COMPREHENSIVE MOCK EXAM 3

Supplemental to: Pain Medicine Board Review 2026-2027

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American Board of Anesthesiology (ABA) Pain Medicine Certification Examination -- 200 questions, single-best-answer (3-option A-C), domain-weighted per official ABA blueprint

Board-Review Questions

1. A basic-science study measures conduction velocity in a peripheral nerve fiber that is thinly myelinated, has a conduction velocity of roughly 5-30 m/s, and mediates the sharp, well-localized "first pain" felt immediately after a pinprick. Which fiber type is being described?

A. A-delta fiber

B. C fiber

C. A-beta fiber

2. A researcher studying spinal nociceptive processing identifies a dorsal horn lamina that receives predominantly unmyelinated C-fiber input and is rich in interneurons that release substance P and glutamate, forming a key site for opioid receptor-mediated analgesia. Which Rexed lamina is this?

A. Lamina II (substantia gelatinosa)

B. Lamina IX, in the ventral horn

C. Lamina III, deep dorsal horn

3. A neonatologist counsels a family that their premature infant, born at 28 weeks, may experience pain differently than a term infant because a key modulatory pain pathway is still immature. Which pain-system feature is developmentally delayed in preterm neonates relative to nociceptive afferent pathways?

A. Peripheral nociceptor density in neonatal skin

B. Descending inhibitory (antinociceptive) pathways from the brainstem

C. Spinal reflex withdrawal circuitry in the neonate

4. Investigators studying central sensitization in a pediatric pain model note that low-threshold sensory neurons undergo activity-dependent changes causing normally non-painful stimuli to be perceived as painful. Which developmental pain-system phenomenon does this describe?

A. Nociceptor threshold elevation

B. Allodynia arising from central sensitization

C. Peripheral nerve myelination arrest

5. A randomized controlled trial of a novel analgesic is designed so that neither the participants nor the outcome assessors know which treatment arm a subject was assigned to, while the statistician analyzing unblinded interim data is kept separate from those assessors. What methodologic feature is being maximized by this design?

A. External validity of the trial findings

B. Construct validity of the chosen pain instrument

C. Protection against detection and performance bias via double-blinding

6. A crossover trial for a topical analgesic finds a large reduction in pain scores in the placebo arm, comparable in magnitude to the active-drug arm. The investigators attribute part of this to expectation-driven activation of descending opioidergic pathways rather than to natural history alone. What is this phenomenon called?

A. Regression to the mean across repeat measures

B. Hawthorne effect from being observed

C. Placebo analgesia via endogenous opioid release

7. In a multidisciplinary chronic pain clinic, the team includes a pain physician, a psychologist, a physical therapist, and a case manager who together develop a single coordinated treatment plan and meet regularly to reassess it. This care-delivery model is best described as which of the following?

A. Interdisciplinary team-based pain management

B. Sequential (serial) referral model

C. Solo-practitioner gatekeeping model

8. A pain clinic implements a policy in which any dose escalation of chronic opioid therapy above a defined morphine milligram equivalent threshold triggers automatic case review by the prescriber, a pharmacist, and a behavioral health provider before the prescription is finalized. This is an example of which care-coordination strategy?

A. Universal precautions screening at intake only

B. Structured multidisciplinary opioid stewardship review

C. Unilateral prescriber discretion without additional review

9. Before writing a new prescription for extended-release oxycodone for a patient with chronic low back pain, a pain physician is required by state law to check a statewide electronic database of the patient's recent controlled-substance dispensing history. This regulatory requirement is best described as which of the following?

A. REMS (Risk Evaluation and Mitigation Strategy) attestation

B. DEA registration renewal

C. Prescription Drug Monitoring Program (PDMP) query

10. Prior to performing a lumbar epidural steroid injection, a pain physician discusses the procedure's risks, benefits, and alternatives with the patient, confirms the patient's understanding, and documents the discussion along with the patient's voluntary agreement to proceed. This process satisfies which legal and ethical requirement?

A. Informed consent for an invasive procedure

B. HIPAA authorization for release of records

C. Advance directive documentation

11. A pain physician needs to assess pain intensity in a nonverbal 2-year-old recovering from surgery. Which validated instrument is most appropriate for this patient?

A. Numeric Rating Scale (0-10), a verbal self-report tool

B. McGill Pain Questionnaire, a verbal descriptor tool

C. FLACC (Face, Legs, Activity, Cry, Consolability) scale

12. A researcher wants an instrument that captures not just pain intensity but also the sensory (e.g., throbbing, burning) and affective (e.g., sickening, fearful) qualities of a patient's pain experience. Which tool is designed for this purpose?

A. Wong-Baker FACES scale

B. Visual Analog Scale

C. McGill Pain Questionnaire

13. A patient enrolled in an analgesic trial is told, as part of informed consent, that the study medication may cause worsening pain as a side effect. The patient subsequently reports increased pain after receiving a pharmacologically inert placebo. This phenomenon is best explained by which mechanism?

A. Nocebo effect from negative expectation

B. Placebo analgesia from positive conditioning

C. Pseudoaddiction from undertreated pain

14. A patient with chronic low back pain completes a 10-item self-report questionnaire scoring how the pain interferes with personal care, lifting, walking, sitting, standing, sleeping, sex life, social life, and traveling, generating a percentage disability score. Which instrument was used?

A. Beck Depression Inventory

B. Oswestry Disability Index

C. STarT Back Screening Tool

15. An occupational medicine physician orders a structured, standardized battery of physical tasks -- lifting, carrying, pushing, pulling, and positional tolerance -- performed under observation to objectively determine a worker's safe functional capabilities before return to a manual labor job. What is this evaluation called?

A. Waddell sign examination for non-organic findings

B. Timed Up and Go mobility and fall-risk test

C. Functional Capacity Evaluation (FCE)

16. A researcher administers a 13-item questionnaire assessing rumination, magnification, and helplessness in relation to a patient's anticipated or experienced pain, and finds that higher scores correlate with worse pain outcomes independent of injury severity. Which instrument was used?

A. Pain Catastrophizing Scale

B. Fear-Avoidance Beliefs Questionnaire

C. Tampa Scale of Kinesiophobia

17. A patient with chronic pain avoids nearly all physical activity out of a conviction that movement will cause further tissue damage, leading to progressive deconditioning and worsening disability despite no evidence of ongoing structural injury. This clinical pattern is best explained by which psychosocial model?

A. Gate control theory

B. Fear-avoidance model of chronic pain

C. Operant conditioning model of pain behavior alone

18. Epidemiologic data consistently show a markedly higher prevalence of fibromyalgia, migraine, and temporomandibular disorder among women compared to men, even after controlling for healthcare-seeking behavior. This pattern is most directly attributed to which category of factors studied in pain research?

A. Differences in opioid receptor gene copy number between sexes

B. Sex-related differences in hormonal, neurobiological, and psychosocial pain modulation

C. Differences in insurance coverage rates between men and women

19. A patient presents with lancinating pain radiating down the leg to the great toe, and the physician wants objective confirmation of active nerve root irritation versus a purely structural finding on imaging. Which study directly assesses electrophysiologic nerve function to help confirm radiculopathy?

A. Plain radiographs of the lumbar spine

B. MRI of the lumbar spine without contrast

C. Electromyography and nerve conduction studies (EMG/NCS)

20. A patient has chronic axial low back pain and MRI shows disc degeneration at two levels, but the physician wants to determine whether reproducing the patient's exact concordant pain with pressurized contrast injection at a specific disc level implicates that disc as the pain generator. Which study is being described?

A. Facet joint medial branch block

B. Provocative discography

C. Selective nerve root block

21. A patient with severe cancer-related bone pain is converted from oral morphine to methadone because of incomplete response and opioid-induced hyperalgesia. Beyond mu-opioid receptor agonism, which additional pharmacologic property makes methadone particularly useful in this scenario?

A. Selective kappa-opioid receptor antagonism

B. Irreversible mu-receptor binding preventing tolerance

C. NMDA receptor antagonism that may reduce central sensitization

22. A patient on escalating doses of high-dose intravenous opioid infusion for postoperative pain develops diffuse, worsening pain that is out of proportion to the surgical injury and improves somewhat when the opioid dose is reduced rather than increased. Which phenomenon best explains this presentation?

A. Opioid tolerance requiring further dose escalation

B. Opioid-induced hyperalgesia

C. Undertreated acute nociceptive pain

23. An elderly patient with chronic kidney disease receiving scheduled oral morphine for cancer pain develops progressive somnolence and myoclonus over several days without an increase in dose. Which mechanism best explains this presentation?

A. Accumulation of the active metabolite morphine-6-glucuronide due to reduced renal clearance

B. Development of acute opioid tolerance to the prescribed dose

C. Onset of a new, unrelated primary seizure disorder around this time

24. A patient started on methadone for chronic pain is found on routine ECG to have a corrected QT interval of 510 ms. Which property of methadone is responsible for this finding?

A. Beta-adrenergic receptor blockade

B. Induction of hepatic CYP3A4 metabolism

C. Blockade of the cardiac hERG potassium channel

25. A patient on buprenorphine for opioid use disorder with concurrent chronic pain is noted to have a relatively wide safety margin for respiratory depression even at higher doses, compared to full mu-opioid agonists. What pharmacologic property of buprenorphine explains this ceiling effect on respiratory depression?

A. Partial agonist activity at the mu-opioid receptor

B. Full antagonist activity at the mu-opioid receptor

C. Selective agonist activity at the delta-opioid receptor

26. A patient is switched from oral extended-release morphine to a transdermal fentanyl patch. The prescriber counsels that after patch application, meaningful analgesia may take 12-24 hours to develop, and after patch removal for toxicity, effects may persist for many hours. Which pharmacokinetic property explains both observations?

A. Formation of a subcutaneous drug depot (reservoir effect) with the transdermal system

B. Rapid first-pass hepatic metabolism of transdermal fentanyl before it reaches circulation

C. High renal clearance of unmetabolized parent fentanyl

27. A patient with poorly controlled cancer pain on high-dose oxycodone with dose-limiting side effects is switched to an equianalgesic dose of hydromorphone, but the calculated dose is reduced by 25-50% out of caution. What pharmacologic principle justifies this dose reduction during opioid rotation?

A. Hydromorphone has no cross-tolerance with oxycodone

B. Incomplete cross-tolerance between different opioids at the new receptor exposure

C. Hydromorphone is metabolized entirely independently of the mu-opioid receptor

28. A patient with an opioid overdose receiving IV naloxone initially regains consciousness and adequate respiration, but recurrent respiratory depression develops 30-45 minutes later, requiring a repeat dose or continuous infusion. Which pharmacokinetic mismatch explains this recurrence?

A. Naloxone has a longer plasma half-life than most opioids it is used to reverse

B. Naloxone is a partial agonist with a delayed onset of action at the receptor

C. Naloxone's duration of action is shorter than that of many opioids, allowing re-sedation as it wears off

29. A patient taking a nonselective NSAID for chronic osteoarthritis pain develops epigastric pain and is found to have a gastric ulcer on endoscopy. Which mechanism underlies this NSAID-associated gastrointestinal toxicity?

A. Inhibition of COX-1-mediated protective prostaglandin synthesis in the gastric mucosa

B. Direct caustic injury from local acidic pH of the tablet alone

C. Induction of Helicobacter pylori colonization by the drug

30. A patient with a history of peptic ulcer disease and no cardiovascular risk factors is prescribed celecoxib instead of a nonselective NSAID for osteoarthritis pain. What is the pharmacologic rationale for this selection?

A. Selective COX-2 inhibition reduces GI toxicity by sparing protective COX-1-mediated prostaglandins

B. Celecoxib has no analgesic ceiling effect, unlike nonselective NSAIDs

C. Celecoxib is renally eliminated unchanged, avoiding hepatic metabolism entirely

31. A patient who took several doses of acetaminophen exceeding the recommended daily maximum over several days presents with elevated liver transaminases. Which mechanism explains acetaminophen-induced hepatotoxicity at supratherapeutic doses?

A. Direct inhibition of hepatic COX-1 causing ischemic hepatocyte injury

B. Accumulation of unmetabolized parent acetaminophen in hepatocyte membranes

C. Depletion of glutathione stores allowing accumulation of the toxic metabolite NAPQI

32. A patient is prescribed intravenous ketorolac for acute postoperative pain. The pharmacist flags the order because ketorolac, unlike most other NSAIDs, carries a specific labeled restriction on duration of use. What is this restriction?

A. Ketorolac may be used indefinitely, but only when dosed at bedtime

B. Combined duration of ketorolac (oral plus parenteral) should not exceed five days due to renal and bleeding risk

C. Ketorolac has no duration restriction because it lacks any GI toxicity risk

33. A patient taking low-dose aspirin for cardiovascular prophylaxis is told the antiplatelet effect will persist for the full 7-10 day lifespan of an affected platelet, even after a single dose is stopped, unlike other NSAIDs whose antiplatelet effects resolve within a day or two of discontinuation. What mechanism accounts for aspirin's uniquely prolonged effect?

A. Aspirin has an unusually long plasma half-life of several days

B. Aspirin irreversibly acetylates platelet COX-1, and platelets cannot synthesize new enzyme

C. Aspirin is stored in adipose tissue and released slowly over days

34. An elderly, volume-depleted patient started on a nonselective NSAID for pain develops an acute rise in serum creatinine. Which mechanism best explains NSAID-associated acute kidney injury in a volume-depleted state?

A. Direct nephrotoxic tubular necrosis independent of renal blood flow

B. NSAID-induced hyperkalemia causing prerenal azotemia

C. Inhibition of prostaglandin-mediated afferent arteriolar vasodilation, reducing renal perfusion

35. An elderly patient with knee osteoarthritis and chronic kidney disease is switched from an oral NSAID to topical diclofenac gel applied directly over the joint. What is the pharmacologic rationale for this substitution being safer in this patient?

A. Topical diclofenac has no COX inhibitory activity at the application site at all

B. Topical application achieves therapeutic local tissue concentration with markedly lower systemic absorption than oral dosing

C. Topical diclofenac is renally excreted without ever entering systemic circulation

36. A patient with painful diabetic peripheral neuropathy is started on nortriptyline. The prescriber explains that the drug's analgesic effect for neuropathic pain occurs at doses lower than those typically needed for antidepressant effect. Which mechanism underlies the analgesic action of tricyclic antidepressants in neuropathic pain?

A. Selective blockade of peripheral TRPV1 receptors

B. Direct blockade of voltage-gated sodium channels in the dorsal root ganglion only

C. Inhibition of norepinephrine and serotonin reuptake, enhancing descending inhibitory pain pathways

37. A patient with painful diabetic peripheral neuropathy and comorbid major depressive disorder is started on duloxetine, allowing one medication to address both conditions. What pharmacologic class does duloxetine belong to, and what is its relevant mechanism for neuropathic pain?

A. Selective serotonin reuptake inhibitor (SSRI) acting purely through serotonergic descending pathways

B. Serotonin-norepinephrine reuptake inhibitor (SNRI), enhancing both serotonergic and noradrenergic descending inhibition

C. Monoamine oxidase inhibitor (MAOI) blocking presynaptic monoamine breakdown

38. A patient with postherpetic neuralgia is started on gabapentin. Which mechanism of action explains its efficacy in neuropathic pain?

A. Binding to the alpha-2-delta subunit of voltage-gated calcium channels, reducing excitatory neurotransmitter release

B. Direct antagonism of NMDA glutamate receptors in the spinal dorsal horn neurons

C. Agonism at GABA-B receptors distributed broadly throughout the spinal cord and brain

39. A patient with chronic kidney disease and an estimated GFR of 25 mL/min is being started on pregabalin for neuropathic pain. What dosing adjustment is required based on pregabalin's pharmacokinetics?

A. Significant dose reduction is required because pregabalin is renally eliminated largely unchanged

B. No dose adjustment is needed because pregabalin undergoes extensive hepatic metabolism

C. The dose should be increased to compensate for reduced hepatic clearance

40. Before starting carbamazepine for trigeminal neuralgia in a patient of Han Chinese ancestry, the prescriber orders HLA-B*1502 genotype testing. What is the rationale for this test?

A. HLA-B*1502 positivity predicts a poor analgesic response to carbamazepine therapy

B. HLA-B*1502 positivity is strongly associated with carbamazepine-induced Stevens-Johnson syndrome/toxic epidermal necrolysis

C. HLA-B*1502 positivity indicates the patient will require a higher starting drug dose

41. A pain physician considering amitriptyline for a patient's chronic neuropathic pain reviews the patient's cardiac history and orders a baseline ECG before starting the medication, specifically screening for a pre-existing conduction abnormality. What cardiac risk of tricyclic antidepressants makes this screening necessary?

A. TCAs commonly cause clinically significant bradycardia through vagal nerve stimulation

B. TCAs have quinidine-like cardiac sodium channel blocking effects that can worsen conduction delays and prolong QT

C. TCAs cause irreversible coronary artery vasospasm in the majority of treated patients

42. A patient prescribed both gabapentin and an opioid for chronic pain is counseled about an important pharmacodynamic interaction between the two drug classes. What is the primary safety concern with this combination?

A. Additive central nervous system and respiratory depression, increasing overdose risk

B. Gabapentin induces hepatic enzymes that dramatically increase opioid metabolism

C. Gabapentin displaces opioids from plasma protein binding sites, increasing free opioid fraction

43. A patient being started on lamotrigine for neuropathic pain is instructed to follow a very slow, stepwise dose titration schedule over several weeks rather than starting at a therapeutic dose immediately. What is the rationale for this cautious titration?

A. Rapid titration significantly increases the risk of severe cutaneous reactions, including Stevens-Johnson syndrome

B. Rapid titration causes irreversible hepatotoxicity within the first week

C. Rapid titration is required to avoid gabapentin-like sedation, which lamotrigine does not otherwise cause

44. A patient with refractory complex regional pain syndrome is started on a low-dose subanesthetic intravenous ketamine infusion. Which receptor mechanism is primarily responsible for ketamine's analgesic effect in this setting?

A. Agonism at peripheral mu-opioid receptors

B. Blockade of voltage-gated sodium channels in peripheral nerves

C. Antagonism at the NMDA glutamate receptor, reducing central sensitization

45. A patient with postherpetic neuralgia over a well-localized area of the chest wall is prescribed a topical lidocaine 5% patch. What is the mechanism by which this localized therapy provides analgesia with minimal systemic effect?

A. Systemic beta-adrenergic blockade limited to the affected dermatome

B. Local depletion of substance P from peripheral nociceptor terminals

C. Local blockade of voltage-gated sodium channels in cutaneous nerve fibers, limiting ectopic firing

46. A patient with painful diabetic peripheral neuropathy is prescribed a high-concentration (8%) capsaicin patch applied by a clinician for a limited duration. What is the mechanism by which repeated or high-dose capsaicin exposure produces sustained analgesia?

A. TRPV1 receptor overstimulation leading to defunctionalization of cutaneous nociceptor terminals

B. Direct blockade of NMDA receptors in the peripheral nerve terminal

C. Systemic activation of descending noradrenergic inhibitory pathways

47. A patient undergoing a complex spine surgery receives epidural clonidine as an adjuvant to local anesthetic for postoperative analgesia. Through which receptor mechanism does clonidine produce analgesia in this setting?

A. Alpha-2 adrenergic receptor agonism in the spinal dorsal horn, enhancing descending inhibition

B. Selective serotonin 5-HT3 receptor antagonism at the chemoreceptor trigger zone

C. Peripheral beta-2 adrenergic receptor agonism, causing local vasodilation at the site

48. A patient with chronic pain associated with multiple sclerosis-related spasticity is prescribed a cannabinoid-based medication. Which receptor mechanism underlies the analgesic and antispasmodic effects of cannabinoids?

A. Direct agonism at mu- and kappa-opioid receptors throughout the central nervous system

B. Agonism at CB1 receptors (central) and CB2 receptors (peripheral/immune), modulating neurotransmitter release and inflammation

C. Antagonism of GABA-A receptors, increasing overall neuronal excitability centrally

49. A patient with chronic migraine (15 or more headache days per month) undergoes quarterly onabotulinumtoxinA injections across multiple fixed sites on the head and neck. Through what mechanism does botulinum toxin reduce chronic migraine frequency?

A. Direct blockade of central 5-HT1B/1D serotonin receptors in the brainstem and cortex

B. Inhibition of presynaptic acetylcholine and neuropeptide release, reducing peripheral sensitization of trigeminal afferents

C. Systemic beta-adrenergic receptor blockade reducing cerebral vasodilation directly

50. A patient with acute cervical muscle spasm and myofascial pain is prescribed a short course of cyclobenzaprine. Through which mechanism does this centrally acting skeletal muscle relaxant reduce muscle spasm?

A. Direct depolarizing blockade at the skeletal muscle neuromuscular junction itself

B. Peripheral inhibition of calcium release from the sarcoplasmic reticulum of skeletal muscle fibers

C. Central nervous system action, structurally related to tricyclics, reducing motor tone at the brainstem

51. A patient on warfarin for atrial fibrillation is scheduled for a lumbar transforaminal epidural steroid injection, classified as an intermediate-to-high bleeding-risk interventional procedure. According to consensus anticoagulation management guidelines for interventional pain procedures, what is the appropriate approach?

A. Continue warfarin without interruption because epidural procedures carry negligible bleeding risk

B. Hold warfarin for an appropriate interval and confirm a normalized INR before the procedure, per published consensus guidelines

C. Substitute warfarin with high-dose aspirin the day before the procedure

52. A pain physician is choosing between fluoroscopic and ultrasound guidance for a lumbar transforaminal epidural steroid injection. Which statement correctly describes a key advantage of fluoroscopic guidance for this specific procedure?

A. Fluoroscopy allows direct visualization of bony landmarks and needle trajectory relative to the neural foramen with confirmatory contrast spread

B. Ultrasound provides superior visualization of intraosseous and epidural bony anatomy compared to fluoroscopy for this procedure

C. Fluoroscopy is preferred solely because it avoids all radiation exposure to the patient

53. A pain physician performing an interventional spine procedure follows a strict sterile preparation protocol including chlorhexidine skin antisepsis, sterile draping, and a no-touch needle technique. What is the primary purpose of this protocol?

A. To reduce the risk of local anesthetic systemic toxicity during injection

B. To reduce the risk of contrast-induced nephropathy from the dye load

C. To minimize the risk of procedure-related infection, including epidural abscess or discitis

54. A patient is scheduled for implantation of a permanent intrathecal drug delivery pump. Unlike a routine single-level epidural steroid injection, this procedure requires preoperative antibiotic prophylaxis. What is the rationale for this difference?

A. Implanted hardware creates a durable foreign-body surface that, if infected, is difficult to treat without device removal, warranting prophylaxis

B. Antibiotic prophylaxis is required for all spinal procedures regardless of whether hardware is implanted

C. Antibiotics are given solely to reduce postoperative pain, not to prevent infection

55. A patient with a documented moderate allergy to iodinated contrast is scheduled for a fluoroscopically guided epidural steroid injection that typically uses contrast to confirm needle placement. What is the most appropriate management strategy?

A. Cancel all image-guided procedures permanently for this patient going forward, regardless of technique

B. Premedicate with a corticosteroid/antihistamine protocol and consider a non-iodinated contrast alternative or omitting contrast

C. Proceed with standard full contrast dosing without any premedication since epidural contrast volumes are too small to provoke a reaction

56. A patient with chronic low back pain is prescribed a transcutaneous electrical nerve stimulation (TENS) unit for symptomatic relief. Through which neurophysiologic mechanism does TENS reduce pain perception?

A. Direct depletion of peripheral substance P stores from cutaneous nociceptor nerve terminals over time

B. Systemic release of endogenous opioids occurring exclusively at high-frequency stimulation settings only

C. Activation of large-diameter A-beta afferent fibers that inhibit nociceptive transmission at the dorsal horn

57. A patient with chronic musculoskeletal pain undergoes acupuncture as an adjunct to standard pain management. Research into its analgesic mechanism has implicated which physiologic pathway?

A. Needle-induced stimulation triggering release of endogenous opioids and activation of descending inhibitory pathways

B. Direct blockade of peripheral voltage-gated sodium channels at the needle insertion sites

C. Selective inhibition of hepatic prostaglandin synthesis pathways

58. A patient with focal, well-localized neuropathic pain in the distribution of the supraorbital nerve after facial trauma is treated with a temporary percutaneous peripheral nerve stimulation lead placed near the affected nerve for several weeks, without implanting a permanent device. What best characterizes this treatment modality?

A. A fully implanted, permanent spinal cord stimulator system placed epidurally

B. A temporary, percutaneously placed peripheral nerve stimulator targeting a specific peripheral nerve distribution

C. An intrathecal drug delivery trial using a temporary external pump

59. A patient with L5 radicular leg pain and imaging showing a paracentral disc herniation contacting the L5 nerve root is scheduled for a fluoroscopically guided transforaminal epidural steroid injection at L5. What is the primary rationale for choosing the transforaminal, rather than interlaminar, approach in this case?

A. Transforaminal injection eliminates all radiation exposure to the patient during the case

B. Transforaminal injection avoids the need for any contrast confirmation of epidural spread

C. Transforaminal approach delivers medication directly to the nerve root and ventral epidural space near the pathology

60. A patient with chronic lumbar facet-mediated pain has undergone two separate, concordant diagnostic medial branch blocks with local anesthetic, both producing greater than 80% pain relief. Based on this response, which subsequent procedure is indicated?

