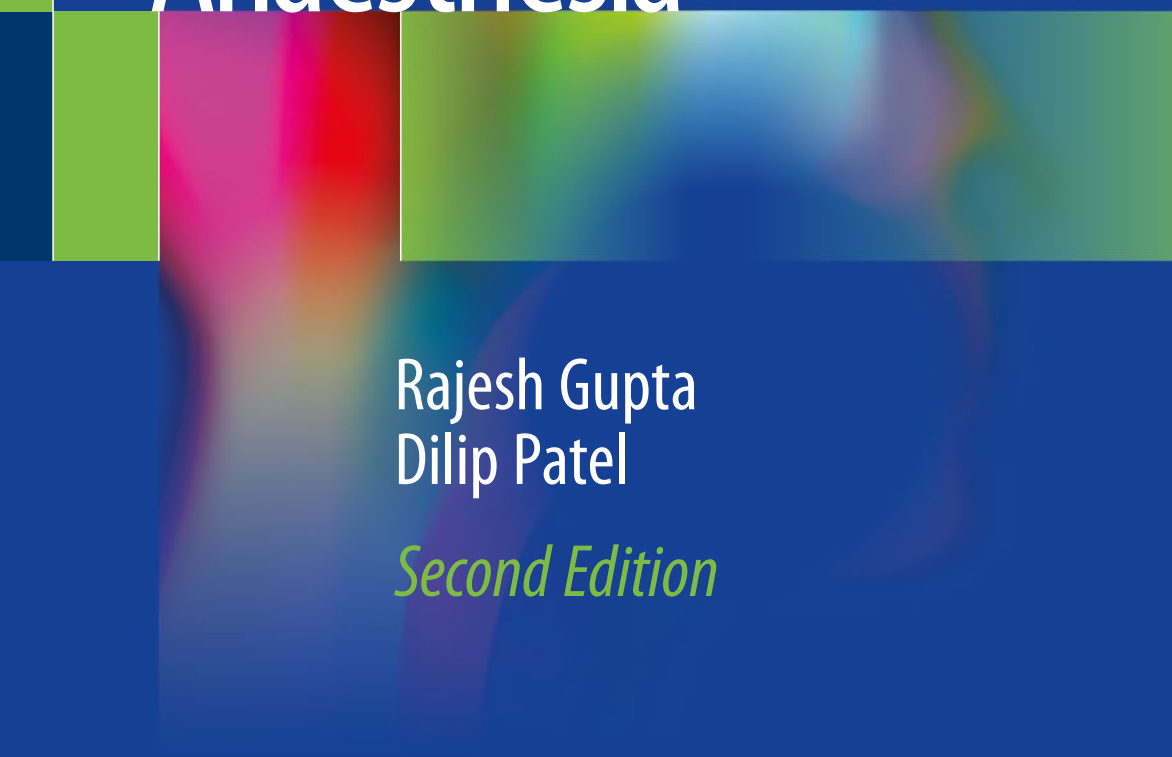
Multiple Choice Questions in Regional Anaesthesia



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Dilip Patel

Second Edition

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Dedicated to my parents
Rajesh Gupta

Contents

Anatomy and Physiology of Acute Pain

1

1. Characteristics of acute pain:
   (a) Acute pain is associated with temporal reduction in intensity.
   (b) Acute pain serves no adaptive purpose.
   (c) Inflammatory pain is classified under nociceptive pain.
   (d) Visceral pain does not radiate in dermatomal pattern.
   (e) Primary hyperalgesia is seen at the site of injury.
2. Gate control theory of pain:
   (a) Sensory fibres stimulate second order spinal neurons.
   (b) Both large and small diameter afferents can activate transmission cells in the dorsal horn.
   (c) Substantia gelatinosa regulates the gate.
   (d) Increased activity in small diameter fibres increases the suppressive effect of substantia gelatinosa cells.
   (e) Central sensitisation within the substantia gelatinosa unlocks the dorsal horn gate and facilitates transmission.
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   (a) Direct nociceptive activators cause transduction.
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   (c) Peripheral sensitisation causes primary allodynia and primary hyperalgesia.
   (d) Secondary sensitisation has no role in neuropathic pain.
   (e) "Wind up" phenomenon relates to increased postsynaptic response to central input.
4. Transduction seen in pain:
   (a) Involves supra-spinal mechanisms
   (b) Calcium channels are involved.
   (c) Primary hyperalgesia is associated with potassium currents.
   (d) CGRP is involved with mechanical and thermal hyperalgesia.
   (e) Increased IL-β results in allodynia.

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5. Conduction in pain:
   (a) Is transfer of action potential from peripheral nociceptive endings via nerve fibers.
   (b) Aβ fibers are non-noxious.
   (c) Initial response to pain is by C fibers.
   (d) Axonal conduction results in release of excitatory amino acids.
   (e) Sodium channels play a major part.
6. Characteristics of pain transmission:
   (a) It is the transfer of noxious impulses from primary nociceptors to cells in the spinal cord dorsal horn.
   (b) Wide dynamic range neurons respond only to noxious stimuli.
   (c) Excitatory amino acids are involved.
   (d) Both AMPA and KAR receptors initiate voltage mediated priming of NMDA receptors.
   (e) Increased prostaglandin E in extracellular and intracellular area is responsible for transcription dependant central sensitisation.
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   (b) It is mediated by endogenous analgesic compounds.
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   (d) Modulatory effects of norepinephrine are mediated by polysynaptic alpha adrenergic receptors.
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   (a) Thalamocortical connections are responsible for sensory qualities (throbbing or burning).
   (b) Limbic system is associated with persistent pain.
   (c) Frontal cortex has a role in learned avoidance.
   (d) Insular cortex is primarily responsible for acute noxious stimulation.
   (e) Opioid induced metabolic suppression involves ipsilateral thalamus and amygdala.
9. Transition from acute to chronic pain:
   (a) Central sensitisation involves activation of NMDA receptors.
   (b) Transcription independent sensitisation can be seen following trauma.
   (c) Downregulation of AMPA receptors can lead to extended pain stimulus.
   (d) Wind up phenomenon is reversible.
   (e) Transcription dependent sensitisation involves alterations in dorsal root ganglion.
10. Peripheral sensitisation:
   (a) Can be increased by increasing efficacy of transducing ion channels.
   (b) Voltage gated ion channels are not involved in sensitisation.
   (c) Neurogenic oedema is contributed by decrease in substance P.
   (d) Extracellular signal regulated kinase is involved in receptor mediated hypersensitivity.
   (e) Mainly involves Aδ and C fibers.

