Local anaesthesia outside the operating room
手術室外的局部麻醉法

An increasing number of minor surgical procedures are performed under local anaesthesia in clinical settings outside the operating room, where monitoring and resuscitation equipment—as well as personnel skilled in resuscitation—may not be readily available. Serious adverse effects and even fatalities may result from the use of local anaesthetic agents, arising from a variety of causes such as systemic toxicity, allergy, vasovagal syncope, and reaction to additives present in the local anaesthetic. This article briefly reviews the pharmacology of local anaesthetic agents, and describes various techniques commonly used for local anaesthesia, with special emphasis on safety. Clinical features of toxicity, and its differential diagnosis and management, are also discussed.

In hand, wiping the process of a small hand, and the potential to avoid and go through, is still not possible. Some adverse effects even result from the use of local anaesthetic agents, arising from a variety of causes such as systemic toxicity, allergy, vasovagal syncope, and reaction to additives present in the local anaesthetic. This article briefly reviews the pharmacology of local anaesthetic agents, and describes various techniques commonly used for local anaesthesia, with special emphasis on safety. Clinical features of toxicity, and its differential diagnosis and management, are also discussed.

Introduction

Since the introduction of cocaine into clinical practice by Koller in 1884, the history of surgery has changed dramatically. Pain-free surgery can now be accomplished under local anaesthesia, with improved patient comfort and cooperation. Local anaesthesia is defined as the reversible loss of sensation in a relatively small or circumscribed area of the body, achieved by topical application or injection of agents that either depress the excitation of nerve endings or inhibit the conduction of impulses along a peripheral nerve. While there are many ways of producing local anaesthesia, such as topical application of ice or vapo-coolant ethyl chloride, local anaesthetics (LAs) are by far the most commonly used.

Traditionally, surgery is performed in the operating room, where monitoring and resuscitation equipment, as well as personnel trained in resuscitation, are readily available. However, because of the increased safety of surgery and anaesthesia, and socio-economic factors, an increasing number of minor surgical procedures are being performed under local anaesthesia outside the operating room. Since potential fatal complications can occur with the use of LAs, a good understanding of LA pharmacology is the key to safe use of these agents. This article reviews the pharmacology, toxicity, and clinical aspects of LAs applicable to settings outside the operating room.

Pharmacology of local anaesthetics

Classification and metabolism

Local anaesthetic agents contain a hydrophobic aromatic ring, which is connected to a hydrophilic secondary or tertiary amine by an intermediate alkyl chain made up of either an amide or ester bond. This linkage to the aromatic ring forms the basis for classifying LAs into amino-esters (eg cocaine, procaine, chloroprocaine,
amethocaine) and amino-amides (eg lignocaine, bupi-
vacaine, etidocaine, ropivacaine), and is also an important
determinant of biodegradation. The amino-ester compounds
are readily hydrolysed in plasma by pseudocholinesterase
to para-aminobenzoic acid (PABA), whereas the amino-
amide compounds are more slowly metabolised by the
hepatic microsomal enzymes to inactive metabolites.

**Physicochemical properties**
All LAs are weak bases. In solution there exists a chemical
equilibrium between the non-ionised form (B) and the ion-
ised form (BH+). At a specific pH (pK_a), the concentration
of the non-ionised form is equal to the concentration of the
ionised form of the LA, and their relationship is defined as:

$$\frac{[BH^+]}{[B]} = 10^{(pK_a-\text{pH})}$$

**Mechanism of action**
There is increasing evidence that LAs impair sodium ion
flux across the neuronal membrane by blocking the sodium
channels. This results in the inhibition of the rate and
amplitude of depolarisation of the neuronal membrane.\(^5\)
After injection of an LA, the non-ionised form of the LA is
released into the relatively alkaline pH of the tissues.

$$B + H^+ \Leftrightarrow BH^+$$

This non-ionised form (B) then diffuses through the nerve
sheath and the neuronal membrane to reach the axoplasm,
where it becomes partially ionised. It is this ionised form
(BH+), which blocks the sodium channel (Fig).