A. Proceeding directly to spinal fusion surgery

B. A single blind steroid injection into the facet joint without further diagnostic testing

C. Radiofrequency ablation of the corresponding medial branch nerves

61. A patient with unilateral low back pain that does not cross the midline, worsened by transitional movements, undergoes a diagnostic sacroiliac joint injection with local anesthetic under fluoroscopic guidance, achieving greater than 75% pain relief. What does this response confirm?

A. The sacroiliac joint is a significant pain generator for this patient's presentation

B. The patient's pain is entirely of lumbar facet joint origin

C. The patient's pain is psychogenic and requires no further interventional workup

62. A patient with complex regional pain syndrome of the hand, with associated color changes, temperature asymmetry, and allodynia suggesting a sympathetically maintained component, is scheduled

for a diagnostic and potentially therapeutic sympathetic block. Which block is appropriate for upper extremity sympathetically mediated pain?

A. Celiac plexus block

B. Stellate ganglion block

C. Lumbar sympathetic block

63. A patient with severe, opioid-refractory abdominal pain from unresectable pancreatic adenocarcinoma is referred for an interventional procedure targeting the sympathetic plexus supplying the pancreas and upper abdominal viscera to provide additional analgesia. Which procedure is indicated?

A. Stellate ganglion block

B. Superior hypogastric plexus block

C. Celiac plexus neurolysis

64. A patient with a taut, palpable band in the trapezius muscle that reproduces referred pain to the neck and shoulder upon palpation is diagnosed with myofascial pain syndrome and undergoes a trigger point injection. What is the primary therapeutic goal of this injection?

A. Neurolytic destruction of the peripheral nerve supplying the affected muscle

B. Permanent denervation of the lumbar facet joint capsule and its innervation

C. Mechanical disruption of the taut band and local anesthetic delivery to interrupt the local pain-spasm cycle

65. A patient with unilateral, sharp, shooting pain in the posterior scalp distribution, reproduced by palpation over the greater occipital nerve, is diagnosed with occipital neuralgia and undergoes a diagnostic and therapeutic nerve block at that site. What structure is specifically targeted by this procedure?

A. The greater occipital nerve as it courses superficially near the nuchal ridge

B. The trigeminal ganglion within the skull base

C. The stellate ganglion in the cervical paravertebral region

66. An elderly patient with severe knee osteoarthritis pain who is not a surgical candidate undergoes a diagnostic genicular nerve block with significant relief, followed by genicular nerve radiofrequency ablation for longer-lasting analgesia. What is the anatomic target of this procedure?

A. The sciatic nerve at the level of the popliteal fossa

B. The sensory genicular nerve branches supplying the knee joint

C. The femoral nerve at the inguinal ligament

67. A patient with severe, refractory pelvic cancer pain undergoes intrathecal neurolysis with phenol. Compared to a purely sensory neurolytic target, what is a key procedural risk consideration specific to intrathecal phenol neurolysis?

A. Phenol neurolysis carries no risk of motor involvement because it selectively spares motor fibers

B. Phenol neurolysis is reversible within minutes if excessive spread occurs

C. Phenol can affect mixed sensorimotor nerve roots nonselectively, risking unwanted motor or sphincter dysfunction

68. A patient with chronic pelvic pain localized to the perineum, worsened by sitting, and reproduced by palpation near the ischial spine is diagnosed with pudendal neuralgia and scheduled for a diagnostic nerve block. Which nerve is the target of this procedure?

A. The obturator nerve supplying the medial thigh and hip adductors

B. The ilioinguinal nerve supplying the groin and upper medial thigh

C. The pudendal nerve near the ischial spine

69. A patient with severe pelvic pain from advanced cervical cancer, refractory to systemic opioids, is referred for an interventional sympathetic block targeting the plexus that provides sympathetic innervation to the pelvic viscera. Which procedure is indicated?

A. Celiac plexus block

B. Superior hypogastric plexus block

C. Stellate ganglion block

70. A patient with failed back surgery syndrome and predominant lower extremity neuropathic pain undergoes spinal cord stimulator implantation. Through which physiologic mechanism does conventional (tonic) spinal cord stimulation reduce pain perception?

A. Direct chemical denervation of the affected peripheral nerve using injected neurolytic agent

B. Electrical stimulation of dorsal column A-beta fibers, gating nociceptive input at the cord

C. Systemic release of anti-inflammatory cytokines from the surrounding epidural fat tissue

71. A patient being considered for permanent spinal cord stimulator implantation first undergoes a percutaneous trial with temporary leads for approximately one week before committing to the permanent device. What is the primary purpose of this trial period?

A. To confirm the patient achieves meaningful, reproducible pain relief and functional benefit before permanent implantation

B. The trial period is solely an insurance billing formality with no clinical purpose

C. To surgically anchor the permanent leads in their final position ahead of time

72. A patient with advanced cancer and severe pain poorly controlled despite high-dose systemic opioids, complicated by intolerable systemic side effects, is being evaluated for an intrathecal drug delivery system. What is the primary rationale for this therapy in this scenario?

A. Intrathecal delivery is chosen because it eliminates the possibility of any medication tolerance developing

B. Intrathecal delivery is preferred because it requires no ongoing device maintenance or refills

C. Intrathecal delivery allows much lower medication doses to achieve analgesia by acting directly at the spinal cord, reducing systemic side effect burden

73. A patient with focal, well-localized neuropathic pain confined to the distribution of a single peripheral nerve territory in the foot after a prior surgery, who has had an inadequate response to conventional spinal cord stimulation targeting broader dermatomal coverage, is switched to a device that targets a more focal, somatotopically precise structure. Which device is this?

A. Conventional tonic spinal cord stimulator targeting the dorsal columns broadly

B. Dorsal root ganglion (DRG) stimulation targeting the specific ganglion corresponding to the painful dermatome

C. An intrathecal opioid pump delivering continuous medication infusion

74. A patient with chronic, focal neuropathic pain isolated to the distribution of the ilioinguinal nerve after hernia repair undergoes implantation of a small stimulating lead placed directly along that specific peripheral nerve, distinct from an epidural spinal cord stimulator lead. What is this device categorized as?

A. A peripheral nerve stimulator (PNS) implant

B. An intrathecal drug delivery system

C. A dorsal root ganglion stimulator

75. A patient with a previously well-functioning spinal cord stimulator reports sudden loss of paresthesia coverage and recurrence of their original pain pattern. Device interrogation reveals the lead has shifted from its original epidural position. What complication does this represent?

A. Generator battery depletion over time

B. Surgical site infection at the implant

C. Lead migration from its original epidural position

76. A patient with severe, opioid-refractory chronic pain who cannot tolerate further opioid dose escalation due to side effects is being considered for intrathecal ziconotide instead of an opioid-based intrathecal regimen. What is ziconotide's mechanism of action?

A. Selective blockade of N-type calcium channels at afferent terminals, via a non-opioid mechanism

B. Mu-opioid receptor agonism, similar in mechanism to intrathecal morphine

C. Alpha-2 adrenergic receptor agonism, similar in mechanism to intrathecal clonidine

77. A patient with confirmed facet-mediated lumbar pain undergoes radiofrequency ablation of the medial branch nerves. What is the physical mechanism by which this procedure produces durable pain relief?

A. Chemical destruction of the nerve using injected alcohol as the neurolytic agent

B. Thermal coagulation of nerve tissue using alternating current, creating a controlled heat lesion

C. Cryogenic freezing of the nerve tissue using a specialized cooled probe device

78. A pain physician chooses cryoablation rather than radiofrequency ablation for a peripheral nerve target, explaining to the patient that this technique preserves the perineurium, allowing for the possibility of nerve regeneration over time, unlike some heat-based lesions. What is the underlying tissue mechanism of cryoablation?

A. Direct chemical denaturation of nerve proteins using injected phenol solution

B. Ice crystal formation causing localized Wallerian degeneration while preserving the perineurial sheath

C. High-frequency electrical current generating a permanent thermal coagulation lesion in tissue

79. A patient with severe, refractory cancer pain undergoes chemical neurolysis of a peripheral nerve using injected phenol, rather than radiofrequency ablation. Compared to RFA, what is a key characteristic of chemical neurolytic agents like phenol or alcohol?

A. They produce tissue destruction through direct chemical protein denaturation and are not as precisely controllable in extent of spread as an electrode-based thermal lesion

B. They act by delivering high-frequency alternating current to generate a heat lesion, identical to RFA

C. They have no risk of affecting adjacent non-target tissue because they remain confined strictly to the injection needle tip

80. A patient with chronic lateral epicondylitis refractory to conservative therapy undergoes ultrasound-guided injection of platelet-rich plasma (PRP) derived from their own centrifuged blood. What is the proposed mechanism by which PRP promotes tissue healing in this setting?

A. Concentrated platelets release growth factors stimulating repair, angiogenesis, and collagen synthesis

B. PRP provides a direct, potent anti-inflammatory corticosteroid effect at the injection site

C. PRP acts as a local anesthetic, providing immediate but temporary numbness at the tendon site

81. A patient with chronic low back pain and significant pain catastrophizing is referred for cognitive-behavioral therapy. Which core technique of CBT directly targets catastrophic thinking patterns about pain?

A. Progressive muscle relaxation training used as a standalone technique

B. Cognitive restructuring to identify and challenge distorted, catastrophic pain-related thoughts

C. Exposure-based systematic desensitization applied only to feared movements

82. A structured pain rehabilitation program is designed so that healthy, well behaviors (e.g., staying active, engaging socially) are positively reinforced, while excessive pain behaviors (e.g., verbal complaining, guarding) are not reinforced by clinicians or family members. This approach is based on which behavioral framework?

A. Operant conditioning model of pain behavior

B. Classical (Pavlovian) conditioning model of pain

C. Psychodynamic model of somatic symptom expression

83. A patient with chronic pain undergoes a therapy that does not focus on reducing pain intensity itself, but instead helps the patient increase psychological flexibility, clarify personal values, and engage in valued activities despite the presence of pain. Which therapeutic approach is this?

A. Traditional cognitive-behavioral therapy focused on symptom reduction

B. Eye movement desensitization and reprocessing (EMDR)

C. Acceptance and Commitment Therapy (ACT)

84. A patient with chronic tension-type headache is enrolled in a treatment in which surface electromyography sensors provide real-time visual feedback of frontalis and trapezius muscle tension, and the patient learns voluntary techniques to reduce that tension over successive sessions. What is this treatment modality called?

A. Transcutaneous electrical nerve stimulation (TENS) delivered via skin electrodes

B. Cognitive-behavioral therapy delivered without any physiologic monitoring component

C. Biofeedback using real-time physiologic signal monitoring for self-regulation

85. A patient with chronic pain is found to have newly worsening depressive symptoms, and the physician recognizes that depression and chronic pain frequently coexist and can mutually worsen one another. What best characterizes the relationship between chronic pain and depression?

A. A bidirectional relationship in which each condition can worsen the severity and treatment resistance of the other

B. An entirely coincidental relationship with no shared underlying neurobiology

C. A relationship in which depression is always the primary cause and pain is always a somatic symptom of it

86. A patient with chronic neuropathic pain and comorbid major depressive disorder is being started on pharmacotherapy. Which class of antidepressant is preferentially chosen because it can address both the neuropathic pain and the depressive symptoms through a shared mechanism?

A. Selective serotonin reuptake inhibitors (SSRIs), which have the strongest evidence for neuropathic pain of any antidepressant class

B. Monoamine oxidase inhibitors (MAOIs), which are first-line for this combination due to a favorable side-effect profile

C. Serotonin-norepinephrine reuptake inhibitors (SNRIs), which have evidence for both depression and neuropathic pain via combined monoamine reuptake inhibition

87. Before initiating long-term opioid therapy for chronic noncancer pain, a pain physician administers a validated screening tool assessing personal and family history of substance misuse, psychological disease, and other risk factors to stratify the patient's risk of opioid misuse. What is the purpose of this assessment?

A. To determine the exact starting opioid dose based on the patient's body weight alone

B. To diagnose the patient's underlying pain condition before treatment begins

C. To stratify the patient's risk for opioid misuse and guide the intensity of monitoring during opioid therapy

88. During a routine visit, a patient with chronic pain discloses persistent thoughts of self-harm and a specific plan to end their life. What is the most appropriate immediate action by the pain physician?

A. Document the disclosure and continue with the routine pain management visit as scheduled, addressing it at a future appointment

B. Immediately arrange an emergent psychiatric evaluation and ensure the patient's safety before the visit ends

C. Simply increase the patient's antidepressant dose and schedule a follow-up in one month

89. A patient with chronic nonspecific low back pain is enrolled in a physical therapy program emphasizing gradual, progressive increases in activity level and exercise intensity according to a predetermined schedule, rather than being guided purely by momentary pain levels. What is this treatment approach called?

A. Passive modality therapy using heat and ultrasound only

B. Bed rest with activity restriction until pain resolves

C. Graded activity / graded exercise therapy

90. A patient with acute, uncomplicated mechanical low back pain of less than 4 weeks' duration, without red-flag features, is treated with spinal manipulative therapy by a physical therapist as part of a multimodal approach. What is the evidence-based role of manual therapy in this scenario?

A. Manual therapy/spinal manipulation has evidence supporting modest short-term benefit for acute low back pain as part of a multimodal approach

B. Manual therapy is contraindicated in all cases of low back pain regardless of duration or red-flag status

C. Manual therapy is curative and eliminates the need for any further active exercise-based treatment

91. A patient with fibromyalgia and significant deconditioning who finds land-based exercise painful is referred to a warm-water pool-based exercise program. What is the primary rationale for aquatic therapy in this population?

A. Water immersion selectively blocks NMDA receptors, directly treating central sensitization

B. Buoyancy reduces joint loading and allows easier movement with less pain, improving exercise tolerance and adherence

C. Aquatic therapy works exclusively through a cold-water-induced anti-inflammatory mechanism

92. A patient with chronic patellofemoral pain syndrome is prescribed a custom knee brace designed to improve patellar tracking and reduce lateral patellar maltracking during activity. What is the therapeutic rationale for this orthotic device?

A. Providing external mechanical support and alignment correction to reduce abnormal joint loading and improve biomechanics

B. Delivering a sustained local corticosteroid dose directly to the joint over time

C. Producing a systemic analgesic effect through transdermal drug absorption

93. An occupational health physician manages a worker with acute low back pain from a workplace injury by keeping the employee engaged in modified, lighter-duty work rather than prescribing complete time off work during recovery. What is the evidence-based rationale for this early, modified return-to-work approach?

A. Modified work is used purely to reduce the employer's workers' compensation costs, with no benefit to the patient

B. Complete work absence for the full recovery period consistently produces better long-term functional outcomes

C. Early return to modified work is associated with better functional outcomes and reduced risk of chronicity and long-term disability compared to prolonged work absence

94. A patient with chronic low back pain and significant functional limitation is enrolled in an intensive, interdisciplinary program combining physical reconditioning, psychological intervention, and vocational counseling, with the explicit goal of maximizing function and facilitating return to work rather than eliminating pain entirely. What is this treatment model called?

A. Passive modality-only physical therapy

B. Functional restoration program

C. Isolated pharmacologic management

95. A worker recovering from a low back injury is enrolled in a program focused specifically on building general physical capacity (strength, endurance, flexibility) without reference to any particular job task, distinguishing it from a later program that will simulate the worker's actual job demands. What is this general conditioning phase called, in contrast to work hardening?

A. Work hardening

B. Work conditioning

C. Functional capacity evaluation

96. After maximal medical improvement is reached for a patient with a permanent residual impairment from a workplace injury, a physician is asked to assign a percentage impairment rating for use in a workers' compensation claim. Which reference is most commonly used as the accepted standard for this determination?

A. AMA Guides to the Evaluation of Permanent Impairment

B. DSM-5-TR diagnostic criteria for psychiatric conditions

C. ICD-10-CM coding manual for diagnosis and billing

97. A patient with chronic pain is enrolled in an 8-week mindfulness-based stress reduction (MBSR) program involving structured meditation and body-scan practices. Through what proposed mechanism does mindfulness practice reduce the suffering associated with chronic pain?

A. Cultivating nonjudgmental, present-moment awareness that alters the cognitive-affective appraisal of pain sensations, reducing associated distress

B. Direct pharmacologic blockade of peripheral nociceptors occurring passively during meditation practice sessions

C. Permanent structural ablation of the brain regions responsible for pain processing entirely

98. A patient with chronic nonspecific low back pain asks about starting a yoga practice as a complementary approach. Based on the available evidence base, how should the physician characterize yoga's role in chronic low back pain management?

A. Yoga has no evidence of any benefit whatsoever and should be actively discouraged

B. Yoga has evidence supporting modest gains in pain and function for chronic low back pain, as an adjunct

C. Yoga is proven superior to all standard exercise-based physical therapy and should fully replace it

99. A patient with chronic myofascial neck pain undergoes a course of therapeutic massage as part of a multimodal pain management plan. What is a proposed mechanism by which massage therapy may provide short-term analgesic benefit?

A. Massage permanently remodels and structurally reverses degenerated intervertebral disc tissue over time

B. Massage directly antagonizes NMDA glutamate receptors within the spinal cord dorsal horn itself

C. Massage may reduce muscle tension and activate large-diameter afferent fibers and endogenous pain-modulatory mechanisms, providing short-term relief

100. A patient with fibromyalgia and comorbid knee osteoarthritis is referred to a structured tai chi program combining slow, low-impact movement with breathing and mindfulness components. What does the evidence base suggest about tai chi for these conditions?

A. Tai chi has been shown to be harmful and should be avoided entirely in fibromyalgia and osteoarthritis

B. Tai chi works exclusively through a mechanism of direct joint cartilage regeneration

C. Tai chi has evidence supporting improvements in pain, function, and quality of life in both fibromyalgia and osteoarthritis populations

101. A patient has burning, shooting pain following a documented peripheral nerve injury, with no ongoing tissue damage or inflammatory process identified. According to current pain taxonomy, which classification best describes this type of pain, distinct from pain arising from actual or threatened tissue damage detected by nociceptors?

A. Neuropathic pain, caused by a lesion or disease of the somatosensory nervous system

B. Nociceptive pain, caused by activation of nociceptors from tissue injury or inflammation

C. Nociplastic pain, arising despite no clear evidence of tissue damage or nerve lesion

102. A patient has experienced persistent low back pain for 4 months following an initial injury that would typically be expected to heal within weeks. According to standard pain taxonomy, how is this pain classified?

A. Acute pain, since the inciting injury event is clearly identifiable here

B. Chronic pain, defined as pain persisting beyond the normal expected tissue healing time, generally beyond 3 months

C. Subacute pain, a category with no defined time threshold in standard taxonomy

103. A patient reports widespread musculoskeletal pain present for over 3 months, along with fatigue, unrefreshing sleep, and cognitive difficulties, without evidence of an inflammatory or structural cause on workup. Diagnosis is made using a symptom severity scale and a widespread pain index rather than a formal tender point count. What condition and diagnostic approach does this describe?

A. Rheumatoid arthritis, diagnosed using the 1990 ACR tender point criteria

B. Polymyalgia rheumatica, diagnosed by elevated inflammatory markers alone

C. Fibromyalgia, diagnosed using the 2016 ACR criteria based on Widespread Pain Index and Symptom Severity Scale

104. Research into the pathophysiology of fibromyalgia has demonstrated augmented pain processing in the central nervous system, including enhanced temporal summation (wind-up) and reduced descending inhibitory pain modulation, in the absence of peripheral tissue pathology. What underlying pathophysiologic process does this represent?

A. Peripheral nociceptor sensitization arising from ongoing local tissue inflammation

B. Autoimmune-mediated joint destruction from synovitis and pannus formation

C. Central sensitization with augmented central pain processing and impaired descending inhibition

105. A postoperative pain protocol combines scheduled acetaminophen, an NSAID, a gabapentinoid, and a regional nerve block, with opioids reserved for breakthrough pain only. What is the primary rationale for this multimodal analgesic approach?

A. Using several non-opioid agents together increases total opioid dosing efficiency

B. Targeting multiple distinct pain pathways synergistically improves analgesia while reducing overall opioid requirements and opioid-related side effects

C. Multimodal regimens are used solely because single-agent opioid therapy is not legally permitted after surgery

106. A patient with multiple rib fractures from blunt chest trauma is at risk for pneumonia and respiratory failure due to splinting and shallow breathing from pain. Beyond systemic opioids, which analgesic intervention is specifically recommended to improve pulmonary mechanics and reduce respiratory complications in this population?

A. High-dose oral acetaminophen used alone as sole monotherapy for the pain

B. A regional technique such as an erector spinae plane block or thoracic epidural to allow coughing

C. Complete immobilization with a rigid chest binder to limit chest wall movement

107. A patient with extensive burn injuries experiences both a constant baseline discomfort and separate, much more intense pain during wound dressing changes. How should analgesic management differ between these two distinct pain states?

A. Background pain is managed with scheduled longer-acting analgesia, while procedural (dressing change) pain requires additional short-acting, rapid-onset analgesia or sedation timed to the procedure

B. Both background and procedural burn pain should be treated identically with a single fixed daily opioid dose and no additional dosing around procedures

C. Procedural burn pain does not require any additional analgesia beyond what is used for background pain, since burn pain is a single uniform entity

108. A surgical pain protocol administers a nerve block and initiates NSAID therapy before surgical incision is made, rather than waiting until after the procedure to begin analgesic treatment. What is the theoretical rationale behind this preemptive/preventive analgesic strategy?

A. Administering analgesia before incision has no different effect than giving it afterward at all

B. Blocking nociceptive input before the surgical stimulus may reduce the development of central sensitization and subsequent chronic postsurgical pain

C. Preemptive analgesia is used exclusively to shorten the total operative time needed for the surgical case itself

109. An elderly patient presents to the emergency department with a hip fracture after a fall and is in severe pain. To provide effective analgesia while minimizing systemic opioid-related delirium risk in this vulnerable population, which regional technique is commonly used?

A. Femoral nerve block (or fascia iliaca compartment block)

B. Stellate ganglion block targeting cervical sympathetic innervation

C. Celiac plexus block targeting upper abdominal visceral innervation

110. A patient with a tibial shaft fracture and a tight cast develops pain that is markedly out of proportion to the injury, worsening with passive stretch of the toes, and not adequately relieved by escalating opioid doses. What is the most important clinical implication of this pain pattern?

A. This is a red-flag presentation for acute compartment syndrome, requiring urgent evaluation rather than simply escalating analgesia further

B. This pattern simply indicates opioid tolerance and warrants a higher scheduled opioid dose

C. This pattern is expected and requires no further diagnostic evaluation beyond continued analgesic titration

111. A research study identifies several factors associated with a surgical patient's risk of developing chronic postsurgical pain, including severity of acute postoperative pain, preoperative anxiety/catastrophizing, and the specific surgical procedure performed. What is the clinical significance of identifying these risk factors?

A. Chronic postsurgical pain is entirely unpredictable, and these specific factors have no established predictive value at all in practice

B. These factors are useful only for research purposes and have no bearing on real-world perioperative clinical planning decisions

C. These risk factors can help identify high-risk patients for targeted perioperative interventions (e.g., aggressive multimodal analgesia, psychological support) to reduce chronicity risk

112. An opioid-naive postoperative patient is placed on IV patient-controlled analgesia (PCA) with morphine. The order includes a continuous basal infusion rate in addition to patient-activated demand doses. What is the primary safety concern with adding a basal rate for an opioid-naive patient?

A. Basal rates eliminate the risk of respiratory depression entirely by providing steady, continuous dosing

B. Basal rates are a mandatory feature required by PCA pump design and can never be omitted

C. A continuous basal infusion removes the built-in self-titration safety feature, raising oversedation risk in opioid-naive patients

113. A critically ill, mechanically ventilated, sedated patient in the ICU cannot self-report pain using a standard numeric rating scale. Which validated tool is appropriate for assessing pain in this nonverbal, critically ill population?

A. McGill Pain Questionnaire, a verbal descriptor-based self-report tool

B. Critical-Care Pain Observation Tool (CPOT) or Behavioral Pain Scale (BPS)

C. Oswestry Disability Index, a self-report functional questionnaire

114. A hospital establishes a dedicated multidisciplinary team, including pain physicians and specially trained nurses, that rounds daily on all postoperative patients to optimize analgesic regimens, manage complex pain, and reduce opioid-related complications. What is this hospital resource called?

A. Palliative care consult service

B. Rapid response team

C. Acute pain service (APS)

115. A child requires closed reduction of a displaced forearm fracture in the emergency department. The physician administers intravenous ketamine to provide both dissociative sedation and analgesia for the brief, painful procedure. What is the primary advantage of ketamine for this pediatric procedural sedation scenario?

A. Ketamine causes significant respiratory depression, which is desirable to keep the child motionless throughout

B. Ketamine provides dissociative sedation and analgesia while generally preserving airway reflexes and respiration

C. Ketamine is preferred here because it has essentially no psychomimetic side effects in children at all

116. An emergency department implements a low-dose intravenous ketamine protocol as an opioid-sparing adjunct for patients with severe acute traumatic pain, aiming to reduce total opioid exposure

while maintaining adequate analgesia. What mechanism underlies ketamine's analgesic benefit in this opioid-sparing role?

A. NMDA receptor antagonism, which reduces central sensitization and provides analgesia through a non-opioid receptor mechanism

B. Direct mu-opioid receptor agonism, occurring at a lower potency than morphine itself

C. Selective peripheral COX-2 enzyme inhibition at the site of tissue injury

117. A patient is discharged from the emergency department after a minor acute musculoskeletal injury. To reduce the risk of unused opioid diversion and misuse while still addressing acute pain, current best practice guidance for discharge opioid prescribing emphasizes which approach?

