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Diabetic ketoacidosis emergency management in children
Children's Health Queensland

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1. Purpose

This procedure provides clinical practice guidelines to clinicians involved in the emergency management of children with diabetic ketoacidosis (DKA).

2. Scope

This procedure relates to staff involved with the care and management of children with DKA.

3. Procedure

This procedure was developed in collaboration with Professor Jennifer Batch and the Paediatric Endocrinology Group. The procedure is based upon the Paediatric Diabetic Ketoacidosis (DKA) Treatment Protocol developed by this group for state-wide use in Queensland.

For the management of DKA, CHS also endorses the use of the *National evidenced-based clinical care guidelines: Type 1 diabetes in children adolescents* and adults—*Chapter 17: Acute complications - diabetic ketoacidosis and sick day management* document by the Australasian Paediatric Endocrine Group for the Australian Government—National Health and Medical Research Council.[1]

4. Introduction

Diabetic ketoacidosis is a life-threatening metabolic disorder and is the leading cause of morbidity and mortality in children and adolescents with type 1 diabetes. Mortality is predominantly due to cerebral oedema, which occurs in 0.3% to 1% of all episodes of DKA in children.[1] DKA accounts for approximately 0.1% of emergency presentations to paediatric emergency services in Brisbane (EDIS data, MCH & RCH).

Diabetic ketoacidosis is caused by a decrease in effective circulating insulin, insulin resistance and increased production of counter-regulatory hormones.[2] The resulting increased hepatic and renal glucose production and impaired peripheral glucose utilisation, causes hyperglycaemia and hyperosmolality. In addition, increased lipolysis with the overproduction of ketones leads to ketonemia and metabolic acidosis. Hyperglycaemia and acidosis causes osmotic diuresis, dehydration and, obligate loss of electrolytes.

The diagnosis of DKA can be made using the following biochemical criteria:[3]

- hyperglycaemia (BSL > 11 mmol/L) with

- venous pH < 7.3 and/or bicarbonate < 15 mmol/L
- presence of ketonemia and ketonuria.

The severity of DKA is categorised by the degree of acidosis:[3]

- Mild: venous pH 7.25 - 7.3 or bicarbonate 10 - 15 mmol/L
- Moderate: pH 7.1 - 7.24 or bicarbonate 5 - 10 mmol/L
- Severe: pH < 7.1 or bicarbonate < 5 mmol/L..

Children may present with DKA at any age, with or without a previous diagnosis of type 1 diabetes. DKA can also occur in newly diagnosed type 2 diabetes. Risk factors for DKA in children with newly diagnosed or established type 1 diabetes are summarised in Table 1.

Table 1: Risk factors for DKA

Newly diagnosed type 1 diabetes	Established type 1 diabetes
■ Young age (< 5 years) ■ First degree relative with type 1 diabetes ■ Lower socio-economic status ■ Medications (high dose glucocorticoids, antipsychotics, diazoxide and immunosuppressants)	■ Poor metabolic control / non-compliance with insulin regimen ■ Previous episodes of DKA ■ Adolescent females e.g. pregnancy ■ Alcohol and/or drugs ■ Psychological stressors e.g. abuse ■ Psychiatric disorders e.g. eating disorders ■ Unstable or difficult family circumstances ■ Limited access to medical services

Adapted from: Dunger et al.[2] and Wollfsdorf et al.[3]

5. Assessment

Emergency assessment and management should always involve a rapid primary survey with evaluation of (and immediate management of concerns with) airway, breathing, circulation and disability (ABCD). Pre-hospital treatment should be taken into consideration.

Clinical Assessment

Clinical assessment of the child with suspected DKA should include the following:

- history
    - polydipsia and polyuria (may be absent in the young child)
    - enuresis and/or wetting 'accidents' in a toilet trained child
    - weight loss and/or increased appetite
    - vomiting
    - abdominal pain
    - and non specific symptoms and signs of general malaise.
- full physical examination
    - weight – caution with obese patients – seek Specialist advice
    - BP, pulse rate, capillary refill time
    - features of acidosis (hyperventilation)
    - assessment of conscious level (AVPU or GCS)
    - severity of dehydration (Table 2).
- biochemical confirmation: glycosuria, ketonuria, BSL (may be inaccurate in the setting of circulatory compromise and acidosis) and acid-base status.

ALERT: Assessment of volume deficit in DKA

Volume deficit is difficult to assess accurately in DKA, particularly in the young child, and may be overestimated because of the subjective criteria used.[5] Dryness of oral mucosa in a patient in DKA may be exacerbated by the tachypnoea of Kussmaul respirations.[6] In addition, vasoconstriction from acidosis may contribute to the appearance of cool extremities in addition to poor perfusion from dehydration.[6] Shock with haemodynamic compromise is uncommon in childhood DKA.

Table 2: Clinical detection of dehydration and assessment of severity

	Increasing severity of dehydration →		
	None	**Clinical Dehydration**	**Clinical Shock (suspected or confirmed)**
Appearance	Well	Unwell or deteriorating	-
Conscious Level	Alert & responsive	Altered responsiveness	↓ level of consciousness
Skin colour	Unchanged	Unchanged	Pale or mottled skin
Extremities	Warm	Warm	Cold
Eyes	Not sunken	Sunken	-
Mucous membranes	Moist	Dry	-
Heart rate	Normal	Normal	↑
Respiratory rate	Normal	↑	↑
Peripheral pulses	Normal	Normal	Weak
Capillary refill	Normal	Normal	Prolonged >2 sec
Skin turgor	Normal	↓	-
Blood pressure	Normal	Normal	↓ (decompensated shock)

- More numerous & more pronounced symptoms & signs of clinical dehydration indicate greater severity.
- For clinical shock, one or more of the symptoms and/or signs will be present.
- If in doubt, manage as if dehydration falls into the more severe category.

