NEW ZEALAND DATA SHEET

1. PRODUCT NAME

Suxamethonium Chloride 50 mg/mL solution for injection.

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

Each 2 mL of solution contains 100 mg of suxamethonium chloride.

For a full list of excipients, see section 6.1.

3. PHARMACEUTICAL FORM

Solution for Injection or Infusion. Clear, colourless solution.

pH of the solution 3.0-4.2.

Osmolality of the product is 300-365 mOsmol/kg.

4. CLINICAL PARTICULARS

4.1 THERAPEUTIC INDICATIONS

For the production of skeletal muscle relaxation in anaesthesia. Suited for procedures requiring only brief relaxation such as endotracheal intubation, endoscopic examinations, orthopaedic manipulations, short surgical procedures and electro-convulsive therapy.

4.2 DOSE AND METHOD OF ADMINISTRATION

Dosage is individualised and its administration should be determined after careful assessment of the patient. The dose of suxamethonium is dependent on bodyweight, the degree of muscle relaxation required and the response of individual patients. Suxamethonium causes paralysis of the respiratory muscles, therefore after administration, respiration must be controlled. It should not be administered to a conscious patient.

Suxamethonium should not be mixed with any neuromuscular blocking agent, nor with general anaesthetics such as short acting barbiturates, nor any other therapeutic agent in the same syringe.

Suxamethonium Chloride contains no antimicrobial agent. It should be used only once and any residue discarded.

An initial test dose of 0.1 mg/kg may be given intravenously to determine the patient's response.

ADULT

For short procedures, such as endotracheal intubation the usual adult dose is 0.6 mg/kg (range 0.3-1.1 mg/kg) administered IV over 10 to 30 seconds. This dose produces muscle relaxation in about 60 seconds and has a duration of approximately 4 to 6 minutes. Larger doses produce more prolonged muscle relaxation.

For more prolonged surgical procedures in an adult, suxamethonium is commonly given by IV infusion at a rate of 2.5 -4.3 mg/minute. When given by intravenous infusion suxamethonium should be diluted to 0.1 to 0.2% (1-2 mg/mL) in 5% dextrose solution or sterile isotonic saline.

CHILDREN

Neonates and premature infants may be relatively resistant to suxamethonium.

The usual paediatric IV dose is 1 to 2 mg/kg. If necessary, additional doses may be administered in accordance with patient's response. Continuous IV infusions of suxamethonium are considered unsafe in neonates and children because of the risk of inducing malignant hyperthermia.

Intravenous bolus in children may result in profound bradycardia or on occasion asystole. This tends to be more common after a second dose. Pre-treatment with atropine can reduce the risk of

bradycardia.

When a suitable vein is inaccessible, suxamethonium may occasionally be given by intramuscular injection. A suggested IM dose for adults and children may be up to 2.5 mg/kg but the total dose should not exceed 150 mg.

Diluted solutions of suxamethonium must be used within 24 hours of preparation. Discard unused solutions.

4.3 CONTRAINDICATIONS

Patients with a personal or familial history of malignant hyperthermia, genetically determined disorders of pseudocholinesterase, myopathies associated with elevated creatinine phosphokinase (CPK) values, Duchenne's muscular dystrophy, known hypersensitivity to suxamethonium, severe hyperkalaemia, acute narrow-angle glaucoma, and the presence of penetrating eye injuries (suxamethonium may cause a slight transient increase in intraocular pressure).

It is also contraindicated in patients after the acute phase of injury following major burns, multiple trauma, renal impairment with a raised plasma-potassium concentration, or in those with extensive muscle degeneration such as recent paraplegia and severe long-lasting sepsis because such patients may become severely hyperkalaemic when given suxamethonium, resulting in cardiac arrhythmia or arrest.

4.4 SPECIAL WARNINGS AND PRECAUTIONS FOR USE

Suxamethonium should only be administered under strict supervision of an anaesthetist familiar with its actions, characteristics and hazards who is skilled in the management of artificial respiration and only when facilities are instantly available for endotracheal intubation and for providing adequate ventilation of the patient, including the administration of oxygen under positive pressure. Be prepared to assist or control respiration.