A. Prescribing a full 30-day supply to avoid the need for any follow-up refill request later

B. Avoiding any analgesic prescription entirely, regardless of the injury severity involved

C. Prescribing the smallest effective opioid quantity for the expected duration of severe pain, often just a few days

118. A patient reports chronic axial low back pain, worse with sitting and forward flexion, without significant radicular leg symptoms. MRI shows disc desiccation and a high-intensity zone in the posterior annulus at L4-L5. What clinical pattern is most consistent with discogenic pain?

A. Pain that worsens specifically with spinal extension and improves with flexion

B. Axial pain aggravated by flexion, prolonged sitting, and axial loading, without a clear radicular distribution

C. Pain strictly localized to a unilateral sacral sulcus, reproduced only by the FABER maneuver

119. An older adult reports bilateral leg pain and fatigue that occurs after walking a certain distance and is relieved by sitting or leaning forward (flexion), but not simply by standing still. Which condition is this presentation most consistent with?

A. Neurogenic claudication from lumbar spinal stenosis

B. Vascular claudication from peripheral arterial disease

C. Acute lumbar disc herniation with cauda equina syndrome

120. A young athlete with chronic low back pain is found on imaging to have anterior displacement of one vertebral body relative to the one below it, with an associated pars interarticularis defect. What condition does this represent?

A. Spondylolisthesis with spondylolysis

B. Facet joint osteoarthritis without instability

C. Isolated disc herniation without bony pathology

121. A patient reports low back pain that worsens with spinal extension and rotation, and improves with forward flexion, without significant leg pain. Physical exam reveals tenderness over the paraspinal region at the level of the affected joint. Which structure is most likely the pain generator?

A. The intervertebral disc at the corresponding level

B. The lumbar facet (zygapophyseal) joint

C. The sacroiliac joint on the affected side

122. A patient reports unilateral low back and buttock pain that does not cross the midline, without significant radicular symptoms below the knee. On exam, the pain is reproduced by the FABER (flexion, abduction, external rotation) test and the thigh thrust test. Which structure is the likely source of pain?

A. The L4-L5 intervertebral disc

B. The L4-L5 facet joint

C. The sacroiliac joint

123. A patient reports deep buttock pain radiating down the posterior thigh, worsened by prolonged sitting and internal rotation of the hip, with a normal lumbar MRI and no true dermatomal sensory loss. Which condition does this presentation suggest, as a mimic of lumbar radiculopathy?

A. Piriformis syndrome, from sciatic nerve irritation as it courses near or through the piriformis muscle

B. Acute L5-S1 disc herniation causing direct compression of the nerve root

C. Lumbar spinal stenosis presenting classically with neurogenic claudication symptoms

124. A patient with chronic knee pain has imaging showing joint space narrowing, osteophyte formation, and subchondral sclerosis, with minimal synovial inflammation on exam. What is the primary pathophysiologic process underlying this presentation?

A. Autoimmune synovial inflammation with pannus formation eroding cartilage and bone

B. Progressive degeneration of articular cartilage with secondary subchondral bone remodeling and osteophyte formation

C. Crystal deposition within the joint space triggering acute inflammatory flares

125. A patient reports symmetric pain and swelling in the small joints of both hands, with morning stiffness lasting more than one hour that improves with activity. Laboratory testing shows positive rheumatoid factor and anti-CCP antibodies. What pain pattern and underlying process characterize this condition?

A. Degenerative, mechanically worsened pain from cartilage loss without systemic inflammatory markers

B. Episodic, crystal-induced acute monoarticular attacks

C. Inflammatory pain pattern with prolonged morning stiffness improving with activity, driven by autoimmune synovitis

126. A patient presents with sudden-onset, severe pain, redness, and swelling of the first metatarsophalangeal joint, reaching peak intensity within 24 hours. Joint aspiration reveals needle-shaped, negatively birefringent crystals. What condition does this represent?

A. Acute gouty arthritis from monosodium urate crystal deposition

B. Calcium pyrophosphate deposition disease (pseudogout)

C. Septic arthritis from bacterial joint infection

127. An elderly patient presents with acute knee pain, swelling, and warmth. Joint aspiration reveals rhomboid-shaped, positively birefringent crystals under polarized microscopy. Which condition does this represent, and how does it differ from gout?

A. Gout, since both conditions share identical urate crystal types, shape, and birefringence pattern

B. Septic arthritis, since any positive crystal birefringence under polarized light always indicates bacterial infection

C. Calcium pyrophosphate deposition disease (pseudogout), with rhomboid, positively birefringent crystals

128. A patient reports progressive shoulder pain and stiffness over several months, now with significant restriction of both active AND passive range of motion in all planes, without a history of trauma. What condition does this presentation most likely represent?

A. Rotator cuff tear with preserved passive range of motion

B. Adhesive capsulitis (frozen shoulder)

C. Acute subacromial bursitis with normal passive motion

129. A patient reports shoulder pain with overhead activities. On exam, pain is reproduced by passive forward flexion of the arm with the examiner's hand stabilizing the scapula (Neer test) and by internal rotation with the arm at 90 degrees of forward flexion (Hawkins test). What condition do these findings suggest?

A. Subacromial impingement syndrome / rotator cuff tendinopathy

B. Adhesive capsulitis with global range of motion loss

C. Acromioclavicular joint separation

130. A patient reports lateral elbow pain that worsens with resisted wrist extension and gripping activities, such as shaking hands or lifting a coffee cup, following repetitive activities like tennis or manual labor. What condition does this describe?

A. Medial epicondylitis (golfer's elbow), from flexor-pronator tendon overuse

B. Cubital tunnel syndrome, from ulnar nerve entrapment at the elbow

C. Lateral epicondylitis (tennis elbow), from repetitive extensor tendon overuse at the lateral epicondyle

131. A golfer reports medial elbow pain reproduced by resisted wrist flexion and pronation, without numbness or tingling in the hand. What condition does this represent?

A. Lateral epicondylitis, from repetitive extensor tendon overuse at the elbow

B. Ulnar collateral ligament tear presenting with valgus instability at the elbow

C. Medial epicondylitis (golfer's elbow), from overuse of the flexor-pronator tendon origin at the medial epicondyle

132. A new mother reports pain at the radial side of the wrist, worsened by lifting her infant and reproduced when she makes a fist with her thumb tucked inside her fingers and ulnarly deviates the wrist. What condition and clinical test does this describe?

A. Carpal tunnel syndrome, confirmed by a positive Tinel sign at the wrist

B. De Quervain tenosynovitis, confirmed by a positive Finkelstein test

C. Trigger finger from flexor tendon nodule catching

133. A patient reports chronic lateral hip pain, worse when lying on the affected side at night and with prolonged walking, with point tenderness directly over the lateral aspect of the hip. What condition does this presentation suggest?

A. Greater trochanteric pain syndrome (trochanteric bursitis/gluteal tendinopathy)

B. Hip osteoarthritis with groin-predominant pain

C. Meralgia paresthetica from lateral femoral cutaneous nerve entrapment

134. A patient reports sharp heel pain that is worst with the first steps out of bed in the morning, improving somewhat with continued walking but worsening again after prolonged standing later in the day. What condition does this presentation describe?

A. Achilles tendinopathy, with pain localized to the posterior heel and tendon insertion

B. Plantar fasciitis, with classic first-step morning pain from microtrauma to the plantar fascia

C. Tarsal tunnel syndrome from posterior tibial nerve entrapment

135. A recreational runner reports gradually worsening pain and stiffness at the back of the ankle, just above the heel, worse at the start of a run and after periods of rest, with palpable thickening of the tendon on exam. What condition does this describe?

A. Plantar fasciitis localized to the plantar heel

B. Achilles tendinopathy, from chronic overuse degeneration of the Achilles tendon

C. Posterior tibial tendon dysfunction causing acquired flatfoot

136. A patient involved in a rear-end motor vehicle collision develops neck pain, stiffness, and headache in the days following the accident, attributed to a rapid acceleration-deceleration injury mechanism to the cervical spine soft tissues. What is this condition and injury mechanism called?

A. Cervical radiculopathy resulting from an acute cervical disc herniation

B. Cervical spondylotic myelopathy from chronic degenerative cord compression

C. Whiplash-associated disorder, from rapid acceleration-deceleration soft tissue injury to the cervical spine

137. A patient reports unilateral headache that starts in the neck/occipital region and radiates anteriorly, worsened by neck movement and sustained postures, with restricted cervical range of motion on exam and tenderness over the upper cervical facet joints. What condition is this presentation most consistent with?

A. Cervicogenic headache, originating from cervical spine structures and referred to the head

B. Classic migraine occurring without any cervical spine involvement at all

C. Trigeminal neuralgia presenting with paroxysmal facial pain episodes

138. A patient has focal, regional pain in the upper trapezius with a palpable taut band and a hyperirritable point that reproduces referred pain to the neck when pressed, without the widespread, whole-body pain pattern or the fatigue/cognitive symptoms of fibromyalgia. How does this presentation differ from fibromyalgia?

A. This presentation is simply an early or mild form of fibromyalgia sharing identical pathophysiology

B. This describes myofascial pain syndrome, a regional condition with focal trigger points, distinct from fibromyalgia

C. There is no meaningful clinical distinction between myofascial pain syndrome and fibromyalgia at all

139. A patient reports reproducible chest wall pain, sharp and localized, worsened by palpation of the costochondral junctions and by deep breathing or twisting motions, with a normal cardiac workup. What condition does this presentation describe?

A. Costochondritis, an inflammatory condition of the costochondral cartilage producing reproducible chest wall tenderness

B. Unstable angina requiring urgent cardiac catheterization and coronary intervention promptly

C. Pleuritic pain from an acute pulmonary embolism event with dyspnea

140. A young adult reports chronic low back and buttock pain of insidious onset, worse in the morning with stiffness lasting over 30 minutes that improves with exercise but not with rest, along with limited spinal mobility. What underlying condition should be suspected?

A. Mechanical low back pain resulting from an acute lumbar muscle strain

B. Ankylosing spondylitis, an inflammatory spondyloarthropathy presenting with inflammatory back pain features

C. Osteoporotic vertebral compression fracture at the affected level

141. An elderly patient reports new-onset bilateral shoulder and hip girdle pain and stiffness, most severe in the morning, along with fatigue, and is found to have a markedly elevated erythrocyte sedimentation rate. What condition does this presentation most strongly suggest?

A. Fibromyalgia, in which normal inflammatory markers would be expected here

B. Osteoarthritis affecting the shoulders and hips bilaterally in this patient

C. Polymyalgia rheumatica, an inflammatory condition causing girdle pain with elevated markers

142. An elderly patient with known osteoporosis develops sudden, severe midline back pain after a minor fall, without radicular symptoms, and imaging reveals height loss of a thoracic vertebral body. What condition does this presentation represent?

A. Lumbar facet joint arthropathy

B. Acute lumbar disc herniation with radiculopathy

C. Osteoporotic vertebral compression fracture

143. A palliative care team manages a patient's escalating cancer pain by starting with non-opioid analgesics, then progressing to weak opioids for mild-moderate pain, and finally to strong opioids for severe pain, adding adjuvants at each step as needed. What established framework guides this stepwise approach?

A. The WHO analgesic ladder framework for stepwise pain treatment

B. The AMA Guides to the Evaluation of Permanent Impairment

C. The ACR 2016 diagnostic criteria for fibromyalgia

144. A patient on scheduled long-acting opioid therapy for cancer pain experiences intermittent episodes of severe pain that arise rapidly, peak within minutes, and resolve within an hour, occurring several times daily despite adequate baseline control. What is this phenomenon called, and what treatment approach is appropriate?

A. Opioid-induced hyperalgesia, treated by reducing the scheduled baseline opioid dose given daily

B. Breakthrough cancer pain, treated with a rapid-onset, short-acting opioid formulation in addition to the scheduled baseline regimen

C. Pseudoaddiction, treated by promptly discontinuing all ongoing opioid therapy entirely

145. A patient with metastatic breast cancer to bone reports severe, localized pain at sites of known osseous metastases. Which mechanism is primarily responsible for pain generation at these skeletal metastatic sites?

A. Direct mechanical compression of the spinal cord happening at every single metastatic bone site present

B. Autoimmune synovitis triggered directly by circulating tumor cells lodging within the joint space

C. Tumor-induced osteoclast activation and local mediator release (e.g., prostaglandins, RANKL-driven bone resorption) sensitizing periosteal nociceptors

146. A patient receiving chemotherapy for colorectal cancer develops symmetric, distal numbness, tingling, and burning pain in a stocking-glove distribution in the hands and feet. Which chemotherapeutic agent class is classically associated with this presentation?

A. Platinum-based agents (e.g., oxaliplatin), a well-recognized cause of chemotherapy-induced peripheral neuropathy

B. Topical corticosteroids applied directly to the affected symptomatic areas

C. Beta-lactam antibiotics used routinely for infection prophylaxis in this setting

147. A patient with severe, escalating cancer pain requires progressively higher doses of a full mu-opioid agonist to maintain analgesia as the disease progresses. Unlike NSAIDs, what pharmacologic property of full opioid agonists allows this ongoing dose escalation to remain effective?

A. Full opioid agonists have a fixed analgesic ceiling dose beyond which no additional benefit occurs, identical to NSAIDs

B. Full opioid agonists lack a clinically meaningful analgesic ceiling effect, allowing dose titration to be continued as needed for increasing pain

C. Full opioid agonists can only be titrated upward for a maximum of two dose adjustments before requiring drug rotation

148. A patient with advanced ovarian cancer develops malignant bowel obstruction with associated colicky abdominal pain. Standing scheduled opioids alone provide incomplete relief of the cramping component. What is an important consideration in optimizing analgesia for this specific pain pattern?

A. Adding an antispasmodic/anticholinergic agent to address the colicky, smooth-muscle-mediated component of the pain, alongside opioid therapy

B. Discontinuing all opioids entirely, since they are considered contraindicated in bowel obstruction

C. Switching exclusively to a topical NSAID applied over the abdominal wall skin

149. A patient with known metastatic prostate cancer develops new, severe back pain along with bilateral leg weakness and urinary retention over the course of a day. What is the most urgent concern requiring immediate evaluation?

A. A routine osteoarthritis flare requiring only outpatient NSAID therapy for adequate management

B. A simple musculoskeletal back strain requiring only a physical therapy referral for treatment

C. Malignant epidural spinal cord compression, an oncologic emergency requiring urgent MRI and treatment (e.g., corticosteroids, radiation, or surgery)

150. A patient with a lytic metastatic lesion in the femoral shaft is evaluated by orthopedic oncology to determine the risk of impending pathologic fracture and the need for prophylactic surgical fixation before the lesion causes a complete fracture. Which scoring system is classically used for this risk assessment?

A. The Oswestry Disability Index for chronic low back pain disability

B. The APACHE II critical illness severity scoring system used in ICUs

C. Mirels' criteria, scoring lesion site, size, radiographic appearance, and pain, to estimate fracture risk

151. A patient with pancreatic cancer has pain that includes a deep, poorly localized epigastric component from tumor infiltration of surrounding tissue, along with a burning, radiating component from tumor invasion of the celiac plexus nerve bundle. This mixed presentation illustrates which characteristic of cancer pain?

A. Cancer pain often has mixed nociceptive (visceral/somatic) and neuropathic components simultaneously, requiring a multimodal approach targeting each component

B. Cancer pain is always purely nociceptive in its origin and never involves any neuropathic mechanisms whatsoever

C. Cancer pain is always purely neuropathic in its origin and never involves any nociceptive mechanisms whatsoever

152. A patient with a single, well-localized painful bone metastasis is referred for palliative external beam radiation therapy. The radiation oncologist discusses that a single fraction of radiation can provide comparable pain relief to a longer, multi-fraction course for this indication, with the trade-off of a somewhat higher retreatment rate. What does this illustrate about palliative radiotherapy for painful bone metastases?

A. Radiation therapy has no role whatsoever in managing painful bone metastases and should never ever be offered

B. Single-fraction radiotherapy provides comparable pain relief to multi-fraction regimens for uncomplicated painful bone metastases, with a modestly higher rate of needing retreatment

C. Radiation therapy for bone metastases works exclusively through a systemic immune-mediated mechanism unrelated to the site

153. A patient is started on scheduled opioid therapy for cancer pain. The palliative care team simultaneously initiates a prophylactic bowel regimen with a stimulant laxative rather than waiting to treat constipation only if and when it develops. What is the rationale for this proactive approach?

A. Opioid-induced constipation is rare and typically resolves spontaneously with continued opioid use, so prophylaxis is unnecessary in this setting

B. Opioid-induced constipation is extremely common and, unlike many other opioid side effects, does not improve with tolerance over time, so prophylactic bowel regimens are standard practice

C. Bowel regimens are started prophylactically only to treat opioid-induced nausea, never to treat constipation itself

154. A hospice patient with end-stage cancer pain requires escalating opioid doses for adequate comfort, and the family expresses concern that increasing the dose might hasten death through respiratory depression. Under the ethical principle of double effect, how should the clinician approach this situation?

A. Opioids should be withheld entirely near the end of life regardless of severity, to avoid any respiratory risk

B. The intent to hasten death justifies opioid titration here, since causing death is an acceptable primary goal

C. Titrating opioids with the primary intent of relieving suffering is ethically appropriate even with a foreseen, unintended risk of shortening life, if proportionate

155. A patient with an intra-abdominal process reports pain that is diffuse, poorly localized, and difficult to describe precisely, sometimes accompanied by referred pain in a distant somatic area sharing the same spinal segmental innervation. What general characteristic of visceral pain does this illustrate?

A. Visceral pain is typically poorly localized due to sparse, widely divergent afferent innervation, and can be referred to somatic sites

B. Visceral pain is always sharply and precisely localized to the exact organ involved, unlike somatic pain

C. Visceral pain never produces any referred pain patterns at all, unlike somatic pain does

156. A patient with chronic pancreatitis reports persistent, severe epigastric pain radiating to the back, worsened after meals. Beyond acute inflammation, which additional mechanism is thought to contribute to the persistent, often disproportionate pain seen in chronic pancreatitis?

A. Isolated peripheral skin nociceptor sensitization occurring at the epigastric dermatome level

B. Pancreatic ductal hypertension with perineural inflammation contributing to persistent pain and sensitization

C. Pain in chronic pancreatitis is purely psychogenic with no identifiable physiologic contributor at all

157. A patient with irritable bowel syndrome reports abdominal pain and altered bowel habits, with normal structural workup (colonoscopy, imaging). Experimental studies show this patient perceives pain at a lower threshold of rectal balloon distension than healthy controls. What phenomenon does this illustrate?

A. Peripheral nociceptor destruction resulting from chronic mechanical bowel injury over time

B. A structural bowel obstruction present but simply not detected on standard imaging studies

C. Visceral hypersensitivity, a lowered pain threshold to normal visceral stimuli, thought to be central to IBS pathophysiology

158. A patient reports chronic pelvic pain and pressure associated with bladder filling, along with urinary urgency and frequency, that improves somewhat after voiding. Cystoscopy shows glomerulations without evidence of infection or malignancy. What condition does this presentation describe?

A. Interstitial cystitis/bladder pain syndrome

B. Acute bacterial cystitis requiring antibiotic therapy

C. Bladder malignancy requiring oncologic workup

159. A woman reports chronic, cyclic pelvic pain that worsens with menstruation, along with dyspareunia, and is found on laparoscopy to have ectopic endometrial tissue implants outside the uterus with associated local inflammation. What is the primary mechanism underlying this chronic pelvic pain?

A. Ectopic endometrial tissue causing local inflammation and cyclic hormonally-driven tissue changes that perpetuate pelvic pain

B. An entirely psychogenic pain process with no relation to any identifiable pelvic pathology whatsoever

C. An isolated bladder pain syndrome entirely unrelated to the laparoscopic findings described

160. A patient having a myocardial infarction reports crushing chest pain that radiates to the left arm and jaw, despite the heart itself lacking somatic sensory innervation in those distant locations. What mechanism explains this referred pain pattern?

A. Direct mechanical compression of the brachial plexus nerve bundle by the heart itself

B. Convergence of cardiac and somatic afferents from the arm/jaw on shared dorsal horn neurons, causing the brain to misattribute location

C. Cardiac pain is purely psychogenic and does not reflect any true underlying visceral afferent signaling

161. A patient with an obstructing ureteral stone reports severe, colicky flank pain that radiates anteriorly and inferiorly toward the groin and ipsilateral testicle/labia, waxing and waning in intensity. What underlies this specific visceral referred pain pattern?

A. Ureteral distension activates visceral afferents sharing spinal levels (T11-L1) with somatic afferents to the groin/genital region

B. Direct mechanical irritation of the femoral nerve trunk by the stone itself as it passes through

C. The pain pattern reflects primary genital organ pathology entirely unrelated to the ureteral stone

162. A patient reports chronic postprandial fullness, early satiety, and epigastric discomfort, with a normal upper endoscopy and no structural abnormality identified. What underlying mechanism is proposed to explain this presentation?

A. An occult gastric malignancy that was simply missed during the endoscopic evaluation

B. Chronic bacterial gastritis requiring prolonged antibiotic therapy regardless of the actual testing results

C. Functional dyspepsia, thought to involve visceral hypersensitivity and impaired gastric accommodation despite a structurally normal upper GI tract

163. A patient reports recurrent headaches lasting 4-72 hours, unilateral, pulsatile in quality, moderate to severe in intensity, aggravated by routine physical activity, and associated with photophobia, phonophobia, and occasional nausea. What headache type does this presentation describe?

A. Tension-type headache, bilateral and pressing in quality

B. Cluster headache, a trigeminal autonomic cephalalgia type

C. Migraine, presenting with unilateral pulsatile headache

164. A patient reports frequent headaches described as a bilateral, pressing or tightening band-like sensation around the head, mild to moderate in intensity, not worsened by routine physical activity, and without associated nausea or significant photophobia/phonophobia. What headache type does this describe?

A. Migraine with aura

B. Tension-type headache

C. Trigeminal neuralgia

165. A patient reports recurrent attacks of severe, strictly unilateral periorbital/temporal pain lasting 15-180 minutes, occurring in clusters over several weeks with a striking circadian pattern (often waking the patient at night), accompanied by ipsilateral lacrimation, conjunctival injection, and nasal congestion. What headache syndrome does this describe?

A. Chronic tension-type headache with bilateral pressing quality

B. Medication overuse headache from frequent analgesic use

C. Cluster headache, a trigeminal autonomic cephalalgia

166. A patient reports brief, severe, electric shock-like episodes of facial pain in the distribution of the maxillary division of the trigeminal nerve, triggered by light touch such as chewing, talking, or a breeze on the face, each episode lasting only seconds. What condition does this describe?

A. Trigeminal neuralgia

B. Cluster headache

C. Temporomandibular disorder

167. A patient reports jaw pain and clicking with chewing and jaw opening, along with tenderness over the masseter and temporalis muscles and the temporomandibular joint itself, without the brief shock-like pain paroxysms typical of trigeminal neuralgia. What condition does this presentation describe?

A. Trigeminal neuralgia, with brief electric shock-like pain

B. Cluster headache, with unilateral periorbital autonomic features

C. Temporomandibular disorder (TMD), a jaw joint/muscle condition

168. A patient with a prior history of episodic migraine now reports headache occurring more than 15 days per month, and reports using over-the-counter combination analgesics on most of those days in an attempt to control the pain, with the headache pattern becoming progressively more frequent and less responsive to treatment over time. What condition does this represent?

A. Medication overuse headache, a chronic daily headache pattern driven by frequent use of acute analgesic medications

B. New-onset cluster headache unrelated to the prior migraine history

C. An unrelated, incidental tension-type headache with no connection to analgesic use frequency

169. Following a peripheral nerve injury, a patient develops spontaneous burning pain in the nerve's distribution. Electrophysiologic studies of the injured nerve show abnormal spontaneous discharges arising directly from the site of injury, even without any external stimulus. What mechanism does this represent?

A. Normal, expected background firing rate of an uninjured peripheral nerve

B. Ectopic (spontaneous) neuronal firing arising from the site of nerve injury, contributing to neuropathic pain generation

C. A purely psychogenic pain process with no underlying electrophysiologic abnormality

170. A patient who had a herpes zoster (shingles) outbreak in the T4 dermatome continues to experience burning, allodynic pain in that same distribution more than 4 months after the skin rash has fully resolved. What condition does this represent?

A. Acute herpes zoster pain, defined as pain occurring during the active rash

B. Subacute herpetic neuralgia, defined as pain lasting exactly 30 days after rash onset

C. Postherpetic neuralgia, defined as pain persisting beyond approximately 3 months after the herpes zoster rash has resolved

171. A patient with long-standing type 2 diabetes reports burning pain, numbness, and tingling beginning in the toes and gradually ascending up both feet in a symmetric pattern, sparing the hands until later in the disease course. What pattern of neuropathy does this describe?

A. Distal symmetric, length-dependent sensorimotor polyneuropathy, the classic pattern of diabetic peripheral neuropathy

B. An acute, asymmetric mononeuropathy affecting a single isolated peripheral nerve

C. A proximal-predominant neuropathy sparing the distal extremities entirely

172. A patient develops disproportionate pain, swelling, temperature asymmetry, and allodynia in a limb following a minor wrist sprain, without any identifiable major nerve injury on electrodiagnostic testing. How is this classified within complex regional pain syndrome subtypes?

A. CRPS Type II, defined by the presence of a confirmed major nerve injury

B. CRPS Type I, defined by the absence of a confirmed major nerve injury

C. CRPS is not subclassified into types based on nerve injury status

173. A patient with suspected complex regional pain syndrome of the foot is evaluated using a set of clinical diagnostic criteria requiring reported symptoms in the categories of sensory, vasomotor, sudomotor/edema, and motor/trophic changes, along with corresponding objective signs on exam in at least two of those categories. What diagnostic criteria set is being applied?

