Management of acute pain

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Abstract

Acute pain is a common feature in the presentation of surgical and traumatic pathology and in postoperative patients. In pathological presentations acute pain may have a protective role serving as a warning sign, with muscle spasm helping to limit movement and prevent further injury. Acute postoperative pain can hinder recovery due to limited mobility and may lead to a range of complications, increasing patient morbidity and mortality. Timely and effective management of acute pain is therefore imperative. An acute pain service (APS) is able to assist in the management of complex patients and those with specific invasive analgesic interventions. However, the immediate prescribing is the responsibility of the admitting surgical doctor and therefore this article aims to give an overview of the considerations needed to ensure safe and effective management of acute pain.

Keywords Acute pain; analgesia; nociceptive pain; postoperative pain; pain management

Introduction

Definition of pain

The International Association for the Study of Pain (IASP) defines pain as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage'.[1]

Description of pain

There are two types of pain that are commonly described. Nociceptive pain is usually described by patients as sharp, aching or throbbing. It is well localized and usually relates to an underlying injury or inflammation. The second type of pain is neuropathic and this is often described as a burning, stabbing or shooting sensation. It can be of a diffuse nature and may radiate away from the main area of pain. This is an important distinction to make as the drugs used to treat pain depend upon these underlying features.

Timing of pain

Acute pain is distinct from chronic pain in that it is provoked by a specific injury or pathology, serves a useful biological purpose and is self-limiting. Acute pain is typically of short duration, rapid or recent onset and may be very severe.[2] Chronic pain is described as pain of greater than 3 months' duration and is likely to have neuropathic qualities.

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Pathophysiology of pain

The sensory experience of pain is complex, involving the peripheral and central nervous systems, with multiple complex neurotransmitter and receptor mediated events. Furthermore, there are significant emotional and psychological modifiers of the acute pain experience.

Peripheral nervous system

The specialized primary afferent nerve endings in the skin and visceral tissue involved in pain sensation are termed nociceptors. These receptors transduce chemical and physical signals so that when a threshold level is reached an action potential is generated, which is propagated along the nerve by the opening of voltage gated sodium channels, through the cell body in the dorsal root ganglia and into the spinal cord. The primary nerve axons are classified as A-delta which are myelinated and rapid conducting, and C fibres which are non-myelinated and slow conducting.

Peripheral sensitization is a process which occurs in response to repeated or prolonged stimulation of nociceptors through tissue injury. This involves the release of inflammatory mediators including prostaglandins, leukotrienes, histamine and bradykinin often collectively called the inflammatory soup, resulting in lowering of the threshold for action potentials and increased pain signalling.

Central nervous system

A complex pathway relays pain inputs from the spinal cord to the cerebral cortex. The afferent nociceptor fibres synapse with secondary neurons in the dorsal horn. The dorsal horn nuclei can be anatomically divided into ten layers known as Rexed laminae. A-delta and C fibres synapse predominantly in Rexed laminae I and II, releasing neurotransmitters including glutamate and substance P. Signal output from the dorsal horn is modified by further neuronal inputs including the action of descending inhibitory pathways from the midbrain and medulla utilizing a range of neurotransmitters including G-aminobutyric acid (GABA), serotonin and noradrenaline. The interplay at the dorsal horn is thought to be key to the process of central sensitization describing increased responsiveness of nociceptor neurons to their normal input or responsiveness to sub-threshold stimuli. Central sensitization is thought to involve activation of the N-methyl D-adenine (NMDA) receptor which causes hyperexcitability of neurons. Other processes involved include microglial activation and reduction of descending inhibition.

Secondary afferent neurones decussate in the spinal cord and travel via the contralateral spinothalamic and spinoreticular tracts. Spinothalamic tract neurons synapse with third-order neurons in the thalamus, relaying the signal to the cortex. Spinoreticular tract neurons predominantly synapse in the brainstem reticular formation with further projections to the thalamus, hypothalamus and cortex, and are involved in the complex emotional and physiological interplay termed pain processing.

