Local and regional anaesthetic techniques in wound management

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Abstract

Adequate pain control is vital in perioperative care. Pain affects respiratory and cardiovascular function with knock-on effects on many other organs and systems. Surgery is also recognized as one of the most frequent causes of chronic pain with surgical approach and level of pain experienced being modifiable risk factors for the development of chronic post-surgical pain.

Local anaesthetic can be used to facilitate pain relief either by local infiltration or nerve blocks, increased mobility and a better perioperative and postoperative experience for the patients. There are two pharmacological types, esters and amides, that have different uses. Commonly used blocks are brachial plexus block, Bier's block, femoral nerve block, spinal block and epidurals. These can be used alone or in combination with a general anaesthetic.

Keywords Block; epidural; infiltration; lipid rescue; local anaesthetic; toxicity

Pain control has a direct impact on the ability to inflict a surgical wound, the site of creation of such a wound, and the subsequent postoperative management of patients with wounds. The development of persistent pain following surgery is receiving growing recognition.[1,2] A number of risk factors for the development of chronic post-surgical pain have been identified such as moderate to severe pain preoperatively or postoperatively and surgical approaches with risk of nerve damage.[3,4] Therefore adequate perioperative analgesia is a priority for all involved in patient management to alleviate patients' suffering and promote their prompt recovery. The management of postoperative pain is getting more organized with acute pain teams in most hospitals. Broad ranges of options are available for different clinical situations, and these must be tailored to individual patient's needs.

Local and regional techniques add substantial additional benefit in pain management of surgical patients compared to general anaesthesia using opioids. This occurs not only through improved pain control, of which the psychological benefits are self-evident, but important physiological benefits arise through the minimization of the stress response during surgery, which affects multiorgan systems.

Good pain relief allows adequate respiratory function by avoiding splinting of the diaphragm and encouraging recruitment of small airways, thereby preventing atelectasis and pneumonia. In terms of cardiac function, sympathetic stimulation is reduced through adequate analgesia, thus decreasing cardiac work and systemic vascular resistance, and subsequently improving splanchnic and renal perfusion, with a consequent minimal reduction in glomerular filtration rate and a shorter duration of ileus. In addition, reduced opiate requirements minimize the incidence of nausea and vomiting in the postoperative period, allowing early feeding. Furthermore, pain management is also of relevance to the process of wound healing itself through improving tissue oxygenation, reducing vasoconstriction and optimizing the immune response. Good analgesia also allows for early mobilization therefore contributing to minimization of postoperative thromboembolic events.

The benefits of local and regional techniques render them key components of enhanced recovery practices and therefore are of economic as well as clinical interest (see *Acute Pain Management: Scientific Evidence; Chapter 5 Regionally and locally administered analgesic drugs*[5]).

Local anaesthesia

Local anaesthesia is the simplest method, which strives to create a small area of anaesthesia through application of a substance (local anaesthetic), which will cause a reversible loss of the ability to conduct nerve impulses. This can be achieved by subcutaneous infiltration, or topical application of, a local anaesthetic drug.

The mechanism of action of local anaesthetic is to block the action potential of nerve impulses by inhibiting the influx of sodium ions through sodium channels (Figure 1). Conduction of a nerve impulse along a nerve fibre is triggered by a noxious stimulus at the sensory nerve terminal and occurs as an influx of sodium ions cause depolarization of the nerve cell. Repolarization occurs rapidly through the loss of potassium ions from the cell and active expulsion of sodium through the sodium/potassium pump. The conduction of the nerve impulse propagates along the nerve fibre as the change in local voltage stimulates opening of the next sodium channel.

Local anaesthetic agents block the sodium channel from within the cell, which prevents sodium ion influx and therefore blocks depolarization. To cross the cell membrane the local anaesthetic agent, which is ionized in solution, becomes non-ionized within the tissues. The local anaesthetic is then re-ionized within the cell to bind to the sodium channel. Local anaesthetics are less effective in infected or inflamed tissues as the acidic environment of these tissues prevents them from becoming non-ionized and therefore reduces their ability to cross the nerve cell membrane.

Local anaesthetic agents

There are two groups of local anaesthetics of differing chemical structure: amides (e.g. lidocaine, prilocaine and bupivacaine) and esters (e.g. amethocaine, benzocaine and cocaine).

Esters are infrequently used, as they are more toxic and more prone to causing allergic responses. Indications for the use of

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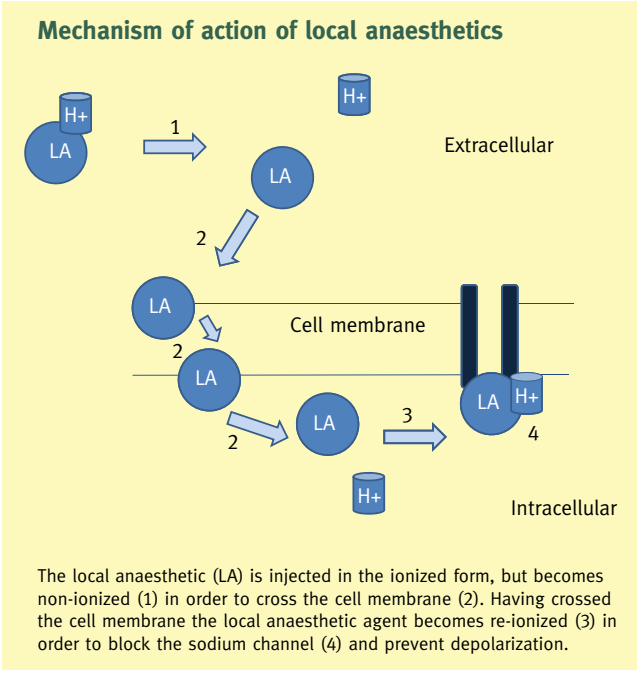