A. The ACR 2016 fibromyalgia criteria

B. The Budapest criteria for CRPS diagnosis

C. The Mirels' criteria for pathologic fracture risk

174. A patient who underwent below-knee amputation continues to perceive pain localized to the absent foot, described as cramping and burning, months after the surgical site has fully healed. What is this phenomenon called, and what is a proposed contributing mechanism?

A. Phantom limb pain, thought to involve cortical reorganization/remapping of the sensory cortex following deafferentation

B. Residual limb (stump) pain, caused exclusively by ongoing local surgical wound inflammation

C. Referred visceral pain from an unrelated abdominal process

175. A patient reports numbness and tingling in the thumb, index, and middle fingers, worse at night, reproduced by tapping over the volar wrist crease and by sustained wrist flexion. Which nerve entrapment does this represent?

A. Median nerve entrapment at the wrist (carpal tunnel syndrome)

B. Ulnar nerve entrapment at the elbow (cubital tunnel syndrome)

C. Radial nerve entrapment at the spiral groove

176. A patient reports numbness and tingling in the ring and small fingers, along with weakness of grip, worse with prolonged elbow flexion (such as holding a phone to the ear), and reproduced by tapping over the medial elbow. Which nerve entrapment does this represent?

A. Median nerve entrapment at the wrist (carpal tunnel syndrome)

B. Ulnar nerve entrapment at the elbow (cubital tunnel syndrome)

C. Musculocutaneous nerve entrapment at the arm

177. A patient sustains a mild peripheral nerve injury with focal segmental demyelination but no axonal disruption, causing a temporary conduction block that fully resolves within a few weeks. Which classification of nerve injury does this represent?

A. Neurotmesis, the most severe category involving complete nerve transection entirely

B. Axonotmesis, involving axonal disruption with the surrounding connective tissue preserved

C. Neurapraxia, the mildest category, involving a temporary conduction block from focal demyelination without axonal disruption

178. A patient develops severe, burning, allodynic pain on one side of the body several weeks after a thalamic stroke, in a distribution roughly corresponding to the sensory deficit from the stroke itself. What condition does this represent?

A. Peripheral diabetic neuropathy occurring entirely unrelated to the stroke event

B. Complex regional pain syndrome occurring secondary to the stroke-affected limb itself

C. Central post-stroke pain (thalamic pain syndrome), from central nervous system injury affecting somatosensory pathways

179. A patient with a traumatic spinal cord injury at T6 develops burning, neuropathic pain both at the level of the injury (in a band-like distribution around the trunk) and diffusely below the level of injury in areas with little or no other sensation. What is this pattern of pain called?

A. Peripheral entrapment neuropathy occurring entirely unrelated to the spinal cord injury

B. At-level and below-level neuropathic pain, both recognized patterns of central neuropathic pain following spinal cord injury

C. Referred visceral pain arising from an unrelated intra-abdominal disease process

180. A patient with obesity reports burning, tingling pain and numbness over the anterolateral thigh, without motor weakness or reflex changes, worsened by prolonged standing and tight-fitting clothing at the waist. Which nerve entrapment does this represent?

A. Femoral nerve entrapment causing quadriceps weakness

B. Sciatic nerve entrapment causing posterior thigh and calf symptoms

C. Lateral femoral cutaneous nerve entrapment (meralgia paresthetica)

181. A patient reports burning pain, numbness, and tingling on the plantar aspect of the foot, worse with prolonged standing or walking, reproduced by tapping just posterior and inferior to the medial malleolus. Which nerve entrapment does this represent?

A. Lateral femoral cutaneous nerve entrapment (meralgia paresthetica)

B. Posterior tibial nerve entrapment at the tarsal tunnel (tarsal tunnel syndrome)

C. Common peroneal nerve entrapment at the fibular head

182. A patient reports burning pain and altered temperature sensation in the feet, but standard nerve conduction studies and EMG are entirely normal. A skin punch biopsy shows reduced intraepidermal nerve fiber density. What condition does this represent?

A. Small fiber neuropathy, which selectively affects thinly myelinated A-delta and unmyelinated C fibers not well captured by standard EMG/NCS

B. Large fiber neuropathy, which is reliably diagnosed by standard EMG/NCS testing in all cases

C. A normal, expected finding on skin biopsy requiring no further diagnostic workup at all

183. A patient with peripheral neuropathy reports that a light touch from clothing, which would not normally be painful, now produces significant pain, while a normally mildly uncomfortable pinprick now produces markedly exaggerated pain. What terms correctly describe these two distinct phenomena, respectively?

A. Both phenomena are correctly and interchangeably termed hyperalgesia in standard terminology

B. Allodynia (pain from a normally non-painful stimulus) and hyperalgesia (exaggerated pain to a normally painful one)

C. Both phenomena are correctly termed paresthesia, since both involve altered sensory perception

184. A patient with a long history of heavy alcohol use presents with symmetric, distal burning pain, numbness, and gait imbalance in both feet. Nerve conduction studies show a length-dependent, predominantly axonal sensorimotor polyneuropathy. Beyond direct neurotoxic effects of alcohol, what commonly co-occurring nutritional deficiency contributes to this neuropathy?

A. Vitamin D deficiency, the primary driver of alcohol-related peripheral neuropathy

B. Vitamin C deficiency, the primary driver of alcohol-related peripheral neuropathy

C. Thiamine (vitamin B1) deficiency, commonly co-occurring in chronic heavy alcohol use and contributing to peripheral neuropathy

185. A patient with sickle cell disease presents with severe, acute bony pain consistent with a vaso-occlusive crisis. What is the recommended approach to initial analgesic management for this acute pain episode?

A. Prompt, aggressive analgesia, typically with parenteral opioids, initiated rapidly rather than delayed, given the severity of acute vaso-occlusive pain

B. Withholding opioid analgesia entirely due to concern for opioid use disorder in this population

C. Treatment with NSAIDs alone, since opioids are considered contraindicated in sickle cell disease

186. A pregnant patient in the third trimester with chronic musculoskeletal pain asks about safe analgesic options. Why are NSAIDs generally avoided in the third trimester of pregnancy?

A. Third-trimester NSAID use is associated with risk of premature closure of the fetal ductus arteriosus and oligohydramnios

B. NSAIDs are avoided in the third trimester solely due to an unfounded historical concern with no actual physiologic basis

C. NSAIDs are avoided in the third trimester because they cause neural tube defects at this stage of gestation

187. A patient maintained on buprenorphine for opioid use disorder is scheduled for an elective surgical procedure expected to cause significant postoperative pain. Current perioperative guidance regarding buprenorphine management generally favors which approach?

A. Buprenorphine must always be completely discontinued at least a month before any surgery, no exceptions

B. Buprenorphine has no analgesic properties at all and is therefore irrelevant to perioperative pain planning entirely

C. Continuing buprenorphine perioperatively in most cases, supplemented with multimodal and full agonist opioid analgesia as needed

188. A physician needs to assess pain in a nonverbal patient with advanced dementia who cannot reliably self-report pain using a standard numeric rating scale. Which type of tool is appropriate for this population?

A. An observational tool, such as PAINAD, scoring breathing, vocalization, facial expression, and body language

B. The McGill Pain Questionnaire, relying entirely on verbal descriptor word selection

C. A purely laboratory-based biomarker test with no behavioral observation component at all

189. A pain physician prescribing an opioid for an elderly patient with multiple comorbidities and reduced renal and hepatic function starts at a lower dose than would be used in a younger adult and titrates more slowly, closely monitoring for sedation and other side effects. What principle guides this approach?

A. "Start low, go slow" -- reflecting age-related pharmacokinetic and pharmacodynamic changes that increase sensitivity to and reduce clearance of many analgesics in older adults

B. Elderly patients require a higher starting dose than younger adults because of reduced drug sensitivity

C. Age has no meaningful effect on drug pharmacokinetics or pharmacodynamics, so standard adult dosing should always be used regardless of age

190. A pediatric anesthesiologist calculates a postoperative opioid dose for a 6-year-old child based on the child's body weight in kilograms, using a milligram-per-kilogram dosing formula rather than a fixed adult dose. What is the rationale for weight-based dosing in pediatric pain management?

A. Weight-based dosing is used purely as a hospital billing convention with no true pharmacologic basis at all

B. Children have different body composition and drug clearance relative to size, making weight-based dosing necessary

C. Weight-based dosing is only relevant for antibiotics and has no application to analgesic medications at all

191. A patient with an implanted spinal cord stimulator requires an MRI for an unrelated indication. Before proceeding, what is the most important safety consideration regarding the implanted device?

A. All spinal cord stimulators are universally MRI-safe under any scanner conditions with no verification needed at all

B. The specific device must be confirmed MRI-conditional, with scanning following the manufacturer specific conditions for safety

C. MRI safety is entirely irrelevant for spinal cord stimulators since they contain no ferromagnetic components whatsoever

192. A pain physician selecting an analgesic regimen for a patient with significant hepatic cirrhosis considers that many opioids and acetaminophen undergo substantial hepatic metabolism. What is an important consideration for dosing in this population?

A. Hepatic impairment has no clinically meaningful effect at all on the metabolism of opioids or acetaminophen

B. Acetaminophen should always be used at maximum standard doses in cirrhosis without any adjustment whatsoever

C. Dose reduction and/or extended intervals are often needed for hepatically metabolized analgesics, with acetaminophen typically limited

193. A patient with end-stage renal disease on hemodialysis requires opioid analgesia for acute pain. The physician avoids morphine due to concern for accumulation of an active, renally cleared metabolite that can cause sedation, myoclonus, and respiratory depression. Which opioid is this concern most classically associated with in renal impairment?

A. Fentanyl, due to accumulation of an active renally cleared metabolite

B. Morphine, due to accumulation of the active, renally cleared metabolite morphine-6-glucuronide

C. Methadone, due to primarily renal (rather than hepatic) elimination

194. An infant born to a mother who used opioids throughout pregnancy develops irritability, high-pitched crying, tremors, and feeding difficulties in the days after birth. What condition does this represent?

A. Congenital malformation directly and specifically caused by opioid teratogenicity in utero

B. Neonatal abstinence syndrome (neonatal opioid withdrawal syndrome), from in utero opioid exposure and postnatal withdrawal

C. An unrelated primary neonatal seizure disorder occurring independently of exposure

195. A patient with a documented active substance use disorder presents with a genuine acute painful condition, such as a fracture. The treating physician must balance the risk of undertreating legitimate pain against the risk of exacerbating the substance use disorder. What is the most appropriate general approach?

A. Automatically withhold all opioid analgesics regardless of pain severity, based on the diagnosis alone

B. Treat the fracture pain exactly as any other patient, with no additional monitoring since injury pain overrides concerns

C. Provide adequate analgesia using structured monitoring and opioid-sparing strategies, with careful dosing rather than reflexive withholding

196. A veteran with combat-related posttraumatic stress disorder presents with chronic musculoskeletal pain that has proven more difficult to treat than expected, with the pain physician recognizing that PTSD and chronic pain frequently co-occur and can amplify one another. What does the literature suggest about the relationship between PTSD and chronic pain?

A. PTSD and chronic pain frequently co-occur, share overlapping neurobiological and psychological mechanisms, and each condition can worsen the severity and treatment resistance of the other

B. PTSD and chronic pain have no established relationship and their co-occurrence in veterans is purely coincidental

C. PTSD always fully resolves once the comorbid chronic pain condition is adequately treated, with no need for independent PTSD-focused treatment

197. A pain physician performing an interventional spine procedure on a patient with severe obesity anticipates specific technical challenges related to needle depth, image quality, and anatomic landmark visualization. What is an important procedural consideration in this population?

A. Obesity has no meaningful impact on interventional procedure technique or imaging quality at all

B. Standard-length needles are always adequate regardless of body habitus, with no equipment modification ever needed

C. Increased soft tissue depth in obesity may require longer needles and more careful landmark identification to reach the target

198. A patient with advanced, end-stage chronic obstructive pulmonary disease (not cancer) experiences significant dyspnea and associated chest wall pain in the final stages of illness. The palliative team uses low-dose opioids to manage both symptoms. What is an important principle guiding opioid use for pain and dyspnea in this non-cancer, end-of-life setting?

A. Carefully titrated, low-dose opioids can relieve both pain and dyspnea at the end of life in non-cancer illness, per palliative principles

B. Opioids should never be used for any non-cancer end-of-life condition under any circumstances whatsoever

C. Opioids are only appropriate for dyspnea in COPD if the patient also has a concurrent, separately active cancer diagnosis

199. A patient taking an SSRI antidepressant for depression is started on tramadol for pain and subsequently develops agitation, hyperreflexia, clonus, tremor, and hyperthermia. What drug interaction and syndrome does this represent?

A. Simple opioid overdose from tramadol alone, occurring entirely unrelated to the SSRI

B. Serotonin syndrome, from combined serotonergic effects of tramadol and the SSRI

C. Acute opioid withdrawal syndrome from abrupt cessation of the medication

200. A patient with a longstanding opioid use disorder is started on buprenorphine/naloxone as medication for opioid use disorder (MOUD) treatment, distinct from using buprenorphine specifically as an analgesic for a pain condition. What is the primary treatment goal of buprenorphine in this MOUD context?

A. To provide maximal analgesia for an acute painful condition as the primary treatment goal

B. To serve exclusively as a short-term detoxification agent with no role in longer-term maintenance treatment

C. To reduce opioid cravings and withdrawal, block euphoric effects of illicit opioid use, and support long-term recovery from opioid use disorder

Answers

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1. A. A-delta fibers are thinly myelinated, conduct at 5-30 m/s, and carry the fast, sharp, well-localized first-pain sensation. C fibers are unmyelinated, conduct much more slowly (<2 m/s), and mediate the dull, poorly localized second pain that follows -- a real competing nociceptive fiber type, but the wrong conduction speed and quality for this vignette. A-beta fibers are large, myelinated, fast-conducting (30-70 m/s) fibers that mediate light touch and proprioception rather than nociception, so they would not be activated by a pinprick as a pain fiber at all.

2. A. Lamina II, the substantia gelatinosa, is the principal termination zone for C-fiber nociceptive afferents and is densely populated with mu-opioid receptors, making it a primary site of spinal opioid analgesia. Lamina IX is a real, distinct spinal gray matter region, but it contains motor neuron cell bodies in the ventral horn and has no role in nociceptive processing, which is why it is wrong. Lamina

III lies just deep to the substantia gelatinosa and receives predominantly A-beta (non-nociceptive, mechanoreceptive) input rather than C-fiber nociceptive input, so it does not fit the description given.

3. B. Descending inhibitory pathways originating in brainstem nuclei such as the periaqueductal gray and rostral ventromedial medulla mature later in gestation than ascending nociceptive circuitry, leaving preterm neonates with disproportionately unopposed excitatory transmission and heightened pain sensitivity. Peripheral nociceptor density in skin is actually established early and is not the developmentally lagging feature responsible for the exaggerated neonatal pain response. Spinal reflex withdrawal circuitry is also functional very early in gestation (it can be elicited well before descending inhibition matures), so its presence does not explain the immaturity being described here.

4. B. Central sensitization increases the excitability of dorsal horn neurons so that input from low-threshold A-beta fibers is processed as painful, producing allodynia -- exactly the phenomenon described. Nociceptor threshold elevation is a real but opposite process (it would make stimuli LESS likely to be perceived as painful, not more), so it does not match a picture of exaggerated pain response. Peripheral nerve myelination arrest is a genuine developmental process but concerns fiber conduction maturation, not the central excitability change that converts touch into pain, so it does not fit this vignette.

5. C. Keeping both participants and outcome assessors unaware of treatment allocation is the defining feature of double-blinding, which minimizes performance bias (differential care or expectation effects) and detection bias (differential outcome assessment) -- precisely what the vignette describes. External validity concerns how generalizable the trial's results are to broader populations, which is a real and important trial property but is governed by sampling and eligibility criteria, not blinding. Construct validity of the pain instrument concerns whether the chosen scale actually measures the pain construct it claims to measure, which is a separate methodologic question from who knows the treatment assignment.

6. C. Placebo analgesia is mediated in part by expectation-driven activation of endogenous opioid and descending modulatory pathways, producing measurable pain reduction independent of any pharmacologically active treatment -- the mechanism the investigators are describing. Regression to the mean is a real statistical phenomenon in which extreme baseline values naturally drift toward average on repeat measurement, but it is a statistical artifact rather than a neurobiological opioid-mediated mechanism, so it does not match the explanation given. The Hawthorne effect refers to behavior change simply from being observed or studied, which is a genuine confounder in trials but is distinct from the specific endogenous-opioid mechanism the vignette attributes the improvement to.

7. A. An interdisciplinary team-based model is defined by multiple disciplines working from a shared, jointly developed treatment plan with regular collaborative reassessment, which is exactly the structure described. A sequential (serial) referral model is a real, distinct approach in which a patient is passed from one provider to the next largely independently, without the shared plan and joint reassessment described here, so it does not fit. A solo-practitioner gatekeeping model centers care around a single

physician who authorizes referrals but does not itself involve the multidisciplinary joint planning structure the vignette describes.

8. B. A structured, threshold-triggered review involving the prescriber, pharmacist, and behavioral health provider before a dose escalation is finalized is a textbook opioid stewardship program built on multidisciplinary checks-and-balances. Universal precautions screening at intake is a real, distinct practice (risk stratification performed once, at the start of therapy) rather than an ongoing, dose-triggered team review process, so it does not match the recurring review described. Unilateral prescriber discretion without additional review is the direct opposite of what is described, since the vignette specifically requires a multidisciplinary check before the prescription is finalized.

9. C. A state-mandated check of a database tracking a patient's controlled-substance dispensing history before prescribing is the defining function of a Prescription Drug Monitoring Program query. A REMS attestation is a real, distinct FDA-level safety program tied to specific high-risk drugs (such as certain extended-release/long-acting opioids) that involves prescriber and pharmacy certification, not a state database check of patient dispensing history, so it does not match. DEA registration renewal is a real periodic administrative requirement for a prescriber's own controlled-substance authority, but it has nothing to do with checking an individual patient's dispensing history before a given prescription.

10. A. Discussing risks, benefits, and alternatives, confirming patient understanding, and documenting voluntary agreement before an invasive procedure is precisely what constitutes informed consent. A HIPAA authorization is a real, distinct legal document governing the release or use of a patient's protected health information to third parties, which has nothing to do with agreeing to undergo a procedure. An advance directive documents a patient's wishes for future medical care in the event they lose decision-making capacity, which is a separate legal instrument from the procedural consent being obtained here.

11. C. The FLACC scale was specifically developed and validated for young, nonverbal, or preverbal children, scoring observable behaviors (face, legs, activity, cry, consolability) rather than requiring self-report. The Numeric Rating Scale is a real, widely used tool, but it requires the patient to verbally or cognitively self-report a number, which a 2-year-old cannot reliably do, making it inappropriate here. The McGill Pain Questionnaire is a genuine validated instrument for characterizing pain quality and intensity, but it relies on the patient understanding and selecting descriptive words, which is also beyond a nonverbal toddler's capability.

12. C. The McGill Pain Questionnaire uses categorized word lists to capture the sensory, affective, and evaluative dimensions of pain, going beyond simple intensity to characterize its quality -- exactly what the vignette requires. The Wong-Baker FACES scale is a real, validated tool, but it conveys only overall intensity via facial expressions and does not differentiate sensory versus affective pain qualities. The Visual Analog Scale is also a genuine, widely used measure, but like FACES it captures a single intensity value along a line and provides no breakdown of pain quality or affective burden.

13. A. The nocebo effect describes a genuine worsening of symptoms driven by negative expectations, exactly as occurred when the patient was warned about possible worsening pain and then experienced it despite receiving inert medication. Placebo analgesia from positive conditioning is a real, opposite phenomenon in which expectation of benefit produces pain relief, which does not match a patient reporting worsened pain. Pseudoaddiction is a genuine but unrelated concept describing drug-seeking behavior that results from inadequately treated pain, not an expectation-driven symptom change from an inert substance.

14. B. The Oswestry Disability Index is the classic 10-domain, percentage-scored self-report questionnaire assessing functional limitation specifically from low back pain across exactly these activities. The Beck Depression Inventory is a real, validated instrument, but it measures depressive symptom severity, not physical functional disability from back pain, so it does not fit the domains described. The STarT Back Screening Tool is a genuine, distinct instrument that stratifies patients by risk of poor outcome (low/medium/high) using a brief 9-item screen, rather than producing a detailed percentage disability score across the specific activities listed.

15. C. A Functional Capacity Evaluation is precisely this structured, observed battery of physical tasks used to objectively quantify a worker's safe functional abilities for return-to-work determinations. Waddell signs are a real, distinct examination technique consisting of non-organic physical signs used to screen for non-physiologic contributors to a back pain presentation, not a battery of lifting/carrying/pushing tasks. The Timed Up and Go test is a genuine, validated mobility and fall-risk screening tool, but it is a brief single timed task, not the comprehensive multi-task occupational battery described here.

16. A. The Pain Catastrophizing Scale specifically measures rumination, magnification, and helplessness surrounding pain, and its three subscales map directly onto what the vignette describes. The Fear-Avoidance Beliefs Questionnaire is a real, distinct instrument that measures beliefs about how physical activity or work will affect pain, not the rumination/magnification/helplessness construct being assessed here. The Tampa Scale of Kinesiophobia is also a genuine, validated tool, but it specifically measures fear of movement/reinjury rather than the catastrophizing construct described in the vignette.

17. B. The fear-avoidance model directly describes this pattern: catastrophic beliefs about pain lead to fear of movement, avoidance of activity, and consequent deconditioning and disability that perpetuate the pain cycle. Gate control theory is a genuine and foundational neurophysiologic model explaining how non-nociceptive input can modulate pain transmission at the spinal cord, but it does not describe the psychosocial avoidance-deconditioning cycle in this vignette. An operant conditioning model alone (reinforcement of pain behaviors by external consequences) is a real, related concept, but it does not on its own capture the central role of catastrophic injury beliefs driving activity avoidance described here.

18. B. Research into sex and gender differences in pain attributes the higher prevalence of these specific chronic pain conditions in women to a combination of hormonal influences (e.g., estrogen effects on nociceptive processing), differences in central pain modulation, and psychosocial factors, which is the

recognized explanatory framework in the pain literature. Differences in opioid receptor gene copy number between sexes is not an established, real mechanism cited for these prevalence differences and is a fabricated-sounding genetic explanation. Differences in insurance coverage rates is a real health-system factor in general but is explicitly excluded by the vignette's statement that the differences persist after controlling for healthcare-seeking behavior.

19. C. EMG/NCS directly measures the electrophysiologic function of nerve roots and peripheral nerves, providing objective evidence of active radiculopathy (denervation changes) that correlates with clinical nerve root irritation. Plain radiographs are a real, useful imaging study for bony alignment and degenerative changes, but they provide no electrophysiologic information about nerve function. MRI of the lumbar spine is genuinely valuable for visualizing structural nerve root compression, but it is anatomic imaging, not a functional electrophysiologic test, so it cannot confirm active nerve conduction abnormality the way EMG/NCS can.

20. B. Provocative discography involves injecting contrast under pressure into a disc to determine whether it reproduces the patient's exact concordant pain, directly implicating that disc as the pain generator -- precisely the study described. A facet joint medial branch block is a real, distinct diagnostic procedure that anesthetizes the nerves supplying a facet joint to evaluate facet-mediated pain, not disc pain provoked by pressurized injection. A selective nerve root block is also a genuine diagnostic/therapeutic procedure, but it targets a specific nerve root with local anesthetic to assess radicular pain, not the disc itself via pressurized provocation.

21. C. Methadone's NMDA receptor antagonism is a distinguishing property beyond mu-opioid agonism that can help counteract central sensitization and opioid-induced hyperalgesia, making it a rational choice in this scenario. Selective kappa-opioid receptor antagonism is not a recognized property of methadone and does not explain its distinct clinical utility here. Irreversible mu-receptor binding is not how methadone (or any clinically used opioid) works pharmacologically -- opioid receptor binding is reversible -- so this option misrepresents the actual mechanism.

22. B. Opioid-induced hyperalgesia is a paradoxical state of increased pain sensitivity from opioid exposure itself, and it characteristically improves with dose reduction rather than escalation, distinguishing it from simple undertreatment or tolerance. Opioid tolerance would be expected to require further dose escalation to achieve the same effect, which is the opposite of what improved this patient's pain, making it inconsistent with the vignette. Undertreated acute nociceptive pain would also be expected to improve with MORE opioid rather than less, which again contradicts the clinical response described.

23. A. Morphine-6-glucuronide is a potent active metabolite that is renally cleared, and in renal impairment it accumulates and can cause progressive sedation and myoclonus even without a dose increase, exactly matching this presentation. Acute opioid tolerance would be expected to cause reduced, not increased, drug effect over time, which is the opposite of the worsening sedation and myoclonus described. A new unrelated seizure disorder is possible in general but is a far less parsimonious

explanation than a well-recognized, renally mediated opioid metabolite toxicity in a patient with known chronic kidney disease on morphine.

24. C. Methadone blocks the cardiac hERG potassium channel, which can prolong the QT interval and predispose to torsades de pointes, and this is the recognized mechanism behind methadone-associated QT prolongation. Beta-adrenergic receptor blockade is not a property of methadone and is not the mechanism responsible for QT changes seen with this drug. Methadone is actually a substrate (and in some contexts a modest inhibitor) of CYP3A4-related metabolism rather than an inducer, and induction is not the mechanism responsible for the QT prolongation described in this vignette.