The importance of effective pain management

Recent studies have illustrated the significant burden of unrelieved acute pain in the USA surgical population, with surveys

finding more than 80% of patients reporting moderate to severe postoperative pain.[3] The consequences of unrelieved acute pain included:

- increased patient morbidity
- increased healthcare costs and utilization
- worsened patient short-term experience
- poor longer term outcomes through delayed recovery
- development of chronic pain syndromes.

The physiological complications of poorly managed acute pain can be found in Table 1.

Assessment of pain

Accurate assessment of pain is central to effective management; however, this is frequently difficult and studies have indicated that it is often performed poorly.[3,4] It is important to remember that pain is a subjective experience and is influenced by emotional and psychological factors.

An accurate pain history from the patient is important this is made simple using the mnemonic SOCRATES:

- site
- onset
- character
- radiation
- associated symptoms
- time course/pattern of pain
- exacerbating/relieving factors
- severity.

The history should also include details of analgesia used by the patient both acutely and chronically. Patients who are long-term users of analgesia, particularly opiate-based medicines are likely to have increased analgesia requirements in an acute setting.

As pain is a subjective experience, measurement of the patient's reported pain using scales represents a useful assessment technique. There are a number of simple uni-dimensional self-reporting scales which have been validated for use in acute pain.[4]

Complications of poorly controlled postoperative pain

Timing	System	Mechanism	Complication
Immediate	Respiratory	Impaired cough, reduced functional residual capacity	Atelectasis Pneumonia
	Cardiovascular	Increased sympathetic activity	Myocardial ischaemia
Early	Gastro-intestinal	Increased sympathetic activity	Ileus Malnutrition
	Metabolic	Inflammation Protein catabolism Reduced blood flow to skin	Poor wound healing
	Other	Venous stasis	Thromboembolism
Delayed	Neurological	Central pain sensitization	Chronic pain syndromes
	Musculoskeletal	Decreased mobility	Muscle wasting

Table 1

An example is numerical rating scale (NRS) using a 0–10 point scale with 10 representing severe pain and 0 no pain. Patients verbalize or write the number that they feel indicates their level of pain.

The use of scales is dependant on the patient's capacity to understand the scale and express their pain accordingly. This can prove challenging with certain groups of patient, commonly paediatric patients and the cognitively impaired elderly.

Pictorial scales such as the Wong-Baker Faces Pain Rating scale[5] (Figure 1) may be more successful at allowing self-reporting in children. Although self-reporting remains a gold standard in pain assessment this may not be possible in very young infants. For children below the age of 3 years observational scales such as the FLACC pain assessment tool may be more suitable. FLACC measures five observed categories of pain behaviours: facial expression, leg movement, activity, cry and consolability against a 0–2 point scale giving an overall score out of 10.

Older adults with cognitive impairment represent another population group in which pain assessment can also be challenging, particularly in the postoperative period or episodes of acute illness. As with infants, observation scales may be more reliable in patients with severe cognitive or communication impairment. The Abbey pain scale combines observed behavioural and physiological parameters to give a numerical pain assessment.[6]

Management of acute pain

Management strategies will vary depending on the exact presentation of acute pain. In certain situations surgery, physiotherapy or adjuvant therapies may represent the most important analgesic approach; however, in the majority of cases the use of pharmacological analgesia remains the mainstay of management.

Pharmacological analgesia

The WHO analgesic ladder (Figure 2) provides a simple, structured starting point for the pharmacological management of pain. This system provides a useful guide for acute nociceptive pain. If patients are experiencing neuropathic pain, then an adjunct medication is usually necessary and it is important to take advice from the APS.

