South Australian Paediatric Practice Guidelines

diabetic ketoacidosis

Note

This guideline provides advice of a general nature. This statewide guideline has been prepared to promote and facilitate standardisation and consistency of practice, using a multidisciplinary approach. The guideline is based on a review of published evidence and expert opinion.

Information in this statewide guideline is current at the time of publication.

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Health practitioners in the South Australian public health sector are expected to review specific details of each patient and professionally assess the applicability of the relevant guideline to that clinical situation.

If for good clinical reasons, a decision is made to depart from the guideline, the responsible clinician must document in the patient's medical record, the decision made, by whom, and detailed reasons for the departure from the guideline.

This statewide guideline does not address all the elements of clinical practice and assumes that the individual clinicians are responsible for discussing care with consumers in an environment that is culturally appropriate and which enables respectful confidential discussion. This includes:

- The use of interpreter services where necessary,
- Advising consumers of their choice and ensuring informed consent is obtained,
- Providing care within scope of practice, meeting all legislative requirements and maintaining standards of professional conduct, and
- Documenting all care in accordance with mandatory and local requirements

This guideline does not address **all** the elements of guideline practice and assumes that the individual clinicians are responsible to:

> Discuss care with consumers in an environment that is culturally appropriate and which enables respectful confidential discussion. This includes the use of interpreter services where necessary

> Advise consumers of their choice and ensure informed consent is obtained

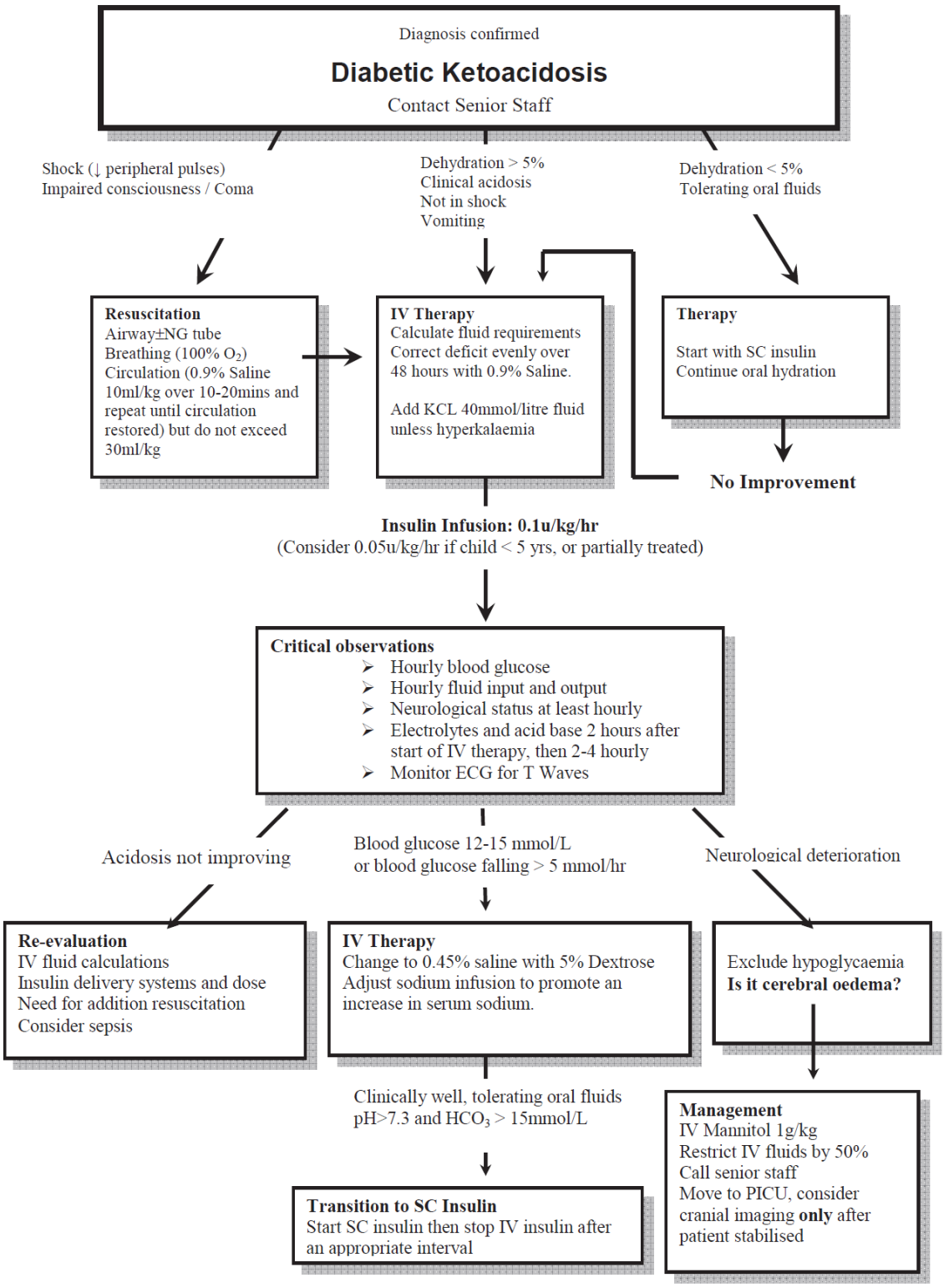
> Provide care within scope of practice, meet all legislative requirements and maintain standards of professional conduct

