perception of "fast pain." ***C fibers*** are unmyelinated fibers that transmit dull, diffuse, aching pain. C fibers carry "slow pain" signals from stimuli like heat and chemicals. ***A-alpha fibers***: These are large-diameter fibers primarily involved in motor function and proprioception, not in pain transmission.

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11. Which of the following best describes the phenomenon of "allodynia"?

a. Increased pain intensity in response to a normally painful stimulus
b. Pain perception from nonpainful stimuli
c. Increased sensitivity to temperature changes
d. Loss of sensation following nerve injury

EXPLANATION

Answer: b. Pain perception from nonpainful stimuli

Allodynia refers to pain resulting from stimuli that do not usually provoke pain, such as light touch or mild pressure, therefore statement ***'Increased pain intensity in response to a normally painful stimulus'*** is correct. Statement ***'Increased pain intensity in response to a normally painful stimulus'*** describes hyperalgesia, not allodynia. Therefore, it is incorrect. Statement ***'Increased sensitivity to temperature changes'*** is incorrect as it is more characteristic of thermal hyperalgesia, not allodynia. The statement '***Loss of sensation following nerve injury'*** is incorrect as it describes hypoesthesia or numbness, not allodynia.

12. What type of receptor is the transient receptor potential cation channel subfamily V member 1 (TrpV1), and what does it primarily respond to?

a. Mechanoreceptor; responds to mechanical deformation
b. Nociceptor; responds to intense cold
c. Thermoreceptor; responds to low temperatures
d. Nociceptor; responds to heat and capsaicin

EXPLANATION

Answer: d. Nociceptor; responds to heat and capsaicin

The transient receptor potential cation channel subfamily V member 1 (TrpV1) is ***nociceptor*** and it primarily ***respond to heat and capsaicin.*** Hence, option d is correct. TrpV1 is not a mechanoreceptor. It responds to thermal and chemical stimuli, not mechanical deformation. Hence, option a is incorrect. TrpV1 is activated by heat, not cold. There are other receptors (e.g., TRPA1) involved in cold sensation. Hence, option b is incorrect. TrpV1 responds to high temperatures, not low ones. Hence, option c is incorrect.

13. Which of the following is NOT a mediator involved in peripheral sensitization?

a. Bradykinin
b. Prostaglandins
c. Norepinephrine
d. Nerve growth factor

EXPLANATION

Answer: c. Norepinephrine

Norepinephrine is a neurotransmitter involved in the sympathetic nervous system and is not directly involved in the sensitization of peripheral nociceptors. ***Bradykinin*** is an inflammatory mediator that increases the sensitivity of nociceptors, contributing to peripheral sensitization. ***Prostaglandins*** are also involved in inflammatory responses and enhance nociceptor sensitivity. ***Nerve growth factor*** plays a significant role in promoting the sensitivity of nociceptors and is involved in peripheral sensitization.

14. Which of the following mechanisms is responsible for increased pain sensation in inflamed or injured tissues?

a. Central sensitization
b. Peripheral sensitization
c. Hyperalgesia
d. Allodynia

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EXPLANATION

Answer: b. Peripheral sensitization

Peripheral sensitization occurs when nociceptors in damaged or inflamed tissues become more sensitive, leading to increased excitability and a lowered threshold for activation. This process underlies the increased pain response in injured tissues. ***Central sensitization*** refers to an increase in the excitability of neurons in the dorsal horn of the spinal cord and contributes to chronic pain, but it is not specific to injured or inflamed tissues. ***Hyperalgesia*** is a clinical manifestation of peripheral sensitization, characterized by an exaggerated response to painful stimuli, but it is not a mechanism itself. ***Allodynia*** is pain from normally nonpainful stimuli, but it is not specific to inflamed or injured tissues.

15. Which of the following inflammatory mediators is known to sensitize nociceptors and lower their threshold for activation?

a. Histamine
b. Acetylcholine
c. Dopamine
d. Glutamate

EXPLANATION

Answer: a. Histamine

Histamine which is, released from mast cells during inflammation, contributes to the sensitization of nociceptors and increases pain perception by lowering the threshold for activation. Hence, option a is correct. ***Acetylcholine*** is a neurotransmitter involved in neuromuscular transmission and is not a key mediator in nociceptor sensitization. Hence, option b is incorrect. ***Dopamine*** is primarily involved in reward pathways and motor control but is not a significant mediator in pain sensitization. Hence, option c is incorrect. ***Glutamate*** is an excitatory neurotransmitter in the central nervous system, contributing to pain signal transmission, but it is not an inflammatory mediator. Therefore, option d is incorrect.

