POTASSIUM CHLORIDE ABORNS

NEW ZEALAND DATA SHEET

1. PRODUCT NAME

POTASSIUM CHLORIDE 0.75 g/10 mL Solution for Injection

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

0.75 g (10 mmol) of Potassium Chloride in 10 mL

Sterile Potassium Chloride Concentrate is a sterile solution of potassium chloride in Water for Injections, containing no preservatives.

For a full list of excipients, see section 6.1.

3. PHARMACEUTICAL FORM

Solution for Injection

4. CLINICAL PARTICULARS

4.1 THERAPEUTIC INDICATIONS

1. The treatment of hypokalaemia
2. Treatment of digitalis intoxication.

The IV route is indicated when the patient is unable to take potassium orally or if hypokalaemia is severe.

4.2 DOSE AND METHOD OF ADMINISTRATION

MUST BE DILUTED BEFORE ADMINISTRATION

This product contains no antimicrobial preservative and should be used in one patient on one occasion only.

Sterile Potassium Chloride Concentrate is a concentrated solution and must be diluted before use. Sterile Potassium Chloride Concentrate is administered intravenously only after dilution in a large volume of parenteral fluid.

The dose and rate of injection are dependent upon the individual patient's condition.

THE USUAL MAXIMUM CONCENTRATION IS 40 mmol/L.

In patients whose serum potassium concentration is above 2.5 mmol/L, the rate of infusion should not exceed 10 mmol/hour. The total dose should not exceed 200 mmol/24 hours.

If urgent treatment is required (serum potassium concentration less than 2 mmol/L with ECG changes or paralysis), infuse potassium in a suitable concentration at a rate of 40 mmol/hour, up to a rate of 400 mmol/24-hour period.

In critical states, potassium may be infused in saline (unless saline is contraindicated) rather than in glucose solutions, as the latter may decrease serum potassium concentrations.

To reduce microbiological hazard, use as soon as practical after dilution. If storage is necessary, hold at 2-8°C for not more than 24 hours.

Diluents Compatibility
Potassium Chloride Injection has been reported to be compatible with the following IV infusion fluids:

- Glucose-Ringer's injection combinations
- Glucose-lactated Ringer's injection combinations
- Glucose 5% in lactated Ringer's injection
- Glucose - saline combinations
- Glucose 5% in sodium chloride 0.9%
- Glucose 2.5% in water
- Glucose 5% in water
- Glucose 10% in water
- Glucose 20% in water
- Ringer's injection
- Lactated Ringer's injection
- Sodium chloride 0.45%
- Sodium chloride 0.9%
- Sodium chloride 3%

Potassium Chloride Injection has been reported to be incompatible with the following IV infusion fluids:

- Mannitol
- Sterile fat emulsions containing soya oil and lecithin.

4.3 CONTRAINDICATIONS

Renal impairment with oliguria or azotaemia, ventricular fibrillation, atrioventricular or intraventricular heart block, untreated Addison's disease, hyperadrenalism associated with adrenogenital syndrome, extensive tissue breakdown as in severe burns, acute dehydration, heat cramps, increased sensitivity to potassium administration as in adynamia episodica hereditaria or congenital paramyotonia, hyperkalaemia of any aetiology and hyperchloraemia.

4.4 SPECIAL WARNINGS AND PRECAUTIONS FOR USE

Only use with specialist advice.

Solutions of potassium chloride MUST BE DILUTED before use according to dilution instructions on individual labels. Careful and thorough mixing of solution after dilution is essential.

In patients with impaired mechanisms for excreting potassium, administration of potassium salts can produce hyperkalaemia and cardiac arrest. This is of particular concern in patients

given IV potassium. Potentially fatal hyperkalaemia can develop rapidly and be asymptomatic. Careful monitoring of serum potassium levels during administration and appropriate adjustment of dosage is essential. It is also recommended that acid-base balance, serum electrolytes, ECG and clinical status of the patient be monitored during therapy.

The use of potassium salts in patients with chronic renal disease, adrenal insufficiency or any other condition which impairs potassium excretion, requires particularly careful monitoring of the serum potassium concentration and appropriate dosage adjustment.

Hypokalaemia should not be treated by the concomitant administration of potassium salts and potassium-sparing diuretic (e.g. spironolactone or triamterene), since the simultaneous administration of these agents can produce severe hyperkalaemia.