Local anaesthetics with a low pK_a are absorbed faster
into nerve tissues, and thus have a more rapid onset in action.
In contrast, LAs with a high pK_a, although they diffuse
into the nerve tissues much more slowly, produce more
effective blockade and have a longer duration of action,
because they also diffuse out slowly. Local anaesthetics are
less effective when injected into tissues which are relatively
acidic (eg abscesses) because of reduced availability of the
non-ionised form.

**Preparation**
Most LAs are bases that are almost insoluble in water.
Solubility is greatly enhanced by their preparation as the
hydrochloride salt, which is usually dissolved in modified
isotonic Ringer solutions. Dilute preparations of LAs are
usually acidic (pH 4.0-5.5), and contain a reducing agent
(eg sodium metabisulphite) to enhance the stability of added
vasoconstrictors. Methylparaben is also added to multidose
vials of lignocaine as a preservative.

**Pharmacological profile**
The pharmacological activity of an LA is determined by its
physicochemical properties. These include lipid solubility,
protein binding, and pK_a, which determine LA potency,
duration of action, and onset time, respectively. Table 1
summarises the pharmacological profiles of LAs commonly
used in Hong Kong.

The effect of an injected LA is terminated mainly by
absorption to the systemic circulation, which is dependent
on local blood flow.\(^5\) Agents with high protein binding, such
as bupivacaine, bind to the local tissue, and hence have
a longer duration of action. Most LAs, except cocaine and
to a certain extent ropivacaine, produce some degree of
vasodilatation, and hence are rapidly absorbed after local
injection. Consequently, vasoconstrictors are frequently
added to reduce systemic absorption, which may result in
reduced systemic toxicity and an increase in the margin
of safety (see below).

**Factors affecting anaesthetic activity**

**Dosage of local anaesthetic solutions**
As the dose of LA is increased, the quality of anaesthesia,
ie the onset, depth, and duration of nerve blockade, is
improved.\(^6\) This can be achieved either by using a large
volume of a less concentrated LA solution, or a small
volume of a more concentrated LA solution. However,
with the same dosage, larger volumes produce greater
spread after epidural injection.\(^7\)

**Site of injection**
The site of injection can influence the onset time and dur-
ation of nerve blockade of an LA. For example, bupivacaine,
when used for intercostal nerve block, has an onset time of
approximately 5 minutes and duration of effect of approxi-
mately 4 hours, and when used for brachial plexus block
has an onset time of approximately 30 minutes and
duration of around 10 hours.\(^6\) These differences can partly
be explained by differences in anatomy, and variable rate
of vascular uptake from the site of injection.

**Addition of vasoconstrictors**
Vasoconstrictors are frequently added to LA solutions to
decrease systemic absorption and thus increase the depth
and duration of nerve blockade. In addition, vasoconstrictors,
by sequestering LAs in tissues, can enhance LA potency
and prolong the duration of nerve blockade. Adrenaline
optimal vasoconstriction with lignocaine, although concentrations ranging from 1 in 80 000 to 1 in 300 000 are also available. Adrenaline also acts as a marker for inadvertent intravascular injection. The effect of adrenaline on the duration of action is most profound with LAs having short-to-moderate duration of action, e.g., lignocaine. Local anaesthetics which are more potent and longer acting, such as bupivacaine, are influenced less by the addition of adrenaline, particularly when such agents are used for epidural blockade. Other vasoconstrictors, such as noradrenaline (norepinephrine) and phenylephrine, have also been used in conjunction with LA solutions. However, studies were unable to demonstrate superiority of these agents to adrenaline.

### Use of additives with local anaesthetics

Attempts have been made to modify LA solutions in a number of ways, in order to improve the onset of action or prolong the duration of nerve blockade. Adding sodium bicarbonate (1 mmol/10 mL LA solution) to lignocaine decreases the onset time after epidural injection by increasing the amount of non-ionised drug (because of the more alkaline medium).