11. Hyperalgesia;
    (a) Is a part of the triple response in acute injury.
    (b) Primary hyperalgesia is due to increased sensitivity of Aβ receptors.
    (c) Allodynia is not mediated by interleukins.
    (d) Secondary hyperalgesia is seen at the spinal level.
    (e) Secondary hyperalgesia is antagonised by inhalational anaesthetics or parenteral opioids.
12. Sympatho-adrenal response to acute injury:
    (a) Manifests as three different stages.
    (b) Highest elevations of sympathetic amines are seen in elderly.
    (c) May be deleterious in coronary artery disease.
    (d) Increased muscle spasms may be seen.
    (e) Hypercoagulation may be seen.
13. Neuroendocrine response to acute injury:
    (a) Increase in anabolic steroids are seen.
    (b) Increased incidence of infections is seen.
    (c) Neuroendocrine response is by decrease in interleukins.
    (d) Immunoglobulin synthesis may decrease.
    (e) Shock may be initiated by β-endorphin.
14. Effect of injury to target organs:
    (a) Perioperative ischaemia mostly occurs within 24 h.
    (b) Myocardial oxygen requirements are decreased.
    (c) Pain following operation on upper abdomen and thoracic musculature is effort dependent.
    (d) Surgically induced pain may cause pulmonary complications in 70% of patients.
    (e) Decrease in functional residual capacity is associated with increase in shunt.
15. Effect of injury to target organs:
    (a) There is increased incidence of deep venous thrombosis and pulmonary embolism.
    (b) Continued alterations in regional blood flow result in sympathetic dystrophy.
    (c) Activation of microglia and neuronal apoptosis may contribute to plastic changes.
    (d) Pain at site of surgery predisposes to persistent pain.
    (e) Limbic cortical response is associated with anxiety and depression.
16. Patient variables influencing acute pain management:
    (a) Advancing age can increase toxicity of opioid administration.
    (b) Visual analogue scale is most effective for detecting age differences in post-operative pain.
    (c) Patient controlled analgesia can be used in paediatric patients as young as 4 years for post-operative pain.
    (d) Ethnicity plays a major role in analgesic response to patient controlled analgesia.
    (e) Females experience more pain in immediate post-operative period than males.

17. Variables affecting acute pain management:
    (a) Patients with passive coping styles consume more morphine.
    (b) Age is an independent risk factor for early post-operative pain.
    (c) Superficial procedures are less painful.
    (d) Pre-emptive analgesia is beneficial even if used only in preoperative period.
    (e) PCA morphine dose is based on body weight.
18. Variables influencing acute pain management:
    (a) Low levels of CSF β endorphin predict a high requirement for postoperative PCA.
    (b) Females respond better to morphine than males in postoperative period.
    (c) Activity of CYP2D6 enzyme is responsible for variations in metabolism for dextromethorphan, tramadol and codeine.
    (d) Buprenorphine is the opioid of choice in renal failure patients.
    (e) Morphine is the only opioid which is safe in liver failure patients.
19. Psychosocial factor associated with acute pain:
    (a) Anxiety has least effect on postoperative pain as compared to depression and anger.
    (b) Pain anxiety symptom scale is validated for acute postoperative pain prediction.
    (c) Kinesiophobia increases the risk of postoperative pain.
    (d) Pain catastrophising increases the incidence of postoperative pain.
    (e) Distraction may help decrease distress in persons experiencing acute pain.
20. Psychological interventions for acute pain:
    (a) Distraction works better in children than adult population.
    (b) Distraction is better than local anaesthetics in managing pain on injections.
    (c) Cognitive behavioural therapy has no role in acute pain.
    (d) Hypnosis can cause reduction in acute pain.
    (e) Virtual reality is effective in acute pain management.

Answers

1. T F T F T
   Acute pain has a protective function as opposed to chronic pain which serves no adaptive purpose. Nociceptive pain is defined as noxious perception resulting from cellular damage following surgical, traumatic or disease related injury. Visceral pain radiating in a particular dermatomal pattern is known as referred pain. It is due to convergence of noxious input from visceral afferents activating second order cells that are normally responsive to somatic sensation.

   Treede RD, Meyer RA, Raja SN, et al. Peripheral and central mechanisms of cutaneous hyperalgesia. Prog Neurobiol. 1992;38(4):397–421.
2. T T T F T
   Sensory fibres stimulate dorsal horn transmission cells or wide dynamic range neurons. Large sensory fibres can activate inhibitory substantia gelatinosa cells.

Increased activity in small diameter fibres decreases the suppressive effect of substantia gelatinosa cells and opens the gate. Peripheral nerve injuries also open the gate by increase small fiber activity and decrease large fiber inhibition.

Melzack R, Wall PD. Pin mechanism: a new theory. Science. 1965;150(699):971–9.

L- light touch mechanoreceptors, S-small diameter unmyelinated pain fibers, SG- substantia

Gelatinosa, T- wide dynamic range neurons

3. T F T F T

Direct activators like potassium, hydrogen ions, ATP and bradykinin causes transduction at peripheral nociceptor ion channel receptors. Nociceptor sensitizers include PGE2, nerve growth factor, bradykinin. They decrease the threshold of activation of ion channel receptors on nociceptor terminals. Secondary sensitisation plays a major role in inflammatory and neuropathic pain. Repetitive stimulus of unmyelinated C fibres can result in prolonged discharge of dorsal horn cells causing wind up.

4. F T T T T

Transduction is the response of peripheral nociceptors to noxious stimuli. Noxious stimuli are converted into a calcium ion mediated electrical depolarisation. Cellular damage is associated with release of intracellular hydrogen and potassium ions. Receptor G-protein complex strengthens inward sodium flux and weakens potassium currents and increased nociceptor excitability causing primary hyperalgesia. Calcitonin gene related protein is 37 amino acid peptide found in the peripheral and central terminals of more than 50% of c fibers and 35% of Aδ fibers.

Ji RR, Woolf CJ. Neuronal plasticity and signal transduction in nociceptive neurons: implications for the initiation and maintenance of pathological pain. Neurolobiol Dis. 2001;8(1):1–10.

5. T T F T F

Largest diameter fibers Aβ and are myelinated. The conduction velocity is 30–50 m/s. Aδ fibers transmit the pain initially, are thinly myelinated with a conduction velocity of 5–25 m/s C fibers are unmyelinated, have a delayed latency and with conduction velocity of <2 m/s. neuronal type calcium channels are in nerve endings and causes a rapid influx of calcium when stimulated.

Fiber type	Function	Diameter (μm)	Myelination	Conduction velocity (m/s)
Type A				
Alpha (α)	Proprioception, motor	12–20	Heavy	70–120
Beta (β)	Touch, pressure	5–12	Heavy	30–70
Gamma (γ)	Muscle spindles	3–6	Heavy	15–30
Delta (δ)	Pain, temperature	2–5	Heavy	12–30
Type B	Preganglionic autonomic	<3	Light	3–15
Type C				
Dorsal root	Pain	0.4–12	None	0.5–2.3
Sympathetic	Postganglionic	0.3–1.3	None	0.7–2.3

6. T F T T T
Second order neurons are of two types- nociceptive specific neurons located in lamina 1 and respond only to noxious stimuli and wide dynamic range neurons which are present in lamina V and respond to both noxious and non-noxious stimuli. Glutamate aspartate is an excitatory amino acid and activates AMPA (ionotropic amino-3-hydroxyl-5 methyl-4 propionic acid) and kainite receptors. NMDA activation, wind up and central sensitisation are responsible for clinical hyperalgesia. Increased intracellular and extracellular prostaglandin E and nitric oxide are responsible for central sensitisation.