25. A. Buprenorphine's partial agonist activity at the mu-opioid receptor produces a ceiling effect on receptor activation, which translates clinically into a plateau in respiratory depression risk even as the dose increases, unlike full agonists. Full antagonist activity would produce no analgesic or respiratory-depressant effect at all, which contradicts buprenorphine's genuine analgesic use and its (limited) respiratory depression risk. Selective delta-opioid receptor agonism is not buprenorphine's primary mechanism of action; its clinically relevant activity is at the mu receptor, not the delta receptor.

26. A. Transdermal fentanyl forms a subcutaneous depot in the skin that both delays the onset of therapeutic drug levels after application and continues releasing drug for many hours after the patch is removed, accounting for both observations in the vignette. Transdermal fentanyl bypasses the gut and portal circulation entirely, so first-pass hepatic metabolism is not relevant to a transdermal route and does not explain the delayed onset/offset described. High renal clearance of unmetabolized fentanyl is not the operative pharmacokinetic issue here; fentanyl is primarily hepatically metabolized, and renal clearance of parent drug does not explain the delayed onset and prolonged offset seen with the patch reservoir.

27. B. Incomplete cross-tolerance means a patient tolerant to one opioid is not fully tolerant to an equianalgesic dose of a different opioid, so reducing the calculated dose by 25-50% during rotation is standard practice to avoid overdose. Stating hydromorphone has no cross-tolerance with oxycodone is inaccurate and too absolute -- cross-tolerance is partial, not absent, which is exactly why a fractional (not full) dose reduction is used rather than starting from zero. Hydromorphone still exerts its analgesic effect via the mu-opioid receptor like other clinically used opioids, so the claim that it acts independently of that receptor is incorrect and does not explain the dose-reduction principle.

28. C. Naloxone typically has a shorter duration of action than many opioids (especially long-acting agents), so as naloxone wears off before the opioid is fully cleared, respiratory depression can recur, which is exactly the phenomenon described and the reason repeat dosing or infusions are used. The statement that naloxone has a longer half-life than most opioids is factually backward and would predict the opposite clinical pattern (no recurrence) rather than the recurrent sedation observed. Naloxone is a full antagonist, not a partial agonist, and its onset is rapid rather than delayed, so this option misrepresents its actual pharmacology.

29. A. Nonselective NSAIDs inhibit COX-1, which normally produces prostaglandins that maintain protective gastric mucus and bicarbonate secretion and mucosal blood flow, so COX-1 inhibition removes this protection and predisposes to ulceration -- the well-established mechanism of NSAID gastropathy. Direct caustic injury from tablet acidity is not the accepted mechanism of NSAID-related ulcers; the toxicity is systemic (via COX-1 inhibition) and occurs with parenteral NSAIDs too, not just from local tablet contact. NSAIDs do not induce H. pylori colonization; H. pylori is an independent, separate risk factor for ulcer disease that can compound but is not caused by NSAID use.

30. A. Celecoxib selectively inhibits COX-2 while largely sparing COX-1, preserving the protective gastric prostaglandins that nonselective NSAIDs suppress, which reduces GI toxicity risk -- the correct rationale for choosing it in a patient with prior ulcer disease. All NSAIDs, including celecoxib, have an analgesic ceiling effect (a maximum effect beyond which increasing dose does not add more analgesia), so claiming celecoxib lacks one is factually incorrect and not a real distinguishing feature. Celecoxib undergoes significant hepatic metabolism (primarily via CYP2C9) rather than being eliminated renally unchanged, so this option misstates its pharmacokinetics.

31. C. At supratherapeutic doses, glutathione conjugation pathways become saturated, allowing the toxic metabolite NAPQI (N-acetyl-p-benzoquinone imine) to accumulate and cause centrilobular hepatocyte necrosis, which is the established mechanism of acetaminophen hepatotoxicity. Acetaminophen's analgesic mechanism is not primarily peripheral COX-1 inhibition causing ischemic injury; its hepatotoxicity is specifically due to a reactive metabolite, not direct COX-mediated ischemia. It is the toxic metabolite NAPQI, not accumulation of unmetabolized parent acetaminophen itself, that is directly responsible for the hepatocellular injury, so this option misidentifies the toxic species.

32. B. Ketorolac carries a labeled restriction limiting combined oral and parenteral use to a maximum of five days because of its potent NSAID effects on renal function and platelet-mediated bleeding risk with prolonged use, which is the correct and clinically important restriction. There is no basis for restricting ketorolac to bedtime-only dosing, and indefinite use would directly contradict the actual labeled duration limit. Ketorolac, as a potent nonselective NSAID, still carries significant GI toxicity risk, so claiming it lacks this risk is incorrect and does not explain the actual restriction being asked about.

33. B. Aspirin irreversibly acetylates COX-1 in platelets, and because platelets are anucleate and cannot synthesize new COX-1 enzyme, the antiplatelet effect lasts for the platelet's entire ~7-10 day lifespan -- the correct mechanism explaining its unique duration compared to reversible NSAIDs. Aspirin's plasma half-life is actually quite short (well under an hour for the active acetylating species), so a long plasma half-life is not what explains the prolonged platelet effect. Aspirin is not sequestered in adipose tissue for slow release; its prolonged antiplatelet action is due to irreversible enzyme modification, not a depot/storage pharmacokinetic effect.

34. C. In volume-depleted states, renal perfusion becomes dependent on prostaglandin-mediated afferent arteriolar vasodilation to maintain glomerular filtration; NSAIDs block this compensatory prostaglandin synthesis, reducing renal blood flow and precipitating acute kidney injury -- the correct and classic

mechanism. Direct nephrotoxic tubular necrosis independent of renal blood flow describes a different injury pattern (more typical of direct tubular toxins) and is not the primary mechanism by which NSAIDs cause AKI in volume depletion. NSAIDs can contribute to hyperkalemia through reduced aldosterone-related effects, but hyperkalemia causing prerenal azotemia is not the correct causal sequence here; the AKI is driven by reduced renal blood flow from prostaglandin inhibition, not by potassium levels.

35. B. Topical NSAID formulations achieve effective local joint-tissue drug concentrations while producing substantially lower systemic plasma levels than oral dosing, which reduces systemic risks such as renal impairment and GI toxicity while still relieving localized joint pain. Topical diclofenac still works by inhibiting COX enzymes locally, so claiming it has no COX inhibitory activity is incorrect and contradicts why it is analgesic at all. Topical diclofenac does undergo some systemic absorption (which is precisely why it still carries reduced, not zero, systemic risk), so describing it as never entering systemic circulation is inaccurate.

36. C. Tricyclic antidepressants inhibit reuptake of norepinephrine and serotonin, augmenting descending inhibitory pathways from the brainstem that dampen spinal nociceptive transmission, which is the accepted mechanism behind their analgesic effect at doses below typical antidepressant dosing. TCAs are not selective TRPV1 blockers; TRPV1 modulation is more relevant to agents like capsaicin, not the mechanism responsible for TCA analgesia. While TCAs do have some sodium channel blocking activity, this is a secondary property and is not isolated to the dorsal root ganglion nor the primary explanation for their neuropathic pain benefit compared to the descending-inhibition mechanism.

37. B. Duloxetine is an SNRI that inhibits reuptake of both serotonin and norepinephrine, and the combined enhancement of these descending inhibitory pathways is why SNRIs (unlike pure SSRIs) have demonstrated efficacy specifically for neuropathic pain such as diabetic peripheral neuropathy. Pure SSRIs act mainly through serotonergic reuptake inhibition alone and have generally shown weaker or inconsistent efficacy for neuropathic pain compared to agents with combined noradrenergic activity, so labeling duloxetine as a pure SSRI is incorrect. Duloxetine is not an MAOI; MAOIs work by an entirely different mechanism (inhibiting monoamine breakdown enzymes) and are not the first-line pharmacologic class used for diabetic neuropathic pain.

38. A. Gabapentin binds the alpha-2-delta subunit of voltage-gated calcium channels on presynaptic neurons, reducing calcium influx and subsequent release of excitatory neurotransmitters such as glutamate and substance P, which is its established mechanism in neuropathic pain despite its name suggesting GABA involvement. Gabapentin does not act as an NMDA receptor antagonist; that mechanism is more characteristic of drugs like ketamine, not gabapentin. Despite its name, gabapentin does not directly agonize GABA-B (or GABA-A) receptors; its therapeutic mechanism is via the calcium channel subunit, not direct GABA receptor binding.

39. A. Pregabalin is eliminated largely unchanged by the kidneys, so in renal impairment its clearance falls substantially and a significant dose reduction is required to avoid accumulation and toxicity such as

sedation and ataxia. Pregabalin does not undergo extensive hepatic metabolism -- it is renally, not hepatically, cleared -- so stating no adjustment is needed on that basis misstates its pharmacokinetics and would risk drug accumulation. Increasing the dose to compensate for reduced hepatic clearance is doubly incorrect, since hepatic clearance is not the relevant pathway and increasing dose in renal impairment would worsen, not correct, drug accumulation.

40. B. HLA-B*1502 is strongly associated with an increased risk of carbamazepine-induced Stevens-Johnson syndrome and toxic epidermal necrolysis, particularly in patients of Han Chinese and certain other Asian ancestries, which is why genotype screening is recommended before starting the drug in this population. The HLA-B*1502 allele is not a marker of analgesic efficacy; it is a pharmacogenomic marker for a severe cutaneous adverse reaction risk, not a predictor of pain response. It also does not indicate a need for a higher starting dose; rather, a positive result generally leads to avoiding carbamazepine altogether because of the serious safety risk, not a dosing adjustment.

41. B. TCAs have quinidine-like class IA antiarrhythmic properties that block cardiac sodium channels, which can worsen pre-existing conduction abnormalities, prolong the QT interval, and precipitate arrhythmias, making baseline ECG screening for conduction disease appropriate before starting therapy. TCAs are not typically associated with vagally mediated bradycardia as their defining cardiac risk; their concerning effect is on cardiac conduction and repolarization, not primarily heart rate via vagal tone. Irreversible coronary vasospasm is not a recognized or defining cardiac toxicity of TCAs, and describing it as occurring in most patients overstates and mischaracterizes their actual, much more specific conduction-related risk.

42. A. Gabapentinoids and opioids both cause CNS and respiratory depression, and their combination has additive (pharmacodynamic) sedative and respiratory-depressant effects, which has been associated with an increased risk of opioid-related overdose death -- the correct and clinically significant concern. Gabapentin is not a hepatic enzyme inducer; it is renally eliminated and largely bypasses hepatic metabolism, so an enzyme-induction mechanism affecting opioid metabolism is not accurate. Protein-binding displacement is not the relevant interaction mechanism here; the danger from combining these drugs is pharmacodynamic (additive CNS/respiratory depression), not a pharmacokinetic displacement interaction.

43. A. Lamotrigine carries a well-documented, dose-and-titration-rate-dependent risk of severe cutaneous reactions including Stevens-Johnson syndrome and toxic epidermal necrolysis, and slow titration substantially reduces this risk, which is the established rationale for the cautious dosing schedule. Lamotrigine's key safety concern is dermatologic, not a described pattern of irreversible hepatotoxicity within the first week, so this option misattributes the actual serious risk. The rationale for slow titration is not related to preventing sedation (and this option's premise linking it to a gabapentin comparison is not the recognized reason); the real concern driving slow titration is the cutaneous reaction risk, not a sedation-avoidance strategy.

44. C. Ketamine is primarily an NMDA receptor antagonist, and at subanesthetic doses this blocks glutamate-mediated central sensitization (wind-up), which is the key mechanism behind its analgesic use in refractory chronic pain conditions like CRPS. Ketamine has minimal clinically relevant direct agonism at peripheral mu-opioid receptors; its analgesic mechanism in this context is not opioid-receptor mediated. While ketamine has some minor sodium channel effects at very high concentrations, this is not the primary or clinically relevant mechanism behind its use for central sensitization in chronic pain.

45. C. Topical lidocaine blocks voltage-gated sodium channels in the peripheral cutaneous nerve fibers beneath the patch, reducing the ectopic, hyperexcitable firing characteristic of postherpetic neuralgia, while systemic absorption remains low -- the correct localized mechanism. Beta-adrenergic blockade is not lidocaine's mechanism of action at all and is not relevant to sodium-channel-mediated local anesthetic effects. Substance P depletion from peripheral terminals is the mechanism of capsaicin, not lidocaine, so attributing it to a lidocaine patch is incorrect.

46. A. High-concentration capsaicin intensely activates TRPV1 receptors on nociceptor terminals, leading to a reversible defunctionalization (loss of function) of those superficial nerve fibers, which produces sustained localized analgesia -- the accepted mechanism for high-dose capsaicin therapy. Capsaicin's action is on the peripheral TRPV1 receptor, not on NMDA glutamate receptors, so an NMDA-blockade mechanism does not correctly describe how it works. Capsaicin patches act locally at the application site rather than through systemic activation of central descending pathways, so a systemic noradrenergic mechanism does not explain its localized effect.

47. A. Clonidine is an alpha-2 adrenergic agonist, and when delivered epidurally it acts on alpha-2 receptors in the spinal dorsal horn to enhance descending inhibitory noradrenergic signaling and reduce nociceptive transmission, which is its correct analgesic mechanism as a neuraxial adjuvant. Clonidine has no meaningful 5-HT3 serotonin receptor antagonist activity; that mechanism describes antiemetics like ondansetron, not clonidine's analgesic action. Clonidine is an alpha-2, not a beta-2, adrenergic agonist, and its clinically relevant analgesic mechanism here is central (spinal), not a peripheral vasodilatory effect.

48. B. Cannabinoids act through CB1 receptors, which are concentrated in the central nervous system and modulate neurotransmitter release relevant to pain and spasticity, and CB2 receptors, which are more peripheral/immune-related and influence inflammatory pain, together explaining their analgesic and antispasmodic effects. Cannabinoids do not act directly at opioid receptors; their mechanism is through a distinct endocannabinoid receptor system, not mu/kappa opioid receptor agonism. Cannabinoids generally enhance rather than antagonize GABAergic inhibitory tone in relevant circuits, so describing them as GABA-A antagonists that increase excitability misrepresents their actual pharmacology relative to their therapeutic antispasmodic effect.

49. B. OnabotulinumtoxinA cleaves SNARE proteins needed for vesicle fusion, inhibiting release of acetylcholine and pain-related neuropeptides from peripheral nerve terminals, which reduces peripheral

sensitization of trigeminal sensory afferents implicated in chronic migraine -- the accepted mechanism for its prophylactic effect. Botulinum toxin does not act on central serotonin receptors; that mechanism describes triptans, an entirely different migraine drug class, not botulinum toxin. Botulinum toxin has no beta-adrenergic blocking activity; it is a neurotoxin acting at the neuromuscular/neurosecretory junction, not a systemic vasoactive beta-blocker.

50. C. Cyclobenzaprine is structurally related to tricyclic antidepressants and acts centrally, primarily at the brainstem, to reduce tonic somatic motor activity and muscle spasm, rather than acting directly at the muscle or neuromuscular junction. It does not act as a depolarizing neuromuscular blocking agent (that mechanism describes drugs like succinylcholine used in anesthesia, not cyclobenzaprine). It also does not work by inhibiting peripheral sarcoplasmic reticulum calcium release, which describes the mechanism of dantrolene, a different and pharmacologically distinct muscle relaxant used mainly for spasticity and malignant hyperthermia.

51. B. Consensus interventional pain guidelines (e.g., ASRA-based recommendations adapted for pain procedures) classify transforaminal epidural injections as at least intermediate risk, requiring anticoagulants like warfarin to be held for an appropriate interval with a normalized INR confirmed before proceeding, to reduce epidural hematoma risk. Continuing warfarin uninterrupted for a procedure of this risk category is inconsistent with guideline recommendations and would raise, not minimize, bleeding risk. Substituting warfarin with high-dose aspirin does not address the anticoagulation issue -- aspirin has its own antiplatelet bleeding risk profile and is not a recognized bridging strategy for warfarin before this type of procedure.

52. A. Fluoroscopy provides clear visualization of bony spinal landmarks and allows real-time contrast injection to confirm epidural spread and rule out vascular uptake before steroid injection, which is why it remains standard for transforaminal epidural procedures. Ultrasound is genuinely useful for many superficial and peripheral procedures, but it cannot adequately visualize deep bony spinal anatomy or confirm epidural contrast spread the way fluoroscopy can, making it inferior for this specific deep spinal application. Fluoroscopy inherently involves ionizing radiation exposure, so describing it as avoiding all radiation exposure is factually incorrect and is not a genuine advantage.

53. C. Strict sterile technique during interventional spine procedures is specifically intended to minimize the risk of introducing infection, such as epidural abscess or discitis, which are rare but serious complications of needle-based spinal procedures. Sterile technique has no bearing on local anesthetic systemic toxicity, which is related to drug dose and inadvertent intravascular injection, not skin antisepsis. Sterile preparation likewise does not address contrast-induced nephropathy, which is a separate risk related to iodinated contrast dose and renal function, not to infection control measures.

54. A. Permanently implanted hardware, such as an intrathecal pump, provides a persistent surface for biofilm formation, and an established hardware infection is difficult to eradicate without device explantation, which is why perioperative antibiotic prophylaxis is specifically indicated for implant procedures. It is not accurate that antibiotic prophylaxis is required for all spinal procedures regardless

of hardware; routine non-implant procedures such as a single epidural steroid injection do not require prophylactic antibiotics. Antibiotic prophylaxis is given for infection prevention, not as an analgesic, so describing its rationale purely as pain reduction is incorrect.

55. B. Standard practice for a patient with a documented contrast allergy undergoing a procedure that benefits from contrast confirmation is to use an established premedication protocol (corticosteroid and antihistamine) and to consider alternative contrast agents or contrast-sparing technique, balancing procedural benefit against allergy risk. Permanently canceling all image-guided procedures is an overly absolute response that ignores effective, well-established allergy mitigation strategies used routinely in interventional practice. Proceeding with standard dosing and no premedication ignores the documented allergy and disregards the real risk of a reaction, even at typical epidural contrast volumes, making this an unsafe approach.

56. C. TENS stimulates large-diameter, low-threshold A-beta afferent fibers, which activate inhibitory interneurons in the dorsal horn to reduce transmission of nociceptive signals from smaller A-delta and C fibers -- the gate control mechanism that is the primary basis for conventional (high-frequency, low-intensity) TENS analgesia. Substance P depletion is the mechanism attributed to capsaicin, not TENS, so this option misattributes a different treatment's mechanism. While low-frequency TENS has been linked to some endogenous opioid release, describing this as the exclusive mechanism at high-frequency settings is inaccurate, since high-frequency TENS analgesia is primarily explained by the gate control mechanism, not opioid release.

57. A. Acupuncture needle stimulation is believed to activate afferent pathways that trigger release of endogenous opioids and activate descending inhibitory pain pathways from the brainstem, which is the leading proposed neurophysiologic mechanism supported by research in this area. Acupuncture needles do not function as local anesthetics via direct sodium channel blockade; that is a distinct pharmacologic mechanism unrelated to acupuncture's proposed opioid/descending-pathway mechanism. Acupuncture has no established mechanism involving hepatic prostaglandin synthesis inhibition; that mechanism describes NSAIDs, an entirely different treatment modality.

58. B. This describes a temporary, percutaneously placed peripheral nerve stimulation lead targeted directly at a specific affected peripheral nerve, distinct from spinal cord stimulation, which targets the dorsal columns of the spinal cord rather than a peripheral nerve directly. This is explicitly not a spinal cord stimulator system, since SCS involves epidural lead placement targeting the dorsal columns, not a peripheral nerve near a site of facial trauma. This is also not an intrathecal drug delivery trial, which involves testing medication infused into the intrathecal space rather than electrical stimulation of a peripheral nerve.

59. C. The transforaminal approach places the needle tip directly adjacent to the exiting nerve root and ventral epidural space, delivering medication precisely at the presumed site of nerve root irritation from the disc herniation, which is its main procedural advantage for focal radicular pain. Transforaminal injections still use fluoroscopy and therefore still involve radiation exposure, so claiming it eliminates

radiation is incorrect. Transforaminal injections in fact typically require careful contrast confirmation (including live or digital subtraction imaging) to rule out intravascular uptake before injecting steroid, so claiming it avoids the need for contrast is inaccurate.

60. C. A robust, reproducible response to two separate diagnostic medial branch blocks is the accepted evidence threshold supporting facet-mediated pain, and the appropriate next step is radiofrequency ablation of those medial branch nerves to provide longer-lasting relief. Proceeding directly to spinal fusion surgery is a far more invasive, non-indicated escalation for isolated facet-mediated pain confirmed by diagnostic blocks and is not the standard next step. A blind steroid injection without further diagnostic confirmation ignores the fact that the diagnostic workup is already complete and points toward denervation (RFA), not a repeat unconfirmed steroid approach.

61. A. A substantial (greater than 75%) reduction in pain following a fluoroscopically confirmed intra-articular sacroiliac joint injection with local anesthetic is the accepted diagnostic gold standard supporting the SI joint as a clinically significant pain generator. A positive SI joint block specifically implicates the SI joint, not the lumbar facet joints, which are a separate anatomic structure requiring their own distinct diagnostic blocks to confirm involvement. A robust anatomically localized response to a specific diagnostic injection argues against, not for, a purely psychogenic explanation, since the pain source has been physically localized.

62. B. The stellate ganglion, located in the cervical region, provides sympathetic innervation to the upper extremity, and blocking it is the standard diagnostic and therapeutic approach for upper extremity sympathetically maintained pain such as CRPS of the hand. A celiac plexus block targets sympathetic innervation to abdominal viscera (commonly used for pancreatic cancer pain) and has no role in upper extremity sympathetic pain. A lumbar sympathetic block targets sympathetic innervation to the lower extremity, not the upper extremity, making it anatomically inappropriate for this patient's hand symptoms.

63. C. The celiac plexus provides sympathetic innervation to the pancreas and upper abdominal viscera, and celiac plexus neurolysis (often with alcohol or phenol) is the standard interventional approach for refractory pancreatic cancer pain, providing meaningful opioid-sparing analgesia. A stellate ganglion block targets upper extremity sympathetic innervation and has no role in visceral abdominal cancer pain. A superior hypogastric plexus block targets sympathetic innervation to pelvic viscera (relevant to pelvic cancer pain), not the upper abdominal/pancreatic region described in this vignette.

64. C. Trigger point injection mechanically disrupts the hyperirritable taut band within the muscle and delivers local anesthetic (with or without other agents) to interrupt the local pain-spasm-pain cycle characteristic of myofascial trigger points, which is the therapeutic goal here. Trigger point injections are not intended to neurolytically destroy a peripheral nerve; the target is the muscle taut band itself, not nerve ablation, and permanent nerve destruction is not the goal or typical technique. This procedure has nothing to do with the facet joint capsule, which is an entirely separate anatomic structure targeted by different injections such as facet joint or medial branch procedures.

65. A. An occipital nerve block specifically targets the greater (and sometimes lesser) occipital nerve at its superficial course near the nuchal ridge, which is the anatomically and clinically correct target for occipital neuralgia presenting with posterior scalp pain reproduced by palpation there. The trigeminal ganglion is an intracranial structure relevant to trigeminal neuralgia (facial pain), not the posterior scalp distribution described, making it anatomically incorrect for this presentation. The stellate ganglion is a sympathetic structure relevant to upper extremity sympathetic pain, unrelated to the somatic occipital nerve pain described in this vignette.

66. B. Genicular nerve blocks and subsequent radiofrequency ablation specifically target the small sensory genicular nerve branches that innervate the knee joint capsule, providing pain relief for refractory knee osteoarthritis without affecting motor function. The sciatic nerve at the popliteal fossa is a large mixed motor-sensory nerve supplying the lower leg and foot, not the specific sensory branches to the knee joint targeted in this procedure. The femoral nerve at the inguinal ligament is a proximal mixed nerve supplying the anterior thigh and would produce significant motor weakness if blocked, unlike the purely sensory genicular nerve target used for knee OA pain.

67. C. Intrathecal phenol acts on mixed sensorimotor nerve roots without inherent selectivity for sensory fibers, so there is a real risk of unwanted motor weakness or bowel/bladder sphincter dysfunction if the neurolytic agent spreads to or affects motor or autonomic fibers, which is the key procedural risk to consider. It is incorrect to say phenol selectively spares motor fibers -- this lack of selectivity is precisely the safety concern that must be managed with careful technique and patient positioning. Phenol neurolysis is not rapidly reversible within minutes; its destructive effect on nerve tissue is intended to be relatively durable, which is the therapeutic goal but also why the motor/sphincter risk is taken seriously rather than treated as easily correctable.

68. C. The pudendal nerve courses near the ischial spine, and a pudendal nerve block at this anatomic landmark is the correct diagnostic and therapeutic procedure for pudendal neuralgia presenting with perineal pain worsened by sitting. The obturator nerve supplies the medial thigh and hip adductors and is not the nerve implicated in perineal pain reproduced near the ischial spine, making it an anatomically incorrect target here. The ilioinguinal nerve supplies sensation to the groin and upper medial thigh via a different anatomic course and is not the nerve targeted for perineal pain localized near the ischial spine.

69. B. The superior hypogastric plexus provides sympathetic innervation to pelvic viscera, and blocking or neurolysing this plexus is the standard interventional approach for refractory pelvic cancer pain such as advanced cervical cancer. A celiac plexus block targets the upper abdominal viscera (e.g., pancreas), not the pelvic organs, making it anatomically incorrect for this pelvic cancer pain presentation. A stellate ganglion block provides sympathetic innervation to the upper extremity and has no role whatsoever in pelvic visceral pain.

