NEW ZEALAND DATA SHEET

1 PRODUCT NAME

Suxamethonium chloride dihydrate 100 mg/2 mL solution for injection.

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

Suxamethonium chloride dihydrate 50 mg/mL

2 mL sterile solution of pH 3.0 – 5.0 containing 100 mg of suxamethonium chloride dihydrate.
For the full list of excipients, see Section 6.1.

3 PHARMACEUTICAL FORM

Injection solution. Clear, colourless solution.

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 Injection contains no antimicrobial agent. Product is for single use in one patient only. Discard any residue.

An initial test dose of 0.1 mg/kg may be given intravenously to determine the patients 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 maybe administered in accordance with the 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 i.m. dose for adults and children may be up to 2.5 mg/kg but the total dose should not exceed 150 mg.

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, or 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 positivepressure. 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.

Anaphylaxis

High rates of cross-sensitivity (greater than 50%) between neuromuscular blocking agents have been reported: allergic and non-allergic severe anaphylactic reactions to neuromuscular blocking agents have been reported during anaesthesia induction, sometimes in subjects who have never been exposed to muscle relaxants. The reactions have in some cases been life-threatening and fatal. See section 4.8. The common clinical manifestations are cutaneous eruption i.e., rash, erythema, which are generalised or limited to the injection site. This may be further complicated by anaphylactic shock and/or bronchospasm. In some cases, the bronchospasm and/or anaphylactic shock are not associated by cutaneous manifestations. The appearance of the first signs requires the definitive discontinuation of the administration of suxamethonium if not already completed, and the initiation of symptomatic treatment. Due to the potential severity of these reactions, the necessary precautions, such as the immediate availability of appropriate emergency treatment, should be taken. Where

possible, before administering suxamethonium, hypersensitivity to other neuromuscular blocking agents should be excluded. Suxamethonium, should only be used when absolutely essential in susceptible patients. Patients who experience a hypersensitivity reaction under general anaesthesia should be tested subsequently for hypersensitivity to other neuromuscular blockers. Allergy tests must be performed (immediate specimen then cutaneous test).

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 Contraindications).

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, anti-malarial or anti-cancer drugs, monoamine oxidase (MAO) inhibitors, contraceptive pill, pancuronium, chlorpromazine,

ecothiopate 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 - 10 mg as a 0.1% solution) should be considered.

Antidysrhythmic drugs

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 narcosis, e.g. nitrous oxide/oxygen mixture.
Neostigmine should not be given when prolonged apnoea follows a single dose of suxamethonium. Neostigmine and other anticholinesterase drugs 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.

Use in hepatic impairment

Use with caution in patients who have hepatic disorders. The action of suxamethonium may be altered in these patients.

Use in renal impairment

Use with caution in patients who have renal disorders. The action of suxamethonium may be altered in these patients. Patients with impaired renal function may occasionally experience prolonged apnoea due to accumulation of succinylmonocholine.

Use in the elderly

No data available.

Paediatric use

Neonates and premature infants may be relatively resistant to suxamethonium.

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.

Effects on laboratory tests

No data available.

4.5 Interactions with other medicines and other forms of interactions

Co-administration of inhaled anaesthetics (cyclopane, diethylether, halothane and nitrous oxide) may increase the incidence of dysrhythmias (especially bradycardia), apnoea and the occurrence of malignant hyperthermia in susceptible persons. Inhaled anaesthetics have little effect on the usual depolarising neuromuscular blockade of suxamethonium but may enhance the Phase II block (nondepolarising) that may be produced by repeated dosage of suxamethonium. Severe bradycardia and asystole have occurred when suxamethonium is used in anaesthetic regimens with propofol and opioids such as fentanyl.

Drugs which may enhance or prolong the effects of suxamethonium include lignocaine, procaine, oxytocin, oral contraceptives, some non-penicillin antibiotics, (streptomycin, neomycin, kanamycin, capreomycin, tobramycin, framycetin, amikacin, gentamicin, colistin and polymyxins), tacrine, beta-adrenergic blockers, trimethaphan, phenelzine, aprotinin, quinidine, promazine, lithium carbonate, phenytoin, carbamazepine, magnesium salts, quinine, chloroquine, cimetidine, terbutaline sulfate, high dose corticosteroids and cytostatic agents such as cyclophosphamide, thiotepa and azathioprine, selective serotonin reuptake inhibitors. Diazepam may reduce the duration of neuromuscular blockade produced by suxamethonium.