16. A patient with sunburned skin feels severe pain when lightly touched. What is the most likely mechanism for this increased pain sensitivity?

a. Central sensitization
b. Peripheral sensitization
c. Hyperalgesia
d. Allodynia

EXPLANATION

Answer: b. Peripheral sensitization

In sunburned skin, inflammatory mediators increase the sensitivity of peripheral nociceptors, leading to pain from normally nonpainful stimuli (like a light touch). Hence, option b ***Peripheral sensitization*** is correct. ***Central sensitization*** occurs at the level of the spinal cord and CNS, typically in chronic pain states. It is not the primary mechanism in this scenario. Hence, option a is incorrect. While ***hyperalgesia*** refers to increased pain in response to painful stimuli, the light touch in this case is normally nonpainful, so this term is not applicable. Hence, option c is incorrect. ***Allodynia*** refers to pain from nonpainful stimuli (like light touch), but in this case, the underlying mechanism is peripheral sensitization. Hence, option d is incorrect.

17. Which of the following is the key receptor involved in nociceptor activation by heat and capsaicin?

a. TRPA1
b. TRPV1
c. NMDA receptor
d. GABA receptor

EXPLANATION

Answer: b. TRPV1

TRPV1(transient receptor potential vanilloid 1) is the key receptor activated by heat, acidic pH, and capsaicin, which leads to the sensation of burning pain. ***TRPA1*** is activated by cold temperatures and irritants, not by heat or capsaicin. ***NMDA receptors*** are involved in synaptic plasticity and pain transmission in the CNS but are not directly activated by heat or capsaicin. ***GABA receptor*** are inhibitory and are not involved in nociceptor activation.

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18. Which of the following mediators is MOST LIKELY to cause vasodilation and mast cell degranulation when released by primary afferent nociceptors?

a. Bradykinin
b. Prostaglandins
c. Substance P
d. Leukotrienes

EXPLANATION

Answer: c. Substance P

Substance P is a neuropeptide released from peripheral terminals of nociceptors, and it causes vasodilation, mast cell degranulation, and the release of other inflammatory mediators. It plays a significant role in neurogenic inflammation. ***Bradykinin*** is a potent inflammatory mediator that causes pain and increases vascular permeability, but it is not the primary mediator for mast cell degranulation and vasodilation initiated by nociceptors. ***Prostaglandins*** sensitize nociceptors to other stimuli, contributing to inflammation and pain, but do not directly cause mast cell degranulation. ***Leukotrienes*** are involved in the inflammatory response and can increase vascular permeability, but they are not typically involved in the processes directly triggered by nociceptor activity, like substance P does.

19. Silent nociceptors become sensitized and responsive to mechanical stimuli due to which of the following mechanisms?

a. Decreased substance P release
b. Inflammatory mediator activation
c. Increased myelination of C fibers
d. Loss of sympathetic innervation

EXPLANATION

Answer: b. Inflammatory mediator activation

Inflammatory mediators such as bradykinin (BK), prostaglandins (PGs), and leukotrienes activate silent nociceptors, which are normally insensitive to stimuli but become responsive in pathological conditions like inflammation. Hence, option b is correct. Decreased substance P release would reduce inflammation and sensitivity, not increase it. Silent nociceptors become active due to an increase in inflammatory mediators, not a decrease. Hence, option a is incorrect. Silent nociceptors are primarily unmyelinated C fibers. Myelination is not involved in their sensitization. Hence, option c is incorrect. While the sympathetic nervous system can influence pain responses, sensitization of silent nociceptors primarily occurs through inflammatory mediators, not loss of sympathetic innervation. Hence, option d is incorrect.

20. Which of the following is the MOST COMMON mechanism underlying peripheral sensitization in inflamed tissues?

a. Increased conduction velocity of A-delta fibers
b. Blockade of voltage-gated sodium channels
c. Upregulation of chemically gated and voltage-gated ion channels
d. Decreased sensitivity of A-beta fibers

EXPLANATION

Answer: c. Upregulation of chemically gated and voltage-gated ion channels

Peripheral sensitization in inflamed tissues occurs due to an increase in ion channels (chemically gated and voltage-gated) on the nociceptors. This makes the nociceptors more responsive to stimuli, lowering their activation threshold. Hence, statement ***'upregulation of chemically gated and voltage-gated ion channels***' is correct. Conduction velocity changes are not a major mechanism of sensitization. Sensitization occurs at the level of threshold changes, not necessarily conduction speed. Hence, statement ***'increased conduction velocity of A-delta fibers'*** is incorrect. Blocking sodium channels would decrease nociceptor activity, not increase it. Sensitization involves enhanced excitability, not blockade. Hence, statement ***'blockade of voltage-gated sodium channels'*** is incorrect. A-beta fibers are not primarily involved in nociceptive signaling under normal conditions. Peripheral sensitization primarily involves nociceptive fibers (A-delta and C fibers). Hence, statement ***'decreased sensitivity of A-beta fibers'*** is incorrect.

