

South Australian Paediatric Clinical Practice Guidelines

Diabetic Ketoacidosis (DKA) in Children

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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

Explanation of the aboriginal artwork:

The aboriginal artwork used symbolises the connection to country and the circle shape shows the strong relationships amongst families and the aboriginal culture. The horse shoe shape design shown in front of the generic statement symbolises a woman and those enclosing a smaller horse shoe shape depicts a pregnant woman. The smaller horse shoe shape in this instance represents the unborn child. The artwork shown before the specific statements within the document symbolises a footprint and demonstrates the need to move forward together in unison.



Cultural safety enhances clinical safety.

To secure the best health outcomes, clinicians must provide a culturally safe health care experience for Aboriginal children, young people and their families. Aboriginal children are born into strong kinship structures where roles and responsibilities are integral and woven into the social fabric of Aboriginal societies.

Australian Aboriginal culture is the oldest living culture in the world, yet Aboriginal people currently experience the poorest health outcomes when compared to non-Aboriginal Australians.

It remains a national disgrace that Australia has one of the highest youth suicide rates in the world. The over representation of Aboriginal children and young people in out of home care and juvenile detention and justice system is intolerable.

The cumulative effects of forced removal of Aboriginal children, poverty, exposure to violence, historical and transgenerational trauma, the ongoing effects of past and present systemic racism, culturally unsafe and discriminatory health services are all major contributors to the disparities in Aboriginal health outcomes.

Clinicians can secure positive long term health and wellbeing outcomes by making well informed clinical decisions based on cultural considerations.

The term 'Aboriginal' is used to refer to people who identify as Aboriginal, Torres Strait Islanders, or both Aboriginal and Torres Strait Islander. This is done because the people indigenous to South Australia are Aboriginal and we respect that many Aboriginal people prefer the term 'Aboriginal'. We also acknowledge and respect that many Aboriginal South Australians prefer to be known by their specific language group(s).