Woolf CJ. An overview of the mechanisms of hyperalgesia. Pulm Pharmacol. 1995;8(4–5):161–7.

7. T T T T T
Modulation is mediated by the inhibitory action of endogenous analgesic compounds (enkephalins, norepinephrine, GABA) released from spinal interneurons and terminal endings of inhibitory areas from locus ceruleous. Balance between excitatory mediators and the inhibitory effects of endogenous analgesics adjusts potassium ion flux and firing frequency of dorsal horn cells.

8. T T T F T
Projections from limbic cortex activates motor cortex, hypothalamus and pituitary gland mediating persistent pain. Frontal cortex and amygdala mediate fear, anxiety, helplessness, learned avoidance associated with acute pain. Primary somatosensory cortex is mainly responsible for acute noxious stimuli while insular cortex is responsible for pain anticipation. Increased blood flow in the parietoinsular cortex corresponds to the physical sensation of pain and its intensity (pain thresholds).

Besson JM. The neurobiology of pain. Lancet. 1999;353(9164):1610–5.

9. T T F T T
Central sensitisation requires activation of spinal and supra-spinal NMDA receptors and increased intraneuronal calcium ion influx. It is divided into transcription dependent and independent and later represents neurochemical and electrical alterations seen in trauma. Upregulation of AMPA receptors leads to increase in synaptic efficacy and firing rate of dorsal horn cells. This leads to tactile allodynia which outlasts the conditioning stimulus for hours e.g. sunburn. Wind up is a form of transcription independent central sensitisation that is rapid and reversible.

Rygh LJ, Svendsen F, Fiska A. long term potentiation in spinal nociceptive systems. How acute pain may become chronic. Psychoneuroendocrinology. 2005;30(10):959–64.

10. T F F T T
Transducing ion channels primarily determine response specificity. Sensitisation occurs by modifying voltage gated channels to reduce firing thresholds and increase the response to supra-threshold stimuli. Increase in substance P and calcitonin gene related peptide (CGRP) causes neurogenic oedema (redness, warmth, oedema and pain). Thermal hyperalgesia is attenuated by ERK inhibitors.

Bhave G, Gereau 4th RW. Posttranslational mechanisms of peripheral sensitisation. J Neurobiol. 2004;61:88–106.

11. T F F F F

Acute surgical traumatic injury may cause increased blood flow (flare), tissue oedema (wheal), sensitisation of peripheral nociceptors (hyperalgesia). Hyperalgesia refers to altered state of sensibility in which intensity of discomfort associated with repetitive noxious stimuli is increased. Primary hyperalgesia involves Aδ and C nociceptors. Allodynia refers to painful perception of ordinarily non noxious stimuli e.g. touch and pressure. It is mediated by IL-1β and IL-6. Sensitizers like substance P and noradrenaline are released causing an increase in sensitivity. Secondary hyperalgesia is the adaptive facilitatory change seen in spinal cord, brainstem and limbic system. Secondary hyperalgesia has wide dynamic range neurons exhibiting enhanced sensitivity for prolonged periods. It is not antagonised by inhalational anaesthetics or parenteral opioids.

Raja SN, Meyer RA, Campbell JN. Peripheral mechanisms of somatic pain. Anesthesiology. 1988;68:571–90.

Primary hyperalgesia	Secondary hyperalgesia
Seen 30–60 min after injury	Develops later
Lasts for several hours or days	Shorter duration
Seen in the area of injury	Seen in the area surrounding the injured area
Non-painful stimuli perceived as painful	Pain is more than normal
Mechanism	
Decreased pain threshold	Convergence facilitation

12. T F T T T

Sympatho-adrenal response manifests in three stages. Initial stage is flight/fight reaction which allows rapid withdrawal from the traumatic event. It is followed by "resistance stage" which maintains blood flow to critical areas. The third is the "exhaustion stage" which limits mobility and improve tissue repair. Sympathetic amines are increased following extensive procedures and in younger individuals. Sympatho-adrenal response may increase oxygen requirements and may cause worsening of coronary artery disease. The response decreases microcirculatory blood flow to non-essential areas causing impaired wound healing, increased visceral spasm, visceral/somatic ischaemia and acidosis. Platelet activation may be increased and may accelerate coagulation.

Breslow MJ. Neuroendocrine responses to surgery. In: Breslow MJ, Miller CF, Rogers MC, editors. Perioperative management. St. Louis, MO: Mosby; 1990.

13. F T F T T

Increased secretion of catabolic hormones is seen e.g. cortisol, glucagon, growth hormone and catecholamines. Anabolic steroids are decreased like insulin and

testosterone. There is increased tendency for infections because of impaired immunocompetence secondary to decreased Ig synthesis and impaired phagocytosis. Neuroendocrine response is increased by interleukins esp. IL-6 and IL-1β which increases ACTH and cortisol levels. B endorphin is an endogenous opioid neuropeptide. It is an agonist of the opioid receptor with affinity for μ receptors.

Chernow B. Hormonal response to a graded surgical stress. Arch Intern Med. 1987;147:1273–8.

14. F F T T F

Perioperative ischaemia occurs between 1 and 3 days. Poorly controlled pain contributes to it. Oxygen requirements are increased while oxygen supply is decreased. Vital capacity is the first parameter to change (40–60%) after thoracic surgery (within 3 h). Decrease in functional residual capacity is associated with progressive arterial hypoxaemia and increase in functional residual capacity causes improvement in physiological shunt.

Ali J, Weisel RD, Layig AB. Consequences of post-operative alterations in respiratory mechanics. Am J Surg. 1974;128:376–82.

15. T T T T T

Injury causes increased stress response (increased catecholamines and angiotensin) which increases the platelet-fibrinogen activation and increased coagulable state. Pulmonary embolism is associated with 20–30% mortality. Risk factors for persistent pain include pain at the site of injury, young population, psychosocial abnormalities, genetic susceptibilities.

Mannion RJ, Woolf CJ. Pain mechanisms and management: a central perspective. Clin J Pain. 2000;16(suppl):144–56.

16. T F T F T

Plasma levels of albumin are decreased with increase in age which causes increase in fraction of unbound or active drug and may cause toxicity. Average postoperative morphine requirements can be calculated as:

$$24 \text{ hour morphine requirement}(\text{mg}) = 100 - \text{age}(\text{years})$$

Most validated scale for detecting age differences in post-operative pain are McGill pain questionnaire and present pain intensity. VAS has insufficient sensitivity. (*Gagliese L, Weizblit N, Ellis W. the measurement of post-operative pain: a comparison of intensity scales in younger and older surgical patients. Pain. 2005;117:412–20*). PCA can be used in children. (*Marchetti G, Calbi G, Vallani A. PCA in the control of acute and chronic pain in children. Paedr Med Chir. 2000;220:9–13*). Ethnicity plays a major role in response to oral analgesics but not PCA. Females experience more pain than males in post-operative period. (*Aubrun F, Salvi N, Coriat P. Sex and age related differences in morphine requirements for post-operative pain relief. Anesthesiology. 2005;103:156–60*).