Source: National Collaborating Centre for Women's and Children's Health[4]

Investigations

The following urgent baseline investigations should be requested:

- BSL and finger prick blood ketones to confirm hyperglycemia and ketonemia—see Table 3 for interpretation
- blood electrolytes including glucose (calculate corrected sodium (Na^+ + [2 x (glucose – 5.5)] / 5.5) and osmolality (calculate by 2 [Na^+ + K^+] + glucose)
- venous pH and acid-base status.
- full blood count and haematocrit (the white blood cell count may be elevated due to stress and should not be interpreted as a sign of infection)
- blood urea and creatinine (creatinine measurements may be spuriously raised by assay interference from ketones). Serum urea >9.0 mmol/L may indicate severe dehydration.
- urinalysis for ketones—see Table 3 for interpretation
- urine microscopy and culture, blood cultures and chest x-ray only if indicated by clinical findings
- Hb A1C (for later analysis).

Blood should be collected for the following investigations in children with newly diagnosed diabetes:

- TSH
- blood for tissue auto-antibodies (insulin antibodies, islet cell antibodies, GAD, IA2) may be collected, particularly if there is any diagnostic uncertainty between type 1 and other types of diabetes.
- Coeliac screen Total IgA and anti-tissue transglutaminase Ab (A-TgA)

Table 3: Ketone readings and probability of DKA (use with BSL and pH)

	Low	Moderate #	High
Urine			
Bayer brand Keto-Diastix	0 mmol/L	0.5 mmol/L to < 1.5 mmol/L	≥ 1.5 mmol/L
Accu-chek brand Keto-Diabur-Test 5000	Negative	< 1.0 mmol/L	≥ 1.0 mmol/L
Blood – capillary			
Bedside meter Abbott	< 0.6 mmol/L	0.6 mmol/L to < 1.5 mmol/L	≥ 1.5 mmol/L

Possible DKA – repeat test and if the reading does not change investigate further to either establish or eliminate a diagnosis of DKA

DKA is not diagnosed on the basis of detection of ketones alone. DKA probability should be assessed in conjunction with elevated BSL levels, evidence of dehydration, and acidosis.

6. Management of Moderate to Severe DKA

General notes on management

- vomiting on presentation should initially be treated with insulin and IV fluids (not antiemetics). Consider potential underlying concomitant conditions (e.g. appendicitis, pancreatitis)
- use of neurotropic antiemetics such as Promethazine hydrochloride (Phenergan), Prochlorperazine, Prochlorperazine maleate, Prochlorperazine mesylate (Stemetil) may interfere with the patient's neurological status; if necessary use Ondansetron (Zofran)
- an IV cannula may be placed for convenient and painless repetitive blood sampling. An IA line may be necessary in some critically ill patients managed in an intensive care unit
- catheterisation of the bladder is usually not necessary, unless the child is unconscious or unable to void on demand (e.g. infants and very ill young children)
- Mannitol 20% should be available in all areas where children with DKA are managed.

Resuscitation

The following aspects need to be considered in the resuscitation of a child or adolescent with DKA:

- assess and maintain airway patency and breathing
- in severely shocked patients give high concentration oxygen via non-rebreather mask
- if shocked, initiate IV fluid administration and volume expansion immediately with an isotonic solution (0.9% NaCl). The volume and rate of administration depends on circulatory status. When clinical signs of shock are present, give 10-20 mL/kg fluid as a rapid bolus and repeat as required.

ALERT:
Most children with DKA who present in shock will not require more than two fluid boluses. If two or more fluid boluses are administered, sepsis should be considered in the differential diagnosis.

Rehydration

Diabetic ketoacidosis is characterised by loss of water and electrolytes. Administration of IV fluid, prior to giving insulin, results in substantial falls in blood glucose because the resultant increase in glomerular filtration rate (GFR) leads to increased urinary glucose excretion.[7,8] The aims of fluid and sodium replacement therapy in DKA are:

- restoration of circulating volume
- replacement of sodium and water deficit over 48 hours
- restoration of GFR with enhanced clearance of glucose and ketones from the blood
- avoidance of cerebral oedema, which may be caused by rapid fluid shifts from the extracellular fluid to the intracellular fluid compartment.

Following initial fluid resuscitation (with 0.9% NaCl), further fluid should be given as 0.9% NaCl + 40 mmol of KCL per litre. Fluid should be commenced one hour before starting insulin therapy. Once BSL falls to ≤15mmol/L or if BSL falls by more than 5 mmol per hour after the first hour, add extra glucose to the IV rehydration fluid (*see Intravenous insulin dosage and administration in Section 6*).

If hypernatraemia is present, consult with a senior specialist in emergency or paediatric medicine or a paediatric intensivist. The use of 0.45% NaCl should be considered.

