

LIGNOCAINE INJECTION

Product Information

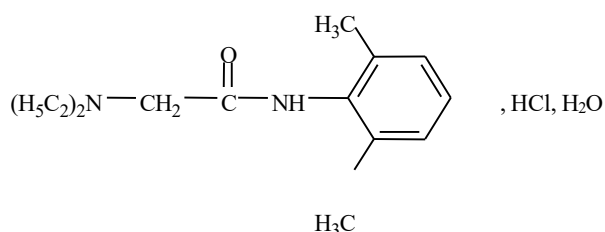
Name of drug

Lignocaine Hydrochloride.

Description

Lignocaine hydrochloride is 2-diethylamino- 2',6'- dimethylacetanilide hydrochloride monohydrate. It appears as a white, crystalline powder that is very soluble in water, freely soluble in alcohol and practically insoluble in ether.

The structural formula is represented below.



Molecular Formula: C₁₄H₂₂N₂O,HCl,H₂O

Molecular Weight: 288.8

CAS Number: 6108-05-0

Lignocaine Injection is a clear, colourless, sterile, isotonic, preservative-free solution containing Lignocaine Hydrochloride 1% or 2% and Sodium Chloride in Water for Injections.

Pharmacology

Class: Local anaesthetic of the amide type and antiarrhythmic drug.

Mechanism of action: Lignocaine stabilises all potentially excitable membranes and prevents the initiation and transmission of nerve impulses. This produces a local anaesthetic effect. Onset of action is rapid and blockade may last from 60 - 90 minutes.

Studies of the effects of therapeutic concentrations of lignocaine on the electrophysiological properties of mammalian Purkinje fibers have shown that lignocaine attenuates phase 4 diastolic depolarisation, decreases automaticity, and causes a decrease or no change in excitability and membrane responsiveness. Action potential duration and effective refractory period of Purkinje fibers are decreased, while the ratio of effective refractory period to action potential duration is increased. Action potential duration and effective refractory period of ventricular muscle are also decreased. Effective refractory period of the AV node may increase, decrease, or remain unchanged, and atrial effective refractory period is unchanged. Lignocaine raises the ventricular fibrillation threshold. No significant interactions between lignocaine and autonomic nervous system have been described and, consequently, lignocaine has little or no effect on autonomic tone.

Pharmacokinetics: Lignocaine has a rapid onset and a medium duration of action. The onset of action is 1 to 5 minutes following infiltration and 5 to 15 minutes following other types of administration.

The rate of absorption depends upon the dose, the route of administration and the vascularity of the injection site. Intercostal blocks give the highest peak plasma concentrations (approximately 1.5 microgram/mL for every 100 mg injected), while abdominal subcutaneous injections give the lowest (approximately 0.5 microgram/mL per 100 mg injected). Epidural and major nerve block produce peak plasma levels intermediate between these.

Absorption of lignocaine from the epidural space occurs in two phases; the first phase is in the order of 9 minutes and the second is approximately 82 minutes. The slow absorption is the rate limiting step in the elimination of lignocaine, which also explains the apparent elimination half-life following epidural injection being longer than after intravenous administration.

The plasma binding of lignocaine is dependent on drug concentration, and the fraction bound decreases with increasing concentration. At free base concentrations of 1 to 4 microgram/mL, 60 to 80% of lignocaine is protein bound. Binding is also dependent on the plasma concentration of the alpha-1-acid glycoprotein.

Lignocaine crosses the blood-brain and placental barriers by passive diffusion. Since the degree of plasma protein binding in the fetus is less than that in the mother, although free lignocaine concentrations will be the same, the total plasma concentration will be greater in the mother.

Lignocaine has a total plasma clearance of 0.95 L/minute, a volume of distribution at steady state of 91 L, an elimination half-life of 1.6 hours and an estimated hepatic extraction ratio of 0.65. Approximately 90% of a parenteral dose of lignocaine is rapidly metabolised in the liver by de-ethylation to form monoethylglycinexylidide (MEGX) and glycinexylidide (GX) followed by cleavage of the amide bond to form xylidine and 4-hydroxyxylidine, which are excreted in the urine. Less than 10% of a dose is excreted unchanged in the urine.