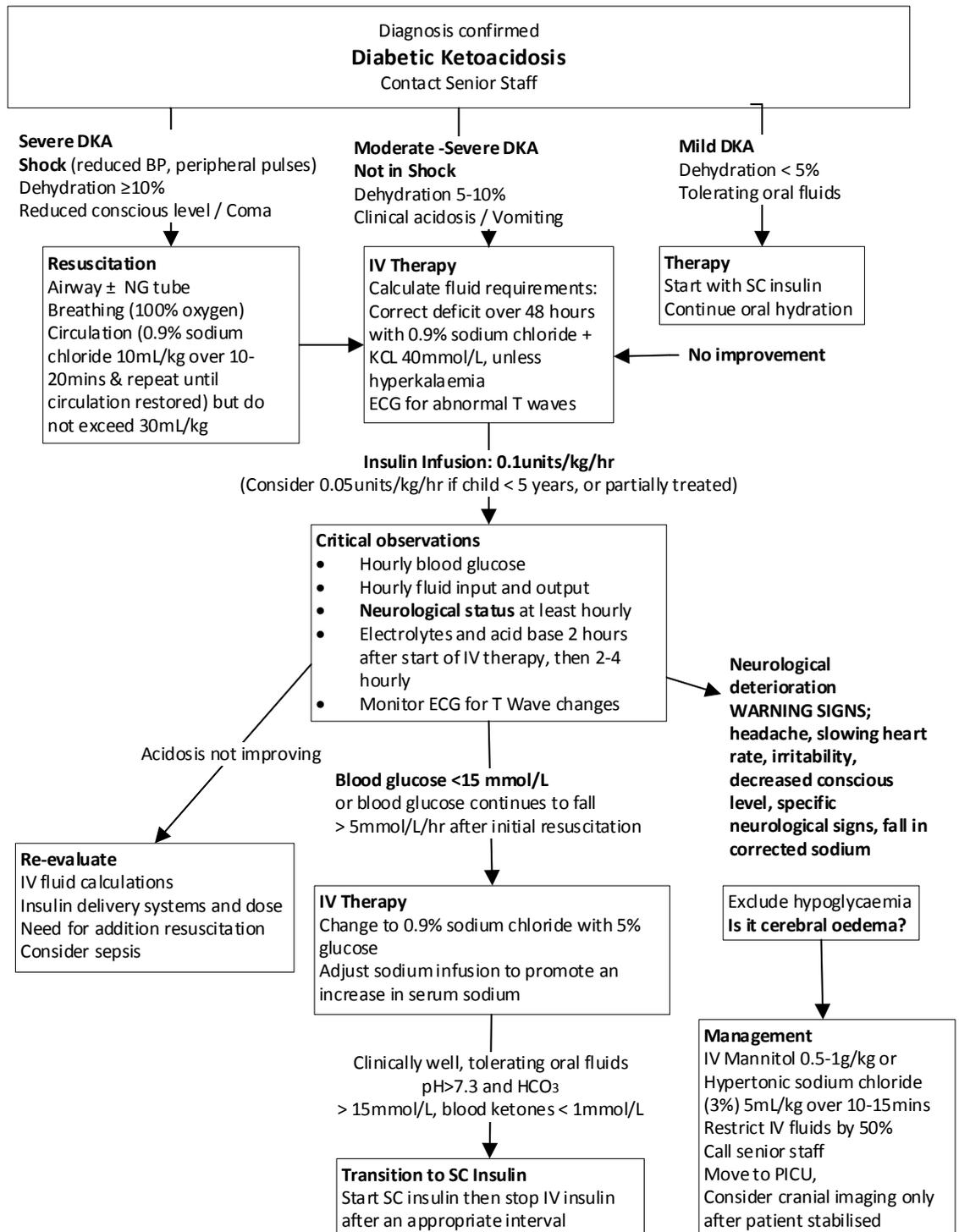


Diabetic Ketoacidosis (DKA) in Children

Purpose and Scope of PCPG

The Diabetic Ketoacidosis (DKA) Guideline is primarily aimed at medical staff working in any of primary care, local, regional, general or tertiary hospitals. It may however assist the care provided by other clinicians such as nurses. The information is current at the time of publication and provides a minimum standard for the assessment (including investigations) and management of Diabetic Ketoacidosis; it does not replace or remove clinical judgement or the professional care and duty necessary for each specific case.

Management Summary Flowchart



Diabetic Ketoacidosis (DKA) in Children

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Abbreviations

DKA	Diabetic Ketoacidosis
HDU	High Dependency Unit
MET	Medical Emergency Team
PICU	Paediatric Intensive Care Unit
SC	subcutaneous
T1D	Type 1 diabetes
T2D	Type 2 diabetes
WCH	Women's and Children's Hospital
HCO ₃	Bicarbonate

Diabetic Ketoacidosis (DKA) in Children

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 the onset of clinical diabetes or in children with established diabetes who have either omitted insulin or had inadequate insulin therapy during illness. Children on insulin pump therapy are also at increased risk of DKA, if monitoring and pump failure management guidelines are not followed. As only rapid acting insulin is used in insulin pumps, interruption of insulin delivery for any reason (most commonly a blocked delivery set) rapidly leads to insulin deficiency and ketosis.

The biochemical criteria for the diagnosis of DKA are:

- > Hyperglycaemia (blood glucose >11mmol/L)
- > Venous pH <7.3 and / or bicarbonate <15mmol/L
- > Ketonaemia* and ketonuria

*Measurement of blood ketone (β OHB) level with a bedside/point of care meter, if available, is useful to rapidly confirm ketoacidosis (β OHB \geq 3mmol/L). Urine ketones should be measured if a blood ketone meter is unavailable.

The severity of DKA is categorised by the degree of acidosis:

- Mild: pH 7.20 - 7.30 HCO₃ 10-15mmol/L
- Moderate: pH 7.10 - 7.20 HCO₃ 5-10mmol/L
- Severe: pH < 7.10 HCO₃ < 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. Independent risk factors for cerebral oedema include; high serum urea and low pCO₂ at presentation, a fall in serum sodium concentration during therapy and the use of bicarbonate therapy.