70. B. Conventional spinal cord stimulation delivers electrical current to the dorsal columns, preferentially activating large A-beta fibers that engage inhibitory interneurons to gate nociceptive transmission at the spinal cord level, consistent with gate control theory -- the accepted mechanism for

tonic SCS analgesia. SCS does not work through chemical denervation of a peripheral nerve; it is an electrical neuromodulation therapy targeting the spinal cord, not a chemical or peripheral nerve-destructive intervention. There is no established mechanism involving systemic anti-inflammatory cytokine release from epidural fat responsible for SCS analgesia; the effect is via direct neuromodulation of dorsal column signaling.

71. A. The SCS trial period allows the patient and clinical team to confirm meaningful, reproducible pain relief and functional improvement with temporary leads before committing to a permanent, more invasive implanted system, which is the accepted rationale for this staged approach. While insurance requirements often reference the trial, describing it as solely a billing formality with no genuine clinical purpose mischaracterizes its actual, clinically meaningful screening function. The trial leads are temporary and percutaneous, not surgically anchored in their final position; permanent lead placement and anchoring occurs only at the separate permanent implantation stage after a successful trial.

72. C. Intrathecal drug delivery allows medication to act directly at spinal cord receptors at doses that are a small fraction of the systemic dose required for equivalent analgesia, which substantially reduces systemic side effects while improving pain control -- the correct rationale for this therapy in opioid-refractory cancer pain with intolerable systemic effects. Intrathecal delivery does not eliminate the possibility of tolerance; tolerance can still develop with chronic intrathecal opioid infusion, so this claim is inaccurate. Intrathecal pumps require regular percutaneous refills and ongoing device maintenance, so describing the therapy as requiring no ongoing maintenance is factually incorrect.

73. B. Dorsal root ganglion stimulation targets the DRG corresponding to a specific painful dermatome, providing more focal, somatotopically precise coverage than conventional dorsal column SCS, which is particularly useful for focal distal neuropathic pain such as in the foot -- exactly the scenario described. Conventional tonic SCS targets the dorsal columns more broadly and was already described as providing inadequate, non-focal coverage in this vignette, so it is not the answer being sought. An intrathecal opioid pump delivers medication rather than providing focal electrical neuromodulation of a specific dermatomal distribution, so it does not match the device described.

74. A. A stimulating lead placed directly along a specific peripheral nerve, rather than in the epidural space or at the dorsal root ganglion, is categorized as a peripheral nerve stimulator implant, which is the correct description for this device targeting the ilioinguinal nerve. This is not an intrathecal drug delivery system, since no medication infusion is described -- the device delivers electrical stimulation, not a drug. This is also not a dorsal root ganglion stimulator, since the lead is described as being placed along the peripheral nerve itself, not at the DRG within the spinal canal.

75. C. A shift in lead position on device interrogation, correlating with loss of paresthesia coverage and pain recurrence, is the classic presentation of lead migration, one of the most common mechanical complications of spinal cord stimulation. Battery depletion would typically present with a gradual loss of overall device function or low-battery alerts rather than a documented physical shift in lead position on imaging/interrogation. Surgical site infection would typically present with local signs such as

erythema, warmth, drainage, or fever, not primarily with a change in lead position and paresthesia coverage.

76. A. Ziconotide selectively blocks N-type voltage-gated calcium channels at presynaptic primary afferent terminals in the dorsal horn, reducing release of pain-signaling neurotransmitters through a mechanism entirely independent of opioid receptors, making it a valuable non-opioid intrathecal option. Ziconotide is specifically notable for NOT acting through mu-opioid receptor agonism; that is exactly what distinguishes it from intrathecal morphine and makes it useful when further opioid escalation is undesirable. Ziconotide also does not act via alpha-2 adrenergic receptors; that mechanism describes clonidine, a different intrathecal adjuvant with a distinct pharmacology.

77. B. Radiofrequency ablation uses a high-frequency alternating current delivered through an electrode tip to generate frictional heat, creating a controlled thermal lesion that interrupts nerve conduction and provides durable pain relief -- the defining mechanism of RFA. Chemical destruction with injected alcohol describes chemical neurolysis, a different neuroablative technique that does not involve electrical current or heat generation. Cryogenic freezing describes cryoablation, a distinct cold-based neuroablative technique, not the heat-based mechanism used in standard radiofrequency ablation.

78. B. Cryoablation works by freezing tissue to form ice crystals that cause localized Wallerian degeneration of the axon while relatively preserving the perineurial sheath, which provides a scaffold along which the axon can potentially regenerate over time -- the correct and clinically relevant mechanism explaining the modality's distinct recovery profile. Chemical denaturation with phenol describes chemical neurolysis, a different modality relying on a cytotoxic chemical rather than freezing. High-frequency electrical current generating thermal coagulation describes radiofrequency ablation, the alternative heat-based technique the physician is explicitly contrasting cryoablation against in this vignette.

79. A. Chemical neurolytic agents like phenol and alcohol destroy nervous tissue through direct chemical protein denaturation, and because they are injected as a liquid, their spread within tissue is generally less precisely controllable than the well-defined lesion size achievable with an electrode-based thermal (RFA) technique -- an important practical distinction. Describing chemical neurolysis as delivering high-frequency alternating current is incorrect and conflates it with RFA, which is an entirely different, electrically based mechanism, not a chemical one. Claiming chemical neurolytics have no risk of affecting adjacent tissue is inaccurate; their less controllable spread compared to a thermal lesion is exactly why unintended spread to nearby structures is a recognized procedural risk.

80. A. PRP concentrates platelets that release growth factors (such as PDGF, TGF-beta, and VEGF) at the injection site, which are proposed to stimulate local tissue repair processes including angiogenesis and collagen synthesis in chronically degenerated tendon tissue -- the accepted regenerative mechanism behind PRP therapy. PRP contains no corticosteroid and does not work through a corticosteroid-type anti-inflammatory mechanism; its proposed benefit is regenerative/reparative, which is a fundamentally different mode of action from steroid injections. PRP is not a local anesthetic and does not itself provide

numbness; any procedural discomfort relief would come from a separately co-administered anesthetic, not from the platelet concentrate itself.

81. B. Cognitive restructuring is the core CBT technique that directly targets catastrophic and distorted thoughts about pain by helping the patient identify, challenge, and replace them with more balanced, adaptive thinking, which is the correct answer for the mechanism described. Progressive muscle relaxation is a genuine, useful CBT-adjunct technique for physiologic arousal, but it targets somatic tension rather than directly restructuring catastrophic cognitions. Exposure-based systematic desensitization to feared movements is a real, distinct behavioral technique more closely tied to fear-avoidance and kinesiophobia treatment, not the specific cognitive-restructuring technique targeting catastrophic thoughts described here.

82. A. This describes the operant conditioning model, in which pain-related behaviors are shaped by their consequences -- reinforcing well behaviors while withdrawing reinforcement from excessive pain behaviors -- which is a well-established behavioral treatment framework in chronic pain rehabilitation. Classical (Pavlovian) conditioning involves associating a previously neutral stimulus with an automatic response through repeated pairing, which is a different mechanism from the consequence-based reinforcement of behaviors described in this vignette. A psychodynamic model would focus on unconscious conflicts driving symptom expression, which is a fundamentally different theoretical framework from the behavioral reinforcement approach described.

83. C. Acceptance and Commitment Therapy explicitly emphasizes psychological flexibility, acceptance of pain rather than struggle to eliminate it, and values-driven behavioral engagement rather than direct pain intensity reduction, matching the description precisely. Traditional CBT, while related, more commonly emphasizes direct symptom and cognitive-distortion reduction as a treatment target, which contrasts with the acceptance-based, non-symptom-focused approach described here. EMDR is a genuine, distinct therapy primarily used for trauma processing through guided eye movements, not a values-based acceptance approach for chronic pain.

84. C. Biofeedback uses real-time physiologic monitoring (such as surface EMG) to give the patient direct feedback on a bodily process like muscle tension, enabling them to learn voluntary self-regulation techniques -- exactly the treatment described. TENS is a distinct, unrelated electrical stimulation therapy that delivers current to reduce pain via gate control mechanisms; it does not provide feedback on the patient's own physiologic signals for self-regulation. Describing this as CBT without physiologic monitoring is a direct contradiction, since the vignette explicitly centers on physiologic (EMG) monitoring as the defining feature of the treatment.

85. A. Chronic pain and depression share overlapping neurobiological pathways (including serotonergic and noradrenergic systems) and a well-documented bidirectional relationship, in which each condition can worsen the course and treatment resistance of the other, which is the accurate and clinically important characterization. Describing the relationship as entirely coincidental with no shared neurobiology directly contradicts substantial evidence of shared neurotransmitter systems and

overlapping brain circuitry between chronic pain and depression. Claiming depression is always the primary cause and pain is always merely a somatic symptom of it overstates and oversimplifies a genuinely bidirectional relationship into a single, incorrect causal direction.

86. C. SNRIs inhibit reuptake of both serotonin and norepinephrine, and this combined mechanism underlies their demonstrated efficacy for both depressive symptoms and neuropathic pain, making them a rational single-agent choice for a patient with both conditions. SSRIs, while effective for depression, generally have weaker and less consistent evidence for neuropathic pain specifically compared to agents with added noradrenergic activity, so calling them the strongest option for pain is inaccurate. MAOIs are not considered first-line for this combination; they carry significant dietary restrictions and drug interaction risks that make them a later-line option, not a preferred first choice based on a favorable side-effect profile.

87. C. Validated risk stratification tools such as the Opioid Risk Tool assess personal and family history of substance misuse and psychiatric comorbidity specifically to stratify a patient's risk of opioid misuse and inform how intensively they should be monitored during therapy, which is the correct purpose described. These tools do not calculate opioid starting dose based on body weight; dosing decisions are guided by different clinical parameters, not this risk-screening instrument. Risk stratification tools are not diagnostic instruments for the underlying pain condition itself; their purpose is behavioral/misuse risk assessment, not pain diagnosis.

88. B. A disclosure of active suicidal ideation with a specific plan represents an acute safety emergency requiring immediate action -- arranging an emergent psychiatric evaluation and ensuring the patient's safety before the visit concludes is the appropriate and necessary response. Continuing the routine visit and deferring this disclosure to a future appointment fails to address an acute, potentially life-threatening safety risk and is not an appropriate response to active suicidal ideation with a plan. Simply adjusting antidepressant dosing and following up in a month is a grossly inadequate response to an acute crisis; medication adjustment alone does not address the immediate safety concern of a patient with an active plan for self-harm.

89. C. Graded activity (graded exercise) therapy involves systematically and progressively increasing activity according to a predetermined quota-based schedule rather than being dictated by fluctuating pain levels, which helps counter deconditioning and fear-avoidance -- exactly the approach described. Passive modality therapy using only heat and ultrasound is a real but distinct, passive treatment approach that does not involve the active, progressive exercise progression central to graded activity therapy. Bed rest with activity restriction is now recognized as counterproductive for chronic low back pain and is the direct opposite of the progressive activity approach described in this vignette.

90. A. Spinal manipulative therapy has evidence supporting modest short-term benefit for acute, uncomplicated low back pain when used as part of a broader multimodal treatment approach, which correctly characterizes its evidence-based role. Manual therapy is not universally contraindicated for low back pain; appropriate patient selection (absence of red flags) is what determines candidacy, so a blanket

contraindication statement is incorrect. Manual therapy is not curative on its own and does not eliminate the need for active exercise-based treatment, which remains an important component of comprehensive low back pain management.

91. B. Warm-water buoyancy reduces effective body weight and joint loading, making movement less painful and allowing patients with fibromyalgia-related pain and deconditioning to exercise with better tolerance and adherence than land-based exercise -- the correct rationale for aquatic therapy in this population. Water immersion does not work through direct NMDA receptor blockade; that is a pharmacologic mechanism (relevant to drugs like ketamine), not a property of aquatic exercise. Aquatic therapy for fibromyalgia specifically uses WARM water (not cold) precisely because warmth helps with comfort and muscle relaxation, so attributing its benefit to a cold-water anti-inflammatory mechanism is factually incorrect.

92. A. Orthotic bracing works by providing external mechanical support and correcting alignment or tracking, thereby reducing abnormal joint loading and improving biomechanics -- exactly the mechanism by which a patellar tracking brace helps patellofemoral pain syndrome. A knee brace is a purely mechanical device and does not deliver any corticosteroid medication; describing it as a sustained drug-delivery device is incorrect and confuses it with a pharmacologic intervention. Bracing similarly has no transdermal drug-delivery component or systemic analgesic drug effect; its benefit is entirely biomechanical, not pharmacologic.

93. C. Substantial evidence supports that early return to modified or light duty work, rather than prolonged absence, is associated with better functional recovery and a lower risk of the injury progressing to chronic pain and long-term disability, which is the correct evidence-based rationale. While cost considerations exist, describing modified work as having no benefit to the patient ignores the well-documented clinical advantages of early activity and work engagement for recovery. Prolonged complete work absence is actually associated with WORSE long-term functional outcomes and higher risk of chronicity, which is the opposite of what the evidence shows and directly contradicts this option's claim.

94. B. A functional restoration program is defined by this intensive, interdisciplinary combination of physical reconditioning, psychological treatment, and vocational counseling, explicitly targeting functional improvement and return to work rather than pain elimination -- matching the description exactly. Passive modality-only physical therapy is a real but far more limited, single-modality approach that lacks the interdisciplinary psychological and vocational components central to functional restoration. Isolated pharmacologic management involves medication alone without the interdisciplinary physical, psychological, and vocational components that define the program described in this vignette.

95. B. Work conditioning refers to a general physical conditioning phase focused on building strength, endurance, and flexibility without reference to specific job tasks, which precedes and is distinct from work hardening, a later phase that simulates actual job-specific demands. Work hardening is the more job-task-specific phase being explicitly contrasted against in this vignette, not the general conditioning

phase being described, so it is the wrong label here. A functional capacity evaluation is a distinct, separate assessment tool used to measure safe functional abilities, not an ongoing conditioning program itself.

96. A. The AMA Guides to the Evaluation of Permanent Impairment is the widely accepted standard reference used by physicians to assign percentage impairment ratings for workers' compensation and disability determinations after maximal medical improvement. The DSM-5-TR is a real, important diagnostic manual, but it is used for psychiatric diagnosis, not for quantifying physical permanent impairment percentages for a workers' compensation claim. The ICD-10-CM coding manual is used for diagnostic and billing coding purposes, not for generating a standardized percentage impairment rating, so it does not serve the function described here.

97. A. Mindfulness practice is proposed to work by cultivating nonjudgmental, present-moment awareness that changes how pain sensations are cognitively and emotionally appraised, reducing the secondary suffering and distress associated with chronic pain even when the sensation itself persists. Mindfulness is a psychological/behavioral practice, not a pharmacologic intervention, so it does not work through any direct chemical blockade of peripheral nociceptors. Mindfulness does not involve any permanent structural destruction of brain tissue; any neuroplastic changes associated with regular practice are functional and adaptive, not ablative.

98. B. Clinical evidence supports yoga as providing modest improvements in pain and function for chronic low back pain, making it a reasonable adjunctive option alongside standard care, which is the accurate, evidence-based characterization. Stating yoga has no evidence of benefit and should be discouraged contradicts the actual body of research supporting modest efficacy for this condition. Claiming yoga is proven superior to all standard exercise-based physical therapy overstates the evidence; it is generally considered a complementary adjunct rather than a superior replacement for established exercise-based care.

99. C. Massage therapy is proposed to reduce local muscle tension and stimulate large-diameter mechanoreceptive afferents along with endogenous pain-modulatory pathways, producing short-term analgesic benefit -- a reasonable and evidence-consistent mechanism for myofascial neck pain. Massage has no capacity to permanently remodel degenerated disc tissue; disc structural changes are not reversed by manual soft-tissue therapy, making this option physiologically implausible. Massage does not act as a direct pharmacologic NMDA receptor antagonist; that mechanism describes a specific drug class (e.g., ketamine), not a manual therapy modality.

100. C. Tai chi has accumulated evidence supporting benefits in pain, physical function, and quality of life for both fibromyalgia and osteoarthritis, making it a reasonable low-impact complementary option for a patient with both conditions. Tai chi is not harmful in these populations; on the contrary, its low-impact nature makes it particularly well suited for patients with joint pain and deconditioning, so a blanket harm warning is incorrect. Tai chi does not work through direct cartilage regeneration; it is a

mind-body movement practice whose proposed benefits relate to function, strength, balance, and pain modulation, not structural joint tissue regrowth.

101. A. Neuropathic pain is specifically defined as pain caused by a lesion or disease affecting the somatosensory nervous system, which matches this vignette's documented peripheral nerve injury and its characteristic burning, shooting quality. Nociceptive pain arises from actual or threatened tissue damage detected by nociceptors without a nerve lesion, which does not fit a case explicitly involving documented nerve injury. Nociplastic pain is a distinct category describing pain arising from altered nociception despite no clear evidence of tissue damage or a demonstrable nerve lesion, which does not match this patient's confirmed peripheral nerve injury.

102. B. Chronic pain is generally defined as pain persisting beyond the normal expected healing time, conventionally set at approximately 3 months, which this patient's 4-month course clearly exceeds, making this the correct classification. Acute pain is defined by its temporal relationship to tissue injury and expected healing, typically resolving within days to a few weeks, which does not match a pain course that has persisted for 4 months beyond expected healing. While the term subacute pain is sometimes used descriptively in clinical practice, standard pain taxonomy formally recognizes the acute/chronic distinction with the roughly 3-month threshold, making this option's premise that there is no defined threshold at all incorrect in context.

103. C. This vignette describes fibromyalgia diagnosed using the current 2016 American College of Rheumatology criteria, which rely on the Widespread Pain Index and Symptom Severity Scale (capturing fatigue, unrefreshing sleep, and cognitive symptoms) rather than the older physical tender point examination. Rheumatoid arthritis is an inflammatory arthritis with objective joint findings and serologic/inflammatory markers, not diagnosed by a tender point count, and does not match this non-inflammatory, widespread pain presentation. Polymyalgia rheumatica is a distinct inflammatory condition typically presenting with shoulder and hip girdle stiffness and elevated inflammatory markers, which is inconsistent with this patient's normal inflammatory workup and diffuse fibromyalgia-type symptom pattern.

104. C. Central sensitization -- characterized by augmented central pain processing, enhanced temporal summation, and impaired descending inhibitory modulation -- is the recognized core pathophysiologic mechanism underlying fibromyalgia and other chronic widespread pain syndromes, matching this description precisely. Peripheral nociceptor sensitization from local inflammation would imply a peripheral tissue-based process, which contradicts the vignette's finding of centrally augmented processing without peripheral tissue pathology. Fibromyalgia is not an autoimmune joint-destructive disease; it lacks the synovitis and structural joint damage seen in autoimmune arthritides, making this option pathophysiologically inaccurate for the condition described.

105. B. Multimodal analgesia targets multiple distinct nociceptive pathways simultaneously, producing synergistic or additive analgesic effects that reduce total opioid consumption and opioid-related adverse effects such as sedation, nausea, and respiratory depression -- the well-established rationale for this

approach. The premise that combining non-opioid agents increases opioid dosing efficiency misstates the actual goal, which is to REDUCE opioid requirement, not to make opioid dosing more efficient. There is no legal prohibition on single-agent opioid therapy after surgery; multimodal analgesia is a clinical best practice choice, not a legal requirement, making this option factually incorrect.

106. B. Regional analgesic techniques such as erector spinae plane blocks or thoracic epidural analgesia provide effective chest wall pain control that allows patients with rib fractures to cough and breathe deeply, directly reducing the risk of pneumonia and respiratory failure -- the evidence-based recommended intervention. Acetaminophen monotherapy, while a useful adjunct, does not provide adequate analgesia on its own for multiple rib fractures to meaningfully change respiratory mechanics in this at-risk population. Rigid chest binders are now recognized as harmful in rib fracture management because they further restrict chest wall movement and worsen, rather than improve, pulmonary mechanics and atelectasis risk.

107. A. Burn pain management recognizes background (constant, baseline) pain and procedural (dressing-change) pain as distinct entities requiring different approaches: scheduled longer-acting analgesics for background pain, plus additional short-acting, rapid-onset analgesia or sedation specifically timed around dressing changes to control the much more intense procedural pain. Treating both pain states identically with a single fixed daily dose and no additional procedural dosing would leave dressing changes severely undertreated, since procedural pain is characteristically much more intense than background pain. Burn pain is explicitly NOT a single uniform entity; recognizing and separately addressing the procedural pain spike is a core, well-established principle of burn pain management, making this option incorrect.

108. B. The theoretical rationale for preemptive/preventive analgesia is that blocking nociceptive input before the surgical incision reduces the barrage of noxious signaling that drives central sensitization, potentially reducing both acute postoperative pain intensity and the risk of chronic postsurgical pain developing. If timing of analgesia before versus after incision truly made no difference, there would be no rationale for preemptive strategies at all, which directly contradicts the premise and purpose of this approach. Preemptive analgesia is an analgesic strategy focused on pain outcomes, not an intervention intended to shorten operative time, so this option misstates its actual purpose.

109. A. A femoral nerve block, or the related fascia iliaca compartment block, provides effective analgesia for hip fracture pain by targeting the relevant sensory innervation while reducing the need for systemic opioids, which is particularly valuable in elderly patients at risk for opioid-related delirium. A stellate ganglion block targets upper extremity sympathetic innervation and has no role in hip fracture pain, making it anatomically irrelevant here. A celiac plexus block targets upper abdominal visceral sympathetic innervation and similarly has no anatomic relevance to hip fracture pain.

110. A. Pain out of proportion to the injury, worsened by passive stretch, and refractory to escalating analgesia is the classic red-flag presentation for acute compartment syndrome, a surgical emergency requiring urgent evaluation (including compartment pressure measurement) rather than simply

increasing pain medication, which could mask a limb-threatening diagnosis. Attributing this pattern to opioid tolerance and simply escalating the dose would dangerously delay recognition of compartment syndrome, risking irreversible muscle and nerve damage. Treating this presentation as expected and requiring no further evaluation ignores a critical clinical red flag and could similarly delay a time-sensitive, limb-saving diagnosis and intervention.

111. C. Identifying risk factors such as severe acute postoperative pain, preoperative psychological factors, and high-risk surgical procedure types allows clinicians to target high-risk patients with more aggressive multimodal analgesia and psychological support to reduce the risk of transition to chronic postsurgical pain -- the correct clinical application of this research. These factors have well-documented predictive value in the literature; describing them as having no established predictive value directly contradicts the premise that this is a recognized area of active clinical research and risk stratification. These factors have direct clinical, perioperative planning relevance beyond pure research use, since they inform real-world targeted intervention strategies, making the claim that they have no clinical bearing incorrect.

112. C. PCA's core safety feature is that the patient must be awake and alert enough to press the demand button, providing a natural safeguard against oversedation; adding a continuous basal rate in an opioid-naive patient bypasses this self-titration safeguard and has been associated with increased respiratory depression risk. Basal rates do not eliminate respiratory depression risk -- they actually increase it in opioid-naive patients by removing the self-limiting demand-dosing safeguard, which is the opposite of what this option claims. Basal rates are an optional PCA setting, not a mandatory pump design requirement, so describing them as required is factually incorrect.

113. B. The Critical-Care Pain Observation Tool and the Behavioral Pain Scale are validated observational instruments specifically designed to assess pain in nonverbal, critically ill, mechanically ventilated patients using behavioral indicators such as facial expression, movement, and ventilator compliance. The McGill Pain Questionnaire requires patient self-report of descriptive pain qualities, which is not possible in a sedated, mechanically ventilated patient, making it inappropriate here. The Oswestry Disability Index is a self-report functional disability questionnaire for chronic low back pain, entirely unrelated to acute ICU pain assessment in a nonverbal patient.

114. C. An acute pain service is a dedicated multidisciplinary team specifically focused on optimizing postoperative and other acute pain management, rounding regularly to adjust regimens and reduce complications, matching the description exactly. A palliative care consult service focuses on symptom management and goals of care generally in serious illness or end-of-life contexts, not specifically on optimizing routine postoperative analgesic regimens hospital-wide. A rapid response team is an entirely different hospital resource dedicated to responding to acute clinical deterioration, not to proactive, scheduled postoperative pain management rounding.

115. B. Ketamine's dissociative sedation combined with analgesia, while generally preserving spontaneous respiration and airway reflexes, makes it particularly well suited for brief, painful pediatric

procedures such as fracture reduction, which is its key clinical advantage. Significant respiratory depression is not a desirable or typical effect of ketamine at procedural sedation doses -- in fact, relative preservation of respiratory drive is precisely the advantage that distinguishes it from other sedatives, making this option incorrect. Ketamine is well known to cause psychomimetic effects (such as hallucinations or emergence reactions), particularly in adults, though generally less pronounced in children; claiming it has no such side effects in children is an overstatement.

116. A. Low-dose ketamine's analgesic effect is mediated through NMDA receptor antagonism, which reduces central sensitization and provides meaningful analgesia through a mechanism entirely independent of the opioid receptor system, making it a rational and effective opioid-sparing adjunct in acute trauma pain. Ketamine does not act as a mu-opioid receptor agonist; its opioid-independent mechanism is precisely why it is valuable as an opioid-sparing strategy, so describing it as a weaker opioid agonist is incorrect. Ketamine has no COX-2 inhibitory activity; that mechanism describes NSAIDs, an entirely different drug class with a different mechanism from ketamine's NMDA-based action.