The principal metabolites, MEGX and GX, also possess pharmacological activity. The rate of metabolism of lignocaine appears to be limited by hepatic blood flow that may be reduced in patients after acute myocardial infarction and/or congestive heart failure. The rate of lignocaine metabolism may also be reduced in patients with hepatic tissue necrosis, possibly because of altered perfusion.

The duration of action depends upon the concentration used, the dose given, the nerves to be blocked and the status of the patient. The 2% solution will produce an effect for 1.5 to 2 hours when given epidurally, and up to 5 hours when given as a peripheral nerve block. When used in a 1% concentration there is less effect on motor nerve fibres and the duration of effect is shorter.

Indications

- Lignocaine is indicated for production of local or regional anaesthesia by nerve block, infiltration injection, caudal or other epidural blocks.
- Treatment or prophylaxis of life-threatening ventricular arrhythmias, including those associated with myocardial infarction, general anaesthesia in patients predisposed to ventricular arrhythmias, digitalis intoxication, or following resuscitation from cardiac arrest.

Contraindications

- Known hypersensitivity to local anaesthetics of the amide type.
- Inflammation or sepsis at the proposed site of injection and in the presence of septicaemia.

- Patients with myasthenia gravis, severe shock or impaired cardiac conduction.
- Epidural or spinal anaesthesia in patients with
 - uncorrected hypotension or
 - coagulation disorders or receiving anticoagulants or
 - serious diseases of the central nervous system or spinal cord such as meningitis, spinal fluid block, cranial or spinal haemorrhage, tumours, poliomyelitis, syphilis, tuberculosis or metastatic lesions of the spinal cord.
- Antiarrhythmic use in patients with
 - supraventricular arrhythmia or
 - Stokes-Adams Syndrome or severe degrees of sinoatrial, atrioventricular or intraventricular block unless the patient has an artificial pacemaker.
- Lignocaine suppresses ventricular pacemaker activity and may cause ventricular standstill in such patients
- General contraindications related to epidural anaesthesia, regardless of the local anaesthetic used, should be taken into account.

Precautions

General Precautions:

- When any local anaesthetic agent is used, resuscitative equipment and drugs, including oxygen, should be immediately available in order to manage possible adverse reactions involving the cardiovascular, respiratory or central nervous systems. Because of the possibility of hypotension and bradycardia following major blocks, an IV cannula should be inserted before the local anaesthetic is injected. Delay in proper management of dose-related toxicity, under-ventilation from any cause and/or altered sensitivity may lead to the development of acidosis, cardiac arrest and death.
- Injection of repeated doses of lignocaine may cause significant increases in blood levels with each repeated dose due to slow accumulation of the drug or its metabolites, or slow metabolic degradation. This is especially relevant in patients with hepatic and/or renal impairment.
- Tolerance varies with the status of the patient. Debilitated, elderly patients, acutely ill patients and children should be given reduced dosages commensurate with their age and physical status.
- Lignocaine should be used cautiously in patients with known drug allergies or sensitivities. Patients showing allergy to ester derivatives of para-aminobenzoic acid such as procaine, benzocaine and tetracaine have not shown cross-sensitivity to
- Lignocaine should be given cautiously to patients with epilepsy, hepatic disease, renal disease, congestive cardiac failure, marked hypoxia, severe respiratory depression, severe shock or hypovolaemia and in patients with any form of heart block or sinus, bradycardia, cardiac conduction disturbances, severe digitalis intoxication. Lignocaine should be administered with great caution to patients with impaired cardiovascular function as they may be less able to compensate for functional changes associated with the prolongation of atrioventricular conduction produced by these drugs. In patients with Stokes-Adams syndrome or Wolff-Parkinson-White syndrome extreme care should be taken to avoid accidental arteriovenous injection.
- Hypokalaemia, hypoxia and disorders of acid-base balance should be corrected before treatment with lignocaine.

