Postoperative analgesia

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Abstract

Pain is a complex experience consisting of sensory, affective, behavioural and physiological components. Pain management is therefore best achieved through an approach which acknowledges the complex interaction between biological, psychological and sociocultural factors. Effective pain management requires preoperative patient engagement and education in order to manage expectations and a structured inpatient service to facilitate evidence-based postoperative pain management and continuous staff education. Multimodal postoperative analgesia, built on an opioid-sparing ethos, is one component of postoperative pain management and is essential for achieving patient satisfaction and enhanced recovery. Effective pain management facilitates early mobilization and a reduction in respiratory and cardiac complications, reducing the stress response to surgery in turn improving wound healing and recovery. Inadequate pain control can lead to higher morbidity and mortality, prolonged hospital stays and the development of chronic postoperative pain.

Keywords Multimodal analgesia; pain physiology; postoperative pain

Royal College of Anaesthetists CPD Matrix: 1A02; 1D01; 1D02; 2E01

Learning objectives

After reading this article, you should be able to:

- explain the mechanisms of acute postoperative pain
- discuss the targets for postoperative analgesics with a multimodal, opioid-sparing approach to the management of pain
- identify patients that may be challenging in terms of postoperative pain and develop potential strategies for effective management

Introduction

Pain is defined by the International Association for the Study of Pain as: 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage'.[1] Pain is also defined as: 'whatever the experiencing person says it is, existing whenever he/she says it does'. Pain is therefore a subjective experience and there are no objective tests or investigations which can prove whether a patient has pain or not.

Pain is a complex integrated response and consists of sensory, emotional, cognitive and behavioural components that may be described on the verbal-subjective, motor-behavioural, and physiological levels. These three levels of responses need to be considered in the analysis of pain. An integrated multidisciplinary approach needs to be taken to pain management that also considers patient preferences and prior experience.

Normally the purpose of pain is to alert an animal about damage from injury or disease and to help the animal make decisions about what to do with regards to the injury/disease. It serves a useful purpose to protect the body from further harm and allow tissue healing. Acute pain normally resolves with healing of the underlying injury.

If pain is not treated, activation of sympathetic efferents can lead to cardiac ischaemia. Severe pain after thoracic and upper abdominal surgery contributes to an ineffective cough, resulting in an increased incidence of pulmonary complications. Patients at greatest risk of adverse outcomes from undertreated acute postoperative pain include those at the extremes of age or who have concurrent medical illnesses. Inadequately treated acute pain is also a risk factor for poor wound healing, delayed discharge and the development of chronic pain.

Pain perception and tissue damage sensing pathways

A useful model for understanding the experience of acute pain due to tissue injury consists of four processes: transduction, transmission, perception and modulation (Figure 1). An understanding of this model provides a cognitive map for implementing effective, bespoke, multimodal opioid-sparing analgesia and can help guide an effective postoperative analgesic strategy.

Transduction

Transduction is the conversion of a chemical, thermal or mechanical stimulus into an electrical signal by nociceptors. Nociceptors exist on free nerve endings of primary sensory neurones present in skin, muscle, joints, viscera and meninges and are unprotected from chemicals secreted into or applied to tissue. They respond to relatively high magnitude or potentially tissue-damaging stimuli. Their cell bodies reside in the dorsal root ganglia (DRG).

Surgical trauma results in inflammation, degranulation of mast cells, secretion of inflammatory cells and induction of enzymes. Chemical mediators including ATP, bradykinin, substance P and prostaglandin E2 are released from these inflammatory cells at the axon terminal. These chemical mediators act either directly on their associated receptor present on the nociceptive afferent terminal to distort or depolarize the membrane of the nociceptor, or via metabotropic receptors, to result in receptor activation or sensitization.