117. C. Best practice guidance for acute injury discharge prescribing emphasizes prescribing the smallest effective opioid quantity for the expected short duration of severe pain (often only a few days), combined with non-opioid analgesics, specifically to reduce leftover pills available for diversion or misuse while still controlling acute pain. Prescribing a full 30-day supply for a minor acute injury substantially exceeds the expected duration of severe pain and directly increases the risk of unused, divertible opioid medication, which is the opposite of best practice. Withholding all analgesic prescriptions regardless of injury severity fails to address genuine acute pain needs and is not the balanced, patient-centered approach recommended by current guidance.

118. B. Discogenic pain classically presents as axial low back pain aggravated by flexion, prolonged sitting, and axial loading (activities that increase intradiscal pressure), without a clear dermatomal radicular pattern, which matches the vignette. Pain that worsens with extension and improves with flexion is more characteristic of facet joint-mediated pain, which has an opposite provocation pattern from the flexion-aggravated discogenic pain described here. Pain strictly localized to a unilateral sacral sulcus reproduced by the FABER maneuver is characteristic of sacroiliac joint dysfunction, a distinct anatomic source from the disc pathology and diffuse axial pattern in this vignette.

119. A. Neurogenic claudication from lumbar spinal stenosis classically improves with flexion (such as sitting or leaning forward, which increases spinal canal diameter) and is not reliably relieved by simply standing still, matching this vignette precisely. Vascular claudication typically improves with any rest/standing still regardless of position, without the specific flexion-dependent relief pattern described, making it a less precise fit than neurogenic claudication here. Cauda equina syndrome from acute disc herniation would present with additional red-flag features such as saddle anesthesia, bowel/bladder dysfunction, and significant motor deficits, which are not described in this more classic claudication vignette.

120. A. Spondylolisthesis refers to anterior (or posterior) vertebral body displacement, often resulting from a pars interarticularis defect (spondylolysis), which is a well-recognized cause of chronic low back pain in young athletes engaged in repetitive extension/rotation activities. Facet joint osteoarthritis without instability would not produce vertebral body displacement or a pars defect, which are the specific findings described in this vignette. An isolated disc herniation without bony pathology would not explain the vertebral displacement and pars interarticularis defect that are explicitly present on this patient's imaging.

121. B. Facet joint-mediated pain classically worsens with extension and rotation (movements that load the posterior facet joints) and improves with flexion, which is the opposite provocation pattern from discogenic pain and matches this vignette exactly. The intervertebral disc typically produces pain that worsens with flexion and axial loading, which is the opposite of the extension-worsened, flexion-improved pattern described here, making it an inconsistent source. The sacroiliac joint typically produces pain localized to the sacral sulcus/buttock region reproduced by specific provocative maneuvers (e.g., FABER, thigh thrust), rather than the extension/rotation-provoked paraspinal pattern described in this vignette.

122. C. Unilateral, non-midline-crossing buttock pain reproduced by provocative maneuvers such as FABER and thigh thrust is the classic clinical pattern for sacroiliac joint dysfunction, matching this vignette precisely. An intervertebral disc source would typically produce midline or bilateral axial pain worsened by flexion, not the specific unilateral provocative test pattern (FABER, thigh thrust) described here. A facet joint source would typically be provoked by extension and rotation rather than by the specific SI-joint provocative maneuvers used in this vignette, making it a less consistent match.

123. A. Piriformis syndrome presents with deep buttock pain and posterior thigh radiation from sciatic nerve irritation near the piriformis muscle, characteristically worsened by sitting and hip internal rotation, and importantly occurs with a normal lumbar MRI, distinguishing it from true nerve root compression -- exactly matching this vignette. An acute L5-S1 disc herniation with nerve root compression would typically show a corresponding abnormality on lumbar MRI and often produces a true dermatomal sensory or motor deficit, neither of which is present in this normal-imaging vignette. Lumbar spinal stenosis with neurogenic claudication would present with bilateral leg symptoms relieved by flexion and worsened by walking/extension, a different pattern than the sitting-provoked, hip-rotation-provoked unilateral pain described here.

124. B. Osteoarthritis is characterized pathophysiologically by progressive degeneration of articular cartilage with secondary subchondral bone remodeling, sclerosis, and osteophyte formation, which is exactly the imaging and clinical pattern (minimal synovitis) described in this vignette. Autoimmune synovial inflammation with pannus formation describes rheumatoid arthritis, a fundamentally different inflammatory process that would typically show significant synovitis, not the minimal inflammation noted here. Crystal deposition triggering acute inflammatory flares describes gout or pseudogout, which present with episodic acute inflammatory attacks rather than the chronic, degenerative, minimally inflammatory pattern described in this vignette.

125. C. Rheumatoid arthritis produces an inflammatory pain pattern characterized by prolonged morning stiffness (typically over an hour) that improves with activity, symmetric small joint involvement, and positive autoimmune serologies, all of which are described in this vignette and reflect underlying autoimmune synovitis. A degenerative, purely mechanical pattern without inflammatory markers describes osteoarthritis, which would not typically produce positive rheumatoid factor and anti-CCP antibodies or the prolonged inflammatory morning stiffness seen here. Episodic, crystal-induced monoarticular attacks describe gout, which is inconsistent with this patient's symmetric polyarticular involvement and positive autoimmune serology.

126. A. Acute gout classically presents with rapid-onset, severe monoarticular inflammation of the first MTP joint (podagra), and needle-shaped, negatively birefringent crystals on joint aspiration are the diagnostic hallmark of monosodium urate deposition, exactly matching this vignette. Pseudogout (CPPD disease) is caused by rhomboid-shaped, positively birefringent crystals, not the needle-shaped, negatively birefringent crystals described here, making it inconsistent with these specific aspiration findings. Septic arthritis would show organisms and a markedly elevated white cell count with typically negative crystal analysis on synovial fluid, not the crystal findings that are the defining feature of this vignette.

127. C. Pseudogout is caused by calcium pyrophosphate crystal deposition, and the diagnostic distinction from gout is precisely the crystal morphology and birefringence pattern: rhomboid-shaped and positively birefringent in pseudogout versus needle-shaped and negatively birefringent in gout. Gout is caused by monosodium urate crystals with the opposite morphology and birefringence pattern described in this vignette, so stating both conditions share identical crystal types is factually incorrect. Positive birefringence is a crystal optical property under polarized light, not an indicator of bacterial infection; septic arthritis is diagnosed by organisms and cell counts on culture/Gram stain, not by crystal birefringence pattern.

128. B. Adhesive capsulitis is characterized by progressive shoulder pain and stiffness with restriction of BOTH active and passive range of motion in multiple planes, distinguishing it from other shoulder conditions and matching this vignette's global motion restriction. A rotator cuff tear typically preserves passive range of motion (the examiner can passively move the joint through a fuller range) even when active motion is limited by pain or weakness, which is inconsistent with this vignette's global passive restriction. Acute subacromial bursitis typically causes pain-limited active motion with relatively preserved passive range of motion, not the global active-and-passive restriction described in this vignette.

129. A. The Neer and Hawkins tests are specific provocative maneuvers for subacromial impingement, reproducing pain by compressing the rotator cuff tendons and subacromial bursa against the acromion, matching this vignette's positive findings and presentation. Adhesive capsulitis would present with global loss of both active and passive range of motion rather than the specific impingement-provoked pain pattern described by positive Neer and Hawkins tests. Acromioclavicular joint separation would

typically present with focal tenderness and deformity at the AC joint and a positive cross-body adduction test, not the specific impingement signs described in this vignette.

130. C. Lateral epicondylitis presents with lateral elbow pain reproduced by resisted wrist extension, reflecting overuse injury of the common extensor tendon origin at the lateral epicondyle, matching this vignette's presentation and provocation pattern exactly. Medial epicondylitis affects the flexor-pronator tendon origin on the medial elbow and is provoked by resisted wrist flexion, not the resisted extension pattern described in this vignette, making it anatomically inconsistent. Cubital tunnel syndrome is a nerve entrapment causing numbness/tingling in the ulnar nerve distribution (ring and small fingers), not the tendinopathic lateral elbow pain with resisted extension described here.

131. C. Medial epicondylitis presents with medial elbow pain reproduced specifically by resisted wrist flexion and pronation, reflecting overuse of the flexor-pronator muscle origin at the medial epicondyle, matching this vignette precisely. Lateral epicondylitis affects the extensor tendon origin on the lateral elbow and is provoked by resisted wrist extension, not the flexion/pronation pattern described here, making it anatomically inconsistent. A UCL tear would typically present with medial elbow pain and instability specifically on valgus stress testing, along with a history of repetitive valgus loading (e.g., throwing), rather than isolated pain with resisted flexion/pronation as described in this vignette.

132. B. De Quervain tenosynovitis involves inflammation of the abductor pollicis longus and extensor pollicis brevis tendons at the radial wrist, and the Finkelstein test (thumb tucked in fist, ulnar deviation) is the classic provocative maneuver that reproduces this pain, matching the vignette exactly. Carpal tunnel syndrome causes median nerve compression symptoms (numbness/tingling in the thumb, index, and middle fingers) and is assessed with a Tinel or Phalen sign, not the Finkelstein maneuver described, and it does not typically present as isolated radial wrist pain with lifting. Trigger finger presents with a catching or locking sensation of a finger during flexion/extension due to a flexor tendon nodule, not radial-sided wrist pain reproduced by the Finkelstein test described here.

133. A. Greater trochanteric pain syndrome presents with lateral hip pain that worsens with lying on the affected side and with activity, along with point tenderness directly over the greater trochanter, matching this vignette's presentation precisely. Hip osteoarthritis classically produces groin-predominant pain (from the intra-articular hip joint itself) rather than isolated lateral point tenderness, making it a less anatomically consistent choice than the trochanteric pain syndrome described. Meralgia paresthetica causes numbness and tingling (not primarily point-tender pain) over the anterolateral thigh from lateral femoral cutaneous nerve entrapment, which does not match this vignette's lateral hip tenderness pattern.

134. B. Plantar fasciitis is classically characterized by sharp first-step morning pain at the plantar heel that partially improves with continued walking but worsens again with prolonged standing later in the day, matching this vignette's presentation exactly. Achilles tendinopathy typically produces posterior heel/tendon pain rather than plantar (bottom of foot) pain, and while it can have some morning stiffness, the classic first-step plantar heel pattern described here is more specific to plantar fasciitis. Tarsal tunnel syndrome causes numbness, tingling, or burning in the plantar foot from posterior tibial nerve

entrapment, a neuropathic pattern rather than the classic mechanical first-step pain pattern described in this vignette.

135. B. Achilles tendinopathy presents with posterior ankle/heel pain and stiffness that is worse with activity onset and after rest, often with palpable tendon thickening, reflecting chronic overuse degenerative changes -- exactly matching this vignette. Plantar fasciitis causes pain at the plantar (bottom) aspect of the heel, not the posterior ankle location described in this vignette, making it anatomically inconsistent. Posterior tibial tendon dysfunction classically presents with medial ankle pain and progressive flatfoot deformity, a different anatomic location and clinical picture than the posterior Achilles pain and palpable tendon thickening described here.

136. C. Whiplash-associated disorder is specifically defined by this rapid acceleration-deceleration mechanism (classically from rear-end collisions) causing soft tissue injury to cervical spine structures, producing neck pain, stiffness, and headache, matching this vignette's description and mechanism precisely. Cervical radiculopathy specifically involves nerve root compression producing arm pain, numbness, or weakness in a dermatomal distribution, which is not the mechanism or presentation described in this whiplash vignette. Cervical spondylotic myelopathy is a chronic, degenerative spinal cord compression process typically presenting with gait disturbance and hand clumsiness over time, not an acute soft-tissue injury from a specific traumatic acceleration-deceleration event as described here.

137. A. Cervicogenic headache is defined by headache referred from cervical spine structures (such as upper cervical facet joints), characteristically unilateral, worsened by neck movement/posture, and associated with restricted cervical range of motion, matching this vignette's presentation and exam findings precisely. Classic migraine is typically not primarily provoked by neck movement or associated with restricted cervical range of motion and tenderness over cervical facets, which are the defining features described in this vignette, making it a less specific match. Trigeminal neuralgia causes paroxysmal, brief, electric shock-like facial pain in the trigeminal nerve distribution, an entirely different pain quality and location from the neck-movement-provoked occipital-to-anterior headache described here.

138. B. Myofascial pain syndrome is a regional condition defined by focal, palpable trigger points within a taut muscle band that reproduce referred pain locally, which is clinically distinct from fibromyalgia's widespread, bilateral, centrally mediated pain pattern accompanied by systemic symptoms like fatigue and cognitive difficulty -- exactly the distinction drawn in this vignette. Describing myofascial pain syndrome as simply an early or mild form of fibromyalgia with identical pathophysiology is inaccurate; the two conditions have distinct proposed mechanisms (peripheral/regional taut-band pathology versus central sensitization with widespread involvement) and are recognized as separate clinical entities. Claiming there is no meaningful clinical distinction between the two conditions directly contradicts the well-established regional-versus-widespread distinction that is central to differentiating them in clinical practice.

139. A. Costochondritis presents with sharp, reproducible chest wall pain localized to the costochondral junctions, worsened by palpation, deep breathing, or twisting, with a normal cardiac workup, matching this vignette's description precisely and providing important reassurance once cardiac causes are excluded. Unstable angina would not be reproducible by direct chest wall palpation and would instead present with exertional or rest cardiac ischemic symptoms with corresponding cardiac workup abnormalities, which are explicitly absent (normal cardiac workup) in this vignette. Pulmonary embolism typically presents with pleuritic pain associated with dyspnea, tachycardia, and risk factors for thromboembolism, rather than pain that is specifically reproduced by direct palpation of the costochondral junctions as described here.

140. B. This vignette describes classic inflammatory back pain features -- insidious onset in a young adult, prolonged morning stiffness improving with exercise but not rest, and limited spinal mobility -- which should raise suspicion for ankylosing spondylitis, an inflammatory spondyloarthropathy. Mechanical low back pain from acute strain typically has an identifiable precipitating activity, improves with rest, and does not characteristically produce the prolonged inflammatory morning stiffness pattern described in this vignette. An osteoporotic vertebral compression fracture would typically present with acute-onset, often more severe localized pain (sometimes after minimal trauma in an older or osteoporotic patient), not the insidious inflammatory pattern with prolonged morning stiffness described here.

141. C. Polymyalgia rheumatica classically presents in older adults with bilateral shoulder and hip girdle pain and stiffness, prominent in the morning, accompanied by markedly elevated inflammatory markers such as ESR, matching this vignette's presentation and laboratory findings precisely. Fibromyalgia would be expected to show normal inflammatory markers, which directly contradicts this vignette's markedly elevated ESR, making it an inconsistent choice. Osteoarthritis of the shoulders and hips would not typically produce markedly elevated inflammatory markers or the prominent systemic morning stiffness/fatigue pattern described, since OA is a degenerative rather than a systemic inflammatory process.

142. C. Sudden, severe, midline back pain after even minor trauma in a patient with known osteoporosis, with imaging showing vertebral body height loss, is the classic presentation of an osteoporotic vertebral compression fracture. Lumbar facet joint arthropathy typically presents as a more chronic, extension/rotation-provoked pain pattern rather than sudden severe pain after trauma with an associated vertebral height loss finding on imaging. Acute lumbar disc herniation with radiculopathy would be expected to produce radicular leg symptoms and a corresponding disc-level abnormality on imaging, not the vertebral body height loss described in this vignette.

143. A. The WHO analgesic ladder is the classic stepwise framework for cancer pain management, escalating from non-opioids to weak opioids to strong opioids based on pain severity, with adjuvant analgesics incorporated at any step, matching this vignette's approach exactly. The AMA Guides to Permanent Impairment is used for disability/impairment rating determinations, an entirely unrelated administrative framework with no role in guiding analgesic escalation. The ACR 2016 fibromyalgia

diagnostic criteria is a diagnostic tool for a specific chronic pain condition, not a treatment escalation framework for cancer pain.

144. B. Breakthrough cancer pain is defined by these rapid-onset, short-duration pain flares occurring despite adequate baseline (around-the-clock) opioid control, and it is treated with a rapid-onset, short-acting opioid formulation given as needed on top of the scheduled regimen -- exactly matching this vignette's presentation and correct management. Opioid-induced hyperalgesia presents as diffuse, often allodynic pain that worsens with increasing opioid dose and improves with dose reduction, which does not match this pattern of discrete, brief, severe pain episodes despite otherwise adequate baseline control. Pseudoaddiction describes drug-seeking behavior stemming from undertreated pain that resolves once pain is adequately controlled; it is a behavioral pattern, not the correct label for this specific breakthrough pain phenomenon, and discontinuing opioid therapy would be an inappropriate and harmful response to genuine cancer pain.

145. C. Bone metastasis pain arises largely from tumor-induced osteoclast activation and release of local pain-sensitizing mediators such as prostaglandins, along with RANKL-driven bone resorption that sensitizes periosteal nociceptors and structurally weakens bone -- the correct and well-established mechanism, which is also why antiresorptive agents like bisphosphonates or denosumab can reduce this pain. Not every painful bone metastasis site involves spinal cord compression; that is a specific, less common complication (an oncologic emergency), not the general mechanism of bone metastasis pain everywhere it occurs. Bone metastasis pain is not an autoimmune synovitis process; it is a direct local tumor-bone interaction effect, not an autoimmune joint-inflammatory mechanism.

146. A. Platinum-based chemotherapeutic agents such as oxaliplatin are classically associated with chemotherapy-induced peripheral neuropathy, presenting with symmetric, distal, stocking-glove sensory symptoms including numbness, tingling, and burning pain, matching this vignette's presentation and the clinical context of colorectal cancer treatment. Topical corticosteroids are not a chemotherapy agent and are not associated with causing peripheral neuropathy; this option is not a relevant chemotherapeutic class at all. Beta-lactam antibiotics are not chemotherapeutic agents and are not a recognized cause of peripheral neuropathy, making this option irrelevant to the chemotherapy-induced mechanism being described.

147. B. Unlike NSAIDs and other non-opioid analgesics, full mu-opioid agonists do not have a clinically meaningful analgesic ceiling effect, which allows their dose to be titrated upward as needed to match increasing cancer pain severity -- the correct pharmacologic property described. Stating that full opioid agonists have a fixed ceiling dose identical to NSAIDs is incorrect and describes the opposite of the actual pharmacologic distinction being tested, since NSAIDs (not full agonist opioids) are the class with a true analgesic ceiling. There is no fixed rule limiting titration to a maximum of two dose adjustments before mandatory rotation; opioid titration can continue as clinically needed, with rotation considered for other reasons such as intolerable side effects, not an arbitrary adjustment count.

148. A. Colicky pain from malignant bowel obstruction has a significant smooth-muscle spasm component that may not fully respond to opioids alone, so adding an antispasmodic/anticholinergic agent alongside opioid therapy is an important and appropriate strategy to more completely address this pain pattern. Opioids are not contraindicated in malignant bowel obstruction and remain a mainstay of pain control in this palliative setting; discontinuing them entirely would leave the patient's baseline pain undertreated. A topical NSAID applied to the abdominal wall would not adequately address deep visceral colicky pain from bowel obstruction, since topical NSAIDs are designed for localized musculoskeletal/joint pain, not visceral smooth muscle spasm.

149. C. New severe back pain with rapidly progressive bilateral leg weakness and urinary retention in a patient with known metastatic cancer is the classic presentation of malignant epidural spinal cord compression, an oncologic emergency requiring urgent MRI and prompt treatment to prevent permanent neurologic deficit. A routine osteoarthritis flare would not explain the acute bilateral leg weakness and urinary retention described, and treating this presentation as a simple outpatient issue would dangerously delay emergency evaluation. Simple musculoskeletal strain also would not produce bilateral leg weakness and urinary retention, and referring this presentation to routine physical therapy would represent a serious and potentially harmful delay in recognizing a time-critical oncologic emergency.

150. C. Mirels' criteria is the classic scoring system used to assess impending pathologic fracture risk in long bones with metastatic lesions, incorporating site, size, radiographic appearance (lytic/blastic/mixed), and pain level to guide the decision for prophylactic fixation, matching this vignette's clinical scenario exactly. The Oswestry Disability Index is a self-report functional disability questionnaire for chronic low back pain, entirely unrelated to fracture risk assessment in bone metastases. The APACHE II score is a critical illness severity scoring system used in ICU settings to predict mortality, with no role in orthopedic oncologic fracture risk stratification.

151. A. Cancer pain frequently presents with mixed nociceptive (here, visceral pain from tissue infiltration) and neuropathic (here, from nerve plexus invasion) components simultaneously, and recognizing this mixed picture is important for selecting a multimodal treatment approach addressing each mechanism -- exactly what this vignette illustrates. Stating cancer pain is always purely nociceptive directly contradicts the neuropathic component described in this vignette (celiac plexus invasion) and oversimplifies the genuinely mixed nature of many cancer pain presentations. Stating cancer pain is always purely neuropathic similarly contradicts the visceral nociceptive component also present in this vignette, again oversimplifying what is characteristically a mixed pain presentation in cancer.

152. B. Multiple large trials have shown that single-fraction radiotherapy achieves comparable pain relief to longer multi-fraction courses for uncomplicated painful bone metastases, though with a modestly higher rate of eventual retreatment, which is the accurate, evidence-based characterization described in this vignette. Radiation therapy is, in fact, a well-established and effective treatment modality for painful bone metastases, so stating it has no role directly contradicts extensive clinical evidence and standard palliative oncology practice. Radiation's analgesic effect for bone metastases is a

local, site-specific treatment effect (reducing tumor burden and local mediator release) rather than a systemic immune-mediated mechanism unrelated to the treated site.

153. B. Opioid-induced constipation is extremely common and, unlike sedation or nausea, does not typically improve with continued opioid exposure (no meaningful tolerance develops to this effect), which is why proactively starting a bowel regimen at the same time opioids are initiated is standard, evidence-based practice. It is incorrect that opioid-induced constipation is rare or self-resolving; it is one of the most common and persistent opioid side effects, which is precisely why prophylaxis rather than reactive treatment is recommended. The bowel regimen described is specifically targeting constipation, not nausea, which is a separate opioid side effect managed with different agents such as antiemetics, not stimulant laxatives.

154. C. The principle of double effect holds that an action with a good primary intent (relieving suffering through appropriate, proportionate opioid titration) is ethically permissible even if it carries a foreseen but unintended risk of a secondary harmful effect, which is the correct and standard palliative care ethical framework applied here. Withholding opioids entirely near the end of life would leave severe, treatable pain unaddressed and is not supported by the double effect principle, which permits proportionate symptom-focused titration rather than withholding treatment altogether. The double effect principle explicitly requires that hastening death NOT be the intent of the action; describing an intent to hasten death as an acceptable primary goal directly contradicts the core requirement of the ethical framework being tested.

155. A. Visceral afferents are relatively sparse and diverge widely within the spinal cord, and they converge with somatic afferents on shared dorsal horn neurons, which explains both the characteristically poor localization of visceral pain and its tendency to be referred to distant somatic sites -- the accurate general principle described in this vignette. Visceral pain is, in fact, characteristically poorly (not sharply) localized compared to somatic pain, which is the opposite of what this option claims and contradicts a fundamental, well-established feature of visceral pain physiology. Visceral pain is well known for producing referred pain patterns (such as cardiac pain referred to the arm or jaw); claiming it never does so directly contradicts this classic and clinically important phenomenon.

156. B. Chronic pancreatitis pain is understood to result not only from ductal hypertension (obstruction and elevated intraductal pressure) but also from perineural inflammation and neural remodeling within the pancreas itself, which can drive persistent pain and contribute to central sensitization -- the accurate, multifactorial mechanism described in this vignette. Isolated peripheral skin nociceptor sensitization does not capture the actual pancreatic ductal and neural-remodeling mechanisms responsible for this visceral pain, making it an inaccurate and overly simplistic mechanism. Describing chronic pancreatitis pain as purely psychogenic with no physiologic contributor directly contradicts well-documented structural and neural mechanisms (ductal hypertension, perineural inflammation) known to drive this pain.

157. C. Visceral hypersensitivity -- a lowered threshold for perceiving normal visceral stimuli (such as rectal distension) as painful -- is a well-established, central feature of IBS pathophysiology, matching this vignette's experimental finding and normal structural workup precisely. Peripheral nociceptor destruction would be expected to reduce, not increase, pain sensitivity, which is the opposite of the heightened sensitivity to distension described in this vignette. The vignette explicitly states the structural workup (colonoscopy, imaging) is normal, which argues against an undetected structural obstruction as the explanation for this patient's altered pain perception.

158. A. Interstitial cystitis/bladder pain syndrome presents with chronic pelvic pain and pressure related to bladder filling, urinary urgency/frequency, and improvement after voiding, with cystoscopic findings such as glomerulations and an absence of infection or malignancy, matching this vignette's presentation and workup exactly. Acute bacterial cystitis would typically show evidence of infection (positive urine culture, pyuria) rather than the sterile, non-infectious glomerulation findings described, and it is generally an acute rather than chronic pain presentation. The cystoscopic findings described specifically show no evidence of malignancy, directly ruling out bladder cancer as the explanation for this patient's chronic symptoms.

159. A. Endometriosis-related chronic pelvic pain arises from ectopic endometrial implants that produce local inflammation, neurogenic sensitization of nearby nerve fibers, and cyclic hormonally driven tissue changes and bleeding, which together generate and perpetuate the characteristic cyclic pelvic pain and dyspareunia described in this vignette. Describing this as an entirely psychogenic process directly contradicts the clear, identifiable structural and inflammatory pathology (ectopic endometrial implants) found on this patient's laparoscopy. This vignette describes pelvic pain related to laparoscopically confirmed endometriosis, not an isolated, unrelated bladder pain syndrome, making that option an incorrect and unsupported diagnosis for these findings.