For local anaesthesia:

- Injection should always be made slowly with frequent aspirations to avoid inadvertent intravascular injection, which can produce cerebral symptoms even at low doses.
- Careful and constant monitoring of cardiovascular and respiratory vital signs and the patient's state of consciousness should be accomplished after each local anaesthetic injection. It should be kept in mind that at such times restlessness, anxiety, tinnitus, dizziness, blurred vision, tremors, depression or drowsiness may be early warning signs of CNS toxicity.
- Low molecular weight heparins and heparinoids - when neuraxial anaesthesia (epidural/spinal anaesthesia) is employed, patients anticoagulated or scheduled to be anticoagulated with low molecular weight heparins or heparinoids are at risk of developing an epidural or spinal haematoma which can result in long-term or permanent paralysis. The risk of these events is increased by the use of indwelling epidural catheters, traumatic or repeated epidural/spinal puncture, and the concomitant use of drugs affecting haemostasis such as NSAIDs, platelet inhibitors or other anticoagulants. Patients should be frequently monitored for signs and symptoms of neurological impairment.
- The safety and effectiveness of lignocaine depends on proper dosage, correct technique and adequate precautions. Standard textbooks should be consulted regarding specific techniques and precautions for various regional anaesthetic procedures.
- The lowest dosage that results in effective anaesthesia should be used to avoid high plasma levels and serious undesirable systemic side effects.
- Local anaesthetics in general should be given cautiously (see Contraindications) to patients with pre-existing abnormal neurological conditions as neurological reactions may occur following administration.
- Inadvertent intravascular or subarachnoid injection of small doses of local anaesthetics in the head and neck area, including retrobulbar, dental and stellate ganglion blocks, may produce adverse reactions similar to systemic toxicity seen with unintentional intravascular injections of larger doses. Clinicians who perform retrobulbar blocks should be aware that there have been reports of cardiovascular collapse and apnoea following the use of local anaesthetic injections for retrobulbar block. Prior to retrobulbar block, necessary equipment, drugs and personnel should be immediately available, as with all other regional procedures. Retrobulbar injections may very occasionally reach the subarachnoid space, causing temporary blindness, cardiovascular collapse, apnoea, convulsions, etc. These must be diagnosed and treated promptly.
- Retro- and peribulbar injections of local anaesthetics carry a low risk of persistent ocular muscle dysfunction. The primary causes include trauma and/or local toxic effects on muscles or nerves. The severity of such tissue reactions is related to the degree of trauma, the concentration of the local anaesthetic and the duration of exposure of the tissue to the local anaesthetic. For this reason, as with all local anaesthetics, the lowest effective concentration and dose of local anaesthetic should be used. Vasoconstrictors may aggravate tissue reactions and should be used only when indicated.
- The likelihood of hypotension and bradycardia following epidural or subarachnoid blockade should be anticipated and appropriate precautions taken.

For cardiac arrhythmia use:

- Constant ECG monitoring is essential during intravenous administration of lignocaine.

If signs of excessive depression of cardiac conductivity (e.g. prolongation of the PR interval or QRS complex), aggravation of arrhythmias or other severe reactions occur, lignocaine should

be promptly discontinued. Severe reactions are often preceded by somnolence and paraesthesia, and these symptoms should not be ignored.

Mortality

In the Cardiac Arrhythmia Suppression Trial (CAST), a long-term, multi-centre, randomised, double-blind study in patients with asymptomatic non-life threatening ventricular arrhythmia who had myocardial infarction more than six days but less than two years previously, an excess mortality and non-fatal cardiac arrest rate was seen in patients treated with encainide or flecainide (56/730), compared with that seen in patients assigned to matched treated groups (22/725). The average duration of treatment with encainide or flecainide in this study was ten months.

While there are no comparable mortality trial data for other class 1 antiarrhythmic agents post myocardial infarction, or in other clinical settings, meta-analysis of small scale clinical trials of these agents in similar populations suggests a trend towards increased mortality compared to placebo and no evidence of benefit.

All class 1 antiarrhythmic agents share the capacity to produce slowing of conduction velocity that can promote tachycardias via re-entry mechanisms. Therefore, the prophylactic use of class 1 antiarrhythmic drugs following myocardial infarction is potentially hazardous. Indeed, the use of these agents for other than life-threatening arrhythmias or severe symptoms due to arrhythmias is not recommended.

The safety and effectiveness of lignocaine depend upon proper dosage, correct technique, adequate precautions and readiness for emergencies.

- Patients with reduced hepatic blood flow or function, and those on prolonged infusions of lignocaine will have a longer half-life and a lower clearance. During congestive cardiac failure, clearance will be reduced and in renal failure, accumulation of lignocaine may occur. Such instances will require a reduction in dosage.
- Lignocaine may increase ventricular rate when it is administered to patients with atrial fibrillation. Ischaemia or necrosis may occur in patients with hypertensive vascular disease or with an exaggerated vasoconstrictor response.
- Lignocaine suppresses ventricular pacemaker activity and may cause severe bradycardia or asystole in patients, including those undergoing epidural anaesthesia.