The most numerous subclass of nociceptor is the C-fibre receptor which is polymodal, responding to a range of mechanical and chemical stimuli. C-fibres are thinly myelinated or unmyelinated with small diameter cell bodies and terminate in laminae I and II of the dorsal horn (DH). They have a slow conduction velocity of less than 3m/second and relate to the perception of a slow 'burning' pain. Aδ fibres are mechanical and thermal

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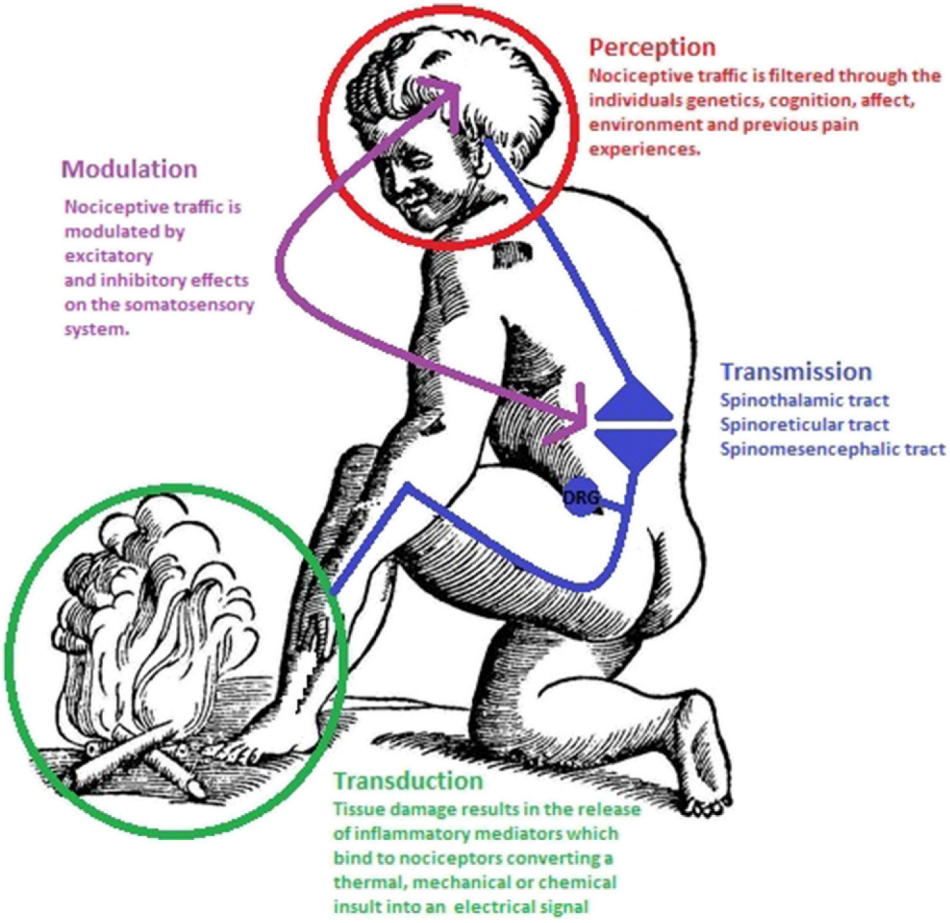

Figure 1 A physiological model of the pain experience

nociceptors that generally terminate in laminae I and III–V. They are the predominant receptor type involved with the perception of 'sharp' pain, and have a fast conduction velocity of 5–30m/sec.

Transmission

Action potentials generated at the nociceptor are transmitted to the structures of the central nervous system concerned with the perception of pain. Impulses generated in the primary sensory neurons are conducted to the dorsal horn of the spinal cord. Here they synapse with second order neurones and are relayed to the brain.

The spinothalamic tract is classically considered the major nociceptive pathway originating from neurons in laminae I and III–V of the DH. Over 85% of spinothalamic axons cross and ascend contralaterally where they project to the thalamus and then to the somatosensory cortex to provide information on the site and type of nociceptive stimulus.

The spinoreticular and spinomesencephalic tracts project to the medulla and brainstem and integrate nociceptive information with homeostatic and autonomic responses as well as projecting to central areas to mediate the emotional component of pain.

Other connections include those to the periaqueductal grey matter of the midbrain and rostroventromedial medulla which are necessary for flight or fight responses, and projections to the reticular formation which play an important role in the regulation of descending pathways to the spinal cord.

Perception

Nociception is the signal produced following tissue damage, but this does not constitute pain. The perception of pain is the conscious awareness of the experience of pain and results from the processing and modulation of the information received via transduction and transmission.