Diabetic Ketoacidosis (DKA) in Children

Assessment

Emergency Assessment:

> Confirm Diagnosis

- History (polyuria, polydipsia, weight loss, vomiting and abdominal pain)
- Biochemical confirmation
 - Immediately measure blood glucose and blood ketone (β OHB) levels with bedside/point of care meter
 - Obtain a blood sample for laboratory measurement of glucose, electrolytes, urea, creatinine, acid base (venous acid base is appropriate in almost all cases)

> Assess Severity of Dehydration and Acidosis

- Weigh if possible
- Clinical assessment of dehydration may be inaccurate in DKA and the degree of metabolic acidosis has been shown to be a more accurate guide
 - In moderate DKA (pH 7.10 - 7.20, HCO_3^- 5-10mmol/L) use 5-7% dehydration for calculations
 - In severe DKA (pH < 7.10, HCO_3^- < 5mmol/L) use 10% dehydration for calculations
- Clinical evidence of acidosis: tachypnea; deep sighing respirations, ketotic odour on breath

> Assess Level of Consciousness

- Glasgow Coma Scale ([see Appendix 2](#))

> Determine the Cause (new onset diabetes, 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

> Non-urgent blood tests for patients with newly diagnosed diabetes

- TSH/FT4
- Coeliac screen and total IgA
- Islet Autoantibodies (GAD/IA2)
- If T2D or MODY are possibilities or diagnosis of T1D is uncertain it is especially relevant to measure islet autoantibodies.

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. Up to 10% of children diagnosed with T2D can present with DKA.



Diabetic Ketoacidosis (DKA) in Children

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 (e.g. hypokalaemia and hyperchloraemic acidosis)

Resuscitation / Supportive Measures

- > **Airway:** Nasogastric tube if vomiting and impaired consciousness
- > **Breathing:** High flow oxygen by face mask*
- > **Circulation:** If shocked (reduced BP / 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

*Intubation should be avoided if possible; a sudden increase in pCO₂ during or following intubation may cause cerebrospinal fluid pH to decrease and contribute to worsening of cerebral oedema.

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

Where the patient should 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.

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

- > Severe DKA (pH <7.10, HCO₃ <5mmol/L).
- > Decreased level of consciousness.
- > Age < 5 years.



Diabetic Ketoacidosis (DKA) in Children

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 second 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 patient unconscious or in infants and very young children with severe DKA).
- > **Hourly venous 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 ketones (β OHB) should be measured every 2 - 4 hours until cleared. Point of Care β OHB measurements correlate well with the reference method up to 3mmol/L, but are not accurate >5mmol/L.

- > ECG monitoring may be helpful to assess for hyperkalaemia or hypokalaemia:
 - In hyperkalaemia: Tall, peaked and symmetrical T waves with shortening of the QT interval.
 - In hypokalaemia: Prolongation of the PR interval, T wave flattening and inversion, ST depression, prominent U waves and apparent long QT interval (due to fusion of T and U waves).

Calculations

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

Anion gap = (Na + K) – (Cl + HCO₃) Normal: <16mmol/L

Effective osmolality = 2x (Measured Na) + Glucose mmol/L



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Fluids and Sodium

Fluid replacement should always begin before starting insulin therapy

- > **Patients with DKA are usually 5-10% dehydrated, but shock with haemodynamic compromise is rare in paediatric DKA.**
- > Clinical estimates of fluid deficit in DKA are often inaccurate and overestimation of fluid deficit in DKA is common. It is therefore recommended that in moderate DKA 5-7% dehydration and in severe DKA 10% dehydration be used for calculations.
- > 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 10-20 minutes, 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 potassium chloride (according to serum potassium) for at least the first 4-6 hours at a rate calculated to replace the fluid deficit evenly over 48 hours (see [Appendix 1 and 3](#)).**
- > **If hypernatraemia is present (corrected Na >150mmol/L) fluid management should also begin with 0.9% sodium chloride with added potassium chloride and correction of fluid and electrolyte deficit should be over 48-72 hours.**
- > If IV fluids have been given elsewhere, prior to assessment, this volume should be included in the fluid calculations
- > Urinary losses should not be added to the calculation of replacement fluid
- > Thereafter, the fluid replacement should be determined by the serum sodium but should always be with a solution that has a tonicity $\geq 0.45\%$ sodium chloride with added potassium chloride. Hartmann's Solution or Plasmalyte can be used at this stage, particularly if there are concerns about hyperchloraemia, but it is important to remember that potassium phosphate is incompatible with Hartmann's Solution.
- > 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.
 - **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



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- > **Effective osmolality = $2 \times (\text{Measured Na}) + \text{Glucose mmol/L}$** may be a useful guide to fluid and electrolyte therapy. A fall in serum osmolality of $>3\text{mosm/kg/hr}$ has been suggested as a risk factor for cerebral oedema
- > **When the blood glucose level falls below 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, and higher glucose concentrations may be required