Effects on ability to drive and operate machinery:

Depending on dosage, local anaesthetic may have a very mild effect on mental function and may temporarily impair locomotion and coordination.

Carcinogenicity and mutagenicity: A two year oral toxicity study of 2,6-xylylidine, a metabolite of lignocaine, has shown that in both male and female rats, 2,6-xylylidine in daily doses of 900 mg/m² (150 mg/kg) resulted in carcinomas and adenomas of the nasal cavity. No nasal tumours were observed in the low dose (15 mg/kg) or control animals. The compound also caused subcutaneous fibromas and/or fibrosarcomas in male and female rats (significant at 150 mg/kg).

The genotoxic potential of 2,6-xylylidine has been studied with mixed results: positive results were reported in assays of gene mutations (weakly positive in the Ames test with metabolic activation and in the mouse lymphoma assay) and chromosomal damage (chromosomal aberrations in Chinese hamster ovary cells at concentrations at which the drug is precipitated from solution). No evidence of genotoxicity was found *in vivo* assays for chromosomal damage (micronucleus assay) and DNA damage (unscheduled DNA synthesis). Covalent binding

studies of DNA from liver and ethmoid turbinates in rats indicate that 2,6-xylidine may be genotoxic under certain conditions in vivo.

Use in pregnancy: Category A: The safe use of lignocaine during pregnancy has not been established. Although lignocaine has been used extensively for surgical procedures during pregnancy with no reports of ill effects to mother or fetus, there are no adequate or well-controlled studies in pregnant women of the effect of lignocaine on the developing fetus.

Lignocaine has been effectively used for obstetrical analgesia and adverse effects on the course of labour or delivery are rare. After epidural administration of lignocaine to women in labour, lignocaine crosses the placental barrier. However, concentrations in umbilical veins are lower than those found in the maternal circulation. It has been suggested that blood glucose levels should be checked in newborns after obstetric regional anaesthesia.

Fetal bradycardia frequently follows paracervical block and may be associated with fetal acidosis and hypoxia. Occasional cases of perinatal morbidity and mortality have been reported. When the recommended dose is exceeded, the risk of fetal bradycardia increases. Careful monitoring of the fetal heart rate is necessary.

Use in lactation: Lignocaine passes into the breast milk although it would be unlikely that the amount passing to the infant would lead to significant accumulation of the parent drug in the infant. The remote possibility of an idiosyncratic or allergic reaction in the breast fed infant from lignocaine remains to be determined.

Interactions with other drugs:

- **Antiarrhythmic drugs:** Use lignocaine cautiously in patients receiving antiarrhythmic drugs as potentiation of cardiac effects may occur.
- **Beta-adrenoreceptor antagonists:** Propranolol and metoprolol reduce the metabolism of intravenously administered lignocaine. It is possible that this effect will be repeated with other beta-adrenergic blockers. If these drugs are administered concurrently, the patient should be closely observed for signs of lignocaine toxicity.
- **Cimetidine:** The clearance of intravenously administered lignocaine is reduced when it is administered in conjunction with cimetidine. Toxic effects due to high serum lignocaine levels have been reported when these two drugs have been administered concurrently.
- **Amiodarone:** Amiodarone has been reported to reduce the clearance of lignocaine in two case reports, although a small prospective study of combined therapy on lignocaine pharmacokinetics found no change in clearance or other pharmacokinetic factors. This combination has been reported to precipitate seizures and lead to severe sinus bradycardia and a long sinoatrial arrest. Until more experience with concurrent use of lignocaine and amiodarone becomes available, patients receiving the combination should be monitored carefully.
- **Anticonvulsant drugs:** Phenytoin, phenobarbitone, primidone and carbamazepine appear to enhance the metabolism of lignocaine possibly due to an induction of microsomal enzymes, but the significance is unknown. Phenytoin and lignocaine have additive cardiac depressant effects.
- **Skeletal muscle relaxants:** Lignocaine and skeletal muscle relaxants, e.g. suxamethonium, lead to excessive neuromuscular blockade, therefore this combination must be used with caution. Lignocaine prolongs the duration of suxamethonium.
- **Inhalational anaesthetics:** Lignocaine decreases the minimum effective concentration of inhalational anaesthetics, e.g. nitrous oxide.