In patients on a low-salt diet particularly, hypokalaemic hypochloraemic alkalosis is a possibility that may require chloride as well as potassium supplementation.

The treatment of potassium depletion, particularly in the presence of cardiac disease, renal disease or acidosis, requires careful attention to acid-base balance and appropriate monitoring of serum electrolytes, the ECG and the patient's clinical status.

Potassium should be used with caution in diseases associated with heart block since increased serum potassium may increase the degree of block.

Initially do not use with glucose infusions as glucose may further decrease potassium levels.

Parenteral potassium chloride solutions may cause pain if given in a small vein.

Sickle cell disease.

Particularly close monitoring is required where potassium salts are given to patients who are taking medicines that may increase potassium levels (see section 4.5).

Pain at the injection site and phlebitis may occur during IV administration of solutions containing 30 mmol potassium or more per litre.

4.5 INTERACTION WITH OTHER MEDICINES AND OTHER FORMS OF INTERACTION

Potassium sparing diuretics, including triamterene, spironolactone and amiloride increase potassium retention by reducing renal elimination of the potassium ion and hence can produce severe hyperkalaemia.

ACE inhibitors including enalapril and captopril elevate serum potassium and may produce hyperkalaemia when administered concurrently with potassium. ACE inhibitors decrease aldosterone secretion, possibly resulting in potassium retention.

Beta-adrenergic blockade increases both peak serum potassium concentration and the time required for serum potassium to return to basal levels in subjects receiving an acute intravenous potassium load.

Nonsteroidal anti-inflammatory drugs (NSAIDs) may cause hyperkalaemia by inducing secondary hypoaldosteronism following inhibition of renal prostaglandin synthesis.

Heparin reduces the synthesis of aldosterone which may result in hyperkalaemia, especially in patients with underlying renal insufficiency or other problems that impair potassium excretion.

Diuretics, such as thiazide, increase the risk of hypokalaemia when a potassium wasting diuretic is discontinued after concurrent use with a potassium supplement.

Potassium supplementation is not recommended with concurrent use of digitalis glycosides in those patients with severe or complete heart block. Careful monitoring is necessary if potassium chloride is used to correct hypokalaemia in such patients.

Concurrent use with insulin may decrease serum potassium.

Concurrent use with sodium bicarbonate may decrease serum potassium.

Concomitant use of other drugs containing potassium or agents having the potential for hyperkalaemia, may lead to accumulation of potassium:

- aliskerin
- angiotension-II receptor antagonists
- cyclosporine
- tacromilus.

4.6 FERTILITY, PREGNANCY AND LACTATION

PREGNANCY
Both potassium and chloride ions are essential constituents of human tissues and fluids. However, supraphysiological levels of potassium are detrimental to maternal and fetal cardiac function. Exogenous potassium may be used as replacement therapy for pregnant women with hypokalaemia; treatment with oral therapy is always preferred. Serum levels should be closely monitored in pregnant women receiving potassium therapy.

BREAST FEEDING
Potassium is excreted into breast milk. Because of the potential for potassium to cause serious adverse effects on the breastfeeding baby, caution should be exercised when potassium therapy is given to a breastfeeding woman.

4.7 EFFECTS ON ABILITY TO DRIVE AND USE MACHINES

Not relevant

4.8 UNDESIRABLE EFFECTS

The symptoms and signs of potassium intoxication include the following:

CARDIOVASCULAR
Fall in blood pressure, cardiac arrhythmias and heart block. Hyperkalaemia may exhibit the following ECG abnormalities: disappearance of the P-wave, widening and slurring of QRS complex, changes of the S-T segment, tall-peaked T-waves.

GASTROINTESTINAL
Nausea, vomiting, diarrhoea and abdominal discomfort.

OTHER
Paraesthesias of the extremities, flaccid paralysis, listlessness, mental confusion, weakness and heaviness of the legs.

Pain or phlebitis may occur if solutions containing more than 30 mmol/L of potassium are given intravenously.

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/.