When the information about tissue damage reaches the brain, it is processed through the individual's genetics, environment, culture, beliefs and previous pain experiences as well as mood and psychological constructs including, for example, self-efficacy and catastrophizing. Self-efficacy is defined as a personal conviction that one can successfully execute a course of action to produce a desired outcome in a given situation. Maladaptive appraisals of pain and its duration and one's personal efficacy may reinforce the experience of demoralization, inactivity, and overreaction to nociceptive stimulation. Catastrophizing consists of extremely negative thoughts about one's plight, even with minor problems being interpreted as major catastrophes. Catastrophizing and self-efficacy are important in determinants one's reaction to pain. Pain is therefore a complex perception requiring activity in higher centres.

Modulation

The degree of tissue injury is not proportional to the pain experienced and pain perception is not always a consequence of tissue injury and nociception. This is due to modulation of the nociceptive traffic at points along the transmission pathway. Pain transmission depends on the balance between inhibitory and

excitatory influences on the somatosensory system. Integration of these influences occurs at multiple levels of the central nervous system (CNS) including the spinal cord, brain stem and multiple cortical regions. The major mechanisms by which this occurs include production of endogenous opioids, local inhibition, and gate control: whereby non-nociceptive afferents are stimulated to inhibit nociceptive signals, or descending inhibition or facilitation from higher centres.

Principles of postoperative pain management

Complex haemodynamic, metabolic, humoral, immune as well as somatosensory responses are triggered by the injury and consequent acute pain. Elevated levels of cortisol, catecholamines and glucagon are seen in experimental studies, together with a decrease in insulin sensitivity. Effective pharmacological pain relief may have a significant impact on these responses. The release of pro-inflammatory cytokines as a result of pain and trauma contributes to physiological responses that hamper patient recovery. Recognition of the importance of postoperative rehabilitation including pharmacological physical, psychological and nutritional components has led to enhanced recovery protocols. If acute pain is prolonged and poorly treated the injury response becomes counter-productive and can have adverse effect on outcomes.

Multimodal analgesia, compared to mainly opioid-based analgesia, improves pain control and reduces opioid consumption and adverse effects thereby enhancing recovery.[2] The concept of multimodal (or 'balanced') analgesia suggests the use of combinations of medications with different modes or site of action (Figure 1). Effective pain management is a core component of enhanced recovery after surgery.

Assessment of postoperative pain

The most reliable assessment of postoperative pain is patient self-reporting which can give accurate information as to the nature, location and intensity of the pain. Frequent assessment in order to determine the response to treatment and recognition of adverse side effects is essential. Communication can be difficult, particularly in the presence of uncontrolled pain when patients may be distressed or agitated and impatient for an intervention to occur.

Unidimensional pain intensity rating scales can be useful in assessing acute pain where the aetiology is clear and when comparing pain intensity before and after an intervention. They can be used quickly in both the recovery room and ward environments. It is also important to assess pain both at rest and during periods of activity such as moving, deep breathing and coughing, as the ability to carry out these actions are useful outcome measures of good postoperative analgesia. Some commonly used unidimensional pain intensity scales include:

- Categorical rating scale: patients are asked to rate pain as none, mild, moderate, severe.
- Numeric rating scale (NRS): patients are asked to rate their pain on a scale of 0–10, with 0 representing no pain and 10 representing the worst pain imaginable.
- The FACES® Pain Scale (FPS) consists of eight faces with varying expressions to represent the severity of pain which may be helpful if the NRS and VAS are unsuitable.

Multidimensional pain scales measure the nature, intensity, and location of pain as well as in some cases assessing the impact that the pain is having on an individual's mood or activity. In the context of postoperative pain this is usually done in the form of a pain history and may be more appropriate in patients with acute on chronic pain.

There will be some patients that cannot be assessed with any of these tools and in these circumstances, pain needs to be estimated indirectly. Careful observation of the patient will reveal behavioural responses to pain such as grimacing, reduced movement or splinting of the operative site. There are also physiological responses to acute pain that may indicate that a patient is experiencing pain. Pain can cause tachycardia and hypertension, as well as sweating, pupillary dilatation and lacrimation.