### Mixtures of local anaesthetics

The advantages of mixing two LAs are, in theory, to allow the use of a smaller amount of any one drug, thus limiting its toxicity, and to combine the rapid onset of one drug with the long duration of the other, e.g., chloroprocaine mixed with bupivacaine. However, this has not been demonstrated consistently. At present there appears to be no significant clinical advantage in using mixtures of LAs.

### Toxicity and adverse effects of local anaesthetics

Adverse reactions to LAs include systemic effects, localised reactions, specific adverse effects related to particular LAs, allergy, and addiction. Other toxic reactions may be related to the additives, such as adrenaline and preservatives, rather than the LA itself. Considering the large number of LAs administered worldwide, the frequency of toxic reactions is extremely low. Moreover, most adverse reactions following LA administration are due to inappropriate use, such as overdose and inadvertent intravascular or intrathecal injection.

### Systemic toxicity

Injected LAs are absorbed into the systemic circulation, and toxic effects on the central nervous system (CNS) or cardiovascular system (CVS) depend on the plasma level of the drug (Boxes 1 and 2). The rate of change, as well as the absolute plasma level of LA, influences toxicity with more rapid accumulation being more toxic. Hence, doses of LA that are considered safe for local infiltration may become toxic when injected intravascularly.

Most of the data on CNS toxicity are based on lignocaine, and are extrapolated to other LAs. Central nervous system toxicity is directly related to the plasma concentration. It is manifested by initial excitation (muscle twitching, convulsion) followed by depression (coma, respiratory arrest).

Cardiovascular system toxicity includes dose-dependent myocardial depression, severe ventricular arrhythmias, ventricular fibrillation (VF), and asystole. Bupivacaine is more cardiotoxic than lignocaine, since doses that cause irreversible CVS collapse are close to those producing CNS toxicity. Acidosis and hypoxia markedly potentiate the cardiotoxicity of bupivacaine, and pregnant patients are more susceptible. Ropivacaine, a new aminoamide LA agent recently introduced for clinical use in Hong Kong, has a sensory block profile very similar to bupivacaine, but produces less motor blockade and is less cardiotoxic than bupivacaine. Levobupivacaine, the S-enantiomer of bupivacaine, which is not yet available in Hong Kong, also exhibits similar potency but is less toxic.

### Box 1. Factors affecting systemic toxicity

- Potency of the local anaesthetic
- Total dose administered
- Addition of exogenous vasoconstrictor
- Vascularity of the tissues
- Rate of systemic uptake
- Patient’s acid-base status

### Box 2. Signs and symptoms of local anaesthetic toxicity (with increasing plasma concentration)

- Numbness of tongue
- Light-headedness
- Visual and auditory disturbances
- Muscular twitching
- Unconsciousness
- Convulsions
- Coma
- Respiratory arrest
- Cardiovascular depression

### Table 1. Pharmacological profile of local anaesthetics available in Hong Kong

<table>
<thead>
<tr>
<th>Agent</th>
<th>Concentration (%)</th>
<th>Onset of action with infiltration</th>
<th>Onset of action with nerve block (mins)</th>
<th>Duration of action with nerve block (mins)</th>
<th>Maximum single dose without adrenaline</th>
<th>Maximum single dose with adrenaline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lignocaine</td>
<td>1-2</td>
<td>Fast</td>
<td>4-10</td>
<td>60-120</td>
<td>4.5 mg/kg</td>
<td>7.0 mg/kg</td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>0.25-0.5</td>
<td>Moderate</td>
<td>8-12</td>
<td>240-480</td>
<td>2.5 mg/kg</td>
<td>2.5 mg/kg</td>
</tr>
<tr>
<td>Ropivacaine*</td>
<td>0.2-1.0</td>
<td>Fast-moderate</td>
<td>8-12</td>
<td>240-480</td>
<td>150-200 mg†</td>
<td>ND‡</td>
</tr>
<tr>
<td>Cocaine§</td>
<td>1-4</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>3 mg/kg</td>
<td>-</td>
</tr>
</tbody>
</table>