Other considerations in fluid management include:

- IV or oral fluids that were given before the initiation of hospital management should be included in calculations of deficit and replacement. Urinary losses should not be added to the initial calculation of replacement fluids. Table 4 summarises the fluid therapy calculation for children with DKA and includes an example of the calculation.
- the patient should initially remain 'nil by mouth' except for ice to suck
- calculation of effective osmolality may be useful to guide ongoing fluid and electrolyte therapy
- if IV fluids are required beyond 24 hours, 0.45% NaCl + 5% glucose may be used.

Table 4: Fluid therapy calculation for children with DKA

Body weight in kg: ..... ❶ _____kg
Total **fluid bolus** given ..... ❷ _____mL

Deficit

No signs of dehydration (tolerating fluids orally)	Continue with oral rehydration
Clinical dehydration (>5% dry but not in shock)	50 mL/kg
Clinical shock (10% dry)	100 mL/kg

Enter deficit estimate (mL/kg) ..... ❸ _____mL/kg
Calculate total deficit: Multiply ❶ by ❸ ..... ❹ _____mL

If **fluid bolus** was given:
then subtract ❷ from ❹ ..... ❺ _____mL

Divide **deficit** over 48hr (divide ❺ by 48) ... ❻ _____mL/hr

Note: Deficit given over 72 hours if Na^+ corrected > 150 mmol/L or hyperosmolality > 310 mosm/L

Maintenance Fluids

Weight:	First 10kg	4 mL/kg/hr	e.g. 36kg child = (4x10) + (2x10)
	Second 10kg	2 mL/kg/hr	+ (1x16) = 76mL/hr
	Every kg after 20kg	1 mL/kg/hr	

Total maintenance fluids..... ❼ _____mL/hr

Calculate total hourly fluid rate: add ❻ and ❼..... _____mL/hr

Worked example: Fluid therapy calculation for children with DKA

Body weight in kg: ..... ❶ 34 kg
Total **fluid bolus** given ..... ❷ 340 mL

Deficit

No signs of dehydration (tolerating fluids orally)	Continue with oral rehydration
Clinical dehydration (>5% dry but not in shock)	50 mL/kg
Clinical shock (10% dry)	100 mL/kg

Enter deficit estimate (mL/kg) ..... ❸ 100 mL/kg
Calculate total deficit: Multiply ❶ by ❸ ..... ❹ 3400 mL

If **fluid bolus** was given:
then subtract ❷ from ❹ ..... ❺ 3060 mL

Divide **deficit** over 48hr (divide ❺ by 48) ... ❻ 64 mL/hr

Note: Deficit given over 72 hours if Na^+ corrected > 150 mmol/L or hyperosmolality > 310 mosm/L

Maintenance Fluids

Weight:	First 10kg	4 mL/kg/hr	**e.g. 34kg child = (4x10) + (2x10)**
	Second 10kg	2 mL/kg/hr	**+ (1x14) = 74mL/hr**
	Every kg after 20kg	1 mL/kg/hr	

Total maintenance fluids..... ❼ 74 mL/hr

Calculate total hourly fluid rate: add ❻ and ❼..... 140 mL/hr

Source: Starship Children's Health[9]

ALERT: IV fluid preparation with 5% glucose

Pre-made solutions containing glucose, e.g. 0.9% NaCl + 5% glucose, are preferred.
To make 0.9% NaCl + 5% glucose, add 50 g glucose (100 mL 50% dextrose) to 1 litre bag of 0.9% NaCl.
To make 0.9% NaCl + 10% glucose, add 100 g glucose (200 mL 50% dextrose) to 1 litre bag of 0.9% NaCl.
Note: Remember to add potassium.

Sodium replacement

Measurement of serum sodium is an unreliable measure of the degree of extracellular fluid contraction as it may be depressed due to the dilutional effect of hyperglycaemia and by fluid shift from the intracellular fluid to the extracellular fluid compartments.

Corrected sodium concentration can be calculated as:[9]

- corrected sodium = measured sodium (mmol/L) + 0.3 (glucose-5.5)

If corrected sodium is >150 mmol/L, a hypernatraemic as well as an independent glucose hyperosmolar state exists and correction of dehydration and electrolyte imbalances should aim to occur over 48-72 hours. The presence of hypernatraemia in DKA requires discussion with a senior specialist in emergency or paediatric medicine or a paediatric intensivist, as it is usually associated with severe DKA.

Normal saline (0.9% NaCl) + 40 mmol KCL per litre is an appropriate initial rehydration fluid to use in this setting. The use of this fluid should continue for the first 12 hours, as hypotonic fluids may be associated raised intracranial pressure.