Suxamethonium has no effect on consciousness, pain threshold or cerebration. It should therefore only be used with adequate anaesthesia.

MALIGNANT HYPERTHERMIA

The abrupt onset of malignant hyperthermia, a very rare hypermetabolic process of skeletal muscle, may be triggered by suxamethonium. Early premonitory signs include muscle rigidity, tachycardia, tachypnoea unresponsive to increased depth of anaesthesia, evidence of increased oxygen requirement and carbon dioxide production, rising temperature and metabolic acidosis.

On evidence of these symptoms the anaesthetic and suxamethonium should be discontinued and supportive measures implemented including administration of oxygen, sodium bicarbonate, lowering of temperature, restoration of fluids and electrolyte balance, maintenance of adequate urinary output and administration of IV dantrolene according to a standard protocol.

HYPERKALAEMIA

Administration of suxamethonium causes an immediate rise in serum potassium. This rise is normally small but may be prolonged and exaggerated in patients taking beta-blockers.

Great caution should also be observed in patients with pre-existing hyperkalaemia or electrolyte imbalance, uraemia, hemiplegia, paraplegia, extensive burns, massive trauma, diffuse intracranial lesions (head injury, encephalitis, ruptured cerebral aneurysm), tetanus, acute anterior horn cell disease, extensive denervation of skeletal muscle due to disease or injury of the CNS, or who have degenerative neuromuscular disease and in severe long-lasting sepsis. Such patients may become severely hyperkalaemic when given suxamethonium, resulting in cardiac arrhythmia or arrest (See section 4.3).

With burns or trauma the period of greatest risk is from about 10-90 days after the injury, but may be prolonged further if there is delayed healing or persistent infection. These patients may still react abnormally to suxamethonium 2 years after the injury. In neuromuscular disease the greatest risk period is usually from 3 weeks to 6 months after onset, but severe hyperkalaemia may occur after 24 to 48 hours or later than 6 months. Patients with severe sepsis for more than a week should be considered at risk of hyperkalaemia and suxamethonium should not be given until the infection has

cleared.

HYPERKALAEMIA RHABDOMYOLYSIS

There is a risk of cardiac arrest from hyperkalaemia due to rhabdomyolysis, particularly in male patients with muscular dystrophy.

LOW PLASMA PSEUDOCHOLINESTERASE

Recovery from suxamethonium may occasionally be delayed possibly due to a low serum pseudocholinesterase level; this may occur in patients suffering from severe liver disease, cancer, malnutrition, severe dehydration, collagen diseases, severe anaemia, myxoedema, burns, pregnancy and the puerperium, severe infections, myocardial infarction, renal impairment and abnormal body temperature.

Also exposure to neurotoxic insecticides or weed killers, antimalarial or anti-cancer agents, monoamine oxidase (MAO) inhibitors, the contraceptive pill, pancuronium, chlorpromazine ecothiapate or neostigmine may result in low levels of pseudocholinesterase.

Suxamethonium should be administered with extreme caution and in reduced doses in such patients. If low pseudocholinesterase concentration is suspected slow administration of a small test dose of suxamethonium (5 to 10 mg as a 0.1% solution) should be considered.

ANTIDYSRHYTHMIC AGENTS

Suxamethonium should be administered with great caution in patients receiving quinidine and those who have been digitalised or who may have digitalis toxicity. In these circumstances the rise in serum potassium due to suxamethonium may possibly cause arrhythmias.

DELAYED RECOVERY

When recovery from suxamethonium is delayed, assisted respiration sufficient for full oxygenation, yet avoiding excessive elimination of carbon dioxide, should be maintained until paralysis ceases. This should be combined with light narcoisis, e.g. nitrous oxide/oxygen mixture.

Neostigmine should not be given when prolonged apnoea follows a single dose of suxamethonium. Neostigmine and other anticholinesterase agents may have the effect of intensifying the depolarisation block caused by suxamethonium.

NONDEPOLARISING BLOCKADE

If suxamethonium is given repeatedly or over a prolonged period the depolarising block may change to one with characteristics of a nondepolarising block. This may be associated with prolonged respiratory depression and apnoea. Following a positive diagnosis of a nondepolarising blockade the administration of neostigmine preceded by atropine may be considered.