Routes of drug delivery

Factors which influence the route of analgesic medicine administration include the severity and type of pain as well as the patient's overall condition and the characteristics of the chosen administration technique. Speed of analgesic onset as well as reliability of effect and duration of action together with patient accessibility and costs are also issues which must be taken into account. Frequent assessment of the patient's pain and their response to treatment must be borne in mind rather than protocoled adherence to particular dosing regimens. This is important if adequate analgesia is to be attained.

Oral

The oral route has good efficacy in most settings except for severe acute pain and is generally acceptable to patients. A few points of importance: paracetamol combined with codeine or paracetamol combined with tramadol is more effective than either method alone. There is no advantage in giving non-steroidals parentally or rectally compared with the oral route and it may be prudent to administer oral paracetamol later on in the postoperative phase since early postoperative oral administration results in highly variable plasma concentrations.

Immediate release oral opioids should be used for breakthrough pain and the use of oral controlled release opioids as the sole agent in the early management of acute pain is discouraged because of difficulties in the short-term dose adjustments needed for titration.

Intravenous

Intermittent intravenous bolus doses of opioids are appropriate for the management of severe acute pain and allow more rapid titration of effect. The intravenous route for titration of opioids for severe acute pain avoids the uncertainty of medicines absorbed by other routes. Intravenous infusions of opioids where required should be administered in a critical care setting as they are associated with an increased risk of respiratory depression.

Intramuscular/subcutaneous

Subcutaneous morphine injection is as effective as intramuscular injections and has greater patient acceptance. Absorption may be impaired in conditions of poor perfusion and may result in suboptimal analgesia. Late absorption of opioids administered via this route when perfusion is restored places the patient at risk of adverse effects.

Transdermal

There are safety concerns with regards to the use of transdermal fentanyl in the management of acute pain. Long-acting opioids are to be discouraged in the management of acute pain due to difficulties in short-term dose adjustments needed for titration. This is particularly concerning in opioid naive patients.

Transmucosal

Submucosal routes are appropriate in the management of cancer pain where intranasal fentanyl provides fast and better analgesia for breakthrough pain. This route is superior to the oral transmucosal route.

Epidural

Epidural analgesia provides superior postoperative pain relief compared with the intravenous route, including PCA opioid administration. Thoracic epidural analgesia for abdominal aortic surgery reduces the duration of tracheostomy and mechanical ventilation. This form of postoperative analgesia also reduces the incidence of myocardial infarction, acute respiratory failure, gastrointestinal complications and renal insufficiency when compared with intravenous opioids. Bowel recovery is aided by thoracic epidural analgesia after abdominal surgery; however, the reduction in perioperative mortality with epidural analgesia must rebalance against the hypotension produced by this form of analgesia. Epidural opioids have to be combined with low concentrations of local anaesthetic agents for epidural analgesia to provide superior pain relief.

The benefits of the epidural route however must be balanced against the invasive nature of this intervention and associated risks including permanent neurological injury. A risk-benefit assessment for the individual patient should be made by the clinician.

Intrathecal (opiates)

Intrathecal morphine improves analgesia and is opioid-sparing for up to 24-hours, especially following abdominal surgery. Their use shows a lower risk of major adverse effects; however this route is associated with a higher incidence of opioid-induced respiratory impairment and pruritus than oral or intravenous opioids. Patients should be monitored for respiratory depression which may then necessitate the need for a critical care bed.

Other regional and local analgesic techniques

Paravertebral blocks provide superior analgesia for breast surgery compared to systemic analgesia within the first 48 hours, with a lower incidence of postoperative nausea and vomiting.

Ultrasound-guided blocks are more likely to be successful as well as being faster to perform compared with the use of a peripheral nerve stimulator. Continuous interscalene analgesia is superior to intravenous PCA or single injection interscalene block after open shoulder surgery. Femoral nerve block provides superior analgesia and decreased nausea compared with intravenous opioid-based techniques after total knee replacement.

Intra-articular morphine does not improve analgesia after knee arthroscopy and there is no additional benefit for local infiltration of a total hip arthroplasty compared with conventional multimodal analgesia. There is insufficient evidence to support the use of local anaesthetic catheters in total knee and hip arthroplasty.

