NEW ZEALAND DATA SHEET

1. PRODUCT NAME
Xylocaine Jelly topical gel 2% w/w.

2. QUALITATIVE AND QUANTITATIVE COMPOSITION
Each gram of Xylocaine Jelly contains lidocaine (lignocaine) hydrochloride 20 mg.

Excipients with known effect: methyl hydroxybenzoate, propyl hydroxybenzoate.

(Note: Lidocaine is another name for lignocaine. Lignocaine is mostly used in this document)

For the full list of excipients, see section 6.1.

3. PHARMACEUTICAL FORM
Xylocaine Jelly is a clear to almost clear, slightly coloured highly viscous liquid.

4. CLINICAL PARTICULARS

4.1 Therapeutic indications
Xylocaine Jelly is indicated as a surface anaesthetic and lubricant for:

- The male and female urethra during cystoscopy, catheterisation, exploration by sound and other endourethral procedures.
- Nasal and pharyngeal cavities in endoscopic procedures such as gastroscopy and bronchoscopy.
- During proctoscopy and rectoscopy.
- Tracheal intubation.

To relieve pain after circumcision in children.

4.2 Dose and method of administration
Xylocaine Jelly provides prompt and profound anaesthesia of mucous membranes, giving effective anaesthesia of long duration (approximately 20-30 minutes). Anaesthesia usually occurs rapidly (within 5 minutes depending upon the area of application).

As with any local anaesthetic, the safety and effectiveness of lignocaine depend on the proper dosage, the correct technique, adequate precautions and readiness for emergencies.

The following dosage recommendations should be regarded as a guide. The clinician's experience and knowledge of the patient's physical status are of importance in calculating the required dose.

Absorption from mucous membranes is variable but especially high from the bronchial tree. The absorption of lignocaine jelly from the nasopharynx is usually lower than with other lignocaine products. Blood concentrations of lignocaine after

instillation of the jelly in the intact urethra and bladder in doses up to 800 mg are fairly low and below toxic levels.

Debilitated or elderly patients, acutely ill patients or patients with sepsis should be given doses commensurate with their age, weight and physical condition.

In children under the age of 12 years the dose should not exceed 6 mg/kg.

Children over 12 years of age should be given doses commensurate with their age and weight.

No more than four doses should be given in a 24 hour period.

Urethral anaesthesia

Surface anaesthesia of the **male** adult urethra: for adequate analgesia in males 20 mL (equivalent to 400 mg lignocaine hydrochloride) jelly is required. The jelly is instilled slowly until the patient has a feeling of tension or until almost half the tube (10 mL equivalent to 200 mg lignocaine hydrochloride) has been emptied. A penile clamp is then applied for several minutes at the corona, after which the rest of the jelly is instilled.

When anaesthesia is especially important, e.g. during sounding or cystoscopy, a larger quantity of jelly (e.g. 30-40 mL) may be instilled in 3-4 portions and allowed to act for 10 minutes before insertion of the instrument. The jelly instilled into the bladder is also effective for procedures in this region.

Surface anaesthesia of the **female** adult urethra: instil 5-10 mL in small portions to fill the whole urethra. In order to obtain adequate anaesthesia, several minutes should be allowed to elapse prior to performing urological procedures.

Endoscopy

The instillation of 10-20 mL is recommended for adequate analgesia and a small amount may be applied to the lubricating instrument. When combined with other lignocaine products (e.g. for bronchoscopy), the total dose of lignocaine should not exceed 400 mg.

Proctoscopy and rectoscopy

Up to 20 mL can be used for anal and rectal procedures. The total dose should not exceed 400 mg lignocaine.

Lubrication for endotracheal intubation

About 2 mL applied to the surface of the tube just prior to insertion. Care should be taken to avoid introducing the product into the lumen of the tube.

4.3 Contraindications

Known history of hypersensitivity to local anaesthetics of the amide type, or other components of the jelly.

Hypersensitivity to methyl and/or propyl hydroxybenzoate (methyl-/propyl paraben), or to their metabolite para amino benzoic acid (PABA). Formulations of lignocaine containing parabens should be avoided in patients allergic to ester local anaesthetics or their metabolite PABA.

4.4 Special warnings and precautions for use

Excessive doses of lignocaine products or short intervals between doses, can result in high plasma levels and serious adverse effects. Patients should be instructed to adhere strictly to the recommended dosage (the management of serious adverse reactions may require the use of resuscitative equipment, oxygen and other resuscitative medicines (see **OVERDOSE**).

Absorption from wound surfaces and mucous membranes is relatively high and especially high in the bronchial tree. The absorption of lignocaine jelly from the nasopharynx is variable but usually lower than with other lignocaine products. Following instillation in urethra and bladder, adsorption is low. Lignocaine jelly should be used with caution in patients with traumatised mucosa and/or sepsis in the region of the proposed application.