Potassium replacement

Serum potassium levels in DKA at presentation are not a reliable indicator of total body potassium stores. Serum potassium may be reduced, normal or elevated at the time of presentation. The administration of insulin and the correction of acidosis will drive potassium back into the cells, decreasing serum levels. *Plan for the predictable fall in the serum potassium concentration after insulin therapy commences and the ketoacidosis starts to reverse***.**

The following issues need to be considered in potassium replacement:

- potassium should be added after restoring normal circulation, concurrent with starting insulin therapy.
- replacement therapy should be based on serum potassium measurements:
  - If hypokalaemic (< 3.5 mmol/L), start potassium replacement immediately.
  - If hyperkalaemic (> 5.5 mmol/L) or anuric (after insertion of a urinary catheter), defer potassium replacement until urine output is documented and electrolytes are available.
- commence replacement with KCL 40 mmol added to 1000 ml 0.9 % Sodium Chloride
- do not exceed a maximal potassium infusion rate of 0.3 mmol/kg/hour without consultation and cardiac monitoring.
- potassium replacement should continue throughout IV fluid therapy.
- potassium phosphate salts may be used as an alternative or in combination with potassium chloride/acetate to avoid hyperchloraemia. If hyperchloraemia is present, discuss management with the paediatric endocrinologist/intensivist. Administration of phosphate may induce hypocalcaemia and prospective studies have failed to show significant clinical benefit from phosphate replacement.[11-15]

ALERT: Preparation of IV fluids with potassium

Pre-made solutions containing potassium, e.g. 0.9% NaCl + 20 mmol/L KCL, are preferred.
If adding potassium, ensure that the total amount of potassium per litre is 40 mmol, not 60 mmol.

Bicarbonate replacement

Severe acidosis is usually reversible by fluid and insulin administration. Sodium bicarbonate should not be used routinely in the management of DKA.

Prospective controlled trials of sodium bicarbonate in children and adults with DKA have failed to demonstrate a clinical benefit.[16-18] Bicarbonate therapy may cause paradoxical CNS acidosis and rapid correction of acidosis may cause hypokalaemia, accentuate sodium load and contribute to serum hypertonicity. It may also increase hepatic ketone production, thus slowing the rate of recovery from the ketosis.[19]

Some patients may benefit from cautious bicarbonate therapy, such as those with severe acidaemia (pH <6.9 ± serum bicarbonate <5 mmol/L), which causes decreased cardiac contractility, peripheral vasodilatation and impaired tissue perfusion, and patients with potentially life-threatening hyperkalaemia. If bicarbonate is given, it should be administered at a dose of 1-2 mmol/kg by an IV infusion over 60 minutes. Cardiac monitoring should be performed in these patients due to the risk of inducing hypokalaemia.

ALERT: Administration of Sodium Bicarbonate

The decision to administer sodium bicarbonate to a child with DKA must be made in consultation with a paediatric intensivist and a paediatric endocrinologist at a Level 6 facility.

Aim to replace half the calculated deficit over 1-2 hours then repeat measurements.
ie. Bicarbonate dose = 0.5 x (Base Excess x Wt in kg x 0.3)

Insulin

Rehydration alone will decrease the BSL to some extent; however insulin therapy is required to normalise the BSL and to suppress lipolysis and ketogenesis. The desired rate of falling BSL should be approximately 4-5 mmol/L per hour, but initial rehydration alone will cause the BSL to fall, so a greater drop in BSL can be accepted in the first few hours of treatment.

Subcutaneous insulin infusion therapy is the usual initial treatment for mild DKA. In moderate and severe DKA, intravenous insulin is required. In circumstances where IV administration is not possible, IM or SC insulin has been used effectively and has been demonstrated to be safe.[20] It should be noted that poor perfusion will impair insulin absorption.[21]

Children on existing subcutaneous insulin pump therapy should have the pump discontinued until DKA has resolved.

If biochemical parameters of ketoacidosis (pH, anion gap) do not improve, reassess the patient, review insulin therapy, and consider other possible causes of impaired response to insulin (for example infection, errors in insulin preparation, adhesion of insulin to tubing with very dilute solutions).

ALERT: Intravenous insulin boluses

Boluses of intravenous insulin ***should not*** be given and may be harmful.

Intravenous insulin infusion preparation:

- only short-acting insulins (eg Actrapid or HumulinR) should be used for intravenous insulin administration.[1]
- the insulin infusion must be clearly labelled to avoid confusion with the rehydrating solution.[1]
- the insulin infusion set should be changed every 24 hours due to the potential for the insulin to denature.[1]

Table 4: ***Intravenous insulin infusion preparation***

If using syringe pump:	If no syringe pump available:
Add 50 units (0.5 ml) short-acting insulin (Actrapid or Humulin R) to 49.5 ml of 0.9 % NaCl in a syringe so that the solution contains an insulin concentration of *1 unit/ml*. The infusion should be delivered by a syringe pump into the side arm of the IV line.	Add 50 units (0.5ml) short-acting insulin (Actrapid or Humulin R) to a 500 ml bag of 0.9 % NaCl so that the solution contains an insulin concentration of *0.1 unit/ ml*. The infusion should be delivered using a volumetric pump into the side arm of the IV line. If a volumetric pump is not available a separate IV site may be required for low infusion rates.

ALERT: Calculating the dose of insulin

Very serious errors have occurred when calculating the dose of insulin. The dose of insulin should always be checked by a colleague.