Pharmacotherapy

The aim in postoperative pain management is to reduce the use of opioids through a multi-modal analgesia strategy including the use of regional anaesthetic techniques when appropriate. There are no universal analgesic guidelines however the regimen chosen should be tailored to the individual patient. By focusing on more than one possible site and mechanism of anti-nociceptive action, it is more likely that greater analgesic efficacy will be achieved with reduced adverse effects (Figure 2).

Opioids

Opioids may be natural or synthetic, act upon opioid receptors and mimic the effects of endogenous opioids. The opioid receptors are G-protein-linked and compromise four subtypes MOP, DOP, KOP and NOP receptors. All receptor types are widely distributed throughout the central and peripheral nervous systems as well as in endocrine and immune cells. Binding of the ligand with the receptor produces hyperpolarization of the cell membrane due to the effect on potassium channels. The inhibitory action on peripheral and central neurons is the fundamental mechanism by which opioid analgesia is achieved. MOP receptors are responsible for the predominant actions of most clinically used opioid drugs, while the other receptor types can modulate nociception either directly or indirectly via modulation of MOP mediated effects.

Prescription opioid use has been increasing over the past 20−30 years and perioperative high-dose opioids are associated with longer hospital stay, higher healthcare costs, readmission and increased risk of inpatient mortality. A multi-modal opioid-sparing approach has been demonstrated to enhance recovery after surgery.[4]

The negative effects of opioids are apparent with both acute and chronic use. Opioids may be thought of as a ‘broad spectrum analgesic’, given that they affect many organ systems within the body. There should therefore be a conscious and considered approach to their prescription in the acute post-operative period with patient consent and involvement.[4]

Common acute effects of opioids include respiratory depression, sedation, dizziness, nausea and vomiting, constipation, delayed gastric emptying and urinary retention. Prolonged usage can lead to tolerance and physical dependence. Less well-known consequences of opioid use are their propensity to cause immunomodulatory and hormonal dysfunction as well as hyperalgesia. Central and peripheral changes in the immune system occur with both acute and chronic opioid use. Endogenous opioids and endorphins cause immunological activation; however, exogenous opioids have a negative effect on peripheral immunological activity by reducing cytokine activation, phagocytic response and natural killer activity. Exogenous opioids are also thought to modulate the immunological response centrally by exerting a negative effect on the hypothalamo−pituitary axis. These immunomodulatory effects can therefore lead to an increased risk of post-operative infection. Testosterone, oestrogen, luteinizing hormone and gonadotrophin releasing hormone production are all decreased in the presence of opioids leading to decreased libido, erectile dysfunction, reduced bone mineral density, osteoporosis and amenorrhoea. Reduced bone mineral density and osteoporosis are of particular importance as this can lead to frequent falls and fragility fractures.[4]