17. T T T F F

Highly aggressive and angry patients tend to consume more morphine than passive patients. (*Bachiocco V, Morselli AM, Corli G. Risk factors for early post-operative pain includes age, preoperative neuroticism, sensitivity to cold*

pressure-induced pain. J Pain Symptom Manage. 1993;8:205–14). Thoracotomies, nephrectomies, spinal fusion, upper abdominal surgery, amputation are more painful than herniorrhaphy. Pre-emptive analgesia is not much beneficial if the regional anaesthesia technique is not continued in the postoperative period. (*Soler Company E, Faus Soler M, Montaner Abasolo M, et al. Factors affecting post-operative pain. Rev Esp Anestesiol Reannim. 2001;48:163–70*). Age is a better predictor than weight for PCA morphine dosage. (*Macintyre PE, Jarvis DA. Age is the best predictor of postoperative morphine requirements. Pain. 1995;64:357–64*).

18. T F T T F

Females respond better to nalbuphine (κ opioid agonist) than morphine (μ agonist). (*Gear RW, Miaskowski C, Gordon NC, et al. The kappa opioid nalbuphine produces gender and dose dependent analgesia in patients with postoperative pain. Pain. 1999;83:339–45*). Haemodialysis does not affect buprenorphine levels allowing for stable analgesia and is the drug of choice in renal failure patients. Liver failure mostly affects oxidation while morphine metabolism follows glucronidation which is less affected. Morphine clearance is decreased and oral bioavailability is increased. Methadone is contraindicated in liver failure. (*Tegeder I, Lotsch J, Geisslinger G. Pharmacokinetics of opiods in liver disease. Clin Pharmacokinet. 1999;37:17–40*).

19. F T T T T

Anxiety is the most important factor predicting postoperative pain. State anxiety significantly contributes to the prediction of pain. Pain anxiety symptom scale has four subscales- fear, cognitive anxiety, somatic anxiety, escape/avoidance. (*McCraken LM, Zayfert C, Grass RT. The pain anxiety symptoms scale: development and validation of a scale to measure fear of pain. Pain. 1992;50:67–73*). Kinesiophobia is excessive and irrational fear of movement and injury/reinjury. Catastrophising is tendency to ruminate on and magnify pain sensation and to feel helpless when confronted with pain. Increased activity is seen in brain areas related to anticipation of pain (medial frontal cortex and cerebellum). (*Pavlin DJ, Sullivan MJL, Freund PR. Catastrophising: a risk factor for post-surgical pain. Clin J Pain. 2005;21:83–90*).

20. T T F T T

Distraction produces significant reduction in distress and increase in coping behaviour. (*Cohen LL, Blount RL, Cohen RJ. Comparative study of distraction versus topical anaesthesia for paediatric pain management during immunisations. Health Psychol. 1999;18:591–8*). Cognitive behavioural therapy is effective especially in reducing behavioural distress. Hypnosis causes reduction in acute pain along with decrease in drug usage and haemodynamic stability. (*Long EV, Barbaum KS, Fainluch S, et al. Adjunctive self-hypnotic relaxation for outpatient procedures: a prospective randomised trial with women undergoing large core breast biopsy. Pain. 2006;126:155–64*). Hypnosis causes decrease in involuntary sympathetic response to pain, increase in endogenous opioid release, change in brain activity (anterior cingulate gyrus) and inhibition of pain at spinal cord.

Anatomical and physiological characteristics of nerve fibers:

Nerve type	Function	Diameter	Conduction velocity (m/s)	Myelination	Sensitivity to local anaesthetics
A fibres					
Aα	Motor	12–20	70–120	+++	++
Aβ	Touch, pressure	5–12	30–70	+++	++
Aγ	Proprioception, muscle tone	1–4	15–30	++	+++
Aδ	Pain, temperature	1–4	12–30	++	+++
B fibres	Preganglionic autonomic	1–3	3–15	+	++
C fibres	Postganglionic autonomic, pain, temperature	0.5–1	0.5–2		+

Assessment and Monitoring of Pain

2

1. Acute pain assessment:
   (a) Visual analogue scale is equally effective as numeric rating scale and verbal categorical rating scale (VRS).
   (b) Faces pain scale is well validated.
   (c) Assessment of pain during mobilisation is more effective for pain control than at rest.
   (d) Mechanical allodynia is assessed by von frey filaments.
2. Visual analogue scale:
   (a) Uses a 10 cm line with end point descriptors.
   (b) Measures subjective characteristics or attitudes that can be directly measured.
   (c) Is inferior to likert scale.
   (d) Can be used in the assessment of parameters other than the pain.
   (e) Can be used to compare pain intensity between two individuals.
3. Verbal numerical rating scale:
   (a) Assessment is by a number between 0 and 10.
   (b) Pain intensity is not adequately measured.
   (c) There is low interchangeability between the scales.
   (d) Not useful in language barriers.
   (e) No special instruments are required.
4. Assessment of acute pain:
   (a) VAS is superior than NRS and VRS for assessment of pain intensity.
   (b) The scales only measure patient's subjective feeling of pain intensity.
   (c) Four point VRS underestimates the most intense pain as compared to VAS.
   (d) Faces pain scale can be used in infants.
   (e) Categorical pain scales measure accurately pain intensity.
5. Brief pain inventory:
   (a) Has 9 items in inventory.
   (b) Is of benefit in younger population only.
   (c) Can be used for research purposes.

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(d) Is only used for non cancer pain.
(e) Can be used in patients with disability related pain.

6. Visual analogue scale:
   (a) Is validated for clinical use.
   (b) Is a form of likert scale.
   (c) Easy to administer.
   (d) Scale can only be used face to face.
   (e) Is better than verbal descriptor scale.
7. Numerical rating scale:
   (a) Horizontal VAS may be more useful in elderly population.
   (b) Disadvantage is less psychometric properties.
   (c) NRS is more effective when shown visually along with asking the patient to rate verbally.
   (d) Is not used in people who cannot read or write.
   (e) Gold standard for pain measurement is self reporting.
8. Wong Baker FACES pain rating scale:
   (a) Can be used only in paediatric patients.
   (b) Is of use in patients with cognitively impaired.
   (c) Is based on age, gender and culture.
   (d) May give falsely high scores.
   (e) Revised faces scale was developed for preschool and school going age.
9. Assessment scales for patients who cannot self report:
   (a) Checklist of non verbal indicators has high sensitivity.
   (b) Pain assessment in advanced dementia scale is used in patients with advanced dementia.
   (c) Abbey pain scale measures only acute pain.
   (d) Elderly pain causing assessment 2 measures both persistent and acute pain.
   (e) Mobilisation observation behaviour intensity dementia pain scale is used for those having musculoskeletal pain.
10. Pain assessment in critically ill:
    (a) Endotracheal tube suctioning causes severe pain.
    (b) The most painful procedure in intensive care is turning of the patient.
    (c) Under treatment of procedural pain is common.
    (d) FLACC is of use in critically ill patients.
    (e) Behavioural pain scale (BPS) is better than critical care pain observation tool (CPOT).
11. Pain assessment in intellectually disables patients:
    (a) Intellectual disability is based on IQ measurement.
    (b) Pain thresholds is lower than normal controls.
    (c) Adults with mental retardation have more acute than chronic pain.
    (d) Moaning during manipulation is an indicator of severe pain.
    (e) Pain and discomfort scale (PADS) is highly sensitive.
12. Pain assessment in schizophrenia:
    (a) Insensitivity to pain is common in patients.
    (b) More post operative complications seen than normal population.