Intravenous insulin dosage and administration:

- The initial IV Insulin dose should be 0.1 units/kg/hr by IV infusion in most cases. Consider 0.05 units/kg/hr in the younger child (less than 5 years), newly diagnosed diabetes, initial BSL <15 mmol/L or severe DKA with pH <7.1. These cases should be discussed with a paediatric endocrinologist.
- The dose of insulin should remain at 0.1 units/kg/hour at least until resolution of ketoacidosis (pH > 7.30, HCO3 > 15 mmol/L and/or closure of anion gap). Do not stop the insulin infusion or decrease below 0.05 units/kg/hour because continuous supplies of both insulin and glucose substrate are required to promote anabolism and reduce ketosis. In practice, this rate is usually continued for the first 12 hours in order to provide enough insulin to reverse the metabolic acidosis.
- In obese patients it may be prudent to start insulin infusion based on ideal body weight. Maximum starting dose should not exceed 5 units/hour. Be aware that there is a close association between obesity and insulin resistance. Further titration of insulin should be done in consultation with a paediatric endocrinologist.
- When the BSL falls to ≤15 mmol/L, glucose should be added to the IV fluid to prevent hypoglycaemia. A solution of 0.9% NaCl + 5% glucose + 40 mmol KCl per litre is commonly used.
- The glucose concentration in the IV fluid may be further increased to 0.9% NaCl + 10% glucose + 40 mmol KCl per litre, if needed to maintain the BSL between 5-10 mmol/L. The insulin infusion rate should only be decreased if the BSL remains below the target range despite glucose supplementation.
- Use the statewide *Diabetic Ketoacidosis (DKA) Insulin Intravenous Infusion Order and Blood Glucose Level Record Form* [add form number]

ALERT: Glucose warnings

50% glucose is extremely hypertonic and should NOT be administered without dilution.

0.9% sodium chloride with 10% glucose is very hypertonic and the site should be monitored for local reactions.

Subcutaneous insulin (with IV fluid hydration):

- if you are unable to administer insulin via IV infusion it is acceptable to use SC insulin provided that peripheral perfusion is adequate. Use the *statewide Diabetic Ketoacidosis (DKA) Subcutaneous Insulin Order and Blood Glucose Level Record Form* [add form number].
- use short or ultra-short acting insulin analog (Humalog [lispro] or NovoRapid [aspart]) at an initial dose of 0.3 units/kg. Further SC insulin may be given two hourly at a dose of 0.15-0.20 units/kg. This

regimen is for children receiving IV rehydration therapy (for mild DKA that does not require IV rehydration—*See Section 7—Management of mild DKA*).

- usual injection sites: Abdomen or leg (all children); or for small children (<2 years of age) may use upper outer quadrant of the buttocks.

Intra-muscular insulin (with IV fluid hydration):

- the intramuscular route is not usually used
- if you wish to administer insulin via this route seek expert advice (contact RSQ 1300799127).

Monitoring

The management of DKA in childhood is dependent on careful clinical observation of progress. Throughout treatment, hourly clinical observation, IV and oral medication, fluids and electrolytes, and laboratory results should be documented. Use the *statewide Diabetic Ketoacidosis (DKA) Insulin Intravenous Infusion or Subcutaneous Insulin Order and Blood Glucose Level Record Form* [add form number]

Signs of severe DKA that should prompt consideration for treatment in a Level 6 high dependency unit or paediatric intensive care unit include:[24]

- long duration of symptoms
- cardiovascular compromise
- depressed level of consciousness
- increased risk for cerebral oedema (including < 5 years of age, new onset)

Clinical reassessment of the child at frequent intervals is mandatory. Clinical and laboratory monitoring should include:

- hourly heart rate, respiratory rate, BP.
- 2nd to 4th hourly temperature.
- hourly accurate fluid input and output. If the level of consciousness is impaired, a urinary catheter may be necessary. Fluid balance should be reassessed regularly (at least 2nd hourly) because continuing polyuria may worsen dehydration.
- hourly (or more frequent) neurological observations for warning signs and symptoms of cerebral oedema.
    - headache
    - inappropriate slowing of heart rate
    - recurrence of vomiting
    - change in neurological states (restlessness, irritability, increased drowsiness, incontinence) or specific neurological signs (such as cranial nerve palsies, pupillary response)
    - rising BP, decreased oxygen saturation.

  Note: It may be difficult clinically to discriminate cerebral oedema from other causes of altered mental status.
- hourly capillary (finger prick) BSL measurement. Laboratory confirmation with venous glucose should be performed every 2 to 4 hours because capillary methods may be inaccurate in the presence of poor peripheral circulation and acidosis
- blood ketones are monitored to assist in determining resolution of DKA. Note that the bedside meter (Abbott brand) ketone readings at values >4 mmol/L are less accurate therefore this is not an appropriate measure of severity of ketosis. Do NOT use either blood or urinary ketones alone as the indicator for changes to fluid or insulin regimes.
- laboratory investigations: 2 to 4 hourly electrolytes, urea, haematocrit, venous BG level and blood gases. In severe DKA it may be necessary to monitor electrolytes hourly
- in severe DKA, ECG monitoring may be helpful to assess T-waves for evidence of hyperkalaemia or hypokalaemia

- urinalysis for ketones until negative
- daily weight is recommended.

See flow chart Appendix 1—Emergency management of children with diabetic ketoacidosis.
See flow chart Appendix 2—Medications and IV fluids for children with diabetic ketoacidosis.

7. Management of Mild DKA

The following management should only be considered in a child who:

- is clinically well
- is tolerating oral fluids
- is less than 5% dehydrated
- has a pH between 7.25 and 7.3
- has normal perfusion.

Once the diagnosis of DKA is confirmed insulin pump therapy should be discontinued for even mild DKA.

Insulin therapy in mild DKA

Subcutaneous insulin:

Short acting insulin (Actrapid or Humulin R) or ultra-short acting insulin analog (Humalog [lispro] or NovoRapid [aspart]) should be used.