Figure 1

esters include amethocaine lozenges for the oropharynx and cocaine for nasal surgery.

Amides, such as lidocaine, are more commonly used, and feature in a variety of techniques. Lidocaine has a relatively fast onset and its duration of action is between 60 and 180 minutes. The maximum dose of lidocaine is 3 mg/kg, maximum 200 mg (without adrenaline) and 7 mg/kg, maximum 500 mg (with adrenaline).

The maximum safe dose of lidocaine (without adrenaline) for a 50 kg individual can be calculated as follows:

- $3 \times 50 = 150$ mg
- 1% solution contains 10 mg/ml
- therefore 15 ml of 1% would be the maximum dose
- if you were using lidocaine with adrenaline the maximum dose would be 35 ml of 1% solution.

This change in dosage applies to lidocaine, as it is a very good vasodilator increasing the blood levels sooner than when mixed with adrenaline, which reduces the absorption due to its vaso constrictive effect.

Bupivacaine (Marcain) is more potent than lidocaine and has a longer duration of action but slower speed of onset. The maximum safe dose with or without adrenaline is 2 mg/kg, maximum 150 mg in 4 hours. In the example given above this would be 20 ml of 0.5% bupivacaine = 2 mg/kg for a 50 kg individual. Levo-bupivacaine (Chirocaine) is the S(−) enantiomer of bupivacaine and has a better safety profile. In addition it has a slightly longer duration of anaesthesia. Caution should be taken in patients with reduced liver function where lower doses of amides should be used as these agents are metabolized by the liver.

Adrenaline is used as a vasoconstricting agent in local anaesthetic preparations to prolong their rate of absorption and therefore the duration of analgesia with reduced toxicity. Adrenaline is typically used in local infiltration and nerve blockade techniques. The maximum safe adult dose is 50 ml of 1:200,000 solution, although this should be reduced in patients with ischaemic heart disease. Adrenaline is absolutely contraindicated when infiltrating the extremities (digits etc) due to the risk of tissue ischaemia.

Overdose

Toxicity occurs following administration of an incorrectly calculated dose, accidental intravenous injection or rapid absorption of a normally safe dose. Toxicity can be local or systemic, affecting the central nervous or cardiovascular systems. Local toxic effects of ester agents include an inflammatory response, particularly if the pH of the solution is less than five. Lidocaine can cause neurotoxicity in high concentrations and locally can be the cause of cauda equina syndrome following spinal infiltration. Neuropathy can occur as the infiltrating needle penetrates the nerve and intraneural injection occurs.

Central nervous system effects occur as local anaesthetics easily cross the blood–brain barrier. Large doses can block inhibitory neurons causing perioral parasthesia, tinnitus, agitation, restlessness, and tremors, even convulsions and coma. Management focuses on oxygenation and seizure control with paralysis, ventilation and anticonvulsants.

Cardiovascular system effects occur as local anaesthetics decrease the excitability of cardiac muscle causing slow impulses manifesting as bradycardia with a prolonged PR and QRS on an ECG. Cardiac output is also reduced, particularly with bupivacaine which blocks calcium channels in addition to sodium channels, thereby enhancing this effect.

The management of local anaesthetic overdose consists of:

- AIRWAY – maintenance of a patient's airway, which may involve intubation
- BREATHING – administration of high flow oxygen, ventilation may be required
- CIRCULATION – supine position, intravenous fluids, atropine to treat bradycardia, inotropes and vasopressors if necessary, cardiopulmonary resuscitation in the event of cardiac arrest
- DISABILITY – convulsions should be treated with diazepam in the first instance.

The Association of Anaesthetists of Great Britain and Ireland (AAGBI) have promoted the use of lipid emulsions, such as Intralipid, against the cardiotoxic effects of local anaesthetics, with the technique being called lipid rescue. Although the mechanism of action of lipid rescue is not completely under-stood, it may be that the added lipid in the blood stream acts as a sink, allowing for the removal of lipophilic toxins from affected tissues.

Regional anaesthesia

Regional anaesthesia encompasses techniques that result in anaesthesia of a large area of the body such as a limb. This is achieved by several means:

- nerve block – during which local anaesthetic agents are infiltrated directly around the nerves
- Bier's block – intravenous application of local anaesthetic using a tourniquet resulting in a limb block
- epidural – anaesthetic agents are injected into the extradural space
- spinal – anaesthetic agents are injected into the subarachnoid space.