- **Alcohol:** Acute, severe alcohol intoxication can centrally depress the cardiovascular system and may thereby prolong lignocaine elimination half-life.
- **Structurally related local anaesthetics:** Lignocaine should be used with caution in patients receiving agents structurally related to local anaesthetics.

Laboratory tests

Creatinine: Creatinine measurements in patients with therapeutic plasma levels of lignocaine are about 15 to 35% higher when measured by an enzymatic method versus the Jaffe method. This appears to be due to assay interference from N-ethylglycine, a metabolite of lignocaine.

Creatine kinase: The intramuscular injection of lignocaine may result in an increase in creatine kinase levels for up to 48 hours. This may interfere with the diagnosis of myocardial infarction.

Adverse reactions

Adverse effects may be due to high plasma levels as a result of excessive dosage, rapid absorption, delayed elimination or metabolism or inadvertent intramuscular injection during local anaesthetic use, or may result from a hypersensitivity, idiosyncrasy or diminished tolerance on the part of the patient.

Pronounced acidosis or hypoxia may increase the risk or severity of toxic reactions. Such reactions are systemic and may involve the central nervous system and/or the cardiovascular system.

Inadvertent subarachnoid injection during anaesthetic procedures may lead to cardiovascular collapse, CNS depression and respiratory arrest.

Systemic reactions of the following type have been reported:

Central nervous system: CNS manifestations are excitatory and/or depressant and may be characterised by light-headedness, disorientation, confusion, psychosis, nervousness, agitation, drowsiness, dizziness, apprehension, euphoria, tinnitus, blurred or double vision, nausea, vomiting, difficulty swallowing, dyspnoea, slurred speech, sensations of heat or cold, numbness, twitching, tremors, convulsions, unconsciousness, seizures, coma, respiratory depression and arrest.

The excitatory manifestations may be very brief or may not occur at all, in which case the first manifestation of toxicity may be drowsiness merging into unconsciousness and respiratory arrest. Drowsiness following administration of lignocaine is usually an early sign of a high blood level of the drug and may occur as a result of rapid absorption. In unconscious patients circulatory collapse should be watched for, as CNS effects may not be apparent, as an early manifestation of toxicity may in some cases progress to frank convulsions and ultimately lead to respiratory depression and/or arrest. It is crucial to have resuscitative equipment and anticonvulsant drugs available to manage such patients.

Cardiovascular system: Hypotension, cardiovascular collapse, arrhythmias including ventricular tachycardia/ventricular fibrillation, heart block, and bradycardia that may lead to cardiac arrest. Methaemaglobinaemia can occur following IV administration.

Cardiovascular toxic effects are generally preceded by signs of toxicity in the central nervous system, unless the patient is receiving a general anaesthetic or is heavily sedated with drugs

such as a benzodiazepine or a barbiturate. In rare cases, cardiac arrest has occurred without prodromal CNS effects.

In children, early signs of local anaesthetic toxicity may be difficult to detect in cases where the block is given during general anaesthesia.

Haemodynamic: Regional anaesthesia may lead to maternal hypotension.

Allergic: Allergic reactions are characterised by cutaneous lesions, urticaria, oedema or anaphylactoid reactions. Allergy to amide type local anaesthetics is very rare. If such a reaction occurs, it should be managed by conventional means.

The detection of sensitivity by skin testing is of doubtful value.

Neurological system: Adverse neurological reactions occur rarely following the use of local anaesthetics. They may be related to the total dose administered, the particular drug used, the route of administration and the physical status of the patient. Persistent anaesthesia, paraesthesia, weakness, paralysis of the lower extremities and loss of sphincter control may occur.

In a prospective review of 10,440 patients who received lignocaine for spinal anaesthesia, the incidences were reported to be about 3% each for positional headaches, hypotension and backache; 2% for shivering; and less than 1% each for peripheral nerve symptoms, nausea, respiratory inadequacy and double vision. Many of these observations may be related to local anaesthetic techniques, with or without a contribution from the local anaesthetic.

In the practice of caudal or lumbar epidural block, occasional unintentional penetration of the subarachnoid space by the catheter may occur. Subsequent adverse effects may depend partially on the amount of drug administered subdurally. These may include spinal block of varying magnitude (including total spinal block), hypotension secondary to spinal block, loss of bladder and bowel control and loss of perineal sensation and sexual function. Persistent motor, sensory and/or autonomic (sphincter control) deficit of some lower spinal segments with slow recovery (several months) or incomplete recovery have been reported in rare instances when caudal or lumbar epidural block has been attempted. Backache and headache have also been noted following use of these anaesthetic procedures.