21. Which inflammatory mediator is MOST LIKELY responsible for lowering the threshold for activation of nociceptors in sensitized tissue?

a. Histamine
b. Bradykinin
c. Serotonin
d. Adenosine triphosphate (ATP)

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EXPLANATION

Answer: b. Bradykinin

Bradykinin is one of the most potent mediators that lower the threshold for nociceptor activation, especially in inflammatory conditions, thereby contributing to peripheral sensitization. ***Histamine*** is involved in inflammation and allergic responses, but its role in directly lowering nociceptor thresholds is less significant compared to bradykinin. ***Serotonin*** (5-HT) plays a role in pain modulation, but bradykinin is the primary mediator involved in lowering nociceptor thresholds during sensitization. ***Adenosine triphosphate (ATP)*** is a signaling molecule involved in pain pathways but does not play as direct a role in lowering nociceptor thresholds as bradykinin.

22. Which neurotransmitter is primarily responsible for rapid excitation of second-order neurons in the dorsal horn during nociception?

a. Substance P
b. Glutamate
c. CGRP
d. Serotonin

EXPLANATION

Answer: b. Glutamate

Glutamate is the primary excitatory neurotransmitter that is released rapidly from the terminals of primary afferent nociceptors, causing fast excitation of second-order neurons in the dorsal horn. ***Substance P*** is involved in slower, longer-lasting excitation and is released by nociceptors, but it is not responsible for the rapid excitation of second-order neurons. ***CGRP*** (Calcitonin Gene-Related Peptide) is involved in modulating nociception and plays a role in vasodilation, but it does not mediate rapid excitation. ***Serotonin*** is involved in pain modulation but is not the primary neurotransmitter for rapid excitation in nociceptive pathways.

23. Which of the following is MOST associated with the "affective" or emotional aspect of pain perception?

a. Somatosensory cortex
b. Cingulate cortex
c. Thalamus
d. Spinothalamic tract

EXPLANATION

Answer: b. Cingulate cortex

Cingulate cortex is involved in the emotional or affective dimension of pain, which produces suffering and influences behavior. ***Somatosensory cortex*** processes the sensory-discriminative aspect of pain, such as its location, intensity, and quality, but not the emotional aspect. While the ***Thalamus*** relays pain signals to both the sensory and emotional centers, it is not primarily responsible for the emotional response itself. ***Spinothalamic tract*** is essential for transmitting pain and temperature signals to the thalamus, but it is not directly responsible for processing the emotional aspect of pain.

24. Which pathway is responsible for transmitting pain and temperature signals to the contralateral thalamus?

a. Dorsal column-medial lemniscus pathway
b. Spinocerebellar tract
c. Spinothalamic tract
d. Corticospinal tract

EXPLANATION

Answer: c. Spinothalamic tract

Dorsal column-medial lemniscus pathway transmits fine touch, vibration, and proprioception, not pain and temperature. ***Spinocerebellar tract*** transmits proprioceptive information to the cerebellum, not pain or temperature signals. ***Spinothalamic tract*** transmits pain and temperature signals from the spinal cord to the contralateral thalamus. ***Corticospinal tract*** is involved in motor control, not pain transmission.

25. Which of the following best explains the phenomenon of referred pain?

a. Divergence of pain signals to multiple thalamic nuclei
b. Cross-talk between adjacent dorsal root ganglia

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c. Convergence of sensory inputs onto the same spinal neurons
d. Inhibition of A-beta fibers by C fibers

EXPLANATION

Answer: c. Convergence of sensory inputs onto the same spinal neurons

Referred pain occurs when visceral and somatic sensory inputs converge on the same spinal neurons, leading to mislocalization of the pain. Hence, statement ***'convergence of sensory inputs onto the same spinal neurons'*** is correct. ***Divergence of pain signals to multiple thalamic nuclei*** explains how pain signals are processed in the brain but does not directly account for referred pain. Hence, this statement is incorrect. ***Cross-talk between adjacent dorsal root ganglia*** is not the primary mechanism for referred pain. Referred pain is due to the convergence of inputs at the spinal level. ***Inhibition of A-beta fibers by C fibers:*** This mechanism relates more to the gating of pain signals, not referred pain.