> Document all care in accordance with mandatory and local requirements

ISBN number: 978-1-74243-529-9
Endorsed by: South Australian Paediatric Clinical Guidelines Reference Committee. South Australian Child Health Clinical Network
Last Revised:
Contact: South Australian Paediatric Clinical Guidelines Reference Committee: cywhs.paediatricclinicalguidelines@health.sa.gov.au

Government of South Australia
SA Health

Management summary



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Introduction

DKA results from absolute or relative deficiency of insulin and the combined effects of increased levels of counter-regulatory hormones. This leads to both increased production and impaired utilisation of glucose, with resultant hyperglycaemia and hyperosmolality. Increased lipolysis and ketone body production causes ketonaemia and metabolic acidosis. Hyperglycaemia and acidosis result in an osmotic diuresis, dehydration and obligate loss of electrolytes. DKA may occur at diabetes onset or in children with established diabetes who have either omitted insulin or had inadequate insulin therapy during intercurrent illness.

The biochemical criteria for the diagnosis of DKA are:

- hyperglycaemia (blood glucose >11mmol/L)
- venous pH <7.3 and / or bicarbonate <15mmol/L.
- ketonaemia / ketonuria and glycosuria.

DKA is categorised by the severity of the acidosis:

Mild:	pH 7.25 - 7.30, HCO_3 10-15mmol/L
Moderate:	pH 7.10 - 7.25, HCO_3 5-10mmol/L
Severe:	pH < 7.10, HCO_3 < 5mmol/L

Children with mild DKA, who are < 5% dehydrated and not vomiting, usually tolerate oral rehydration and subcutaneous insulin therapy.

Morbidity and Mortality of DKA in Children:

- DKA is the most common cause of diabetes related deaths in children and adolescents
- Most deaths in DKA occur as a result of cerebral oedema.

Cerebral oedema

Cerebral oedema typically occurs 4 -12 hours after treatment is initiated, but can be present before treatment has begun or anytime during treatment. Although the aetiology and pathophysiology of cerebral oedema is poorly understood, it is more likely to occur in those patients with newly diagnosed type 1 diabetes (T1D), younger age and greater severity of DKA. High serum urea and low pCO2 at presentation, a fall in serum sodium concentration during therapy and the use of bicarbonate therapy to correct the acidosis have been shown to be independent risk factors for cerebral oedema.

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Definitions

DKA – diabetic ketoacidosis
T1D – type 1 diabetes
T2D – type 2 diabetes
HDU – high dependency unit
PICU – paediatric intensive care unit
WCH – Women's and Children's Hospital

Assessment

Emergency Assessment:

- **Confirm Diagnosis**
  - History (polyuria, polydipsia, wt loss, vomiting and abdominal pain)
  - Biochemical confirmation
    - glucose, EUC, acid base (venous acid base is appropriate in almost all cases)
    - **blood ketone testing using the Optium Meter or urine ketone testing (blood ketones of > 1.5mmol/L = moderate to large urine ketones)**
- **Assess Severity of Dehydration and Acidosis**
  - Weigh if possible
  - Clinical assessment of dehydration
    - 5% dehydration: reduced skin turgor, dry mucous membranes, tachycardia, deep breathing
    - 10% dehydration: capillary refill ≥3 seconds, sunken eyes
    - >10% dehydration: shock, weak peripheral pulses, hypotension, oliguria
  - Clinical evidence of acidosis: hyperventilation, ketotic breath
- **Assess Level of Consciousness**
  - Glasgow Coma Scale (see Appendix 2)
- **Determine the Cause (new onset, inadequate or omitted insulin?) and exclude predisposing infection**
  - History and examination
  - Full blood count and haematocrit (elevated WBC count is common in DKA and cannot be interpreted as a sign of infection)
  - Urine microscopy and culture
  - Blood cultures and CXR if indicated

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- **Non-urgent blood tests for patients with newly diagnosed diabetes**
    - TSH/FT4
    - Coeliac screen and total IgA
    - Consider Islet Cell Antibodies (ICA/GAD/IA2), C-peptide/Insulin levels and HbA1c if T2D a possibility or diagnosis of T1D uncertain

T2D now accounts for 10% of diabetes in children and adolescents <15years of age and should be considered if the following risk factors are present: age >10 years, overweight, family history of T2D, high risk ethnic group, acanthosis nigricans

Management of DKA

Goals of Treatment

- Restoration of circulating volume
- Replacement of fluid and electrolyte deficit evenly over 48 hours
- Correction of acidosis and hyperglycaemia with low dose insulin infusion
- Avoidance of the complications of DKA and its treatment by frequent monitoring for:
    - cerebral oedema
    - hypoglycaemia
    - electrolyte abnormalities

Resuscitation/ Supportive Measures

- **Airway:** Nasogastric tube if vomiting and impaired consciousness
- **Breathing:** High flow oxygen by face mask
- **Circulation:** If shocked (reduced peripheral perfusion)
    - give 10ml/kg of 0.9% sodium chloride over 10-20 minutes
    - repeat until circulation restored, but **do not exceed 30ml/kg** without consulting Paediatric Intensive Care / MedStar Kids (13STAR) or Endocrine Consultant

Call for help (e.g. Consultant, MET team, MedStar Kids, PICU) if aggressive resuscitation required

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Where should the patient be managed?

All patients requiring an insulin infusion should be managed in a unit that has:

> experienced nursing staff trained in monitoring and management

> clear written guidelines and

> access to laboratories for frequent evaluation of biochemical variables

(i.e. most children should be admitted/transferred to HDU or PICU at WCH).

PICU admission is recommended for those at highest risk of cerebral oedema:

> Severe DKA (pH <7.1, HCO3 <5mmol/L)

> Decreased level of consciousness

> Age < 5 years

Clinical and Biochemical Monitoring

Successful management of DKA requires frequent and meticulous monitoring of the patient's clinical and biochemical response to treatment so that timely adjustments to fluid and electrolyte therapy can be made. A venous sampling line should be inserted where possible.

Monitoring should include the following:

> **Hourly vital signs:** pulse rate, respiratory rate and blood pressure

> **Hourly or more frequent neurological observations** to detect the warning signs of cerebral oedema:

- headache
- inappropriate slowing of pulse rate
- recurrence of vomiting
- change in neurological status or specific neurological signs
- rising blood pressure
- decreased oxygen saturation

> **Hourly accurate fluid input and output** (urinary catheterisation may be necessary if impaired level of consciousness)

> **Hourly blood glucose** (capillary blood glucose may be inaccurate in the presence of poor peripheral perfusion and acidosis)

> **Laboratory tests:** glucose, electrolytes, urea, creatinine and acid base should be measured **2 hours after the initiation of treatment and then every 2-4 hours until acidosis has resolved.** Hourly electrolytes may be necessary as clinically indicated in severe cases.