DEBILITATED PATIENTS

Use with caution in patients who are hypoxic or those who have cardiovascular, hepatic, pulmonary, metabolic or renal disorders or myasthenia gravis. The action of suxamethonium may be altered in these patients. Its use is not advisable in patients with phaeochromocytoma. As suxamethonium produces muscle contractions before relaxation it should be used with caution in patients with bone fractures.

Suxamethonium should be avoided in patients with myotonias, as response is unpredictable.

USE IN EYE SURGERY

Suxamethonium causes a slight transient increase in intraocular pressure immediately after injection and during the fasciculation phase. It should therefore be used cautiously if at all during intraocular surgery and in patients with glaucoma.

4.5 INTERACTION WITH OTHER MEDICINES AND OTHER FORMS OF INTERACTION

Suxamethonium, a depolarising muscle relaxant of short duration, may interact with the following:

Anti-arrhythmics:

Lidocaine, procaine, procainamide, chloroprocaine, cocaine, quinidine and verapamil enhance muscle relaxant effect.

Antibacterials:

Effect of muscle relaxants is enhanced by aminoglycosides such as dibekacin, kanamycin, neomycin, ribostamycin and streptomycin, the effect of suxamethonium is also enhanced by vancomycin, azlocillin, clindamycin, colistin, piperacillin and polymyxin B.

Anticholinesterases:

Cholinesterase and pseudocholinesterase both degrade suxamethonium. Therefore anticholinesterases will enhance suxamethonium. Examples of anticholinesterases include donepezil, galantamine, rivastigmine, aprotinin, cyclophosphamide, dexpanthenol, ecothiopate, metoclopramide (non-selective drug), neostigmine, phenelzine (MAOI), promazine, quinine and chloroquine (antimalarials), tacrine and trimetaphan (ganglion blocking drug). Exposure to pesticides may also reduce pseudocholinesterase activity such as diazinon, malathion and sheep dips.

Blood transfusion:

Blood transfusions may contribute to an increase in plasma cholinesterase levels, as a result of which the therapeutic effect of suxamethonium can be influenced unpredictably.

ACE inhibitors:

Concomitant use of medicines that may increase potassium levels, such as ACE inhibitors, can cause hyperkalaemia (see section 4.3).

Antiepileptics:

Effect of muscle relaxants antagonised by carbamazepine and phenytoin (recovery from neuromuscular blockade accelerated).

Antineoplastics (anticancer drugs):

Cyclophosphamide, chlormethine, thiotepa and tretamine all reduce pseudocholinesterase activity.

Benzodiazepines:

Diazepam and midazolam may alter the depth/duration of suxamethonium.

Calcium-channel Blockers:

Nifedipine and verapamil enhance effect of non-depolarising muscle relaxants; hypotension, myocardial depression, and hyperkalaemia reported with intravenous dantrolene and verapamil.

Cardiac Glycosides:

Arrhythmias if suxamethonium given with digoxin.

Cytotoxics:

Cyclophosphamide and thiotepa enhance effect of suxamethonium.

General Anaesthetics:

Propofol can cause serious bradycardia if given with suxamethonium and fentanyl citrate-droperidol (Innovar) enhances the effects of suxamethonium. Suxamethonium also interacts with halothane, isoflurane, enflurane, cyclopropane, propanidid and ether.

Magnesium Salts:

Parenteral magnesium enhances effect of suxamethonium.

Parasympathomimetics:

Demecarium and ecothiopate eye-drops, neostigmine and pyridostigmine, and possibly donepezil enhance effect of suxamethonium but antagonise effect of non-depolarising muscle relaxants.

Sympathomimetics:

Bambuterol enhances effect of suxamethonium.

4.6 FERTILITY, PREGNANCY AND LACTATION

No studies of the effect of suxamethonium on female fertility or pregnancy have been performed.