26. Which region of the brain is involved in the sensory-discriminative aspect of pain, including its intensity and location?

a. Cingulate cortex
b. Insular cortex
c. Somatosensory cortex
d. Amygdala

EXPLANATION

Answer: c. Somatosensory cortex

Somatosensory cortex is responsible for the sensory-discriminative aspect of pain, helping to identify the intensity, location, and quality of the pain. ***Cingulate cortex*** is involved in the emotional or affective component of pain, not its sensory-discriminative aspect. ***Insular cortex*** processes aspects of emotional pain but does not primarily deal with sensory-discriminative aspects like intensity or location. ***Amygdala*** is involved in processing fear and emotional responses but does not directly handle the sensory-discriminative dimension of pain.

27. Which of the following factors is MOST likely to lead to a decreased perception of pain in individuals?

a. High emotional stress
b. Anticipation of pain
c. Opioid analgesics
d. Focused attention on a distraction

EXPLANATION

Answer: c. Opioid analgesics

Opioids bind to specific receptors in the brain and spinal cord, effectively reducing the perception of pain and providing analgesia. Hence, option c is correct. ***High emotional stress*** is likely to increase pain perception rather than decrease it due to heightened awareness and anxiety. ***Anticipation*** typically increases pain perception, as the expectation can enhance the emotional response to pain. While ***distraction*** can mitigate pain perception, it may not be as potent as the analgesic effects of opioids.

28. The phenomenon where an inert substance is perceived to worsen pain when suggested is known as:

a. Placebo effect
b. Nocebo effect
c. Hyperalgesia
d. Allodynia

EXPLANATION

Answer: b. Nocebo effect

Nocebo effect occurs when negative expectations lead to a perceived worsening of symptoms, including pain. ***Placebo effect*** describes the reduction of pain following a treatment with no therapeutic value due to belief in its efficacy. ***Hyperalgesia*** refers to an increased sensitivity to pain, which is not related to the expectation of pain. ***Allodynia*** involves pain due to stimuli that do not normally provoke pain; it does not pertain to expectation.

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29. Which brain structure is primarily involved in the descending modulation of pain transmission?

a. Cerebellum
b. Periaqueductal gray (PAG)
c. Hippocampus
d. Thalamus

EXPLANATION

Answer: b. Periaqueductal gray (PAG)

The ***periaqueductal gray (PAG)*** is a key area in the brain that is involved in the descending modulation of pain, interacting with structures in the medulla and spinal cord. The ***cerebellum*** is primarily involved in coordination and balance, not in pain modulation. While the ***hippocampus*** is important for memory, it does not play a direct role in pain modulation. The ***thalamus*** relays sensory information, including pain, but does not modulate pain transmission directly.

30. In which scenario would the endogenous opioid system MOST likely be activated?

a. During a minor surgical procedure
b. When receiving a placebo treatment for pain
c. During an intense emotional event
d. Both b and c

EXPLANATION

Answer: d. Both b and c

Placebo treatments can activate the endogenous opioid system, leading to pain relief. Hence, ***option b is correct***. Also, intense emotions can trigger the release of endogenous opioids that help modulate pain. Hence, ***option c is correct***. Endogenous opioids may be released, but the strongest activation occurs in response to suggestion or emotional stimuli. Hence, ***option a is incorrect***.

31. What is the primary mechanism behind the analgesic effect of attention in pain perception?

a. Increased peripheral sensitivity
b. Enhanced descending inhibition
c. Activation of nociceptors
d. Stimulation of A-delta fibers

EXPLANATION

Answer: b. Enhanced descending inhibition

Option b ***'enhanced descending inhibition'*** is correct. This is because focusing attention can activate the descending pain modulatory pathways, increasing inhibition of pain signals at the spinal level. ***Increased peripheral sensitivity*** does not describe the mechanism of attention; it would actually lead to increased pain perception. Hence option a is incorrect. ***Option c is incorrect*** because attention does not activate nociceptors; it modulates the perception of pain. ***Option d is incorrect*** because A-delta fibers are involved in fast pain transmission, but attention does not specifically stimulate them.