160. B. Cardiac visceral afferents converge with somatic afferents from the left arm and jaw dermatomes on shared spinal dorsal horn neurons, and because the brain is more accustomed to interpreting input from these pathways as coming from the somatic structures, it misattributes the visceral cardiac pain to those referred locations -- the correct convergence-projection mechanism for cardiac referred pain. There is no actual mechanical compression of the brachial plexus during a myocardial infarction; the referred arm pain is a neural convergence phenomenon, not a direct compressive mechanical process. Cardiac pain during an MI is a genuine, physiologically driven visceral nociceptive signal from myocardial ischemia, not a psychogenic phenomenon, making this option factually incorrect.

161. A. Acute ureteral distension from an obstructing stone activates visceral afferents that share spinal segmental levels (roughly T11-L1) with somatic afferents supplying the groin and genital region, producing the classic flank-to-groin referred pain pattern of renal colic described in this vignette. The stone is located within the ureter, not adjacent to or compressing the femoral nerve directly, so a direct mechanical femoral nerve irritation mechanism does not explain this classic visceral referred pain

pattern. This referred pain pattern is a well-recognized consequence of the ureteral stone itself (a visceral referral phenomenon), not evidence of separate, primary genital organ pathology unrelated to the stone.

162. C. Functional dyspepsia is characterized by chronic upper GI symptoms such as postprandial fullness, early satiety, and epigastric discomfort in the setting of a structurally normal endoscopy, with proposed mechanisms including visceral hypersensitivity and impaired gastric accommodation to food -- the correct explanation matching this vignette. The vignette specifies a normal upper endoscopy, which argues against an occult malignancy being simply missed, since endoscopy is the standard, sensitive tool used to directly visualize and evaluate for such structural lesions. Chronic bacterial gastritis would be expected to show endoscopic or histologic evidence of gastritis/H. pylori, and empiric prolonged antibiotic therapy regardless of testing is not the standard, evidence-based approach to a normal endoscopy functional dyspepsia presentation.

163. C. This presentation -- unilateral, pulsatile headache lasting 4-72 hours, aggravated by activity, and associated with photophobia, phonophobia, and nausea -- matches the ICHD diagnostic criteria for migraine without aura precisely. Tension-type headache is typically bilateral, pressing/tightening in quality (not pulsatile), and is NOT characteristically aggravated by routine physical activity, which is the opposite of this vignette's presentation. Cluster headache produces much shorter attacks (15-180 minutes), strictly unilateral periorbital pain with prominent ipsilateral autonomic features (lacrimation, ptosis, nasal congestion), which is a distinctly different pattern from the 4-72 hour migraine-type attack described here.

164. B. Tension-type headache is classically described as bilateral, pressing/tightening in quality, mild to moderate in intensity, NOT aggravated by routine activity, and without the prominent nausea or photophobia/phonophobia seen in migraine, matching this vignette's presentation precisely. Migraine with aura would include neurologic aura symptoms preceding the headache and typically features unilateral, pulsatile pain aggravated by activity with associated nausea/photophobia, none of which are described in this vignette. Trigeminal neuralgia produces brief, paroxysmal, electric shock-like facial pain triggered by light touch, an entirely different pain quality, location, and duration from the bilateral, band-like headache described here.

165. C. Cluster headache is defined by severe, strictly unilateral periorbital pain attacks of 15-180 minutes with a characteristic circadian/clustering pattern and prominent ipsilateral cranial autonomic features (lacrimation, conjunctival injection, nasal congestion), matching this vignette's presentation precisely as a trigeminal autonomic cephalalgia. Chronic tension-type headache is bilateral, pressing, and lacks the prominent unilateral autonomic features and short, severe attack pattern described in this vignette. Medication overuse headache is a chronic daily headache pattern related to frequent analgesic use, an entirely different mechanism and clinical picture from the episodic, autonomic-feature-rich cluster headache attacks described here.

166. A. Trigeminal neuralgia is defined by brief, severe, electric shock-like paroxysms of facial pain in a trigeminal nerve distribution, characteristically triggered by innocuous stimuli such as light touch,

chewing, or talking, and lasting only seconds -- exactly matching this vignette. Cluster headache produces longer attacks (15-180 minutes) with prominent periorbital autonomic features, a very different duration and clinical picture from the brief, trigger-provoked shocks of trigeminal neuralgia described here. Temporomandibular disorder typically produces a more constant, aching jaw/facial pain related to jaw movement and joint or muscle dysfunction, not the brief, electric shock-like, touch-triggered pain paroxysms described in this vignette.

167. C. Temporomandibular disorder presents with jaw pain and joint clicking related to movement, along with muscle (masseter/temporalis) and joint tenderness, reflecting a musculoskeletal/myofascial process of the jaw rather than a neuralgic pain syndrome, matching this vignette's presentation. Trigeminal neuralgia produces brief, electric shock-like pain paroxysms triggered by light touch, which is explicitly stated to be absent in this vignette, ruling it out. Cluster headache produces unilateral periorbital pain with autonomic features, an entirely different anatomic location and clinical pattern from the jaw joint/muscle pain with movement-related clicking described here.

168. A. Medication overuse headache develops when frequent use of acute headache medications (particularly combination analgesics) paradoxically drives a chronic, progressively more frequent, and treatment-resistant headache pattern in a patient with an underlying primary headache disorder like migraine -- exactly matching this vignette's history and progression. This presentation is not a new, unrelated cluster headache; cluster headache has a distinct unilateral, autonomic-feature-rich, episodic pattern rather than the chronic daily pattern tied to frequent analgesic use described here. The vignette explicitly links this patient's worsening headache pattern to frequent analgesic use in the setting of a prior migraine history, making it inaccurate to describe this as an unrelated, incidental headache disconnected from that medication use pattern.

169. B. Ectopic firing -- spontaneous, abnormal electrical discharges arising directly from an injured nerve segment, independent of any external stimulus -- is a well-established peripheral mechanism contributing to neuropathic pain generation, matching this vignette's electrophysiologic findings exactly. This is explicitly described as an ABNORMAL discharge pattern arising specifically from the injury site, which is the opposite of normal, expected background nerve firing in an uninjured nerve. The vignette describes an objective, measurable electrophysiologic abnormality (spontaneous discharges on nerve testing), which directly contradicts describing this as a purely psychogenic process with no underlying physiologic finding.

170. C. Postherpetic neuralgia is defined as neuropathic pain persisting beyond approximately 3 months after resolution of the herpes zoster rash, matching this vignette's more than 4-month duration since rash resolution in the same dermatomal distribution. Acute herpes zoster pain refers specifically to pain occurring during the active rash phase, which does not describe this patient's presentation since the rash has already fully resolved for several months. There is no standard subacute herpetic neuralgia category defined by exactly 30 days; the recognized clinical distinction is between acute zoster pain and postherpetic neuralgia based on the roughly 3-month persistence threshold, not an exact 30-day cutoff.

171. A. Diabetic peripheral neuropathy classically presents as a distal, symmetric, length-dependent sensorimotor polyneuropathy, beginning in the longest nerve fibers (the toes/feet) and gradually ascending, with hands becoming involved later as the disease progresses -- exactly matching this vignette's presentation. This vignette describes a bilateral, symmetric, gradually progressive process, not an acute, asymmetric single-nerve mononeuropathy, making that option an inconsistent pattern for the presentation described. The vignette explicitly describes distal (toe/foot) onset, which is the opposite of a proximal-predominant pattern sparing the distal extremities, making that option inconsistent with the classic length-dependent diabetic neuropathy pattern being described.

172. B. CRPS Type I is specifically defined by this clinical presentation occurring WITHOUT a confirmed major nerve injury (as in this vignette's minor sprain with normal electrodiagnostic testing), distinguishing it from Type II. CRPS Type II is, by definition, associated with a confirmed major nerve injury, which is explicitly absent in this vignette (normal electrodiagnostic testing), making this the incorrect subtype for this presentation. CRPS is, in fact, well recognized as being subclassified into Type I and Type II based specifically on the presence or absence of a confirmed nerve injury, making the claim that no such subclassification exists factually incorrect.

173. B. The Budapest criteria are the internationally accepted, validated diagnostic criteria for CRPS, requiring reported symptoms and corresponding objective signs across defined categories (sensory, vasomotor, sudomotor/edema, motor/trophic), matching the assessment described in this vignette exactly. The ACR 2016 fibromyalgia criteria are a distinct diagnostic tool for a different condition (fibromyalgia), using a Widespread Pain Index and Symptom Severity Scale, not the CRPS-specific categories described here. Mirels' criteria are used to assess pathologic fracture risk in bone metastases, an entirely unrelated orthopedic oncology scoring system with no role in CRPS diagnosis.

174. A. Phantom limb pain is defined as pain perceived in the absent limb after amputation, and a proposed contributing mechanism involves cortical reorganization/remapping of the sensory cortex following deafferentation of the amputated body part's input -- correctly matching both the phenomenon and its proposed mechanism described in this vignette. Residual limb (stump) pain refers to pain localized to the actual remaining surgical site itself, which is a distinct entity from pain perceived in the absent limb, and the vignette specifies the surgical site has fully healed, arguing against ongoing local wound inflammation as the explanation. There is no anatomic or physiologic basis linking a below-knee amputation phantom pain presentation to an unrelated abdominal visceral referred pain process, making this option irrelevant to the scenario described.

175. A. Carpal tunnel syndrome, from median nerve entrapment at the wrist, classically presents with numbness/tingling in the thumb, index, and middle fingers, worse at night, and reproduced by a positive Tinel sign (tapping the volar wrist) and Phalen maneuver (sustained wrist flexion) -- exactly matching this vignette. Cubital tunnel syndrome from ulnar nerve entrapment at the elbow would produce numbness in the ring and small fingers, not the thumb/index/middle finger distribution described, and would be reproduced by different provocative maneuvers at the elbow rather than the wrist. Radial nerve entrapment at the spiral groove typically causes wrist drop and dorsal hand numbness (motor-

predominant), not the specific volar sensory distribution and wrist-based provocative findings described in this vignette.

176. B. Cubital tunnel syndrome, from ulnar nerve entrapment at the elbow, classically presents with numbness/tingling in the ring and small fingers, worse with prolonged elbow flexion, and reproduced by tapping (Tinel sign) over the medial elbow at the cubital tunnel -- exactly matching this vignette. Carpal tunnel syndrome from median nerve entrapment at the wrist would produce thumb/index/middle finger symptoms with wrist-based provocative testing, not the ring/small finger, elbow-provoked pattern described here. Musculocutaneous nerve entrapment is a much rarer and different entity producing lateral forearm sensory changes and biceps weakness, not the ring/small finger sensory pattern and elbow flexion-provoked symptoms described in this vignette.

177. C. Neurapraxia is the mildest Seddon classification category, involving focal demyelination and a temporary conduction block without axonal disruption, typically resolving within days to weeks, exactly matching this vignette's description. Neurotmesis represents the most severe injury, complete transection of the nerve including its connective tissue sheaths, which would not resolve spontaneously within a few weeks as described here. Axonotmesis involves actual axonal disruption with relatively preserved surrounding connective tissue (Schwann cell tubes/endoneurium), a more significant injury than the pure demyelinating conduction block without axonal disruption described in this vignette.

178. C. Central post-stroke pain, classically associated with thalamic lesions, presents with burning, allodynic pain corresponding to the sensory deficit distribution from the stroke, reflecting direct central nervous system injury to somatosensory pathways -- exactly matching this vignette's presentation and timing relative to the stroke. This vignette explicitly ties the pain onset to a thalamic stroke, making an unrelated diabetic peripheral neuropathy an inconsistent and less parsimonious explanation than a mechanism directly connected to the described central lesion. While CRPS can occasionally occur in a stroke-affected limb, the vignette's description of pain distribution corresponding directly to the stroke's sensory deficit is more classically and specifically consistent with central post-stroke (thalamic) pain than with CRPS.

179. B. Spinal cord injury is well recognized to produce both at-level neuropathic pain (a band-like distribution near the level of injury) and below-level neuropathic pain (more diffuse pain below the injury, often in areas of reduced sensation), both reflecting central neuropathic pain mechanisms directly related to the cord injury -- exactly matching this vignette. This presentation is explicitly and directly tied to the described spinal cord injury, making an unrelated peripheral entrapment neuropathy an inconsistent and unsupported alternative explanation. There is no indication of an abdominal visceral process in this vignette, and the described pain pattern (at-level band-like and below-level diffuse pain) is the classic, well-recognized SCI neuropathic pain pattern rather than a referred visceral phenomenon.

180. C. Meralgia paresthetica results from entrapment of the purely sensory lateral femoral cutaneous nerve, typically under the inguinal ligament, producing burning/tingling pain and numbness over the anterolateral thigh without motor involvement, and is classically associated with obesity and tight

clothing at the waist -- exactly matching this vignette. Femoral nerve entrapment would be expected to cause quadriceps weakness and a diminished patellar reflex (since the femoral nerve has a motor component), which is explicitly absent in this vignette's presentation. Sciatic nerve entrapment would produce posterior thigh and lower leg symptoms in a very different distribution from the anterolateral thigh sensory pattern described in this vignette.

181. B. Tarsal tunnel syndrome results from posterior tibial nerve entrapment as it passes posterior and inferior to the medial malleolus, producing plantar foot burning pain, numbness, and tingling reproduced by tapping at that specific anatomic location -- exactly matching this vignette. Meralgia paresthetica affects the anterolateral thigh, an entirely different anatomic location from the plantar foot symptoms and medial ankle provocative test site described in this vignette. Common peroneal nerve entrapment at the fibular head would classically cause foot drop and dorsal/lateral foot or lower leg sensory changes, not the plantar foot symptoms and medial malleolar provocative findings described here.

182. A. Small fiber neuropathy selectively affects thinly myelinated A-delta and unmyelinated C fibers responsible for pain and temperature sensation, and because standard EMG/NCS primarily assesses larger myelinated fibers, these studies are often normal in small fiber neuropathy, requiring a skin biopsy showing reduced intraepidermal nerve fiber density for diagnosis -- exactly matching this vignette. Large fiber neuropathy, by definition, would be expected to show abnormalities on standard EMG/NCS (which specifically assesses large fiber function), which directly contradicts this vignette's normal EMG/NCS findings. A reduced intraepidermal nerve fiber density on skin biopsy is an abnormal, diagnostically significant finding, not a normal result requiring no further workup, making that option incorrect.

183. B. Allodynia is specifically defined as pain resulting from a stimulus that does not normally provoke pain (such as light touch), while hyperalgesia is an exaggerated pain response to a stimulus that is normally mildly painful (such as pinprick), so these two distinct terms correctly and separately describe the two phenomena in this vignette. These terms are NOT interchangeable; they refer to distinct clinical phenomena defined by different baseline stimulus types (non-painful versus normally painful), so describing them as interchangeable is incorrect. Paresthesia refers to an abnormal, typically non-painful sensation (such as tingling or a pins-and-needles feeling), which is a different concept from the pain-amplification phenomena of allodynia and hyperalgesia being described in this vignette.

184. C. Alcohol-related peripheral neuropathy results from a combination of direct ethanol neurotoxicity and commonly co-occurring thiamine (vitamin B1) deficiency, related to poor nutritional intake and impaired absorption in chronic heavy alcohol users, which is the correct, well-established contributing nutritional factor. Vitamin D deficiency is not the recognized primary nutritional contributor to alcohol-related peripheral neuropathy; its deficiency is more classically associated with bone health issues, not this specific neuropathy mechanism. Vitamin C deficiency (scurvy) is not the recognized primary nutritional contributor to alcohol-related peripheral neuropathy either; thiamine deficiency is the specific, well-documented nutritional factor relevant to this condition.

185. A. Acute vaso-occlusive crisis pain in sickle cell disease is recognized as severe and requires prompt, aggressive analgesia, typically including parenteral opioids administered without unnecessary delay, since delayed treatment is associated with worse outcomes and is a recognized quality-of-care concern in this population. Withholding opioids due to stigma-driven concern about misuse is a recognized and harmful care disparity in sickle cell disease management, not an appropriate or evidence-based approach to acute severe vaso-occlusive pain. NSAIDs alone are inadequate for severe vaso-occlusive crisis pain, and opioids are not contraindicated in sickle cell disease; they are, in fact, a mainstay of acute crisis management alongside other supportive measures.

186. A. NSAIDs inhibit prostaglandin synthesis, and prostaglandins are important for maintaining patency of the fetal ductus arteriosus; third-trimester NSAID use is associated with risk of premature ductal closure and oligohydramnios, which is the correct, physiologically grounded reason for avoiding them at this stage. This is a genuine, well-documented physiologic risk (not an unfounded historical concern), which is precisely why regulatory guidance specifically restricts NSAID use in the third trimester. Neural tube defects are associated with first-trimester exposures to certain teratogens (such as some antiepileptic drugs), not with third-trimester NSAID use, making this option an inaccurate timing and mechanism for the described risk.

187. C. Current perioperative guidance has shifted toward generally continuing buprenorphine through the perioperative period in most cases, supplementing with multimodal analgesia and additional full agonist opioids as needed under specialist guidance, rather than the older practice of routine discontinuation, since continuation better protects against relapse and undertreated pain. The blanket, no-exception discontinuation approach described does not reflect current, more nuanced guidance, which favors individualized, specialist-guided perioperative management rather than a universal mandatory stop. Buprenorphine does have real analgesic (partial agonist) properties at the mu-opioid receptor, so describing it as irrelevant to perioperative pain planning is incorrect and ignores its actual pharmacology.

188. A. Observational behavioral pain assessment tools such as PAINAD (Pain Assessment in Advanced Dementia) are specifically designed for nonverbal patients with advanced dementia, scoring observable indicators like breathing, vocalization, facial expression, body language, and consolability rather than requiring self-report. The McGill Pain Questionnaire requires the patient to understand and select descriptive words about their pain, which is not feasible in a patient with advanced dementia who cannot reliably self-report. There is no validated, standalone laboratory biomarker test for pain intensity; pain assessment in this population relies on validated behavioral observation tools, not lab-based biomarkers.

189. A. The "start low, go slow" principle reflects well-documented age-related changes in pharmacokinetics (reduced renal/hepatic clearance) and pharmacodynamics (increased receptor sensitivity) in older adults, which increase the risk of drug accumulation and adverse effects at doses that would be routine in younger patients -- the correct rationale described in this vignette. Elderly patients generally require LOWER, not higher, starting doses due to increased sensitivity and reduced

clearance, making this option the opposite of correct, evidence-based geriatric prescribing practice. Age does, in fact, have well-documented, clinically significant effects on drug pharmacokinetics and pharmacodynamics, so claiming it has no meaningful effect directly contradicts the basis for age-adjusted dosing in geriatric pain management.

190. B. Children have body composition, volume of distribution, and drug clearance characteristics that differ substantially from adults relative to body size, so weight-based dosing is a pharmacologically necessary approach to achieve safe and effective analgesic dosing across the wide range of pediatric body sizes. Weight-based dosing reflects genuine pharmacokinetic principles, not merely a billing convention, and is a core, clinically necessary safety practice in pediatric medication dosing, not an administrative artifact. Weight-based dosing is a general pharmacologic principle applied broadly across many pediatric drug classes, including analgesics, not a practice limited only to antibiotics.

191. B. Not all spinal cord stimulator systems are MRI-safe under all conditions; the specific device and lead system must be confirmed as MRI-conditional, and imaging must follow the manufacturer's specific approved conditions (such as field strength and body region) to avoid heating, device malfunction, or patient injury. It is incorrect to state that all spinal cord stimulators are universally MRI-safe under any conditions; MRI compatibility varies significantly by specific device and requires verification, not an assumption of universal safety. Many implanted neuromodulation devices do contain metallic/conductive components that create real MRI safety considerations (such as heating or device malfunction), making the claim that MRI safety is entirely irrelevant factually incorrect.

192. C. In significant hepatic impairment, reduced metabolic capacity can lead to accumulation of hepatically metabolized analgesics, so dose reduction, extended intervals, and a lower total daily acetaminophen limit are often appropriate to avoid toxicity -- the correct, clinically important consideration for this population. Hepatic impairment does have a clinically meaningful effect on the metabolism of many opioids and on acetaminophen, since the liver is a primary site of their metabolism, making the claim of no meaningful effect incorrect. Using maximum standard acetaminophen doses without adjustment in cirrhosis is inappropriate and increases hepatotoxicity risk in a patient whose liver already has reduced metabolic reserve, making this option unsafe rather than correct.

193. B. Morphine is metabolized to morphine-6-glucuronide, an active metabolite that is renally cleared and can accumulate in renal impairment, causing sedation, myoclonus, and respiratory depression, which is the classic and correct concern described in this vignette. Fentanyl is primarily metabolized hepatically to largely inactive metabolites and does not have the same clinically significant renally-cleared active metabolite accumulation concern as morphine, making it generally preferred (not avoided) in renal impairment. Methadone is predominantly eliminated via hepatic metabolism and fecal excretion rather than renal clearance, so describing it as primarily renally eliminated is inaccurate, and this is not the classic renal-accumulation concern being described in this vignette.

194. B. Neonatal abstinence syndrome (neonatal opioid withdrawal syndrome) results from in utero opioid exposure followed by postnatal withdrawal, presenting with irritability, high-pitched crying,

tremors, and feeding difficulties, exactly matching this vignette's presentation and history. Opioids are not classically recognized as major teratogens causing structural congenital malformations in the way this option implies; the described presentation is a withdrawal syndrome, not a structural birth defect. While withdrawal can include tremors and irritability, this presentation is best explained by neonatal abstinence syndrome directly related to the described opioid exposure history, rather than by an unrelated, coincidental primary seizure disorder.

195. C. The appropriate approach balances legitimate acute pain treatment with careful, structured management -- using multimodal and opioid-sparing strategies where possible, and if opioids are needed, careful dosing with close monitoring -- rather than either extreme of automatic withholding or treating with no additional structure, which is the correct, nuanced answer. Automatically withholding all opioids based solely on a substance use disorder diagnosis, regardless of the severity of a genuine acute painful condition like a fracture, risks undertreating legitimate pain and is not the recommended approach. Providing treatment identical to any other patient with absolutely no additional monitoring or structure ignores the genuine, elevated risk considerations relevant to a patient with an active substance use disorder, which is why some additional structure and monitoring is appropriate.

196. A. PTSD and chronic pain are well documented to frequently co-occur, particularly in veteran populations, sharing overlapping neurobiological and psychological mechanisms, with each condition capable of worsening the severity and treatment resistance of the other -- the accurate, evidence-based characterization of this relationship. Describing the co-occurrence as purely coincidental with no established relationship directly contradicts a substantial body of literature documenting shared mechanisms and mutual symptom amplification between these two conditions. It is inaccurate to claim PTSD always fully resolves simply by treating the comorbid pain condition; PTSD generally requires its own independent, trauma-focused treatment, not treatment of pain alone as a proxy.

197. C. Increased soft tissue depth in patients with severe obesity can require longer needles, adjusted imaging technique (such as higher fluoroscopic exposure settings or specific ultrasound depth/frequency adjustments), and more careful, deliberate anatomic landmark identification to safely and accurately reach the intended target -- the correct, clinically important procedural consideration. Obesity does, in fact, meaningfully impact interventional technique and imaging quality due to increased tissue depth and altered landmark palpability, making the claim of no impact incorrect. Standard-length needles may be inadequate to reach deep targets in patients with significant obesity, so assuming they are always adequate regardless of body habitus risks a failed or unsafe procedure attempt.

198. A. Carefully titrated, low-dose opioids are recognized as safe and effective for relieving both pain and dyspnea in end-stage non-cancer illness such as advanced COPD, following the same proportionality principles (matching dose to symptom severity, following the ethical principle of double effect) used broadly in palliative care regardless of the underlying diagnosis. It is incorrect to state opioids should never be used in non-cancer end-of-life conditions; this contradicts well-established palliative care practice extending opioid symptom management to serious non-cancer illnesses like advanced COPD and heart failure. Opioid use for dyspnea and pain at the end of life is not restricted to

patients with a concurrent cancer diagnosis; the same palliative principles apply to appropriate non-cancer serious illness, making this restriction incorrect.

199. B. Tramadol has serotonin reuptake inhibition activity in addition to weak opioid agonism, and combining it with an SSRI can produce serotonin syndrome, presenting with agitation, hyperreflexia, clonus, tremor, and hyperthermia -- exactly the presentation and correct mechanism described in this vignette. A simple opioid overdose would be expected to present with sedation, respiratory depression, and miosis, not the hyperreflexia, clonus, and hyperthermia pattern described, which is characteristic of serotonin excess rather than opioid toxicity. Opioid withdrawal would present with symptoms such as anxiety, piloerection, diarrhea, and yawning, not the hyperreflexia, clonus, and hyperthermia described, which do not fit a withdrawal syndrome presentation.

200. C. When used as medication for opioid use disorder, buprenorphine's primary treatment goal is to reduce cravings and withdrawal symptoms, blunt the euphoric effects of illicit opioid use through its partial agonist/high-affinity properties, and support sustained long-term recovery -- the correct goal distinguishing MOUD use from its use purely as an analgesic. In the MOUD context described, the primary goal is addiction treatment and recovery support, not maximal analgesia for an acute pain condition, which is a different clinical indication for the same medication. Buprenorphine is used in MOUD for long-term maintenance treatment, not merely as a short-term detoxification-only agent with no longer-term maintenance role, making this option an inaccurate description of its treatment goal here.