Amphotericin B and thiazide diuretics may increase the effects of suxamethonium secondary to induced electrolyte imbalance. Patients with hypokalemia or hypocalcaemia require reduced doses of suxamethonium.

Inhibitors of plasma cholinesterases such as neostigmine, pyridostigmine bromide, rivastigmine, donepezil, metoclopramide, physostigmine and phospholine iodide can considerably prolong the depolarising action of suxamethonium. It is recommended that long-acting anticholinesterase inhibitor (ecothiopate) eye drops should be discontinued several months prior to administration of suxamethonium.

Administration of suxamethonium prior to or with a nondepolarising muscle relaxant e.g. pancuronium, mivacurium can alter the intensity and/or duration of neuromuscular blockade.

Simultaneous administration of suxamethonium and atracurium significantly reduces the duration of suxamethonium.

Concomitant digoxin or verapamil, and suxamethonium therapy has been reported to result in cardiac arrhythmias.

4.6 Fertility, pregnancy and lactation

Use in pregnancy – Category A

Safety of the use of suxamethonium in pregnancy has not been established with respect to effects on foetal development. Therefore, suxamethonium should not be administered to pregnant women unless the potential benefit outweighs the possible hazards.

Plasma pseudocholinesterase levels are decreased in pregnancy and several days postpartum by approximately 25%, therefore a high proportion of these patients may be expected to show prolonged apnoea.

Suxamethonium crosses the placenta, but generally only in small amounts. Residual neuromuscular blockade may occasionally occur in the neonate after repeated high doses of suxamethonium to the mother during delivery by caesarean section.

4.7 Effects on ability to drive and use machines

The effects of this medicine on a person's ability to drive and use machines were not assessed as part of its registration.

4.8 Adverse effects (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	
Common	Anaphylactic reactions. Anaphylactic shock.
Not known	Hypersensitivity 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 registration of the medicinal product is important. It allows continued monitoring of the benefit-risk balance of the medicinal product. Healthcare professionals are asked to report any suspected adverse reactions at https://pophealth.my.site.com/carmreportnz/s/.

4.9 Overdose

For information on the management of overdose, contact the National Poison Centre on 0800 POISON (0800 764766).

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 nondepolarising 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.

5 PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Mechanism of action

Suxamethonium is an ultra short-acting depolarising-type neuromuscular blocking agent.

Suxamethonium combines with the cholinergic receptors of the motor end plate to produce depolarisation. Neuromuscular transmission is inhibited so long as an adequate concentration of suxamethonium remains at the receptor site.

Suxamethonium has no direct action on smooth muscle structures, including the uterus.

Suxamethonium may produce slowing of heart rate via vagal stimulation.

When suxamethonium is administered over a prolonged period the characteristics of the neuromuscular block may change from the characteristic depolarising type to one resembling a nondepolarising block.

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 1/2 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

Water for injection

Hydrochloric acid for pH adjustment

Sodium hydroxide for pH adjustment

6.2 Incompatibilities

Incompatibilities were either not assessed or not identified as part of the registration of this medicine.

6.3 Shelf life

18 months

6.4 Special precautions for storage

Store between 2° - 8°C. Refrigerate, do not freeze.

6.5 Nature and contents of container

2 mL Type I clear glass vials with grey bromobutyl rubber stoppers, red aluminium seal and red flip-off plastic caps in packs of 10 or 50. Red aluminium seal and red flip-off plastic caps have PARALYZING AGENT printed on with black ink.

6.6 Special precautions for disposal

In Australia, any unused medicine or waste material should be disposed of by taking to your local pharmacy.
To reduce microbiological hazard, use as soon as practicable after preparation. If storage is necessary, hold at 2-8°C for not more than 24 hours.

7 MEDICINE SCHEDULE

Prescription medicine

8 SPONSOR

Medsurge Pharma Ltd
PO Box 331054
Takapuna
Auckland 0622

Marketed and distributed by Medsurge Healthcare Pty Ltd
Telephone: 0800 788 261
Website: www.medsurgehc.com

9 DATE OF FIRST APPROVAL

19 December 2024

10 DATE OF REVISION

26 Jan 2026

Summary table of changes

Section changed	Summary of new information
4.4	Addition of information on Anaphylaxis
4.8	Change in frequency of *Anaphylactic reactions* from very rare to common. Addition of *Anaphylactic shock* as a common adverse reaction. Addition of *Hypersensitivity reactions* as a not known adverse reaction.
6.5	Change in flip top and seal colour