> **Blood or urine ketones until cleared**

> ECG monitoring may be helpful to assess T waves for evidence of hyperkalaemia or hypokalaemia

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Calculations:

Anion gap = Na – (Cl + HCO_3) **Normal: 12 ± 2**

Corrected Na = Measured Na + 2x {(Glucose – 5.5) ÷ 5.5} mmol/L

Effective osmolality = 2 x ($Na_{(uncorrected)}$ + K) + Glucose mmol/L

Fluids and Sodium:

- Patients with DKA are usually 5-10% dehydrated.
- Clinical estimates of fluid deficit in DKA have been shown to be inaccurate and may overestimate the deficit, therefore in moderate DKA use 5-7% and in severe DKA use 10% dehydration
- If needed, volume expansion to restore peripheral circulation should begin immediately with 0.9% sodium chloride. The volume and rate of administration depends on the circulatory status
- **If shocked give 10ml/kg of 0.9% sodium chloride over 10-20minutes, this may be repeated if necessary but should not exceed 30ml/kg.** Clinical improvement should be apparent within minutes of the bolus being completed.
- **Subsequent fluid management should begin with 0.9% sodium chloride with added KCl (for at least the first 4-6 hours), at a rate calculated to replace the fluid deficit evenly over 48 hours** (see Appendix 1)**.** Thereafter, the fluid replacement should be determined by the serum sodium but should always be with a solution that has a tonicity equal to or greater than 0.45% sodium chloride with added KCl
- **If hypernatraemia is present (corrected Na >150mmol/L) fluid management should also begin with 0.9% sodium chloride and correction of fluid and electrolyte deficit should be over 48-72 hours**
- If IV fluids have been given elsewhere, prior to assessment, the volume should be included in the fluid calculations
- Urinary losses should not be added to the calculation of replacement fluid
- In severe dehydration and acidosis only allow sips of water or ice to suck (include in fluid balance)
- **Serum sodium is an unreliable measure of the degree of ECF contraction** due to the dilutional effect of the hyperglycaemia and the resultant fluid shift from the ICF to the ECF. (The serum urea and haematocrit are more useful markers of severe ECF contraction)
- **Calculate and monitor corrected sodium throughout therapy**
- Corrected Na = Measured Na + 2 x [(glucose – 5.5) ÷ 5.5] mmol/L
- As the plasma glucose concentration falls, measured and corrected sodium should rise steadily

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- A fall in serum sodium is one of the few biochemical correlates of impending cerebral oedema
- **If the corrected sodium fails to rise, and particularly if it falls, a careful re-evaluation of the fluid replacement is required as the concentration of sodium chloride may need to be increased**
- **Effective osmolality = 2($Na_{uncorrected}$ + K) + glucose mmol/L** may be a useful guide to fluid and electrolyte therapy. A fall in serum osmolality of >3mosm/kg/hr has been suggested as a risk factor for cerebral oedema

- **When the blood glucose level falls below 12-15mmol/L, add 5% glucose to the rehydration fluids**, rather than reducing the insulin infusion rate. Insulin therapy must be continued to correct the acidosis.

Fluids:

The following fluids are commercially available but not always stocked at each hospital:

- 0.9% sodium chloride
- 0.45% sodium chloride
- 0.9% sodium chloride with 5% glucose
- 0.45% sodium chloride with 5% glucose
- 0.45% sodium chloride with 5% glucose with 20mmol/L KCl
- 0.9% sodium chloride with 20mmol/L KCl

To make 0.9% sodium chloride with 5% glucose:

Remove 100ml of 0.9% sodium chloride from a 1000ml bag of 0.9% sodium chloride and add 100ml of 50% glucose.

Potassium:

- **Potassium replacement is always required in DKA, as total body potassium is substantially depleted**
- Serum potassium levels at presentation may be normal, increased or decreased. (hypokalaemia at presentation represents a significant total body potassium deficit, whereas hyperkalaemia implies reduced renal function)
- Insulin administration and the correction of acidosis will drive potassium back into the cells, decreasing serum levels**. Therefore, potassium replacement should always <u>precede</u> insulin therapy, unless hyperkalaemia or anuria is present**.
- **If serum K^+ < 2.5 mmol/L** discuss with Intensivist on call for advice as cardiac monitoring will be required
- **If serum K^+ 2.5 - 3.5 mmol/L** administer 40-60mmol KCl per litre of IV fluids and monitor K^+ hourly

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- **If serum K^+ 3.5 - 5.0 mmol/L** administer 30-40mmol KCl per litre of IV fluids to maintain K^+ at 3.5-5.0 mmol/L
- **If serum K^+ > 5.0 mmol/L** do not give IV KCL. Monitor K hourly until K^+ < 5.0mmol/L, then administer 30-40mmol KCl per litre of IV fluids to maintain serum K^+ at 3.5-5.0 mmol/L

The maximum recommended rate of intravenous potassium replacement is 0.5mmol/kg/hour.