Pregnancy

Suxamethonium has no direct action on the uterus or other smooth muscle structures. In normal therapeutic doses it does not cross the placental barrier in sufficient amounts to affect the respiration of the infant. The benefits of the use of suxamethonium as part of a rapid sequence induction for general anaesthesia normally outweighs the possible risk to the foetus. Plasma cholinesterase levels fall during the first trimester of pregnancy to about 70 to 80% of their pre-pregnancy values; a further fall to about 60 to 70% of the pre-pregnancy levels occurs within 2 to 4 days after delivery. Plasma cholinesterase levels then increase to reach normal over the next 6 weeks. Consequently, a high proportion of pregnant and puerperal patients may exhibit mildly prolonged neuromuscular blockade following suxamethonium injection. Suxamethonium is not embryotoxic or teratogenic in two animal species. The use of suxamethonium may be considered during pregnancy, if necessary.

Breast-feeding

It is not known whether suxamethonium or its metabolites are excreted in human milk. However, because the drug is rapidly hydrolyzed by plasma cholinesterase (pseudocholinesterase) to an inactive metabolite, no effects on the breastfed newborns/infants are anticipated.

Fertility

There is no data from the use of suxamethonium on fertility. However, because the drug is rapidly hydrolyzed by plasma cholinesterase (pseudocholinesterase) to an inactive metabolite, no effects on fertility are anticipated once the pharmacological effect is over.

4.7 EFFECTS ON ABILITY TO DRIVE AND USE MACHINES

Not relevant

4.8 UNDESIRABLE EFFECTS

Adverse reactions are listed below by system organ class and frequency. Estimated frequencies were determined from published data. Frequencies are defined as follows: very common (≥1/10); common (≥1/100 to <1/10), uncommon (≥1/1,000 to <1/100); rare (≥1/10,000 to <1/1,000); very rare (<1/10,000).

Immune system disorders	
Very rare	Anaphylactic reactions.
Eye disorders	
Common	Increased intraocular pressure.
Cardiac disorders	
Common	Bradycardia, tachycardia.
Rare	Arrhythmias (including ventricular arrhythmias), cardiac arrest[1]
Vascular disorders	
Common	Skin flushing.
Not known	Hypertension and hypotension
Respiratory, thoracic and mediastinal disorders	
Rare	Bronchospasm, prolonged respiratory depression[2], apnoea[2]
Gastrointestinal disorders	
Very common	Increased intragastric pressure

Unknown	Excessive salivation
Skin and subcutaneous tissue disorders	
Common	Rash
Musculoskeletal and connective tissue disorders	
Very common	Muscle fasciculation, post-operative muscle pains
Common	Myoglobinaemia[3], myoglobinuria[3]
Rare	Trismus
General disorders and administration site conditions	
Very rare	Malignant hyperthermia
Investigation	
Common	Transient blood potassium increase

1 There are case reports of hyperkalaemia-related cardiac arrests following the administration of suxamethonium to patients with congenital cerebral palsy, tetanus, Duchenne muscular dystrophy, and closed head injury. Such events have also been reported rarely in children with hitherto undiagnosed muscular disorders.

2 Individuals with decreased plasma cholinesterase activity exhibit a prolonged response to suxamethonium. Approximately 0.05% of the population has an inherited cause of reduced cholinesterase activity (please refer to section 4.4).

3 Rhabdomyolysis has also been reported

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://pophealth.my.site.com/carmreportnz/s/.

4.9 OVERDOSE

The most serious effects of overdosage are apnoea and prolonged muscle paralysis. It is essential to maintain the airway and adequate ventilation until spontaneous respiration is fully restored.

The use of neostigmine to reverse a non-depolarising block is a clinical decision which depends on the subject, the experience, and the judgment of the clinician. If neostigmine is used, its administration should be accompanied by an appropriate dose of atropine.

For risk assessment and advice on the management of overdose please contact the National Poisons Centre on 0800 POISON (0800 764766).