WHO step 1: Simple analgesia

Paracetamol: is a widely used first-line simple analgesic and antipyretic. It is commonly used in oral preparation for mild to moderate pain but is also effective when used as part of a multimodal approach to treat severe acute pain. Proposed mechanisms of action include inhibition of the synthesis of prostaglandins by cyclooxygenase and activation of descending inhibitory serotonin pathways. Oral and rectal administration of paracetamol produce analgesia within 40 minutes with peak effect at 1 hour. However large variations in bioavailability make onset time and duration of action unpredictable. The intravenous route provides an onset of analgesia within 5 minutes, peaking at 30–40 minutes with a duration of 4–6 hours. Efficacy is comparable to that of standard doses of NSAIDs and weak opioids. Paracetamol has a synergistic action in combination with a

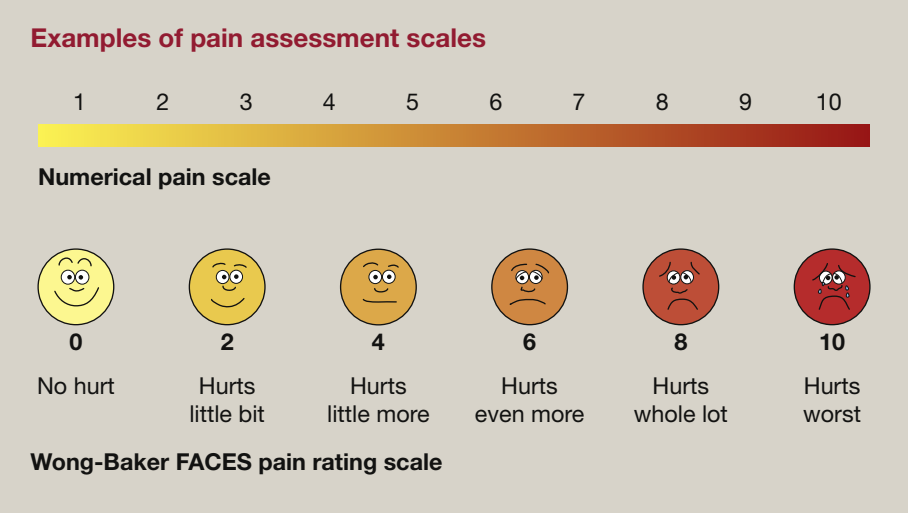

Figure 1

number of other analgesics including NSAIDs and opioids, and is therefore useful as an opioid sparing agent. Paracetamol is safe in the recommended doses with minimal side effects. The main toxic effect of overdose is hepatotoxicity which can cause acute liver failure.

NSAIDs: are widely used analgesic and anti-inflammatory medicines which inhibit the cyclooxygenase enzymes COX-1 and COX-2 to disrupt synthesis of prostaglandins including PGE2, and thromboxane A2 which have a role particularly in peripheral sensitization of pain. COX-1 is a widely expressed isoform of the COX enzyme in contrast to the inducible isoform COX-2 which is undetectable in most tissues but significantly upregulated in inflammation.

Traditional NSAIDs such as ibuprofen and diclofenac are non-selective for COX-1 or COX-2. Recent formulations such as parecoxib and celecoxib are selective for COX-2. COX-2 selective inhibitors are hypothesized to have safer side effect profiles in comparison to traditional NSAIDs[8] (Table 2).

NSAIDs are highly protein bound and should be used in caution with other highly protein bound medications such as Phenytoin or Warfarin as their effects may be potentiated.

For a summary of these simple analgesics including doses and routes of administration see Table 2.

WHO Step 2: weak opioids

Opioid analgesics have been in use for centuries and remain the mainstay in treatment of acute pain. An increasing array of medications are available varying in their potency, speed of onset, duration of action, route of administration and opioid receptor affinity. It is important to keep up-to-date with new formulations to ensure patients get safe, effective analgesia.

Definition: the term *opioid* describes all medications which exert an action at opioid receptors, while *opiate* refers to those which are naturally occurring, such as morphine.

Opioid receptors are a family of G-protein coupled receptors. There are four recognized receptors with the current nomenclature MOP, KOP, DOP and NOP. Activation of these receptors