Give 0.1 – 0.2 units/kg every 4-6 hours depending on the response. For young children < 4-5 years, a smaller dose of 0.05units/kg may be used. If the BGL remains elevated, a further dose of 0.05units/kg can be given after 2-3 hours. Use the *statewide Diabetic Ketoacidosis (DKA) Subcutaneous Insulin Order and Blood Glucose Level Record Form* [add form number]

Intramuscular insulin:

It is usually preferred to give either intravenous insulin via an infusion, or intermittent subcutaneous insulin. If intramuscular insulin is used, give 0.1 unit/kg/hour. Change to subcutaneous insulin once BGL < 12 mmol/L. Use only short-acting insulin (Actrapid or Humulin R).

Monitoring

Clinical reassessment of the child at frequent intervals is mandatory.

- vital signs – hourly (or more frequently if clinically indicated)
- temperature – at least 4th hourly
- blood glucose - finger prick or venous BGL at a minimum 1 hourly and more frequently if BGL is fluctuating
- ketones – preferably blood (capillary) hourly
- strict fluid balance – 1 to 2 hourly – in mild DKA it is still CRUCIAL to monitor fluid balance (in particular fluid intake) as even mild DKA can develop cerebral oedema at any time.

8. Management of Hypoglycaemia

- if BGL <5 mmol/L suspend insulin infusion, continue IV fluids, and review insulin infusion rate
- if blood glucose <4 mol/l give IV 10% glucose 2 ml/kg over 3 minutes
- do not discontinue the insulin infusion permanently
- change intravenous rehydration fluids to 0.9% NaCl + 10% glucose + 40 mmol KCl per litre until BSL stabilises at > 4 mmol/L.

9. Management of Cerebral Oedema

Cerebral oedema is a rare but devastating complication of diabetes, occurring in ~1% of children being managed for DKA. It is typically described as having a sudden onset and manifesting as rapidly progressive neurological deterioration (altered/fluctuating conscious level, headache, vomiting, bradycardia, hypertension, cranial nerve palsy, abnormal posturing, etc).

Clinical cerebral oedema can occur suddenly and at any time. The most common period for this to occur is 4-12 hours after commencement of treatment.

Risk factors include:

- new onset Type 1 diabetes
- elevated serum urea nitrogen
- severe dehydration
- severe DKA (pH ≤ 7.1)
- lower bicarbonate levels
- age ≤ 5 years
- reduced level of consciousness

Excess administration of IV fluids may contribute to the development of cerebral oedema.

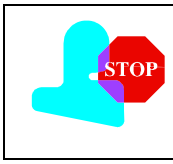
Biochemical red flags associated with the development of cerebral oedema include:

- a rapid fall in the calculated osmolarity with treatment. Usually the serum sodium rises as the glucose falls resulting in a relatively stable calculated osmolarity.
- development of hyponatraemia during therapy
- an initial corrected sodium in the hypernatraemic range.

If any of these biochemical red flags is detected consult a paediatric intensivist or paediatric endocrinologist/paediatrician for further advice.

Treatment should be initiated as soon as the condition is suspected. Management should involve:

- raise the head of the bed to 30^0
- give oxygen via O2 mask
- reduce the rate of fluid administration
- give IV Mannitol (0.5-1.0 g/kg over 20 mins) in patients with signs of cerebral oedema before impending respiratory failure. Repeat mannitol administration in 2 hours if there is no initial response.[22]
- hypertonic saline (3%) 5-10 mL/kg over 30 minutes may be an alternative to mannitol
- intubation and ventilation may be necessary, but aggressive hyperventilation has been associated with poor outcome in retrospective studies of DKA related cerebral oedema.[23]
- the patient should be transferred to an intensive care facility and a neurological assessment and MRI or CT scan arranged.



ALERT:

Emergency management of suspected cerebral oedema must be initiated as a matter of extreme urgency, and must not be delayed by requests for neurology consultation or performance of a CT or MRI.

10. Disposition

Mild and moderate cases of DKA may be managed in a general paediatric ward. A paediatrician with training and expertise in the management of DKA should direct inpatient management. The child with mild to moderate DKA should receive care in a unit that has:

- experienced nursing staff trained in the monitoring and management of DKA
- written guidelines for DKA management in children
- access to laboratories that can provide frequent and timely measurements of biochemical variables.

If appropriate staff and facilities are unavailable, the child should be transferred to a Level 6 facility.

Children presenting with severe DKA must be nursed in a Level 6 high dependency unit or paediatric intensive care unit, with a paediatric endocrinologist and/or intensivist directing management.

See flowchart Appendix 2—Admission criteria for children presenting with acute asthma.