5. PHARMACOLOGICAL PROPERTIES

5.1 PHARMACODYNAMIC PROPERTIES

Pharmacotherapeutic group: Peripherally acting muscle relaxants, choline derivatives
ATC code M03AB01

Suxamethonium is closely related in structure to acetylcholine. Suxamethonium is quickly hydrolysed by plasma cholinesterase. Suxamethonium acts on the skeletal muscle motor endplate just like acetylcholine as an agonist, to cause flaccid paralysis of muscle (phase 1 block). Suxamethonium diffuses slowly to the endplate and the concentration at the endplate persists for long enough to cause loss of electrical excitability. The depolarization of the muscle endplate establishes a voltage gradient and this causes opening of voltage-dependent ion channels of the muscle leading to transient contraction of the muscle. Although the end-plate stays depolarised, the muscle membrane accounts for this depolarization and remains flaccid. If suxamethonium is kept continuously present during infusion, the junctional membrane slowly regains its resting potential with the return of neuromuscular transmission; to maintain the effect, a higher infusion rate is required (tachyphylaxis). With continued

infusion, neuromuscular transmission will fail again (phase 2 block) even though the membrane potential of the end-plate stays unchanged and normal or near normal. A phase 2 block has clinical characteristics of a non-depolarizing block. A phase 2 block may be associated with prolonged neuromuscular blockade and apnoea. The mechanism of this block is not known but channel blocking by penetration of suxamethonium into the sub-end plate cytoplasm, intracellular accumulation of calcium and sodium, the loss of intracellular potassium, and activation of Na,K-ATPase all contribute.

Neuromuscular-blocking drugs are used mainly in anaesthesia to produce muscle relaxation. Although complete relaxation can be produced by anaesthetic drugs alone, the concentrations needed to obliterate spinal reflexes are high and it is much more satisfactory to produce paralysis by blocking neuromuscular transmission. The drugs are given intravenously, and act within about 30 to 60 seconds. Suxamethonium acts for about 2 to 6 minutes, being hydrolysed by plasma cholinesterase (pseudocholinesterase).

5.2 PHARMACOKINETIC PROPERTIES

Absorption

Suxamethonium has a rapid onset and a short duration of action. Following intravenous (IV) administration of a single therapeutic dose in healthy adults, complete muscle relaxation occurs within ½ to 1 minute, persists for about 2 - 3 minutes, and gradually dissipates within 10 minutes.

Following intramuscular (IM) administration the onset of action occurs in about 2-3 minutes, with a duration ranging from 10-30 minutes.

The duration of action is prolonged in patients with low plasma pseudocholinesterase concentration.

Distribution

Suxamethonium crosses the placenta, generally in small amounts.

Elimination

Plasma pseudocholinesterases hydrolyse suxamethonium to succinylmonocholine (relatively inactive) and choline. Approximately 10% of drug is excreted unchanged in the urine.

Patients with impaired renal function may occasionally experience prolonged apnoea due to accumulation of succinylmonocholine.

6. PHARMACEUTICAL PARTICULARS

6.1 LIST OF EXCIPIENTS

Hydrochloric acid (for pH adjustment)
Water for injection

6.2 INCOMPATABILITIES

Suxamethonium should not be mixed with any neuromuscular blocking agent, nor with general anaesthetics such as short acting barbiturates, nor any other therapeutic agent in the same syringe.

6.3 SHELF-LIFE

18 months

Once opened, use immediately.

6.4 SPECIAL PRECAUTION FOR STORAGE

Store between 2°- 8°C. REFRIGERATE - DO NOT FREEZE.

Keep in the outer carton to protect from light.

For storage conditions after first opening, see section 6.3.

6.5 NATURE AND CONTENTS OF CONTAINER

Type 1 clear glass 2 mL ampoule.

Pack size of 10 ampoules.

6.6 SPECIAL PRECAUTIONS FOR DISPOSAL

Any unused medicine or waste material should be disposed of in accordance with local requirements.

7. MEDICINE SCHEDULE

Prescription Medicine.

8. SPONSOR

Max Health Ltd
PO Box 44452
Pt Chevalier, Auckland 1246

Telephone: (09) 815 2664.

9. DATE OF FIRST APPROVAL

2 May 2013

10. DATE OF REVISION OF THE TEXT

8 April 2025

SUMMARY OF CHANGES TABLE

Section changed	Summary of new information
All	• Editorial
4.8	• Updated AE reporting URL as per Medsafe template
4.9	• Added ‘risk assessment’ wording as per Medsafe template.
6.3	• Corrected to include ‘Once opened, use immediately’.
6.4	• Correced to include ‘protect fom light. For storage conditions after first opening, see section 6.3.’