* Data from Mather and Chang
† Manufacturer’s (AstraZeneca, Södertälje, Sweden) quoted maximum dose
‡ ND data not available
§ Cocaine is indicated only for topical anaesthesia of mucosal membranes

(epinephrine) is the most commonly used vasoconstrictor, at a concentration of 1:200 000 (5 mg/mL), which provides optimal vasoconstriction with lignocaine, although concentrations ranging from 1 in 80 000 to 1 in 300 000 are also available.
**Direct local tissue toxicity**

Under experimental conditions, high concentrations of LAs, and various buffers and preservatives added to LAs, are neurotoxic. Under clinical conditions, intraneural injection, by causing pressure effects on the nerve fascicles, may contribute to some of the observed nerve injuries. Full thickness skin necrosis has been reported after subcutaneous infiltration of 1% lignocaine, which is presumably due to the added adrenaline. Nevertheless, commercially available LAs are generally safe for clinical use.

**Allergy**

Although reports of allergic reactions, hypersensitivity, or anaphylactic reactions to LA agents appear periodically, it is often found that these ‘reactions’ have been confused with systemic toxicity, vasovagal syncope, or allergy to the preservatives methylparaben or metabisulphite. In addition, the majority of LA allergies are associated with the ester agents, and are related to the metabolite PABA. True allergy to amide-type LAs is extremely rare. Allergic cross-reactivity between amide and ester agents is weak. Patients sensitive to one ester are likely to be sensitive to the entire group, whereas patients who are allergic to one amide LA may be able to tolerate an alternative amide. In patients with a history of an anaphylactic reaction to LAs, skin testing and subcutaneous challenge tests, with and without preservatives and/or adrenaline, is recommended.

**Adverse effects of specific local anaesthetics**

Prilocaine, in doses exceeding 600 mg, can cause oxidation of the ferrous form of haemoglobin to the ferric form (methaemoglobin). Methaemoglobin cannot carry oxygen and the skin develops a bluish discolouration, in most cases benign and resolving spontaneously. However, when symptoms of tissue anaemia do occur, it can be promptly treated with intravenous methylene blue.

Cocaine, which causes vasoconstriction and inhibits the reuptake of catecholamines, can cause hypertension, tachycardia, arrhythmia, myocardial ischaemia, and sudden death. Although the maximum recommended dose of cocaine in adults is 1-3 mg/kg, fatalities have been reported following the use of less than 200 mg. Management of cocaine toxicity is mainly supportive, with benzodiazepines for the treatment of convulsions. Although propanolol has been used successfully to treat cocaine-induced cardiac toxicity, and the clinical situation. Bupivacaine, for example, should not be used for intravenous regional anaesthesia (IVRA), and prilocaine should be avoided in patients with low levels of methaemoglobin reductase, or glucose-6-phosphate dehydrogenase deficiency.

**Topical anaesthesia**

Topical anaesthesia of mucosal membranes can be achieved with a number of agents, eg cocaine and amethocaine. Cocaine, because of its unique properties of both local anaesthesia and vasoconstriction, remains popular among otorhinolaryngologists. Caution must be exercised when combining cocaine with adrenaline, as cumulative toxicity can occur. Alternatives, such as 1 to 2% lignocaine with adrenaline or other vasoconstrictors, may be considered.