Insulin:

- Insulin therapy is essential to normalise blood glucose concentration and suppress lipolysis and ketone production.
- Children with mild DKA (<5% dehydration, pH 7.25-7.3, bicarbonate 10-15mmol/L) who are not vomiting can usually be managed with oral rehydration and subcutaneous insulin therapy.
- **Insulin infusion therapy should not be started until the circulating volume has been restored, the serum potassium is known and appropriate potassium replacement has commenced.**
- Insulin should be administered by continuous low-dose IV infusion using an electronic infusion pump. The infusion may then be run as a sideline to the rehydrating fluid.
- Prime the IV tubing by flushing the insulin infusion solution through all IV tubing before connecting to the patient (to saturate the insulin binding sites in the tubing)
- **Insulin Dose: 0.1units/kg/hr (50units soluble insulin (Actrapid, Humulin R) diluted in 50mls of 0.9%Saline, 1unit = 1ml)**
- **A lower insulin dosage of 0.05u/kg/hr** should be considered in children <5yrs of age (who may be more sensitive to insulin) in children with known diabetes who have a lower blood glucose due to partial insulin treatment prior to presentation, and in children with severe DKA (pH <7%, bicarbonate <5mmol/L).
- During the first 60-90 minutes of rehydration, the blood glucose may fall substantially even without insulin therapy
- **After resuscitation, the desired rate of fall in blood glucose is 4-5 mmol/hour**
- When the BGL falls to 12-15 mmol/L, add 5% Dextrose to the IV fluids to keep blood glucose in the desired range of 8-12mmol/L. If necessary more dextrose may be added to the IV fluids
- **The insulin infusion should not be stopped or reduced below 0.05u/kg/hr until the acidosis has resolved**

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- If the biochemical parameters of DKA (pH, HCO_3 and Anion Gap) do not improve, reassess the patient, recalculate the IV fluid replacement, review the insulin therapy (delivery and dose) and consider possible causes of impaired response to insulin (e.g. infection, errors in insulin preparation)
- The insulin infusion should be replaced every 24 hours to avoid inactivation of insulin
- In unusual circumstances where IV administration is not possible, the use of hourly IM or SC injections of short or ultra short acting insulin (0.1units/kg/hour) has been shown to be effective

Phosphate:

- There is a depletion of intracellular phosphate in DKA and insulin administration results in a fall in plasma phosphate as phosphate re-enters the cells
- There is no evidence however that phosphate replacement has any clinical benefit in DKA.
- **Administration of phosphate may induce hypocalcaemia**
- KPO_4 may be used as an alternative to or in combination with KCl to avoid hyperchloraemia, provided careful monitoring for hypocalcaemia is performed

Acidosis:

- Even severe acidosis is reversible by fluid and insulin replacement
- **Bicarbonate therapy has not been shown to confer clinical benefit in DKA and may increase the risk of cerebral oedema**
- Cautious alkali therapy may be required however in selected patients with severe acidaemia (pH <6.9) in whom decreased cardiac contractility and peripheral vasodilatation can further impair tissue perfusion and in patients with potentially life threatening hyperkalaemia.
- The use of large amounts of 0.9% Saline has been associated with the development of a hyperchloraemic metabolic acidosis after the clinical status has improved and ketosis has resolved. Treatment with $NaHCO_3$ may be considered.

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Treatment of Cerebral Oedema:

Mannitol should always be immediately available during the treatment of DKA

Warning signs of cerebral oedema include:

> Headache
> Inappropriate slowing of pulse rate
> Recurrence of vomiting
> Change in neurological status or specific neurological signs
> Rising BP
> Decreased oxygen saturation
> Fall in serum sodium concentration

If cerebral oedema is suspected, URGENT action is required:

> **Exclude hypoglycaemia**
> Halve the rate of IV fluid administration until the situation has stabilised
> **Give IV Mannitol 1 gm/kg over 20 minutes (i.e. 5ml/kg of 20% mannitol solution)**
> Consider continuation of mannitol infusion 0.25g/kg/hour to prevent rebound increase in intracranial pressure, or repeat boluses every 4-6 hours
> Hypertonic Saline (3%), 5-10 ml/kg over 30 minutes may be an alternative to Mannitol
> Intubation and ventilation may be necessary, however aggressive hyperventilation has been associated with poor outcome. If assisted ventilation is required maintain pCO_2 above 3.5kPa
> Cranial imaging should only be considered after the child has been stabilised. Intracranial events other than cerebral oedema can occur (e.g. haemorrhage or thrombosis)