(c) Response to experimental pain is diminished.
(d) Pain insensitivity may be seen as a familial trait.
(e) Pain is mostly a part of hallucination.

13. Pain assessment in post traumatic stress disorder:
(a) Symptoms take long time for resolution.
(b) Autonomic instability may be a useful marker of pain.
(c) Early treatment helps in the management.
(d) Patients are less sensitive to heat stimuli.
(e) May be seen with fibromyalgia.

Answers

1. F T T T
Verbal rating scale is less useful. It should be used only as a coarse screening instrument. Four point VRS instrument underestimates intense pain as compared to VAS. (*Breivik EK, Bjornsson GA, Skovland E. A comparison of pain rating scale by sampling from clinical trial data. Clin J Pain. 2000;16:22–8*). Faces pain scale is validated for more than 3 years of age. (*Hicks CL, Von Baeyer CL, Spafford PA, et al. The faces pain scale revised: toward a common metric in paediatric pain measurement. Pain. 2001;(93):173–83*). Von frey filaments are made up of nylon hairs, of the same length but will different diameters to provide different range of forces especially from 0.008 gms force up to 300 gms force.
2. T F F T F
Visual analogue scale uses a 10 cm line with no pain at left end of line and worst pain imaginable marked at the right end. The characteristics cannot be measured directly. VAS have superior material characteristics than discrete scales such as likert scale. (*Grant S, Aitchison T, Henderson E, et al. A comparison of the reproducibility and the sensitivity to change of visual analogue scales, borg scales, and likert scales in normal subjects during submaximal exercise. Chest. 1999;116(5):1208–17*). VAS can be used in assessment of parameters other than pain like assessment of loudness and annoyance of acute and chronic tinnitus. (*Adamchic I, Langguth B, et al. Psychometric evaluation of visual analogue scale for the assessment of chronic tinnitus. Am J Audiol. 2012;21:215–25*).
3. T F T T T
One-dimensional scales are least suited to assess pain intensity.
4. F F T F F
VAS is equally effective as NRS and VRS is least effective. The scales also measure unpleasantness of pain and impact of pain on function. (*Breivik EK, Bjornsson GA, Skovlond E. A comparison of pain rating scales by sampling from clinical trial data. Clin J Pain. 2000;16:22–8*). Faces pain scale can be used for more than 3 years. (*Hicks CL, Von Baeyer CL, Spafford PA, et al. The*

faces pain scale-revised. Toward a common metric in paediatric pain management. Pain. 2001;93:173–83). Categorical scales are good as a coarse screening test, whereas accurate pain assessment is by NRS or VAS.

5. T F T F T
   Brief pain inventory measures pain severity and rates level of pain interference with 7 key areas of function (general activity, mood, walking ability, normal work life, relation with people, sleep and enjoyment of life). Brief pain inventory can be used in elderly population. It can be used for cancer pain, cardiac surgery, traumatic stress, diabetic neuropathy. (*Evdemoglu AK, Koc R. Brief pain inventory score identifying and discriminatory neuropathic and nociceptive pain. Acta Neurol Scand. 2013;128(5):351–58*).
6. F T T T F
   Visual analogue scale is validated for research only and not for clinical use. Though it is easy to administer, the scoring is time consuming and takes time. Patients make more mistakes with visual analogue scale (*Peters ML, Patijin J, Lame I. Pain assessment in younger and older pain patients: psychometric properties and patient preference of five commonly used measures of pain intensity. Pain Med. 2007;8(7):601–10*).
7. F F T F T
   There are two types of VAS—horizontal and vertical. Vertical VAS is more useful in patients with narrowed visual field, elderly, those having difficulties with horizontal scale. Numerical rating scale has good psychometric properties as compared to verbal descriptor scale, horizontal VAS, vertical VAS. NRS has higher reliability in illiterate patients when compared to VAS or verbal rating scale (*Ferraz MB, et al. Reliability of pain scales in the assessment of literate and illiterate patients with rheumatoid arthritis. J Rheumatol. 1990;17(8):1022–4*).
8. F T F T T
   Adults also prefer the scale because of the cartoon like features.
9. F T F T T
   Checklist of nonverbal indicators uses six behaviour items in cognitively impaired older adults. Fifty percent of patients have no indicators of pain so sensitivity is low. Pain assessment in advanced dementia scale measures breathing, negative vocalisation, facial expression, body language and consolibility. Abbey pain scale is an informant based tool that measures pain intensity in late stage dementia and measures acute pain, chronic pain and acute or chronic pain. MOBD scale guides a patient through five structured activities (mobilisation of both hands, both arms, both legs, turning in bed, sitting at bedside).
10. T T T T F
    Mean pain intensity during endotracheal suctioning is 4–5 where as some patients report up to 7–8. (*Puntillo KA. Dimensions of procedural pain and its analgesic management in critically ill surgical patients. Am J Crit Care. 1994;3(2):116–22*). Painful procedures in intensive care include turning, wound drain removal, wound care, tracheal suctioning, central line placement,

femoral sheath removal. Under treatment of procedural pain is seen up to 63%. (*Puntillo KA, et al. Practice and procedures of analgesic intervention for adults undergoing painful procedures. Am J Crit Care. 2002;11(5):415–29*). Face, legs, activity, cry, consolibility scale. Other measures include behavioural pain rating scale, behavioural pain scale, non verbal pain scale, critical care pain observation scale. CPOT is better than BPS as it evaluates 4 domains instead of 3. Behavioural pain scale includes facial expression, movements of upper limbs, compliance with ventilation. CPOT also addresses both ventilated and non ventilated patients whereas BPS only addresses ventilated patients.

11. T T F T T

An IQ of 50–70 is mild cognitive impairment, 35–49 is moderate cognitive impairment, 20–34 is severe cognitive impairment and less than 20 is profound impairment. Disables patients are more sensitive to some types of pain. Adults with mental retardation have more chronic than acute pain on a daily basis. (*Bodfish J, et al. Issues in pain assessment for adults with mental retardation. From research to practice. In: Oberlander TF, Symons FJ, editors. Pain in developmental disabilities. Baltimore, MD: Paul H Brookes*). Indicators for severe pain include crying during manipulation, painful facial expression, swelling, screaming, not using affected body part. PADS is sensitive to nonverbal signs of pain in adults with severe intellectual disability and is sensitive to everyday pain, acute pain response, chronic pain and effect of treatment on acute pain.