Topical anaesthesia of the skin, because of poor penetration by the LA, is more difficult to achieve. A mixture of the crystalline bases of lignocaine and prilocaine, by reducing their melting points, produces a liquid at room temperature, which is called a ‘eutectic mixture of local anaesthetics’ (EMLA). A eutectic mixture of LAs 1 to 2 g/10 cm² of skin is applied under an occlusive dressing. An EMLA patch (AstraZeneca, Södertälje, Sweden) is currently available; this consists of an adhesive disc impregnated with EMLA cream 1 g intended for anaesthesia of 10 cm² of skin. When EMLA cream is applied under an occlusive dressing, an anaesthetic depth of 3 mm is achieved after 60 minutes. The depth increases by 1 mm per 30 minutes up to 5 mm at 120 minutes. However, in highly vascular sites, such as the face or damaged skin, the onset is much more rapid. Adverse effects of EMLA are minimal, and include local erythema, pallor, oedema, pruritus, and potentially methaemoglobinemia attributable to prilocaine.

**Local infiltration**

This technique involves injection of LA directly into the tissue; pain during injection is therefore common. Several approaches to reducing pain on injection have been suggested: deep intradermal rather than superficial dermal injection, slow injection using a long needle of the smallest possible calibre, administration of the LA solution at body temperature, the LA buffered with sodium bicarbonate in a 10:1 ratio, use of the smallest amount of the lowest concentration, and use of the smallest volume syringe that can provide the desired amount of local anaesthesia.

**Field block**

When local infiltration of the surgical margin is deemed inappropriate, such as with a contaminated wound, or when it is necessary to avoid tissue distortion (eg the ear), local anaesthesia can be achieved by injecting the LA around the area under consideration.

**Tumescent technique**

Tumescent anaesthesia is the technique of anaesthetising large areas of adipose tissue. It is commonly used for liposuction, but more recently has also been used for
procedures such as hair transplantation, ambulatory phlebectomy, wide cutaneous excision, and facial resurfacing. This technique involves infiltrating adipose tissue with large volumes of a dilute lignocaine solution (0.05 to 0.2%) with adrenaline (1:1 000 000). Sodium bicarbonate (1 mEq/100 mL LA solution) may also be added to buffer the LA solution. Large total doses of lignocaine up to 55 mg/kg are frequently used; although this exceeds the maximum recommended dose for infiltration anaesthesia, toxic complications are rare. This may be due to the slow systemic absorption of lignocaine from adipose tissues, resulting in plasma concentrations of lignocaine below the toxic range. However, several deaths have been reported following liposuction, and LA toxicity masked by the concomitant use of sedatives has been suggested as a possible cause.

**Intravenous regional anaesthesia**

This is the technique of producing anaesthesia by administering an LA intravenously in an exsanguinated and tourniquet-occluded limb. Proper monitoring and immediate availability of resuscitation facilities are essential for management of toxic reactions that may occur in the event of a tourniquet failure. The tourniquet should not be released for at least 30 minutes after the LA injection. Prilocaine, because of its high therapeutic ratio, is recommended for IVRA. However, since it is unavailable in Hong Kong, 0.5% lignocaine is used. Bupivacaine, because of its cardiotoxicity, should not be used for IVRA. Contraindications include sickle cell disease or trait, untreated heart block, and local infection.

**Haematoma block**

This is the technique of injecting LA directly into the haematoma, which has been used in management of fractures such as Colles’ fracture. However, IVRA has been shown to be superior to haematoma block in terms of efficacy, radiological result, and remanipulation rate.

**Peripheral nerve block**

Simple peripheral nerve blocks, such as digital and penile nerve block, can be used for minor surgery. More complex nerve blocks, such as brachial plexus block, require a thorough understanding of the anatomy and adequate training. The use of adrenaline-containing LA must be avoided in areas supplied by end-arteries (eg in digital nerve block, penile block), because of the potential risk of producing ischaemia. Ropivacaine has intrinsic vasoconstrictor properties, and in a recent case report was implicated as a cause of ischaemia of the glans penis after penile nerve block. Thus, until further evidence is available, it may be wise to exercise caution when using ropivacaine for local anaesthesia in areas where there is potential for end-artery ischaemia.