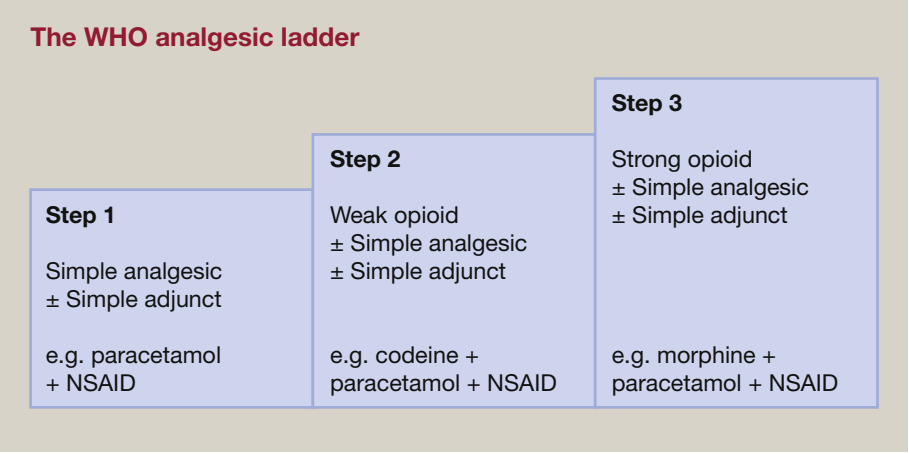

Figure 2

Properties of simple analgesics

Drug	Routes of administration	Dosage	Mechanism	Adverse effects
Paracetamol	PO, IV, Rectal	1G QDS	Uncertain	Hepatotoxicity in overdose
Ibuprofen	PO	400 mg TDS	Nonselectice COX inhibitor	Gastric ulceration Renal impairment Exacerbation asthma Platelet dysfunction
Diclofenac	PO, IV, Rectal	75–150 mg daily in divided doses	Nonselective COX inhibitor	
Parecoxib	IV	40 mg Max 80 mg day	COX-2 inhibitor	Reduced risk upper GI complications Possible increased risk MI

Table 2

leads to reduced neuronal cell excitability and a reduction in transmission of nociceptive nerve impulses. The majority of opioid drugs exert their analgesic action as well as their broadly similar adverse effects through the MOP receptor. The adverse effects of opioids include respiratory depression, sedation, euphoria, nausea and vomiting, gastric stasis, constipation, pruritis, meiosis and urinary retention. An overview of the pharmacology of the opioids can be found in Table 3.

Codeine: is a weak opioid prodrug which is metabolized to morphine by the cytochrome p450 enzyme CYP2D6. Genetic heterogeneity exists in CYP2D6 metabolism such that a small proportion of the population are non-metabolizers in which codeine is ineffective. More importantly, a small proportion are ultrarapid metabolizers in which the prodrug is rapidly converted to morphine placing the individual at an increased and unpredictable risk of respiratory depression. For this reason, codeine is contraindicated in children under 12 years, or with any history of sleep apnoea, and in breast feeding mothers as it is secreted in breast milk. Another common side effect is constipation which particularly limits its use in the elderly and in bowel surgery.

Tramadol: is a weak agonist at opioid receptors, inhibits reuptake of synaptic noradrenaline and serotonin, and activates descending inhibitory pain pathways. Tramadol causes less

Properties of common opioid drugs used in acute pain management

Drug	Route	Typical starting dosage	Conversion factor (to oral morphine)	Characteristics	Adverse effects/cautions
Codeine	PO	30–60 mg QDS	0.13	Slow onset Weak opioid	Variation in metabolism Constipation
Tramadol	PO, IV	50–100 mg QDS	0.2	Additional pain modulation through inhibition of serotonin/ noradrenaline reuptake	Dysphoria/hallucinations Risk of serotonin syndrome
Morphine	PO Immediate Mod-release IV/IM	2.5–20 mg 10–20 mg Titrated to effect 2.5–10 mg PCA 1 mg bolus 5 min lockout	1 3	Onset 30 mins Onset 5–10 mins Duration approx. 4 hours	Metabolites accumulate in renal failure
Diamorphine	SC, IM, IV	2.5–5 mg	6	Rapid onset Highly fat soluble so useful neuraxial adjunct	
Oxycodone	PO Immediate Mod.release IV	2.5–5 mg 10 mg BD 1–10 mg Titrated PCA 1 mg bolus 5 min lockout	1.5 3	Onset 15 mins Rapid onset May be useful in patients intolerant of morphine	
Fentanyl	IV	25–100 microg PCA 15–25 microg bolus 5 min lockout	0.2 (micg)	Very rapid onset Short duration of action Better tolerated in renal disease	Potent respiratory depression

Table 3

respiratory depression and gastrointestinal adverse effects than morphine but can cause dysphoria and hallucinations particularly in the elderly. It has potential to interact with serotonin reuptake inhibitors and tricyclic antidepressants causing serotonin syndrome therefore Tramadol should be avoided in these patients.