Dosage and administration

Lignocaine Injection is for one dose in one patient only. Discard any remaining contents.

As a local anaesthetic for infiltration and nerve block:

The dosage varies and depends upon the area to be anaesthetised, vascularity of the tissues, number of neuronal segments to be blocked, individual tolerance and the technique of anaesthesia. The lowest dose needed to provide effective anaesthesia should be administered. For specific techniques and procedures, refer to standard textbooks.

It is recommended that the dose of lignocaine at any one time should not exceed 3 mg/kg. However, the dose administered must be tailored to the individual patient and procedure, and the maximum doses here quoted should be used as a guide only.

The normal recommended dose of lignocaine for various anaesthetic procedures in an average, healthy 70 kg adult patient are as follows:

Procedure	Concentration (%)	Volume (mL)	Dose (mg)
Infiltration	1.0	20	200
PERIPHERAL NERVE BLOCKS			
Paravertebral	1.0	3 - 5	30 – 50
Pudendal (each side)	1.0	10	100
Paracervical	1.0	10	100
SYMPATHETIC NERVE BLOCK			
Cervical (stellate ganglion)	1.0	5	50
Lumbar	1.0	10	100
EPIDURAL BLOCKS (2-3mL per segment)			
Thoracic	1.0	10 - 20	100 – 200
Lumbar - analgesia	1.0	10 - 20	100 – 200
- anaesthesia	2.0	5 - 10	100 – 200
Caudal - obstetric analgesia	1.0	10 - 20	100 – 200

The doses suggested above are only a guide. Toxic levels vary widely between patients so doses should be individualised and blood levels should be monitored.

Epidural injections should be administered slowly with frequent aspirations. Subarachnoid and intravascular injections are two of the most serious complications of this technique. If blood or spinal fluid become manifest during aspiration, the needle must be withdrawn and relocated.

The technique of epidural anaesthesia should only be attempted by physicians skilled in this area and readiness for emergencies must be ensured. During spinal anaesthesia the positioning of the patient is very important and the patient's pulse and blood pressure should be monitored. During thoracic, lumbar and caudal epidural anaesthesia, a marked fall in blood pressure and/or intercostal paralysis may be seen, possibly due to the use of excessive doses, improper positioning of the patient or accidental disposition of the anaesthetic within the subarachnoid space. Hypotension and bradycardia may occur as a result of sympathetic blockade.

For continuous epidural or caudal anaesthesia and paracervical block for obstetric analgesia the maximum dose should not be repeated at intervals of less than 1.5 hours.

Adults: The dose should not exceed 200 mg. For spinal anaesthesia, the dose should not exceed 100 mg.

Children: For children, a reduced dosage based on body weight or surface area should be used. The dosage should be calculated for each patient individually and modified in accordance with the physician's experience and knowledge of the patient. Early signs of local anaesthetic toxicity may be difficult to detect in cases where the block is given. In order to minimise the

possibility of toxic effects, the use of Lignocaine Injection 1% solution is recommended for most anaesthetic procedures involving paediatric patients. The dose should not exceed 3 mg/kg.

For intravenous use in cardiac arrhythmias:

Patients with congestive heart failure or cardiogenic shock may require smaller bolus doses.

Adults: The usual dose is lignocaine 50 to 100 mg administered intravenously under ECG monitoring. The dose may be injected at a rate of approximately 25 to 50 mg (2.5 to 5.0 mL of the lignocaine 1% solution or 1.25 to 2.5 mL of the 2% solution) per minute. A sufficient period of time should be allowed to enable a slow circulation to carry the drug to the site of action. If the initial dose of 50 to 100 mg does not produce the desired response, a second dose may be given after five minutes. No more than 200 to 300 mg of lignocaine should be administered during a one hour period.

Following a single injection in those patients in whom arrhythmia tends to recur and who are incapable of receiving oral antiarrhythmic therapy, intravenous infusions of lignocaine may be administered at a rate of 1 to 4 mg/minute (20 to 50 mcg/kg/minute).