**Safety aspects with clinical use of local anaesthetics**

In order to avoid inadvertent intravascular injection and toxicity, the syringe should be intermittently aspirated for blood, the smallest volume of the lowest concentration of LA producing anaesthesia should be used, and the LA should be administered slowly in small aliquots. Patients should lie supine during the block, in case unexpected syncope occurs. In the event that the patient becomes unconscious, LA administration should be discontinued immediately, and basic cardiopulmonary resuscitation (CPR) initiated, with oxygen supplementation as indicated. At the same time, the physician must distinguish between vasovagal reaction, systemic toxicity, and anaphylactic reaction (Table 2). Subsequent management depends on the cause of the syncope.

**Dosage of local anaesthetics**

Local anaesthetic agents currently available in Hong Kong, together with their clinical profile and the maximum recommended doses, are summarised in Table 1. As these doses are based on animal studies, and as the plasma concentration of an LA varies considerably with the site of injection, this ‘maximum recommended dose’ has been challenged. Lignocaine has been used in doses of 900 mg (approximately 18 mg/kg) for brachial plexus block, and in dosages up to 55 mg/kg for tumescent anaesthesia, without producing toxic plasma levels, or clinical signs or symptoms of toxicity. Clinicians should exercise clinical judgement, depending on experience and available facilities, when deciding on the dose of an LA agent.

**Monitoring and resuscitation equipment**

Recommendations on office-based surgical facilities for surgical procedures outside the hospital setting were published recently. For surgical procedures performed under local anaesthesia, and in cases where oral or
intramuscular sedation may be administered, the following
should be available:

1. a blood pressure measuring device, such as a sphygm-
omanometer and stethoscope;
2. a source of airway maintenance, such as a mouth-to-
mouth resuscitation device, or a self-inflating bag and
mask; and
3. a source of oxygen delivery up to 5 L/min.

In addition, adrenaline 1:1000 for subcutaneous admin-
istration and injectable antihistamines should be readily
available for possible allergic reactions. The physician
and appropriate staff should be trained in basic CPR, and
there should be a well-established plan of action to deal with
unexpected emergencies.

Management of systemic toxicity
When signs and symptoms of CNS toxicity occur, the
injection should be stopped immediately. Minor symptoms
seldom require treatment, other than constant verbal
contact, CVS monitoring, and oxygen supplementation as
indicated. Airway maintenance and assisted ventilation with
oxygen supplementation is essential if respiration stops
(Box 3). Should convulsions develop, diazepam 5 to 10 mg
or thiopentone 50 to 100 mg (if available) should be
administered intravenously. Prevention of hypoxia and
acidosis are of utmost importance during treatment.9

Hypotension should be treated with intravenous fluid,
leg elevation, correction of hypoxia, and vasopressors (eg
ephedrine 15 to 30 mg). Cardiopulmonary resuscitation
should be initiated when profound CVS depression is
present. Ventricular tachycardia or fibrillation should be
treated with cardioversion (higher energy may be required).
As bupivacaine dissociates from sodium channels slowly,
CPR should be continued for at least 60 minutes or more in
patients with bupivacaine-induced VF. The medical
treatment for bupivacaine-induced ventricular arrhythmias
remains controversial. Lignocaine, paradoxically, has been
used.49 Bretylium may facilitate cardioversion in refractory
VF.50 Ventricular arrhythmias resistant to bretylium have
been reverted successfully by phenytoin in neonates.51 In
anaesthetised animals, dobutamine and clonidine have
been found to be beneficial in correcting the haemodynamic
and electrophysiological abnormalities.52