WHO Step 3: strong opioids

Morphine: is available in a range of preparations for administration via numerous routes. The oral route has a 30% bioavailability due to first pass metabolism, with an onset of 15–60 minutes and duration of 3–6 hours. Various slow release preparations of oral morphine are also available. Morphine is rarely used in the UK for epidural or intrathecal use as its low lipid solubility leads of increased risk of delayed respiratory depression.

Morphine is metabolized by glucuronidation to morphine-3-glucoronide and morphine-6-glucuronide. These metabolites are both renally excreted and hence may accumulate in renal disease.

Diamorphine: is a synthetic prodrug which is inactive prior to deacetylation to morphine and 6-monoacetylmorphine. Subsequent metabolism is as for morphine. In acute pain settings diamorphine is typically given by the intravenous or intramuscular routes with a potency of twice that of morphine. It has much greater lipid solubility than morphine and therefore has a more rapid onset of action particularly when given via the intramuscular route. It is also available as an intranasal spray which is useful in situations where intravenous access may be difficult. The increased fat solubility compared to morphine confers a reduced risk of delayed respiratory depression when used via the intrathecal or epidural routes. It is used as a common adjunct to neuraxial anaesthesia in obstetrics and as a primary intrathecal analgesic in abdominal surgery.

Fentanyl: is a synthetic phenylperidine derivative which is approximately 100 times as potent as morphine. It is most commonly used via the intravenous route for perioperative analgesia at a dose of 1–2 μg/kg. It is also frequently used as an adjunct in epidural and intrathecal anaesthesia and is available in sublingual and transdermal formulations. Its low oral bioavailability precludes its use by this route. Fentanyl undergoes hepatic metabolism to inactive norfentanyl which is cleared by urinary excretion. For this reason, fentanyl particularly when administered via patient-controlled analgesia (PCA) represents a useful analgesic option in patients with renal impairment.

Alfentanil and remifentanil are compounds related to fentanyl, which have progressively more rapid onsets and shorter durations of action. These pharmacokinetic properties combined with their increased risk of respiratory depression have almost exclusively confined these drugs to anaesthetic intraoperative practice.

Oxycodone: is a synthetic opioid with a high selectivity for the MOP receptor. It is available in various oral formulations of immediate or delayed release. It is also available as an intravenous formulation and is frequently used in PCA.

Analgesic adjunct medications

Several medicines are commonly used as adjuncts to opioids in multi-modal analgesic strategies (Table 4). These are often used intraoperatively by anaesthetists or prescribed by the APS. The most commonly prescribed adjunct are the gabapentinoids, gabapentin and pregabalin which can form part of a multi-modal protocol. Common side effects include sedation, dizziness, somnolence and nausea and vomiting. Gabapentin has a greater evidence base for its ability to reduce pain scores and opiate usage, but the newer pregabalin provides more potent analgesia and a more favourable pharmacokinetic profile with increased bioavailability and duration of action.

It is important postoperatively to note if patients have been prescribed a short course of an adjunct, for example, pregabalin following orthopaedic surgery. These short courses are for the initial postoperative period and it is unusual for patients to require this medication upon discharge.

Timing of medications

A smooth analgesic profile is achieved using a regular, timed administration of medications. Any peaks in pain can then be effectively managed with a PRN medication. It is important to make a daily assessment of pain looking at the last 24 hours of recorded pain scores, analgesia administered and the patients perceived benefit including side effects. It is then possible to titrate the analgesia to the patient experience and monitor the patient for side effects and prescribe any adjuncts needed. For example, a patient who is prescribed codeine should always be prescribed a PRN laxative as the constipating effect of codeine is widely known and can lead to severe abdominal pain.