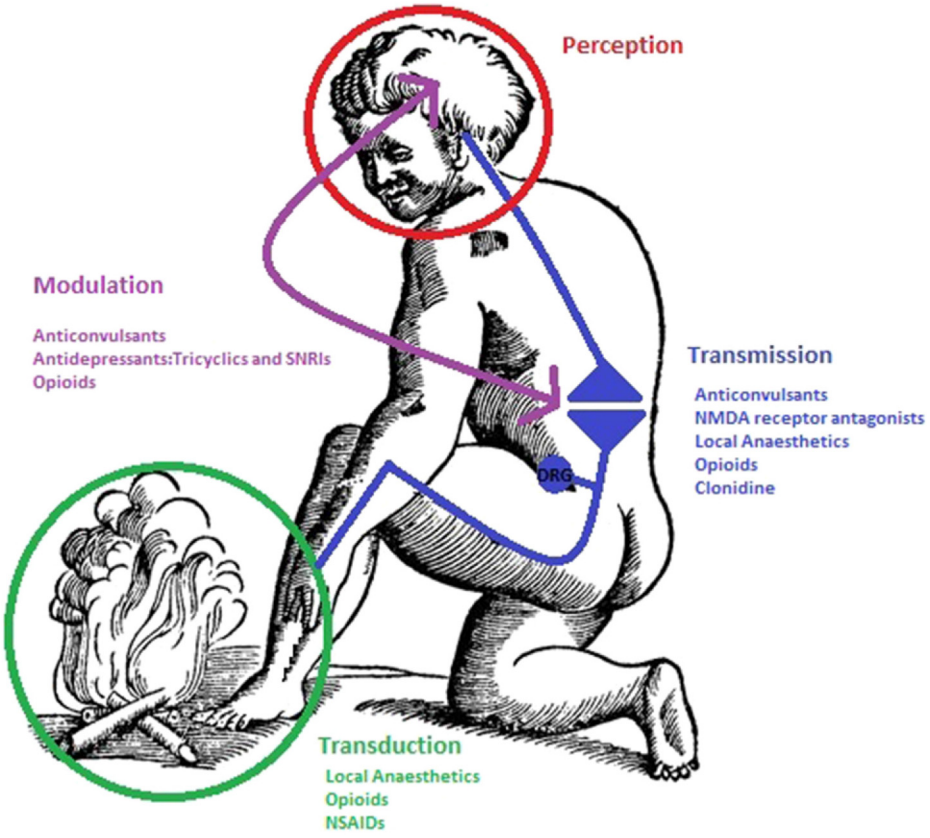

Figure 2 Sites of analgesic drug action NMDA, N-methyl-D-aspartate; NSAID, non-steroidal anti-inflammatory drug; SNRI, serotoninenoradrenaline reuptake inhibitor.

Morphine is the most widely used opioid drug for acute pain and is a pure MOP agonist. It is commonly administered intravenously as a bolus injection, continuous infusion, or via a patient-controlled analgesia (PCA) device. It can also be given intramuscularly, orally (although oral bioavailability is poor at approximately 25%), and into the epidural or intrathecal space. Morphine is metabolized to morphine-6-glucuronide (M6G) and morphine-3-glucuronide (M3G), of which M6G is active at the MOP receptor to add further analgesic effect and excreted in the urine. Impaired renal function, increased patient age and higher doses are associated with higher risk of long-lasting sedation and respiratory depression.

Codeine is a prodrug of morphine and is metabolized in the liver, primarily by glucuronidation to norcodeine. Only a small proportion of codeine is metabolized to morphine, accounting for its low potency of 20% or less when compared to morphine. It is available in a liquid preparation suitable for intramuscular (IM) injection. However, it is the improved bioavailability of 50%, that when compared to morphine makes it a popular choice of analgesic for the treatment of mild to moderate pain in the postoperative period via the oral route.

Tramadol is a racemic mixture of two stereoisomers. It is centrally acting with weak affinity for the MOP receptor and inhibits uptake of noradrenaline and 5-HT within the nervous system which is thought to account for some of its analgesic activity. It is available for administration orally or intravenously and has good oral bioavailability. While it has a reduced incidence of respiratory depression it can produce side effects such as nausea, vomiting, dizziness and sedation which may limit its use in some patient groups.

Fentanyl is a phenylypiperidine derivative and primarily a MOP agonist that is 100 times more potent than morphine. It is highly lipid soluble which gives fentanyl its rapid onset of action, and short duration of action as it is rapidly metabolized by N-dealkylation to inactive compounds that are excreted in the urine. These properties make it particularly useful when treating severe postoperative pain, particularly in PCAs and in patients with renal impairment where accumulation of morphine metabolites could lead to toxicity.

Oxycodone has a faster onset of action than morphine. It is metabolized to noroxycodone and oxymorphone with the latter metabolite being more potent. There are fewer concerns with regards to oxycodone metabolites than with morphine and the drug also works faster and has a higher oral bioavailability and longer duration of action than morphine.

Opioid-sparing adjuvants

Paracetamol is a para-aminophenol that is widely used as an analgesic for mild to moderate pain and as an antipyretic. Its mechanism of action is unclear and as yet there are no known endogenous binding sites. However, it may have some effect on COX inhibition as well as prevention of prostaglandin production that is independent of its COX activity.

It is available for oral administration with a bioavailability of up to 90%. It can also be given rectally and IV. It is an effective

postoperative analgesic that has opioid sparing effects. Caution is needed in patients with low body weight and liver impairment.[4]

Non-steroidal anti-inflammatory drugs (NSAIDs) The principal action of NSAIDs is via inhibition of cyclo-oxygenase (COX) and the consequences are seen in many organ systems potentially causing bleeding, renal impairment and bronchospasm. The incidence of renal impairment is low with perioperative NSAID use when patients are selected and monitored appropriately.