The oropharyngeal use of topical anaesthetic agents may interfere with swallowing and thus enhance the danger of aspiration. Numbness of the tongue or buccal mucosa may increase the danger of biting trauma.

When used for endotracheal tube lubrication, care should be taken to avoid introduction of the jelly into the lumen of the tube. The jelly may dry on the inner surface leaving a residue which tends to clump with flexion, narrowing the lumen. There have been rare reports in which this residue has caused the lumen to occlude.

Patients treated with anti-arrhythmic drugs class III (e.g. amiodarone) should be kept under close surveillance and ECG monitoring considered, since cardiac effects may be additive.

If the dose or administration is likely to result in high blood levels, some patients require special attention to prevent potentially dangerous side effects:

- Patients with partial or complete heart block.
- The elderly and patients in poor general health.
- Patients with advanced liver disease or severe renal dysfunction.

Xylocaine Jelly is probably porphyrinogenic and should only be prescribed to patients with acute porphyia on strong or urgent indications. Appropriate precautions should be taken for all porphyric patients.

4.5 Interactions with other medicines and other forms of interaction

Lignocaine should be used with caution in patients receiving agents structurally related to local anaesthetics, since the toxic effects are additive.

Specific interaction studies with lignocaine and anti-arrhythmic drugs class III (e.g. amiodarone) have not been performed, but caution when treating patients is advised (see **Special warnings and precautions for use**).

Medicines that reduce the clearance of lignocaine (e.g. cimetidine or betablockers) may cause potentially toxic plasma concentrations when lignocaine is given in repeated high doses over a long period of time. Such interactions should be of no clinical importance following short term treatment with lignocaine (e.g. Xylocaine Jelly) at recommended doses.

4.6 Fertility, pregnancy and lactation

It is reasonable to assume that a large number of pregnant women and women of child-bearing age have been given lignocaine. No specific disturbances to the

reproductive process have so far been reported, e.g. no increased incidence of malformations.

Like other local anaesthetics, lignocaine may enter the mother's milk, but in such small amounts that there is generally no risk of this affecting the neonate.

4.7 Effects on ability to drive and use machines

Depending on the dose, local anaesthetics may have a very mild effect on mental function and may temporarily impair locomotion and co-ordination.

4.8 Undesirable effects

LOCAL REACTIONS

An increased incidence of postoperative "sore throat" has been reported following endotracheal tube lubrication with lignocaine jelly.

ALLERGIC REACTIONS

Allergic reactions (in most severe instances anaphylactic shock) to local anaesthetics of the amide type are rare (<1/1000). Other constituents of the jelly e.g. methyl hydroxybenzoate and propyl hydroxybenzoate also cause this type of reaction.

ACUTE SYSTEMIC TOXICITY

Lignocaine may have acute toxic effects if high systemic levels occur due to fast absorption or overdosage (see **Pharmacokinetic properties** and **OVERDOSE**).

Reporting of suspected adverse reactions

Reporting suspected adverse reactions after authorisation of the medicine is important. It allows continued monitoring of the benefit/risk balance of the medicine. Healthcare professionals are asked to report any suspected adverse reactions https://nzphvc.otago.ac.nz/reporting/.

4.9 Overdose

For information on the management of overdose, contact the National Poisons Centre, ph: 0800 POISON (0800 764 766).

ACUTE SYSTEMIC TOXICITY

Toxic reactions originate mainly in the central nervous system and the cardiovascular system.

Central nervous system toxicity is a graded response, with symptoms and signs of escalating severity. The first symptoms are circumoral paraesthesia, numbness of the tongue, light-headedness, hyperacusis and tinnitus. Visual disturbance and muscular tremors are more serious and precede the onset of generalised convulsions. Unconsciousness and grand mal convulsions may follow, which 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. In severe cases apnoea may occur. Acidosis increases the toxic effects of local anaesthetics.

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

Cardiovascular effects are only seen in cases with high systemic concentrations. Severe hypotension, bradycardia, arrhythmia and cardiovascular collapse may be the result in such cases.

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 barbiturate.

TREATMENT OF ACUTE TOXICITY

Should symptoms of systemic toxicity occur, the signs are anticipated to be similar in nature to those following the administration of local anaesthetics by other routes. Local anaesthetic toxicity is manifested by symptoms of nervous system excitation and, in severe cases, central nervous and cardiovascular depression.

Severe neurological symptoms (convulsions, CNS depression) must be treated symptomatically by respiratory support and the administration of anticonvulsive drugs.

If circulatory arrest should occur, immediate cardiopulmonary resuscitation should be instituted. Optimal oxygenation and ventilation and circulatory support as well as treatment of acidosis are of vital importance.

5. PHARMACOLOGICAL PARTICULARS

5.1 Pharmacodynamic properties

Xylocaine Jelly provides prompt and profound anaesthesia of mucous membranes and lubrication which reduces friction. Its water-miscible base, characterised by high viscosity and low surface tension, brings the anaesthetic into intimate and prolonged contact with the tissue, giving effective anaesthesia of long duration (approximately 20-30 minutes). Anaesthesia usually occurs rapidly (within 5 minutes, depending upon the area of application).