Components of multi-modal analgesia include regular simple analgesia and the use of non-opioid adjuncts such as ketamine, gabapentinoids and systemic infusions of local anaesthetic for pain with neuropathic features. Pain protocols for major orthopaedic and abdominal surgeries often initiate these medicines in the preoperative period.

Route of administration

Mild to moderate pain is typically well managed with oral analgesia following the WHO pain ladder. Severe pain and those patients who are truly nil by mouth will need an alternative strategy.

Intravenous PCA: a PCA is an infusion pump through which patients can self-administer bolus doses of opioid with or without a background infusion. Morphine, oxycodone and fentanyl are the most common analgesics given by PCA. A Cochrane review found that PCA use was associated with better pain control, better patient satisfaction and higher opioid use.[9] Possible disadvantages include long periods of inadequate analgesia during sleep, inappropriate use by relatives and the possibility of respiratory depression. As with any patient receiving intravenous opioids, supplemental oxygen should be supplied with regular monitoring of respiratory rate and blood pressure. Many hospital protocols mandate the prescription of an opioid receptor antagonist such as Naloxone alongside PCA.

Intrathecal: opioids can provide high-quality analgesia for surgery involving the abdomen, pelvis or lower limbs. Highly lipid

Properties of analgesic adjunct medications

Drug	Receptor	Route	Typical starting dosage	Uses	Adverse effects/cautions
Ketamine	NMDA	IV, IM Oral	0.1 mg/kg 20 mg QDS	Perioperative pain Hyperalgesic states Non response to opioids	Dysphoria hallucinations Tachycardia Hypertension
Magnesium	NMDA	IV	50 mg/kg	Intraoperative analgesic adjunct	Hypotension
Clonidine	$\alpha 2$	IV PO	25 micg 50-100 micg TDS	Perioperative pain Hyperalgesic states Neuraxial block adjunct	Hypotension Bradycardia Sedation
Gabapentin	GABA	PO	300 mg OD (Max 3.6G daily)	Adjunctive postoperative analgesia for major surgery Neuropathic pain	Sedation dizziness nausea
Pregabalin	GABA	PO	75 mg BD (Max 300 mg Daily)	Adjunctive postoperative analgesia for major surgery Neuropathic pain	Sedation dizziness nausea
Lignocaine (IV infusion)	Voltage gated Na channels	IV	1 mg/kg/hr infusion	Perioperative analgesic adjunct	Risk of LA toxicity. Only for use in fully monitored anaesthetic recovery areas.

Table 4

soluble opioids such as diamorphine or fentanyl are frequently added as an adjunct to spinal anaesthesia for obstetric, orthopaedic and general surgery procedures. Increasingly, intrathecal administration of diamorphine is being utilized where postoperative analgesia rather than anaesthesia is the primary aim. Intrathecal opioids mechanism of action is through pre- and postsynaptic opioid receptors in the dorsal column of the spinal cord. Adverse effects are identical to those for systemically administered opioids and caution should be used in administering supplemental opioids when a patient has received an intrathecal opioid in theatre.

Location of patient

The location of the patient is important when deciding the analgesia to prescribe and how to prescribe it. For an inpatient, the type and frequency of observations that can be conducted on the ward will dictate which analgesia can be safely given on that ward. It is also important to note the level of training of the nurses present on the ward. Many wards will not be able to nurse patients on PCAs, or those with an epidural in-situ, if the nursing staff have not had appropriate training in managing these complex interventions. These patients may have to be monitored in a high-dependency environment. Patients should be moved to the clinical area considered safe for them to have the analgesic plan judged most suitable.

For day-case patients and those discharged from the emergency department, it is important they do not get strong opiates such as morphine to take home if this is not a usual prescription. This is due to the serious potential side-effects and lack of monitoring. It is appropriate to keep a patient in for overnight observation if, following day-case surgery, their acute pain requires strong opiates.

Acute pain service (APS)

In the perioperative context the use of multi-modal analgesic approaches with the input of dedicated APS leads to improved patient outcomes including reduced pain scores, reduced opiate usage and earlier mobilization. The APS will usually review postoperative patients with severe pain or an analgesic device or specialist anaesthetic technique (e.g. epidural).