COX-1 is expressed in most tissues including platelets and is involved in cell-to-cell signalling and tissue homeostasis. COX-2 is induced in inflammatory cells and is responsible for the production of prostanoid mediators of inflammation. Traditional NSAIDs block both COX-1 and COX-2. Newer preparations or 'coxibs' show selectivity for the COX-2 enzyme. However, there is interdependence between COX-1 and COX-2 inhibition and a reduction in major adverse gastrointestinal adverse events has only been shown in a few studies.

Gabapentin and pregabalin are anticonvulsants which are now established in the management of chronic neuropathic pain and their use in the perioperative setting has been investigated for the prevention of chronic postsurgical pain and for their potential opioid-sparing effects. Although there is good evidence for an effect on neuropathic pain, their role in postoperative pain is less certain. Evidence to support their use in postoperative pain is limited because of the poor quality of evidence from clinical trials.

The analgesic effects of gabapentinoids may be attributed to depression of dorsal horn sensitivity through interactions with the a2d-1 calcium channel subunit. They stimulate descending inhibition, inhibit descending serotonergic facilitation and inflammatory mediators, and influence the affective component of pain. They are not currently thought to be useful as sole analgesics but can be effective in selected groups of patients as part of a multi-modal analgesic strategy. They are anxiolytic and have sleep modulating effects potentially adding to their effectiveness as adjuvants in postoperative pain management.[4]

Clonidine is an alpha-2-adrenorecptor agonist that is a useful adjuvant in the management of postoperative pain. The primary site of action of clonidine is in the spinal cord where it inhibits pain transmission by binding to the alpha-2-adrenoreceptor to activate inhibitory G-proteins, but it is also recognized as having supraspinal and peripheral sites of action. At a supraspinal level, it inhibits $\alpha 2$ adrenorecptors at the locus coeruleus in the brainstem which is known to be an important modulator of nociceptive transmission. Its central actions are responsible for the sedative effects, and peripheral action on vascular smooth muscle leads to vasodilatation and reduction in blood pressure. Clonidine is available as both oral and IV preparations and its use perioperatively has been shown provide appropriate levels of anxiolysis and sedation, as well as reducing intra and postoperative opioid requirements. It can also be used as an adjuvant to neuraxial local anaesthetic analgesia.[4]

Ketamine Nociceptive stimuli cause glutamate to be released from excitatory neurons. This causes activation of NMDA receptors in the CNS and PNS. In sub-anaesthetic doses ketamine exerts its principle effects by prevention or reversal of NMDA-mediated central sensitization, wind up and pain memory which clinically manifest themselves as hyperalgesia and allodynia.

Perioperative ketamine infusions may be useful for postoperative analgesia in opioid-tolerant patients and in acute neuropathic pain. It is an opioid-sparing adjuvant and reduces the incidence of chronic postsurgical pain. It should not be used in patients with severe coronary or vascular disease, injuries to the globe of the eye, history of psychosis, hepatic dysfunction, recent liver transplant or porphyria.[5]

Magnesium is an antagonist of the NMDA receptor, and like ketamine, may be useful for its inhibition of central sensitization.

Local anaesthetics (LA) play an important role in the provision of effective postoperative analgesia, primarily through their effects in neuraxial blocks and peripheral nerve blocks, but also via localized wound infiltration and infusion.

Perioperative intravenous lidocaine infusions are opioid-sparing and have been shown to significantly reduce pain scores at rest and during activity. Nausea, vomiting, duration of ileus after abdominal surgery and length of hospital stay are also reduced. The additional benefit of lidocaine is that it has anti-hyperalgesic properties. Intravenous lidocaine is given as an initial bolus intra-operatively followed by a continuous infusion.[6]

Inpatient pain services

The delivery of effective postoperative pain management requires education of medical, nursing, allied health care professionals as well as patients. Attention to the organizational aspects involved in the delivery of pain relief including appropriate postoperative pain protocols will ensure that analgesia is delivered in a timely and safe manner. This may be spearheaded by an inpatient pain service the complexity of which will vary depending on the surgical and trauma services being delivered. Collaboration between anaesthetists, pain specialists, surgeons and general practitioners as well as specialist in addiction medicine, nurses, physiotherapists and psychologists are important for the provision of effective inpatient pain management.