Lignocaine, like other local anaesthetics, causes a reversible blockade of impulse propagation along nerve fibres by preventing the inward movement of sodium ions through the nerve membrane. Local anaesthetics of the amide type are thought to act within the sodium channels of the nerve membrane.

Local anaesthetic drugs may also have similar effects on excitable membranes in the brain and myocardium. If excessive amounts of drug reach the systemic circulation rapidly, symptoms and signs of toxicity will appear, emanating from the central nervous and cardiovascular systems.

Central nervous system toxicity (see **OVERDOSE**) usually precedes the cardiovascular effects as it occurs at lower plasma concentrations. Direct effects of local anaesthetics on the heart include slow conduction, negative inotropism and possibly cardiac arrest.

5.2 Pharmacokinetic properties

Lignocaine is absorbed following topical administration to mucous membranes, its rate and extent of absorption being dependent upon concentration and the total dose administered, the specific site of application, and the duration of exposure. In general, the rate of absorption of local anaesthetic agents following topical application is most rapid after intratracheal and bronchial administration. Lignocaine

is also well-absorbed from the gastrointestinal tract, although little intact drug appears in the circulation because of biotransformation in the liver.

Normally about 65% of the lignocaine is bound to plasma proteins. Amide local anaesthetics are mainly bound to alpha-1-acid glycoprotein but also to albumin.

Lignocaine crosses the blood-brain and placental barriers, presumably by passive diffusion.

The main elimination pathway of lignocaine is by liver metabolism. The primary route of lignocaine in human is N-dealkylation to monoethylglycine xylidine(MEGX), followed by hydrolysis to 2,6-xylidine and hydroxylation to 4-hydroxy-2,6-xylidine. MEGX can also be further dealkylated to glycine xylidine (GX). The pharmacological/toxicological actions of MEGX and GX are similar to, but less potent than, those of lignocaine. GX has a longer half-life (about 10 hours) than lignocaine and may accumulate during long-term administration. Approximately 90% of the lignocaine administrated intravenously is excreted in the form of various metabolites, and less than 10 % is excreted unchanged in the urine. The primary metabolite in urine is a conjugate of 4-hydroxy-2,6-xylidine, accounting for about 70-80% of the dose excreted in the urine.

The elimination half-life of lignocaine following an intravenous bolus injection is typically 1.5 to 2.0 hours. Because of the rapid rate at which lignocaine is metabolised, any condition that affects liver function may alter lignocaine kinetics. The half-life may be prolonged two-fold or more in patients with liver dysfunction. Renal dysfunction does not affect lignocaine kinetics but may increase the accumulation of metabolites.

Factors such as acidosis and the use of CNS stimulants and depressants affect the CNS levels of lignocaine required to produce overt systemic effects. Objective adverse manifestations become increasingly apparent with increasing venous plasma levels above 6.0 µg free base per mL.

5.3 Preclinical safety data

In animal studies the toxicity noted after high doses of lignocaine consisted of effects on the central nervous and cardiovascular systems. No drug related adverse effects were seen in reproduction toxicity studies, neither did lignocaine show a mutagenic potential in either *in vitro* or *in vivo* mutagenicity tests. Cancer studies have not been performed with lignocaine, due to the area and duration of therapeutic use for this drug.

Genotoxicity tests with lignocaine showed no evidence of mutagenic potential. A metabolite of lignocaine, 2,6-xylidine, showed weak evidence of activity in some genotoxicity tests. The metabolite 2,6-xylidine has been shown to have carcinogenicity potential in preclinical toxicological studies evaluating chronic exposure. Risk assessments comparing the calculated maximum human exposure from intermittent use of lignocaine, with the exposure used in preclinical studies, indicate a wide margin of safety for clinical use.

6. PHARMACEUTICAL PARTICULARS

6.1 List of excipients

Hypromellose, sodium hydroxide, hydrochloric acid, methyl hydroxybenzoate, propyl hydroxybenzoate and purified water.

6.2 Incompatibilities

Not applicable.

6.3 Shelf life

24 months.

6.4 Shelf life

Store at or below 25°C.

6.5 Nature and contents of container

30 mL aluminium tube.

6.6 Special precautions for disposal and other handling

Not applicable.

7. MEDICINE SCHEDULING

Pharmacy only medicine.

8. SPONSOR

Pharmacy Retailing (NZ) Limited
Trading as Healthcare Logistics
58 Richard Pearse Drive
Airport Oaks
Auckland
New Zealand

9. DATE OF FIRST APPROVAL

31/12/1969

10. DATE OF FIRST REVISION OF THE TEXT

11 January 2018

SUMMARY TABLE OF CHANGES

Section changed	Summary of new information
	Update to the SPC-style format