Transfer to Oral Fluids and Subcutaneous Insulin:

Oral fluids

> In severe dehydration and acidosis only allow sips of water or ice to suck (include in fluid balance)
> Oral fluids should only be offered after substantial clinical improvement and cessation of vomiting (mild acidosis and ketosis may still be present).
> When oral fluids are tolerated the IV fluids should be reduced
> The insulin infusion can be increased to cover oral carbohydrate intake prior to the commencement of subcutaneous (SC) insulin. The basal insulin infusion rate is usually doubled for 30 minutes for snacks and 60 minutes for meals

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Transfer to SC insulin can be made when the acidosis has resolved and oral intake is tolerated

- The SC insulin dosage and regimen will vary with the patient's age and circumstances, and should be discussed with the Endocrine Team
- The most convenient time to change to SC insulin is before meals

The insulin infusion should be discontinued:

- As soon as the 1st injection of **ultra short acting insulin (Novorapid, Humalog)** is given
- 30 minutes after the 1st injection of **short acting insulin (Actrapid, Humulin R)** is given
- **Blood glucose monitoring should continue at 2- 4 hourly intervals**

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Appendix 1 - Fluid Calculations

Fluid Calculation [Method 1]

Requirements = Deficit + Maintenance

- Calculate **DEFICIT (*) = est. % dehydration x body weight (kg and equivalent in ml)**
- Calculate **MAINTENANCE (ml)** fluid requirements based on weight as follows:
    - for first 10kgs give 4ml/kg/hr
    - for next 10kgs add 2ml/kg/hr
    - for additional weight over 20kgs add 1ml/kg/hr
- For example, a child weighing 34 kg receives: 40 + 20 + 14 = 74ml/hr
- Then add **DEFICIT** to **48 h MAINTENANCE** and replace this volume evenly over 48 h as **0.9% sodium chloride with KCL initially**

FLUID CALCULATION [Method 2]

Covers **MAINTENANCE + 10% DEFICIT (*)** given evenly over 48 h in children of all sizes

- 6 ml/kg per h for children weighing 3–9 kg
- 5 ml/kg per h for children weighing 10–19 kg
- 4 ml/kg per h for children weighing >20 kg (up to maximum of 250 ml/h)

***When calculating deficit, subtract any fluid boluses administered during resuscitation**

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Appendix 2 - Glasgow Coma Scale

Glasgow Coma Scale or Score (GCS)
The GCS consists of three parameters and is scored between 3 and 15; 3 being the worst and 15 the best. One of the components of the GCS is the best verbal response, which cannot be assessed in non-verbal young children. A modification of the GCS was created for children too young to talk.

Best eye response	Best verbal response	Best verbal response (nonverbal children)	Best motor response
1. No eye opening	1. No verbal response	1. No response	1. No motor response
2. Eyes open to pain	2. No words, only incomprehensible sounds; moaning and groaning	2. Inconsolable, irritable, restless, cries	2. Extension to pain (decerebrate posture)
3. Eyes open to verbal command	3. Words, but incoherent**	3. Inconsistently consolable and moans; makes vocal sounds	3. Flexion to pain (decorticate posture)
4. Eyes open spontaneously	4. Confused, disoriented conversation*	4. Consolable when crying and interacts inappropriately	4. Withdrawal from pain
	5. Orientated, normal conversation	5. Smiles, oriented to sound, follows objects and interacts	5. Localises pain 6. Obeys commands

Attention can be held; responds in a conversational manner, but shows some disorientation.
**Inappropriate words, no sustained conversational exchange

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2. Craig ME, Twigg SM, Donaghue KC, Cheung NW, Cameron FJ, Conn J, Jenkins AJ, Silink M, for the Australian Type 1 Diabetes Guidelines Expert Advisory Group. National Evidence-Based Clinical Care Guidelines for Type 1 Diabetes in Children, Adolescents, and Adults, Australian Government Department of Health and Ageing, Canberra 2011

Version control and change history

PDS reference: OCE use only

Version	Date from	Date to	Amendment
1	August 2013	current	Original version

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