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 potassium levels. **Therefore, potassium replacement should always precede insulin therapy, unless hyperkalaemia or anuria is present.**
- > **If serum $K^+ < 2.5 \text{ mmol/L}$** discuss with Intensivist on call for advice as cardiac monitoring will be required
- > **If serum $K^+ 2.5 - 3.5 \text{ mmol/L}$** administer 40-60mmol potassium chloride per litre of IV fluids and monitor K^+ hourly
- > **If serum $K^+ 3.5 - 5.0 \text{ mmol/L}$** administer 30-40mmol potassium chloride per litre of IV fluids to maintain K^+ at 3.5-5.0 mmol/L
- > **If serum $K^+ > 5.0 \text{ mmol/L}$** do not give IV potassium chloride. Monitor K hourly until $K^+ < 5.0\text{mmol/L}$, then administer 30-40mmol potassium chloride 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
- > Potassium phosphate may be used together with potassium chloride or acetate (e.g. 20mmol/L potassium phosphate and 20mmol/L potassium chloride) to reduce the risk of hyperchloraemic metabolic acidosis, however hypocalcaemia can result if potassium phosphate is used alone

Insulin

- > Children with mild DKA ($<5\%$ dehydration, pH 7.20-7.30, HCO_3^- 10-15mmol/L) **who are not vomiting** may 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
- > 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 of regular insulin (Actrapid) diluted in 50mLs of 0.9% sodium chloride; 1unit = 1mL)**
- > **A lower insulin dosage of 0.05units/kg/hr** may be used in children $<5\text{yrs}$ of age (who

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may be more sensitive to insulin) or in children with known diabetes who have a lower blood glucose due to partial insulin treatment prior to presentation, provided that the metabolic acidosis continues to resolve.

- > 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 4-5mmol/hour

- > **When the BGL falls below 15mmol/L, add 5% Glucose to the IV fluids** (0.9% sodium chloride) to keep blood glucose in the desired range of 8-12mmol/L. If necessary more glucose may be added to the IV fluids to prevent hypoglycaemia while correcting the metabolic acidosis
- > **The insulin infusion should not be stopped or reduced below 0.05units/kg/hr until the acidosis has resolved** (pH > 7.30 and HCO₃ > 15mmol/L, βOHB < 1mmol/L)
- > If the biochemical parameters of DKA (pH, HCO₃ 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 insulin infusion is not possible and provided peripheral perfusion is not impaired, the use of 1-2 hourly IM or SC injections of rapid acting insulin (0.1units/kg/hour) has been shown to be effective.

Phosphate

- > Intracellular phosphate is depleted 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 and **administration of phosphate may induce hypocalcaemia.**
- > If severe, symptomatic hypophosphataemia is present potassium phosphate may be safely used as an alternative or in combination with potassium chloride, provided careful monitoring of serum calcium is performed.

Bicarbonate

- > 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 potentially life-threatening hyperkalaemia or with severe acidaemia (pH < 6.9) in whom decreased cardiac contractility and peripheral vasodilatation can further impair tissue perfusion.
- > The use of large amounts of 0.9% sodium chloride has been associated with the development of a hyperchloraemic metabolic acidosis after the clinical status has improved and ketosis has resolved. Treatment with sodium bicarbonate may be considered in this situation.



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Treatment of Cerebral Oedema

Mannitol or Hypertonic Saline (3%) should always be immediately available during the treatment of DKA and the dose to be given calculated beforehand.

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 0.5-1 gm/kg over 10-15 minutes (i.e. 2.5 - 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 sodium chloride (3%) 5mL/kg over 10-15 minutes may be used as an alternative to Mannitol.
- > Elevate the head of the bed to 30 degrees.
- > Intubation and ventilation may be necessary however aggressive hyperventilation has been associated with poor outcome. If assisted ventilation is required maintain pCO₂ above 3.5kPa (26.2mmHg).
- > Cranial imaging should only be considered after the child has been stabilised. Intracranial events other than cerebral oedema can occur which may require emergency neurosurgery (e.g. intracranial haemorrhage) or anticoagulation (cerebrovascular 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 accordingly.
- > 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 doubled for 60 minutes for meals.