When a decision is made to transfer a child to Level 6 facility, referral should be made through RSQ.[25]

Activation of the QLD emergency medical system coordination centre (QCC)

Further information on the preparation of a child prior to transport can be obtained through RSQ *Clinical Guidelines* paediatric section (page 31-35).[25]

Statewide RSQ clinical guidelines—Paediatrics

11. Abbreviations

Term	Definition
AVPU	Alert, Voice, Pain, Unresponsive
BG	Blood glucose
BP	Blood pressure
BSL	Blood sugar level
CHS	Children's Health Services
CNS	Central nervous system
CSCF	Clinical Services Capability Framework
DKA	Diabetic ketoacidosis
ECF	Extracellular fluid
ECG	Electrocardiogram
EDIS	Emergency Department Information System
GCS	Glasgow Coma Scale
GFR	Glomerular filltration rate
ICF	Intracellular fluid
IM	Intramuscular
IV	Intravenous
K^+	Potassium
Kussmaul breathing	Deep and labored breathing associated with metabolic acidosis
MCH	Mater Children's Hospital
Na^+	Sodium
NaCl	Sodium chloride
NBM	Nil by mouth
PICU	Paediatric Intensive Care Unit

RCH	Royal Children's Hospital
RSQ	Retrieval Services Queensland
SC	Subcutaneous

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13. Supporting Documents

- Emergency management of children with diabetic ketoacidosis flow chart
- Medications and IV fluids for children with diabetic ketoacidosis
- Admission criteria for children with diabetic ketoacidosis
- Diabetic Ketoacidosis insulin infusion order and blood glucose level record [add form number/s]
- National evidenced-based clinical care guidelines: Type 1 diabetes in children, adolescents and adults – prepared by Australasian Paediatric Endocrine Group for National Health and Medical Research Council

14. Consultation

This clinical procedure was developed and completed in collaboration with Professor Jennifer Batch, Director of Endocrinology and Diabetes, Royal Children's Hospital, Brisbane.

Key stakeholders who reviewed this version are:

- Director of Paediatric Emergency Medicine, Children's Health Services

- Clinicians (medical, nursing, allied health) working within Level 4, Level 5 and Level 6 Children's Health and Metro Children's Health Services in emergency, inpatient and ambulatory services
- Children's Health Services District clinical leaders — medical, nursing and allied health
- District Chief Executive Officers — Children's Health Services, Metro South, Metro North and West-Moreton Health Service Districts
- Queensland Ambulance Services — Manager Clinical Standards.

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- Dr Zaahid Pandie—Staff Specialist, Emergency Services, Logan Hospital
- Dr Natalie Deuble—Registrar, Emergency Services, Royal Children's Hospital
- Shahn Horrocks—Nurse Practitioner, Emergency Services, Gold Coast and Logan Hospitals
- Elizabeth Ruddy—Clinical Nurse Consultant, Emergency Services, Mater Children's Hospital
- Kate Trenoweth—Clinical Nurse, Emergency Services, Royal Children's Hospital
- Tony Hsuing—Clinical Nurse, Emergency Services, Logan Hospital.

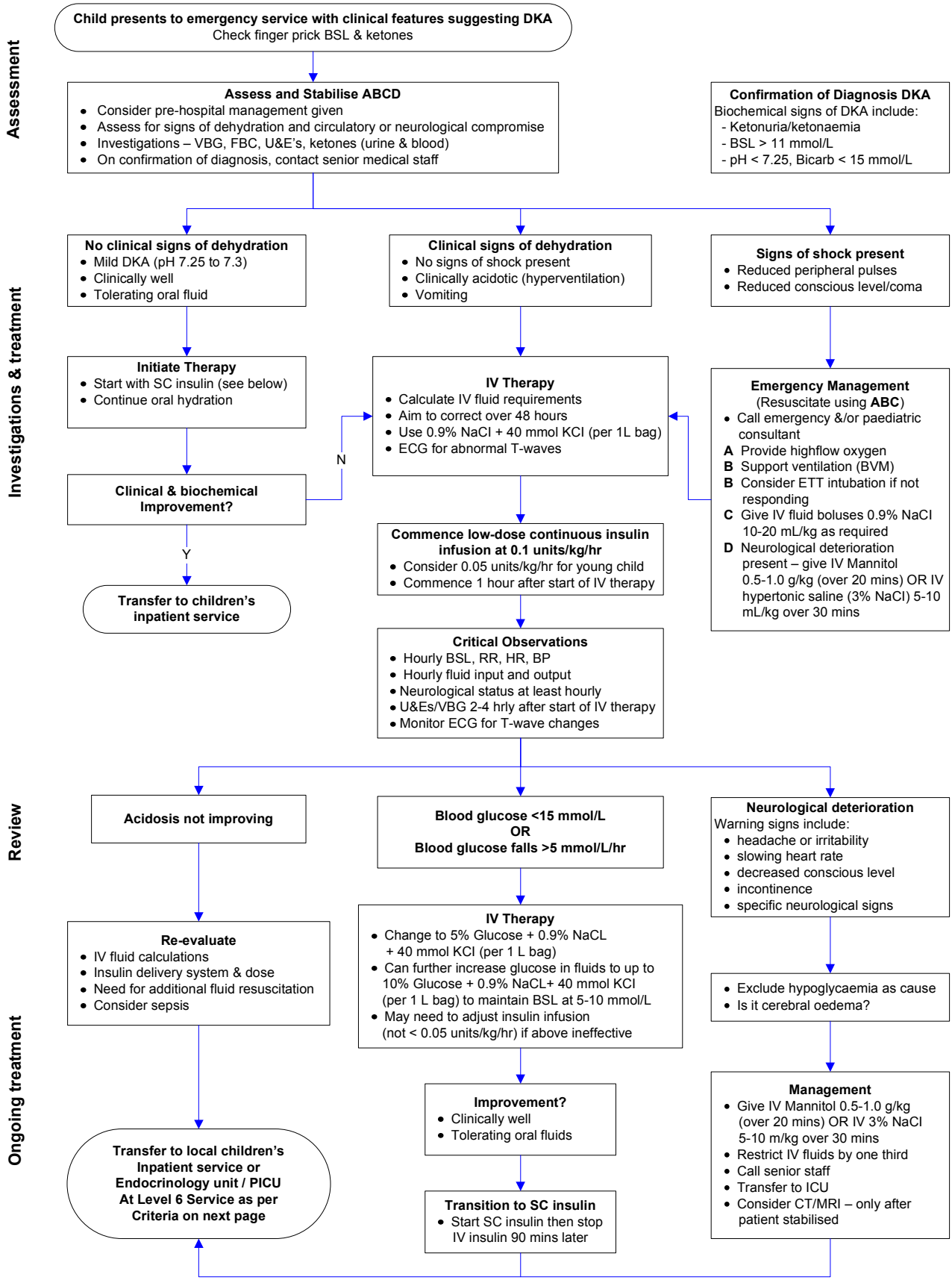
15. Procedure Revision and Approval History