**Special patient groups**
Following the report of 31 maternal deaths after the use
of 0.75% bupivacaine for epidural anaesthesia, this concen-
tration has been withdrawn from obstetric anaesthetic
use in the United States.18 It is suggested that the increased
susceptibility of pregnant patients to the cardiotoxic effects
of bupivacaine is due to the direct effects of progesterone.53
On the other hand, lignocaine, because of its low pKₐ,
and propensity for ion trapping, is not indicated in large
doses for obstetric patients, especially where foetal distress
is present, in order to avoid foetal toxicity.17 Moreover,
concerns have also been raised recently regarding transient
neurological symptoms occurring after spinal anaesthesia
with lignocaine.54 Being less cardiotoxic than bupivacaine,
ropivacaine is gaining popularity among obstetric anae-
thesists. Nevertheless, LAs should be used with caution in
obstetric patients, and foetal monitoring may be required.
Selective surgical procedures should be delayed until after
the period of organogenesis.59

In children, the recommended maximum LA dosages
adjusted to body weight are similar to those in adults
(Table 1).56 However, LA doses in neonates must be
reduced, especially during infusion, because of diminished
protein binding and immature hepatic clearance.56 In
older infants and children, because of their inability to
describe warning symptoms, and the difficulty of reliably
detecting intravascular injection, it is essential that LA
injection occurs slowly, in small increments, with constant
assessment of the child for signs of toxicity.56

**Drug interactions**
Caution should be exercised in patients on concurrent
medications. Monoamine oxidase inhibitors may precipi-
tate a hypertensive crisis when exogenous adrenaline
contained in the LA solution is administered. Phenothiazines,
because of their irreversible α-blocking properties, may
cause hypotension and cardiac arrest, due to additional
vasodilatation caused by the LA.1 Serious hypertension-
bradycardia has been reported in a patient on a β-blocker
because of interaction with adrenaline.57 Cimetidine,
β-blockers, and procainamide, by reducing hepatic blood
flow, may decrease lignocaine metabolism and lead to
accumulation of the LA.58 Hence, a detailed drug history
should be taken before administration.

**Multidose vials**
Four patients were infected with human immunodeficiency
virus (HIV) in a doctor’s surgery in Australia.59 These pa-
ients underwent minor skin surgery after another pa-
tient who was HIV-infected, and no identifiable breach of

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**Box 3. Treatment of systemic local anaesthetic toxicity**

| Airway     | Establish clear airway; suction, if required |
| Breathing  | Oxygen with face mask; encourage adequate ventilation; artificial ventilation, if required |
| Circulation| Elevate legs; increase IV fluids if ↓ blood pressure; CVS support if ↓ blood pressure persists or ↓ heart rate; cardioversion for ventricular arrhythmias |
| Drugs      | Central nervous system depressant: Diazepam 5-10 mg IV, midazolam 2-5 mg; Thiopental 50 mg IV, incremental dosages until seizures cease; CIV support if ↓ heart rate; Ephedrine 15-30 mg IV, to restore adequate blood pressure; Adrenaline for profound cardiovascular collapse |

* IV intravenous
+ CVS cardiovascular system

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infection control guidelines was found. A separate report later described the observation that needles and syringes retained small volumes (25 mL) of fluid after use, which could be transferred to multidose vials, and that active HIV could be isolated from the contaminated multidose vial after up to 4 hours. Hence, use of multidose vials of LA for more than one patient is no longer recommended.

**Conclusion**

Currently available LA agents are safe if used with due care and caution. One must exercise extra caution when using these drugs outside the operating room, as monitoring and resuscitation equipment may not be readily available. Clinicians should have a clear understanding of LA pharmacology, toxicity, and factors predisposing to LA toxicity before administering these drugs. The amount of LA injected should be carefully calculated to avoid unintentional overdose. Inadvertent intravascular injection must be avoided. There should be a well-established plan of action to deal with unexpected complications in settings where LAs are administered.

**References**

44. Burke D, Joypaul V, Thomson MF. Circumcision supplemented by...

Coming in the June issue of the Hong Kong Medical Journal

- Risk factors for preterm delivery in women with placenta praevia and antepartum haemorrhage: retrospective study
- Recall of preoperative anaesthesia information in Hong Kong Chinese patients
- Living donor liver transplantation without the use of blood products