12. T T T T F

Post operative complications are seen more in schizophrenic patients like respiratory failure, deep venous thrombosis. They report less post operative pain and consume less than 60% of analgesic medication. Response to experimental pain is diminished. (*Polvin S, Marchand S. Hypoalgesia in schizophrenia is independent of antipsychotic drugs: a systemic review of experimental studies. Pain. 2008;138(1):70–8*). Pain insensitivity may be seen as a familial trait (*Singh MK, et al. Pain insensitivity in schizophrenia: trait or state marker. J Psychiatr Pract. 2006;12(2):90–102*).

13. F T T T T

PTSD is an anxiety disorder that can occur following an extremely traumatic event that involves being threatened by or witness to a situation that involves death or injury. The symptoms begin within 3 months and 50% recover with 3 months. Early treatment has a protective effect especially with propranolol. (*Pitman RK, et al. Pilot study of secondary prevention of post traumatic stress disorder with propranolol. Biol Psychiatry. 2002;51(2):189–92*). Patients are less sensitive to heat stimuli. They respond more to suprathreshold heat and mechanical stimuli. (*Geuze E, et al. Altered pain processing in veterans with post traumatic stress disorder. Arch Gen Psychiatry. 2007;64(1):76–85*). PTSD like symptoms may be seen with in fibromyalgia (*Cohen H, et al. Prevalence of post traumatic stress disorder in fibromyalgia patients: overlapping syndromes or piost traumatic fibromyalgia syndrome. Semin Arthritis Rheum. 2002;32:38–50*).

Pharmacology

3

1. Characteristics of non opioid analgesics:
   (a) Regular daily use of NSAIDs is better than opioids.
   (b) Non opioids should not be given with opioids at the same time.
   (c) NSAIDs do not cause gastric ulcers if given rectally or parenteraly.
   (d) Antacids with NSAIDs are effective in reducing gastric ulcers.
   (e) NSAIDs affect bone healing for longer period of time.
2. Non-steroidal anti-inflammatory drugs:
   (a) First line analgesics for acute nociceptive pain.
   (b) Single dose of these drugs may be effective.
   (c) Are more effective for somatic nociceptive pain especially involving inflammation.
   (d) Combination of two NSAIDs gives better results.
   (e) Can contribute to opioid sparing effect.
3. Adverse effects of NSAIDs:
   (a) Chronic usage may lead to tolerance.
   (b) Risk factors for liver injury include poor nutrition.
   (c) Advanced age is a risk factor for NSAID induced gastro intestinal adverse effect.
   (d) COX-2 selective NSAIDs have no effect on bleeding time.
   (e) Depression is a known risk factor for acetaminophen toxicity.
4. Adverse effects of NSAIDs:
   (a) High acetaminophen usage is associated with decrease in renal function.
   (b) Acetaminophen is better than NSAIDs for analgesia in renal disease.
   (c) Acetaminophen use may cause hypertension.
   (d) Acetaminophen may be associated with anticoagulant effect.
   (e) Acetaminophen is COX-1 selective.
5. Adverse effects of NSAIDs:
   (a) Long term therapy causes gastrointestinal complications in 80% of patients.
   (b) Nabumetone is the non selective NSAID mostly involved with gastric injury.

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(c) Gastrointestinal complications are more if the medications are used for more than a year.
(d) Helicobacter pylori increase the risk of peptic ulcers with concomitant use of NSAIDs.
(e) COX-2 selective NSAIDs are associated with cardio vascular complications.

6. Adverse effects of NSAIDs:
   (a) Misoprostol is well tolerated in elderly population.
   (b) H2 antagonists are beneficial in patients with Helicobacter pylori infection.
   (c) Ulcer relapse rate is more with misoprostol than omeprazole.
   (d) COX-2 selective NSAIDs along with proton pump inhibitors is more effective than COX-2 alone.
   (e) Celecoxib is associated with less mucosal breaks.
7. Adverse effects of NSAIDs:
   (a) Increased risks of cardiovascular side effects are seen with rofecoxib and valdecoxib.
   (b) COPD is a risk factor for cardiovascular events with COX-2 inhibitors.
   (c) Stroke seen is more common in females.
   (d) Cigarette smoking is protective for cardiovascular events in BSAID users.
   (e) Obstructive sleep apnoea is a modifiable risk factor.
8. Adverse effects of NSAIDs:
   (a) Rofecoxib increases cardiovascular events.
   (b) Valdecoxib is associated with cutaneous hypersensitivity.
   (c) Meloxicam has a better side effect profile.
   (d) Elderly patients taking aspirin should avoid NSDAIDs.
   (e) Celecoxib is contraindicated in bleeding disorders.
9. NSAID effect on renal system and vascular system:
   (a) COX-2 is present in glomerulus and afferent arteriole.
   (b) NSAID induced renal toxicity mostly occurs in use during post operative period.
   (c) Endogenous renal prostaglandin synthesis does not have significant role in maintaining GFR and renal blood flow.
   (d) Heart failure and chronic kidney disease are absolute contraindications for NSAID therapy.
   (e) Celecoxib treatment has the lowest incidence of cardiovascular adverse effects.
10. Topical NSAIDs:
    (a) Are not available in patches.
    (b) Works by inhibiting NMDA and sodium channels.
    (c) Therapeutic effect is due to absorption in systemic circulation.
    (d) Bioavailability is 50–60%.
    (e) Gels are more effective than creams.
11. Topical NSAIDs:
    (a) Diclofenac 1.3% patch causes decrease in pain, morning stiffness in acute pain.

(b) Topical diclofenac has no role in osteoarthritis.
(c) Topical diclofenac is more effective than oral diclofenac in morning stiffness.
(d) Piroxicam topical preparation is better choice for osteoarthritis than diclofenac.
(e) Ibuprofen cream can help in analgesia post DC cardioversion.

12. Acetaminophen:
(a) Increase of the dosage more than 1000 mg adds little to analgesia.
(b) Is safe to use in liver disease.
(c) Liver function tests should be performed.
(d) Has no gastrointestinal effect.
(e) Should be used with caution in G6PD deficiency.

13. NSAIDs for acute and chronic pain:
(a) Ibuprofen at a dose of 400 mg cause both analgesic and anti-inflammatory effect.
(b) Celecoxib efficacy is increased by giving maximum dosage in divided doses.
(c) NSAIDs with longer half life have a slower onset of analgesia.
(d) Caution is required in older population.
(e) Analgesia occurs after few weeks of usage.

14. Ketorolac:
(a) Is effective for severe pain in combination with other analgesics.
(b) An initial loading dose is required.
(c) Duration of analgesia is extended if given by intramuscular route.
(d) Most frequent side effect is headache.
(e) Renal failure is a contraindication.