Version No	Modified by	Amendments authorised by	Approved by
1.0	Greater Brisbane metropolitan area clinical procedures working group	Greater Brisbane metropolitan area clinical procedures editorial group	Chief Executive Officer, Children's Health Services

16. Audit / Evaluation Strategy

Level of risk	High
Audit strategy	1. Staff survey to evaluate awareness of procedure and emergency management practices 2. Observe practice 3. Review documentation, i.e. chart audit, to evaluate compliance with procedure
Audit tool attached	Nil
Audit date	Annual snapshot review (August)
Audit responsibility	Individual Greater Brisbane Metropolitan hospitals, i.e. Ipswich, Logan, Redland, MCH, RCH, TPCH, Redcliffe, Caboolture
Key Elements / Indicators / Outcomes	KPI 1 — greater than 80% staff awareness of procedure KPI 2 — greater than 80% compliance with procedure

17. Appendix 1 – Emergency Management of Children with Diabetic Ketoacidosis



18. Appendix 2 – Medications and IV Fluids for Children with Diabetic Ketoacidosis

Fluid therapy calculations

Body weight in kg: ..... ❶_____ kg
Total **fluid bolus** given ..... ❷_____ mL

Deficit

No signs of dehydration (tolerating fluids orally)	Continue with oral rehydration
Clinical dehydration (> 5% dry but not in shock)	50 mL/kg
Clinical shock	100 mL/kg

Enter deficit estimate (mL/kg) …… ❸_____ mL/kg
Calculate total deficit: Multiply ❶ by ❸ …… ❹_____ mL

If **> 20 mL/kg fluid bolus** given then:
subtract ❷ from ❹ …….. ❹ **B** _____ mL

Divide deficit over 48 hours (divide ❹ or ❹ **B** by 48) ❺ _____ mL/hr

Note: give defecit over 72 hrs if corrected Na+ > 150 mmol/L or hyperosmolality > 310 mosm/L.

Maintenance fluids

Weight:	First 10 kg	4 mL/kg/hr
	Second 10 kg	2 mL/kg/hr
	Every kg after 20 kg	1 mL/kg/hr

Total maintenance fluids …….. ❻ _____ mL/hr

Calculate total hourly fluid rate: add ❺ and ❻ ……. ❼ _____ mL/hr

Insulin (IV) **Dose:** 0.1 units/kg/hour

Syringe pump: Add 50 units of short-acting insulin (Actrapid or Humulin R) to 49.5 mL 0.9% NaCl (~ 1 unit of insulin per mL)
No syringe pump: Add 50 units of 50 units of short-acting insulin (Actrapid or Humulin R) to 500 mL 0.9% NaCl (~ 1 unit of insulin per 10 mL)

Insulin (SC) **Dose:** 0.1-0.2 units/kg, 4-6 hourly depending on response (Actrapid or Humulin R)

IV fluids with potassium (KCL) Add 40 mmol KCL to 1 litre bag of 0.9% NaCl
Note: use pre-mixed bags of NaCl + KCL if available

IV fluids with glucose 0.9% NaCl (1 litre bag) + 40 mmol KCL + 50 g (100 mL) **50% dextrose**
Note: use pre-mixed bags if available

19. Appendix 3 – Admission Criteria for Children with Diabetic Ketoacidosis

Criteria for admission to children's inpatient service

Criteria for admission to the children's inpatient service for a child with DKA includes:

- mild DKA (pH < 7.3 or bicarbonate < 15 mmol/L) or
- moderate DKA (pH < 7.2, bicarbonate < 10 mmol/L)
- no neurological deterioration.

In addition, the children's inpatient unit must have:

- a paediatrician trained and experienced in the management of DKA
- experienced nursing staff trained in the monitoring and management of DKA
- written guidelines for DKA management in children
- access to laboratories that can provide frequent and timely measurements of biochemical variables.

Criteria for admission to Level 6 emergency or PICU service

Consultation with the paediatric specialty team in the current facility and/or discussion with a Level 6 children's health service via RSQ is required when:

- shock not responding to treatment
- appropriate staff / facilities unavailable to care for a child with mild or moderate DKA
- severe cases of DKA
- neurological deterioration/cerebral oedema
- requirement for respiratory support (intubation & venitlation).