The ideas, concerns and expectations of patients and their active participation in decisions about their care pain management are essential. Accurate information about perioperative pain management as well as the risks and benefits of any treatments should be provided. There is evidence that specific pain education in certain surgical settings may result in decreased pain and opioid use post operatively as well as less healthcare utilization. It is important to appreciate that effective perioperative pain management may result more from appropriate education and organizational structures than innovative analgesic techniques.

Inpatient pain services may improve pain relief and reduce the incidence of adverse effects, lower postoperative morbidity and mortality, and reduce the incidence of persistent pain after surgery. An inpatient pain service may also take a role in identifying those patients which are likely to pose more challenges in terms of the management of postoperative pain.

Postoperative analgesia in the patient on chronic opioid therapy

There are increasing numbers of patients presenting for surgery that are receiving high dose opioids. Managing pain post operatively in this group can be challenging as these patients may be tolerant to the effects of opioids. They may also have a physical dependence on opioids which needs to be managed pre- and postoperatively. Ideally any established opioid regime should be continued perioperatively. Postoperatively, opioid requirements are likely to increase, and standard postoperative dosing regimens will need to be modified to reflect any opioid tolerance. The nature and extent of any surgery may affect the function of the gastrointestinal system, which may mean than parental opioids need to be administered. Alternatively, an epidural could be considered depending on patient and surgical factors. Many drugs have an opioid sparing effect, and the use of these adjuvants in this situation can avoid the need for very high dose opioids and their subsequent side effects.

Analgesia challenges in the post anaesthetic care unit (PACU)

The protocol below is a suggestion for therapeutic options in patients who have pain which is difficult to manage in the PACU. It represents a reflection of the evidence-based clinical practice and experience of a large UK university teaching hospital. The protocol begins with the reminder that pain is an unpleasant sensory and emotional experience and a holistic approach taking into account the patients ideas, concerns and expectations around the management of their pain is crucial. Patients should be reassured that a pain score of mild to moderate pain is what is realistically achievable and that postoperative pain does not mean that something untoward has happened.

Gabapentin, ketamine and clonidine all have a reasonable evidence base for analgesia but conventional analgesics provides adequate pain relief to most patients without the need for these adjuvants, all of which have potential side effects.

The difficult pain pathway should be supervised by a senior anaesthetist or critical care doctor

The following options below should be considered in patients in the PACU who have received regular post op paracetamol and if possible an intravenous non-steroidal anti-inflammatory drug and an opiate such as, morphine >40 mg, oxycodone >40 mg or fentanyl >300 μg.

Step 1

(1) Gabapentin 600 mg orally or 300 mg if patient is felt to be frail.
(2) A regional or local anaesthetic technique.

If, after 30 minutes gabapentin is not successful or is contraindicated then other options that can be considered are:

Step 2

(1) Further opiates (consider alternate opiate e.g. fentanyl).
(2) Ketamine up to a maximum dose of 300 μg/kg with a starting dose of no more than 10 mg and repeated to the maximum of 30 mg.
(3) Clonidine 150 μg diluted up into a 20 ml syringe in sodium chloride 0.9% given in 2 ml boluses to a maximum of 3 μg/kg.
(4) Magnesium 8 ml given intravenously over 20 minutes as per difficult pain prescription in electronic prescribing system (EPR).

Any patient who requires adjuvant therapy such as gabapentin or further therapies please alert the acute pain team.

Persistent post-surgical pain

Persistent postsurgical pain, which is often neuropathic, is common and results in significant disability. Factors which may predispose to the development of persistent postsurgical pain include anxiety, pain catastrophizing, depression, psychological vulnerability and stress.[3] Other risk factors include the severity of presurgical chronic pain and the severity of postsurgical acute pain as well as intraoperative nerve injury.

Perioperative ketamine infusions may reduce the incidence of chronic postsurgical pain. Perioperative pregabalin and gabapentin may reduce chronic postsurgical pain although there is controversy with regards to doses timing and duration of treatment. ◆

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