15. Adverse effects of NSAIDs:
(a) Acetaminophen does not cause hematologic abnormalities.
(b) Aspirin should be stopped 5 days preoperatively.
(c) Increased risk of bleeding with ketorolac is seen with advanced age.
(d) NSAIDs should be avoided in renal failure irrespective of half lives.
(e) COX-2 selective NSAIDs inhibit healing more than nonselective NSAIDs.

16. Mechanisms of opioid analgesia:
(a) Therapeutic opioids activate endogenous pain modulating systems and produce analgesia.
(b) Endogenous opioids inhibit pain via the descending modulatory systems.
(c) Opioids decrease the influx of calcium.
(d) Opioids inhibit GABA system leading to pain transmission.
(e) Opioids can cause produce analgesia following local administration.

17. Opioid receptor sites:
(a) Three types are seen.
(b) Antagonists reverse opioid side effects.
(c) Mu receptors are free of respiratory depression.
(d) Most opioids bind to mu receptor sites.
(e) Nalbuphine is a pure mu agonist.

18. Pharmacokinetics of opioids:
    (a) Oral bioavailability of morphine is more than parenteral administration.
    (b) Bioavailability is increased in hepatic dysfunction.
    (c) Hydromorphone has 100% bioavailability by intravenous route.
    (d) Lipid solubility increases bioavailability.
    (e) Protein bound drug is devoid of pharmacological activity.
19. Pharmacokinetics of opioids:
    (a) Morphine metabolites are active.
    (b) Prodrugs are pharmacologically active.
    (c) CYP450 is the only enzyme system responsible for metabolism of opioids.
    (d) CYP450 enzyme system is important for opioid metabolism.
    (e) Poor metabolisers do not get desired analgesic effect.
20. Pharmacokinetics of opioids:
    (a) Liver is the primary organ for elimination.
    (b) Terminal half life is the same as distribution half life.
    (c) Creatinine clearance can alter with age.
    (d) Drug half life increases with age.
    (e) Long term opioid analgesic treatment is based on steady state concentration.
21. Opioid efficacy and potency:
    (a) Receptor occupancy which is required for an agonist to produce a response is inversely proportional to its intrinsic efficacy.
    (b) Efficacy is the same as potency.
    (c) Increased potency means increased therapeutic effect.
    (d) Opioids do not have analgesic ceiling.
    (e) Opioids are the first line of medications for neuropathic pain.
22. Opioid tolerance:
    (a) Continued exposure to the drug is the main cause.
    (b) Tolerance develops due to addiction.
    (c) Tolerance develops more quickly in younger individuals than in older patients.
    (d) Sedation levels are used to monitor opioid induced respiratory depression.
    (e) The first indication of tolerance is decrease in analgesic effect.
23. Opioid tolerance:
    (a) Tolerance to the analgesic effects of opioids is absolute.
    (b) Pain can diminish adverse effects of opioids.
    (c) Drugs that act at the same receptor can produce different levels of tolerance.
    (d) Incomplete cross tolerance is due to different selectivity for the receptor subtype.
    (e) New opioids used in opioid rotation should be increased by 20–50% in dosage for maximum effect.
24. Opioid tolerance:
    (a) Opioid tolerance can develop as early as 7 days of continuous use.
    (b) Develops only on intravenous opioids.

(c) Opioid tolerance patients should have background infusion in PCA post operatively.
(d) Tolerance to side effects develops earlier than analgesia.
(e) Dose of opioids required by opioid tolerant patient with cancer pain is not increased.

25. Physical dependence:
(a) Can be seen with other drugs than opioids.
(b) Is associated with withdrawal.
(c) Can be avoided by avoiding abrupt cessation or administration of an opioid antagonist.
(d) Onset of withdrawal symptoms is independent of half life.
(e) Opioid weaning is dependent on the duration of opioid intake.

26. Breakthrough pain:
(a) Is mostly a continuous type.
(b) Is similar to incident pain.
(c) End of dose failure is treated by decreasing the dosage of opioids.
(d) Most common type of breakthrough pain is neuropathic pain.
(e) It decreases the quality of life.

27. Treatment of breakthrough pain:
(a) Is always pharmacotherapy.
(b) Mostly opioids are prescribed on as required basis.
(c) Oral opioids are the drugs of choice for breakthrough pain.
(d) Transmucosal and intranasal routes are better than oral route.
(e) Intrathecal opioids can be given for breakthrough pain.

28. Patient demand dosing of opioids:
(a) Requires active patient participation.
(b) Is only effective for breakthrough pain.
(c) Risk of under treatment is seen.
(d) There is no risk for under treatment in dementia.
(e) Patient controlled analgesia is a method of as required dosing.

29. Patient controlled analgesia:
(a) Is based on nurse's interpretation of pain.
(b) Can be used for procedural sedation.
(c) Only opioid analgesics can be used.
(d) Better pain control is seen.
(e) Can be used in children.

30. Morphine:
(a) Is a pure mu agonist.
(b) Is first line medication for neuropathic pain.
(c) Cognitive decline is seen with opioid use.
(d) Has good lipid solubility.
(e) Intramuscular morphine is the ideal route.

31. Morphine:
(a) All the metabolites of morphine are active at opioid receptors.
(b) M3G is implicated in opioid induced hyperalgesia.
(c) M3G produces less side effects than morphine.

(d) Morphine has longer duration of action.
(e) Dosage of oral route is the same as intravenous route.

32. Codeine:
(a) Efficacy of codeine increases as the dosage increases.
(b) The ideal route to administer is intramuscular route.
(c) Is a prodrug.
(d) Metabolism depends on the presence of cytochrome P4502D6.
(e) Is not secreted in the breast milk.

33. Codeine:
(a) Is absorbed from gastrointestinal tract.
(b) Tolerance does not develop.
(c) Sudden abstinence is life threatening.
(d) Hypogonadism can occur in male patients.
(e) Has an inherent antitussive effect.

34. Fentanyl:
(a) Ventilation may be difficult on rapid intravenous administration.
(b) Is less potent than morphine.
(c) Lipophilicity decreases the absorption.
(d) The patch's efficacy is dependent on steady state.
(e) Can be given by intrathecal route.

35. Fentanyl:
(a) Transmucosal route is used in opioid tolerant patients.
(b) Works at both Presynaptic and post synaptic levels.
(c) Steady state is achieved with patch after 12 h.
(d) Patches need to be stored at room temperature.
(e) Transmucosal Fentanyl is ideal for breakthrough pain.

36. Methadone:
(a) Can be given intrathecally.
(b) Can be used as patient controlled epidural analgesia.
(c) Acts only at mu receptors.
(d) Pharmacokinetics is similar to morphine.
(e) Is metabolised by hepatic pathway.

37. Methadone:
(a) Oral bioavailability is lesser than morphine.
(b) Has toxic metabolites.
(c) Causes inhibition of reuptake of serotonin and nor epinephrine at central synapses.
(d) Is ideal for sublingual and topical administration.
(e) Elimination is primarily via faeces.