4.9 OVERDOSE

SYMPTOMS
If excretory mechanisms are impaired or if IV potassium is administered too rapidly, potentially fatal hyperkalaemia can result (See sections 4.3 and 4.4). However, hyperkalaemia is usually asymptomatic and may be manifested only by an increased serum potassium concentration and characteristic ECG changes (peaking of T-waves, loss of P-wave, depression of S-T segment and prolongation of the QT interval). Late manifestations include muscle paralysis and cardiovascular collapse from cardiac arrest. Other symptoms that may occur are paraesthesia of the extremities, listlessness, mental confusion, weakness or heaviness of the legs, cold skin, grey pallor, peripheral vascular collapse, fall in blood pressure, cardiac arrhythmias and heart block, due to which patients may deteriorate rapidly. Should any of these manifestations occur, discontinue potassium administration immediately.

Extremely high plasma potassium concentrations (8-11 mmol/litre) may cause death from cardiac depression, arrhythmias or arrest.

TREATMENT
If hyperkalaemia develops, the following measures should be considered: elimination of foods and medications containing potassium and of potassium-sparing diuretics; IV administration of 300 to 500 mL/hour of 10% glucose solution containing 10 to 20 units of insulin/1000 mL; correction of acidosis, if present, with IV sodium bicarbonate, use of exchange resins, haemodialysis, or peritoneal dialysis. Cardiac arrhythmias or a serum concentration above 6.5 mmol/L require immediate attention and may be treated by intravenous administration over 1-5 minutes of 10-20 mL of 10% calcium gluconate solution. Continuous ECG monitoring is mandatory.

In treating hyperkalaemia in digitalised patients, too rapid a lowering of the serum potassium concentration can produce digitalis toxicity.

MONITORING

- Measure urea, electrolytes and creatinine
- Monitor potassium levels regularly (2 to 3 hourly if raised)
- Continuous 12 lead ECG
- Observe asymptomatic patients for at least 6 hours.

For advice on the management of overdose please contact the National Poisons Centre on 0800 POISON (0800 764 766).

5. PHARMACOLOGICAL PROPERTIES

5.1 PHARMACODYNAMIC PROPERTIES

Potassium ion is the principal intracellular ion of most body tissues. Potassium ions are involved in a number of essential physiological processes, including the maintenance of intracellular tonicity, the transmission of nerve impulses, the contraction of cardiac, skeletal, and smooth muscle and the maintenance of normal renal function.

Excretion of potassium occurs mainly via the kidneys and normally any amounts given in excess of intracellular requirements are rapidly eliminated.

5.2 PHARMACOKINETIC PROPERTIES

After intravenous administration, potassium is actively transported from extracellular fluid into cells where concentrations reach up to 40 times that of extracellular fluid. It is excreted mainly by the kidneys and is secreted in the distal tubule where it is involved in the sodium- potassium exchange process. Some potassium is excreted in the faeces and small amounts may also be excreted in the sweat, saliva, bile and pancreatic juice.

5.3 PRECLINICAL SAFETY DATA

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY
No data are available. Both potassium and chloride ions are essential constituents of human tissues and body fluids. At physiological levels, neither of these ions is known to have a carcinogenic or genotoxic activity or to cause an adverse effect on fertility.

6. PHARMACEUTICAL PARTICULARS

6.1 LIST OF EXCIPIENTS

Hydrochloric acid

Water for injection

6.2 INCOMPATABILITIES

Potassium chloride solution has been reported to be incompatible when diluted in solutions containing the following medicines:

- Amikacin sulfate
- Amphotericin B
- Amoxicillin sodium
- Benzylpenicillin
- Diazepam
- Dobutamine hydrochloride
- Ergotamine tartrate
- Etoposide with cisplatin and mannitol
- Methylprednisolone sodium succinate
- Phenytoin sodium
- Promethazine hydrochloride
- Sodium nitroprusside
- Streptomycin sulphate

The above list may not be complete.

6.3 SHELF-LIFE

36 months.

6.4 SPECIAL PRECAUTION FOR STORAGE

Store below 25°C

6.5 NATURE AND CONTENTS OF CONTAINER

Polypropylene ampoules in packs of 50

6.6 SPECIAL PRECAUTIONS FOR DISPOSAL

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

7 MEDICINE SCHEDULE

General Sale Medicine

8 SPONSOR

Aborns Pharmaceuticals Ltd
Level 4, 35 Grey Street,
Tauranga 3110

9. DATE OF FIRST APPROVAL

7 September 1989

10. DATE OF REVISION OF THE TEXT

28 May 2024

SUMMARY TABLE OF CHANGES

Section changed	Summary of new information
Header	Change to Aborns
Product name	Change Juno to Aborns
8.	Change to Aborns name & address
10.	Changed to 28 May 2024