32. Functional imaging studies have shown that the placebo analgesic response is associated with activity in which brain region?

a. Amygdala
b. Insular cortex
c. Dorsolateral prefrontal cortex (DLPFC)
d. Occipital lobe

EXPLANATION

Answer: c. Dorsolateral prefrontal cortex (DLPFC)

Activity in the ***dorsolateral prefrontal cortex (DLPFC)*** has been associated with the cognitive modulation of pain and the placebo analgesic response. The ***amygdala*** is more related to the emotional response to pain rather than the placebo effect. The ***insular cortex*** is involved in the emotional and sensory processing of pain but is not primarily linked to the placebo analgesic effect. The ***occipital lobe*** is primarily involved in visual processing and is not related to pain modulation.

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33. Which of the following mechanisms is involved in the pathogenesis of neuropathic pain?

a. Increased sodium channel density in damaged neurons
b. Activation of COX-2 enzymes
c. Hypoxia of nerve fibers
d. Autoimmune attack on nociceptors

EXPLANATION

Answer: a. Increased sodium channel density in damaged neurons

Option a is correct because neuropathic pain involves hyperexcitability of damaged neurons due to an increased number of sodium channels, leading to spontaneous activity. ***Activation of COX-2 enzymes*** is a mechanism involved in inflammatory pain, not neuropathic pain. ***Hypoxia*** may cause tissue damage, but it is not directly related to neuropathic pain mechanisms. ***Autoimmune attack on nociceptors*** may occur in some autoimmune diseases but is not the main mechanism of neuropathic pain.

34. Which of the following is an appropriate first-line treatment for postherpetic neuralgia with prominent allodynia?

a. Topical capsaicin
b. Oral morphine
c. Topical 5% lidocaine patch
d. NSAIDs

EXPLANATION

Answer: c. Topical 5% lidocaine patch

Option c is correct as ***lidocaine patches*** are effective for treating allodynia in postherpetic neuralgia. ***Capsaicin*** is sometimes used for neuropathic pain, but it is not the first-line treatment for postherpetic neuralgia. While ***opioids*** can be used for severe pain, topical treatments are preferred initially for localized neuropathic pain. Hence, option b is incorrect. ***NSAIDs*** are primarily used for nociceptive or inflammatory pain and are not as effective in neuropathic pain.

35. Complex Regional Pain Syndrome (CRPS) is characterized by which of the following?

a. Immediate onset of pain after injury
b. Spontaneous pain with signs of sympathetic dysfunction
c. Pain confined to the site of the injury
d. Decreased skin sensitivity over the affected area

EXPLANATION

Answer: b. Spontaneous pain with signs of sympathetic dysfunction

Complex Regional Pain Syndrome (CRPS) is often associated with spontaneous pain, swelling, and other signs of sympathetic nervous system involvement. Hence, option b is correct. CRPS typically has a delayed onset, not immediate. Hence, option a is incorrect. CRPS pain often extends beyond the site of the initial injury. Hence, option c is incorrect. CRPS is often associated with allodynia (increased sensitivity), not decreased sensitivity. Hence, option d is incorrect.

36. Which of the following describes CRPS Type I (Reflex Sympathetic Dystrophy)?

a. Occurs after identifiable nerve injury
b. No identifiable nerve injury is present
c. More common in the upper limbs
d. Can only be treated with NSAIDs

EXPLANATION

Answer: b. No identifiable nerve injury is present

CRPS Type I occurs without clear nerve injury and is often termed reflex sympathetic dystrophy. Hence, option b is correct. CRPS Type II (causalgia). occurs after identifiable nerve injury, not Type I. Hence, option a is incorrect. CRPS can affect any limb, not specifically the upper limbs. Hence, option c is incorrect. Treatment options of CRPS Type I include sympathetic blocks, physical therapy, and medications beyond NSAIDs. Hence, option d is incorrect.

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37. **Which of the following non-narcotic analgesics poses the lowest cardiovascular risk among NSAIDs?**

a. Ibuprofen
b. Naproxen
c. Indomethacin
d. Celecoxib

EXPLANATION

Answer: b. Naproxen

Naproxen is considered the NSAID with the lowest cardiovascular risk. While effective,***Ibuprofen*** has a higher cardiovascular risk than naproxen. ***Indomethacin*** is associated with significant gastrointestinal and cardiovascular risks. ***Celecoxib***, a COX-2 inhibitor, is associated with cardiovascular risks, especially at higher doses.