Intravenous infusions must be given under ECG monitoring to avoid potential overdosage and toxicity. The infusion should be terminated as soon as the patient's cardiac rhythm appears to be stable or at the earliest signs of toxicity. It should rarely be necessary to continue the infusion beyond 24 hours. As soon as possible, patients should be changed to an oral antiarrhythmic agent for maintenance therapy.

Paediatrics: For children, a reduced dose based on body weight or surface area should be used. It is recommended that the 1% solution be used to minimise the possibility of toxic effects. Experience with lignocaine is limited.

A suggested paediatric dose is a loading dose of 0.5 to 1 mg/kg repeated if necessary up to 3-5 mg/kg, followed by continuous infusions of 10 to 50 micrograms/kg/min.

Geriatrics: A reduction in dosage may be necessary for elderly patients, particularly those with compromised cardiovascular and/or hepatic function.

Impaired hepatic function: Although lignocaine is metabolised by the liver, dosage reduction for local anaesthesia is probably not warranted. However, caution should be exercised with repeated doses or prolonged infusion.

Impaired renal function: Impairment of renal function is unlikely to affect lignocaine clearance in the short term (up to 24 hours). However, toxicity due to accumulation may develop with prolonged or repeated administration.

Incompatibilities: Although no specific incompatibilities have been reported the introduction of any additive requires caution. Do not administer unless the solution is clear.

Overdosage

In anaesthesia, acute emergencies associated with the use of lignocaine are normally related to high plasma levels, or unintended subarachnoid injection (see Adverse reactions).

With accidental intravascular injections, the toxic effect will be obvious within one to three minutes. With overdosage, peak plasma concentrations may not be reached for 20 to 30 minutes depending on the site of injection, and toxic signs will be delayed. Toxic reactions mainly involve the central nervous and cardiovascular systems.

Symptoms

In children, early signs of local anaesthetic toxicity may be difficult to detect in cases where the block is given during general anaesthesia.

Acute toxicity: CNS toxicity is a graded response with symptoms and signs of escalating severity. The first symptoms are circumoral paraesthesia, numbness of the tongue, lightheadedness, hyperacusis and tinnitus. Visual disturbance and muscular tremors are more serious and precede the onset of generalised convulsions. These signs must not be mistaken for neurotic behaviour.

Unconsciousness and grand mal convulsions may follow. These may last from a few seconds to several minutes. Hypoxia and hypercarbia occur rapidly following convulsions due to the increased muscular activity, together with the interference with normal respiration and loss of the airway. In severe cases, apnoea may occur. Acidosis increases the toxic effects of local anaesthetics.

Recovery is due to redistribution of the local anaesthetic drug from the central nervous system and metabolism. Recovery may be rapid unless large amounts of the drug have been injected.

Cardiovascular toxicity: Cardiovascular toxicity indicates a more severe situation. Hypotension, bradycardia, decreased cardiac output, heartblock, arrhythmia and even cardiac arrest may occur as a result of huge systemic concentrations of local anaesthetics.

Cardiovascular toxic effects are generally preceded by signs of toxicity in the central nervous system, unless the patient is receiving a general anaesthetic or is heavily sedated with drugs such as a benzodiazepine or a barbiturate. In rare cases, cardiac arrest has occurred without prodromal CNS effects.

Management of adverse effects: Treatment of toxicity should be to discontinue administration of lignocaine. Institute emergency resuscitative procedures and administer the emergency drugs necessary to manage the situation. Adequate ventilation of the patient (including oxygen if necessary) should be ensured and convulsions should be arrested if present. For severe convulsions, small increments of diazepam or a short acting barbiturate such as thiopentone should be administered. Suxamethonium will stop the muscle convulsions rapidly but will require tracheal intubation and controlled ventilation, and should only be used by those familiar with these procedures. If ventricular fibrillation or cardiac arrest occurs, effective cardiovascular resuscitation treatment must be instituted and maintained for a prolonged period if necessary. Optimal oxygenation and ventilation, and circulatory support as well as treatment of acidosis are of vital importance. Dialysis is of negligible value in the treatment of acute overdosage with lignocaine.

Presentation

Lignocaine Injection 1% 50 mg in 5 mL sterile ampoules. (50s)

Storage

Store below 30°C.

Single use only. Discard unused portion.

The expiry date (month/year) is stated on the package after EXP.

Manufacturer:

Bridgewest Perth Pharma Pty Ltd

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