The APS may take referrals for patients with chronic pain who present with a flare up or an acute unrelated pathology. These patients can be challenging to manage as they may already be on strong analgesia for chronic pain. Patients who have an analgesic dependence problem will often need the advice of the APS.

Adjuvant therapies

In addition to pharmacological analgesia there is evidence for many adjuvant therapies including:[7]

- transcutaneous electrical nerve stimulation (TENS) (e.g. acute back pain)
- distraction techniques such as listening to music, reading, watching television (e.g. post-surgical)

However, there is no robust scientific evidence to support the use of:

- management of expectations in preoperative information sessions (e.g. orthopaedic joint replacement programmes)
- relaxations techniques (e.g. postoperative)

Specialist local anaesthetic techniques

Local anaesthetics can be defined as drugs which reversibly prevent nerve impulse transmission in the area to which they are applied. Their mechanism of action is to reversibly block voltage gated sodium channels in neuronal cells and prevent nerve impulse propagation. They may be used to provide analgesia for surgical procedures alone or as part of a multimodal strategy. Local anaesthetics are classified as amides or esters depending on their chemical structures. Amide local anaesthetics are more frequently encountered in clinical practice and include Lignocaine, bupivacaine, levo-bupivacaine and ropivacaine. Cocaine and amethocaine are examples of ester local anaesthetics.

Techniques for use of local anaesthetics include local wound infiltration, peripheral nerve blockade, neuraxial blockade and systemic intravenous infusion.

Peripheral nerve blockade

Peripheral nerve blockade involves the deposition of local anaesthetic around nerves to elicit regional anaesthesia. Typically this involves the use of ultrasound to identify nerves, nerve plexi or fascial compartments through which nerves run. Single shot blocks are frequently performed to provide analgesia for an increasing array of surgeries on all parts of the body. A disadvantage of this approach is that frequently the block will wear off at an inconvenient time in the postoperative period resulting in inadequate analgesia. An increasingly prevalent solution to this problem is the placement of continuous infusion or intermittent bolus nerve catheters, examples of these include:

- fascia iliaca catheters for hip surgery
- interscalene catheters for shoulder surgery
- paravertebral catheters for major breast surgery and rib fractures
- rectus sheath catheters for laparotomies
- femoral and sciatic nerve catheters for lower limb amputations.

The use of these catheters may extend for several days into the perioperative period and therefore they are likely to be encountered on surgical wards. Management is generally carried out by anaesthetists and the APS; however, all clinicians should be aware of the potential complications of nerve catheters which can include local anaesthetic toxicity, motor block and the masking of surgical complications such as compartment syndrome or thromboembolism.

Neuraxial blockade

The technique most likely to be encountered on surgical wards is epidural analgesia, the placement of a continuous infusion catheter into the epidural space. They are used for surgeries on the trunk and abdomen where prolonged analgesia is required including:

- major abdominal surgery
- major vascular surgery such as aortic aneurysm repair
- major orthopaedic surgery such as hip revision
- thoracic surgery.

Epidural analgesia is particularly useful for patients in whom respiratory compromise would be especially undesirable such as those with pre-existing respiratory disease. As with peripheral nerve catheters, epidurals are typically managed by anaesthetists and the APS; however, there are issues all clinicians should be aware of. Chief among these is the management of anticoagulation, in particular around the removal of catheters, where local protocols should be observed. A dense motor block in these patients warrants an urgent call to an anaesthetist as it may indicate an underlying epidural haematoma or abscess, both of which need urgent imaging and treatment.

Conclusion

Surgeons admit patients with acute pain and are often the first port of call for postoperative pain problems. These patients need a rapid but thorough assessment of their pain. It is important to use a systematic approach when taking a history and deciding if the pain is acute or chronic, nociceptive or neuropathic as this will affect the analgesia prescribed. It is important to consider the pharmacological interactions with medications the patient is currently taking, allergies and the patients location. Safety is paramount and it is always better to titrate analgesia slowly and monitor for adverse events. The APS play a significant role in the management of acute surgical pain and can support the management of complex patients. ◆

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