38. **Which of the following analgesics is contraindicated in patients with renal disease?**

a. Fenoprofen
b. Acetaminophen
c. Ketorolac
d. Ibuprofen

EXPLANATION

Answer: a. Fenoprofen

Fenoprofen is contraindicated in patients with renal disease due to its nephrotoxic potential. ***Acetaminophen*** is relatively safe for use in renal disease, as it has minimal renal toxicity. While ***Ketorolac*** is used parenterally, it should be used with caution in renal impairment but is not contraindicated. Though NSAIDs like ***Ibuprofen*** generally carry a risk in renal impairment, Fenoprofen is specifically contraindicated.

39. **Which of the following opioid analgesics is poorly absorbed orally and has a toxic metabolite, normeperidine?**

a. Methadone
b. Meperidine
c. Oxycodone
d. Hydromorphone

EXPLANATION

Answer: b. Meperidine

Meperidine is poorly absorbed orally and has a toxic metabolite, normeperidine, which can cause seizures with prolonged use. ***Methadone*** is well absorbed orally and has a long half-life, but respiratory depression is a concern. ***Oxycodone*** is well absorbed orally and commonly combined with acetaminophen or aspirin. ***Hydromorphone*** is well absorbed and acts shorter than morphine.

40. **Which of the following is an opioid that has both opioid and adrenergic action?**

a. Codeine
b. Tramadol
c. Fentanyl
d. Buprenorphine

EXPLANATION

Answer: b. Tramadol

Tramadol has a mixed mechanism of action, involving both opioid receptor agonism and inhibition of norepinephrine and serotonin reuptake. ***Codeine*** is a traditional opioid with no adrenergic activity. ***Fentanyl*** is a pure opioid receptor agonist without adrenergic effects. ***Buprenorphine*** is a partial opioid agonist but does not have adrenergic activity.

41. **Which of the following narcotic analgesics is most associated with the risk of delayed respiratory depression due to its long half-life?**

a. Morphine
b. Methadone
c. Hydromorphone
d. Fentanyl

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EXPLANATION

Answer: b. Methadone

Methadone has a long half-life, which can result in cumulative effects and delayed respiratory depression, even after analgesic effects wear off. ***Morphine*** has a shorter half-life compared to methadone, so it does not have the same risk of delayed respiratory depression. ***Hydromorphone*** is shorter-acting than methadone. While ***Fentanyl*** can cause respiratory depression, it has a shorter half-life compared to methadone.

42. Which of the following opioid analgesics is available in a 72-hour transdermal patch?

a. Buprenorphine
b. Fentanyl
c. Oxycodone extended-release
d. Morphine sustained release

EXPLANATION

Answer: b. Fentanyl

Fentanyl is available in a 72-hour transdermal patch and is used for chronic pain management. ***Buprenorphine*** is available in a 7-day transdermal patch. ***Oxycodone*** is available in oral extended-release formulations, not as a transdermal patch. ***Morphine*** is available in oral slow-release forms but not as a transdermal patch.

43. Which of the following analgesics should not be initiated at doses >40 mg/day due to the risk of respiratory depression and sedation?

a. Methadone
b. Oxycodone
c. Levorphanol
d. Morphine

EXPLANATION

Answer: a. Methadone

Methadone has a long and variable half-life and initiating therapy at doses higher than 40 mg/day increases the risk of respiratory depression and sedation. ***Oxycodone*** does not carry the same dosing restrictions as methadone for respiratory depression. ***Levorphanol*** has a long half-life, but the respiratory depression risk is not as pronounced as with methadone. While ***Morphine*** can cause respiratory depression, it is dosed differently from methadone.

44. Which antidepressant has the highest potency for serotonin (5-HT) reuptake blockade?

a. Doxepin
b. Amitriptyline
c. Venlafaxine
d. Nortriptyline

EXPLANATION

Answer: b. Amitriptyline

Amitriptyline has the highest potency for serotonin reuptake blockade among the listed drugs. ***Doxepin*** has moderate serotonin reuptake blockade compared to Amitriptyline. ***Venlafaxine*** has moderate serotonin reuptake blockade but not as high as Amitriptyline. ***Nortriptyline*** has a moderate effect but is not as potent as Amitriptyline for serotonin reuptake.

45. Which antidepressant is least likely to cause orthostatic hypotension?

a. Doxepin
b. Amitriptyline
c. Desipramine
d. Imipramine

EXPLANATION

Answer: c. Desipramine

Desipramine has the lowest risk of orthostatic hypotension. ***Doxepin*** has moderate risk for orthostatic hypotension. ***Amitriptyline*** has the highest risk for orthostatic hypotension. ***Imipramine*** has a higher risk of orthostatic hypotension compared to Desipramine.

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