38. Sufentanil:
(a) Less lipid soluble than Fentanyl.
(b) Quality of analgesia via epidural route is better than Fentanyl.
(c) Can be used intraspinally.
(d) Can be used as a patch.
(e) Can displace buprenorphine from its binding sites.

39. Opioid therapy:
    (a) Weight has direct correlation with analgesic requirements.
    (b) Opioid initiating dose should be lowered in elderly.
    (c) Equianalgesic chart is helpful when switching from one drug to another.
    (d) Breakthrough pain may require boluses of op to every 15–30 min.
    (e) Dose of breakthrough pain is normally 20–30%.
40. Opioid therapy:
    (a) Titration is aimed at finding the lowest possible effective dose.
    (b) Increase in breakthrough doses is an indicator for increasing sustained release medication.
    (c) Multimodal therapy leads to less titration of opioids.
    (d) Fentanyl is ideal for acute titration of post operative pain.
    (e) Short acting drugs should be used in conjunction with long acting medicines.
41. Patient controlled analgesia:
    (a) Can be given only via the intravenous route.
    (b) Large doses with long lock out period are ideal.
    (c) Analgesia requirement increases with age.
    (d) Use of opioid infusions in opioid naive patients is not recommended.
    (e) PCA by proxy is safe.
42. Adverse effects of opioids:
    (a) Are dose dependent.
    (b) Dose reduction should be 50% to avoid side effects.
    (c) Constipation is due to delayed gastric emptying.
    (d) Opioid agonists are helpful in refractory constipation.
    (e) Risk factors for constipation include advanced age, immobility, abdominal disease, concurrent medications.
43. Adverse effects of opioids:
    (a) Bulk laxatives are ideal for treating opioid induced constipation.
    (b) Initial treatment in constipation is a combination treatment.
    (c) Continuous thoracic epidural can prevent paralytic ileus.
    (d) Opioid antagonists induced withdrawal symptoms is maximally seen with naloxone.
    (e) Post operative ileus is true obstruction.
44. Adverse effects of opioids:
    (a) Routing nasogastric decompression should be used in paralytic ileus.
    (b) Oral intake should be started as early as possible to prevent ileus.
    (c) Excess fluids can decrease gastro-intestinal motility and increases ileus.
    (d) Laparoscopy technique can reduce ileus.
    (e) Opioids should be avoided in post operative ileus.
45. Adverse effects of opioids:
    (a) Nausea usually develops after weeks of opioid treatment.
    (b) Female sex is a risk factor for opioid induced nausea and vomiting.
    (c) Incident pain is associated with higher incidence of post operative nausea and vomiting.

(d) Single drug antiemetic prophylaxis has a high success rate.
(e) Prophylactic antiemetics should be given to all patients.

46. Adverse effects of opioids:
(a) Billiary spasm by opioids can increase pain.
(b) Meperidine has no effect on sphincter of oddi.
(c) Pruritus is an uncommon complication.
(d) Pruritus can be measured on a numerical scale.
(e) Post operative opioid induced Pruritus patients have well controlled pain.

47. Adverse effects of opioids:
(a) Intravenous patient controlled analgesia has the highest incidence of hypotension.
(b) All opioids cause bradycardia.
(c) Incidence of hypotension can be minimised by administering opioid slowly.
(d) High incidence of urinary retention is seen in post operative period in elderly men.
(e) Addition of local anaesthetic to opioids intrathecally can decrease the evidence of urinary retention.

48. Adverse effects of opioids:
(a) Tolerance to opioid induced urinary retention is seen.
(b) Myoclonus is rare in patients taking opioids.
(c) Myoclonus is seen mostly with Meperidine.
(d) Mental status changes may be seen in majority.
(e) Post operative delirium occurs in older patients.

49. Adverse effects of opioids:
(a) Poorly managed pain is a risk factor for post operative pain.
(b) Meperidine causes most amount of post operative delirium.
(c) Fentanyl PCA has less cognitive impairment post operatively than morphine PCA.
(d) Opioids cause sedation due to its anticholinergic activity.
(e) Donezepil is useful in opioid induced sedation.

50. Adverse effects of opioids:
(a) Severe respiratory depression may be seen.
(b) Post operative hypoxaemia is oxygen saturation less than 95%.
(c) Tolerance to respiratory depression is seen.
(d) Respiratory depression is maximally seen 2–3 days after the surgery.
(e) End tidal CO2 is an early indicator of impending respiratory depression.

51. Addiction of opioids:
(a) High risk is seen after the post operative period.
(b) Physical dependence can be seen within hours of administration.
(c) Tolerance is appropriately treated by decreased opioid dosage.
(d) Increase in opioid dosage can cause increase in pain.
(e) COX-2 inhibitors can prevent opioid induced hyperalgesia.

52. Adverse effects of opioids:
(a) Immune function is suppressed as early as 2 weeks of opioid initiation.
(b) Long term opioids can cause hypogonadism.
(c) Pain in persons with addictive disease is undertreated.

(d) Methadone maintenance treatment should be continued in surgeries requiring opioids.
(e) Adding opioids to patients with history of addiction problems may lead to relapse.

53. Opioid use in special conditions:
(a) Meperidine is ideal in pregnancy.
(b) Infants may show addiction behaviour born to mothers who take opioids.
(c) Meperidine is contraindicated in lactating mothers.
(d) Most painful procedure responsive to opioids in intensive care unit is simple turning of the patient.
(e) Increased dose of analgesics and sedatives are associated with shortened survival.

54. Adjuvant analgesics:
(a) Are reliable in providing pain relief on its own.
(b) Are only effective for neuropathic pain.
(c) Can be used for acute pain.
(d) Are more time consuming.
(e) Treatment of depression may give pain relief.

55. Adjuvant analgesics during pregnancy:
(a) Steroids should be avoided during first trimester.
(b) Anticonvulsants are safe during pregnancy.
(c) Usage of phenytoin has high incidence of teratogenic effect.
(d) Local anaesthetics dose may be decreased in pregnancy.
(e) Tricyclic antidepressants are not recommended in pregnancy.

56. Multimodal analgesia:
(a) Has a major role in acute pain and post operative pain.
(b) Achieves pain relief with minimal side effects.
(c) May help in treatment symptoms related to pain.
(d) May cause increase in side effects.
(e) Most adjuvant medications have hepatic metabolism.

57. Clonidine:
(a) Is an alpha-2 agonist.
(b) Can be used via neuraxial route.
(c) Transdermal patch is useful in post operative pain.
(d) Causes sedation.
(e) Transdermal patch can be cut into pieces.

58. Ketamine:
(a) Can be used for post operative pain.
(b) Can be used for treating acute opioid induced hyperalgesia.
(c) Perioperative Ketamine decreases opioid usage and complication rate.
(d) Has better analgesic profile in depressed patients in acute pain.
(e) Epidural route is the preferred route for post operative pain management.

59. Mode of local anaesthetics:
(a) Blocks impulses by inhibiting sodium channels.
(b) Local anaesthetics cause complete neural blockade.
(c) Has analgesic effect when given systematically.