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

- > The subcutaneous 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 a meal.
- > The insulin infusion should be discontinued 10-15 minutes after the 1st injection of rapid acting insulin (Novorapid, Humalog) is given.
- > Blood glucose monitoring should continue at 2- 4 hourly intervals.

References

1. Wolfsdorf JI et al. Pediatric ISPAD Clinical Practice Consensus Guidelines 2018: Diabetic ketoacidosis and the hyperglycemic hyperosmolar state. *Pediatric Diabetes* October 2018; 19 (Suppl. 27): 155–177



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Appendix 1 - Fluid Calculations

Fluid Calculation [Method 1]

Fluid Requirements = Maintenance + Deficit

- > Calculate **MAINTENANCE (mL/hr)** 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, in a child weighing 30 kg maintenance fluid = 40 + 20 + 10 = 70mL/hr*
- > Calculate total **DEFICIT*(mLs) = body weight (kg) x % estimated dehydration x 10**
- > *For example in a 30kg child who is 10% dehydrated: Deficit = 30 x 10 x 10 = 3000mls*
- > Then replace the total **DEFICIT** evenly over 48hours
- > *For example in a 30kg child who is 10% dehydrated, receives maintenance 70mL/hr + deficit (3000mL) over 48 hours*

Fluid calculation [Method 2]

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

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

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



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Appendix 2 - Glasgow Coma Scale

Glasgow Coma Scale (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. None 2. To pain 3. To speech 4. Spontaneous	1. None 2. Incomprehensible sounds 3. Inappropriate words** 4. Confused conversation* 5. Fully orientated	1. None 2. Inconsolable, irritable, restless, cries 3. Inconsistently consolable and moans; makes vocal sounds 4. Consolable crying, interacts inappropriately 5. Smiles, orients to sound, follows objects and interacts	1. None 2. Extension to pain (decerebrate posture) 3. Flexion to pain (decorticate posture) 4. Withdrawal from pain 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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Appendix 3 – IV fluid rates (mL/hr) for maintenance plus replacement of deficit over 48hours

Weight (kg)	Maintenance + 3% dehydrated (mL/hr)	Maintenance + 5% dehydrated (mL/hr)	Maintenance + 7% dehydrated (mL/hr)	Maintenance + 10% dehydrated (mL/hr)
5	24	26	28	31
6	29	31	34	37
7	34	36	39	44
8	38	42	45	50
9	43	47	51	56
10	48	52	56	63
11	50	55	60	67
12	53	58	63	71
13	56	61	67	75
14	59	65	70	79
15	61	68	74	83
16	64	71	78	88
17	67	74	81	92
18	70	77	85	96
19	72	80	88	100
20	75	83	92	104
22	78	87	96	110
24	81	91	101	116
26	84	95	105	122
28	87	98	110	128
30	90	102	115	133
32	93	106	119	139
34	95	110	124	145
36	98	113	128	151
38	101	117	133	157
40	104	121	138	163
42	107	125	142	168
44	110	128	147	174
46	113	132	151	180
48	116	136	156	186
50	119	140	160	192
52	122	143	165	198
54	125	147	170	203
56	128	151	174	209
58	130	155	179	215
60	133	158	183	221
62	136	162	188	227
64	139	166	193	233
66	142	170	197	238
68	145	173	202	244
70	148	177	206	250



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Acknowledgements

The South Australian Paediatric Clinical Practice Guidelines gratefully acknowledge the contribution of clinicians and other stakeholders who participated throughout the guideline development process particularly:

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Developed by: SA Child & Adolescent Health Community of Practice
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Endorsed by: SA Health Safety and Quality Strategic Governance Committee
Next review due: 04/07/2024
ISBN number: 978-1-74243-901-3
PDS reference: **CG092**
Policy history: Is this a new policy (V1)? **N**
Does this policy amend or update an existing policy? **Y**
If so, which version? **V1**
Does this policy replace another policy with a different title? **N**
If so, which policy (title)?

Approval Date	Version	Who approved New/Revised Version	Reason for Change
04/07/19	V2	SA Health Safety and Quality Strategic Governance Committee	Formally reviewed in line with 1-5 year scheduled timeline for review.
01/07/13	V1	SA Health Safety & Quality Strategic Governance Committee	Original approved version.

