DIABETIC KETOACIDOSIS (DKA)

PRACTICE GUIDELINE ©

DOCUMENT SUMMARY/KEY POINTS

- Refer to One page Summary and Checklist [next page] for key points.
- Appropriate aseptic technique and hand hygiene is applied for all procedures involving intravenous infusions or medication injection.
- This guideline is intended for use within SCHN, including ward areas, emergency departments, intensive care units and other clinical areas.

  It is recognised that variations from this guideline may be required for individual patients but this should only occur under consultant supervision and in the intensive care setting.
- These guidelines have evolved as an approach to the management of DKA based mainly on clinical experience and physiological principles. There is little high quality scientific evidence comparing different regimens, with only a few randomised trials published. Most published studies are retrospective and non-randomised. Therefore clinical judgement must be exercised with each individual patient.

CHANGE SUMMARY

- New SCHN document – NO changes in practice.
- Replaces:
  - CHW Guideline No: 0/C/08:8061 ***Diabetic Ketoacidosis: Management - CHW***
  - SCH document C.16.D.4 ***Diabetes Ketoacidosis (DKA) Protocol SCH***

READ ACKNOWLEDGEMENT

- Clinical staff, nurses and medical officers, in Emergency Departments, Intensive Care Units and other Ward areas where diabetic patients are managed should read and acknowledge they understand the contents of this document.

Approved by:	Director Clinical Governance and Medical Admin	
Date Effective:	1st October 2015	**Review Period:** 3 years
Team Leader:	Staff Specialist	**Area/Dept**: Endocrinology SCH

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DKA Management - one page summary and checklist

1. **Initial Assessment**
   - Airway, Breathing, Circulation
   - level of consciousness (Glasgow Coma Scale, see Appendix 1)
   - hydration
   - measure blood glucose (and blood ketones, if available) with bedside meter
   - test urine for ketones and glucose
   - obtain patient's weight
   - Send baseline blood sample (at same time as siting IV cannula):
     - glucose, UEC, CMP, osmolality, venous pH, FBC
     - if newly diagnosed diabetes (and sufficient blood available) add autoantibodies against insulin /GAD /IA-2, TSH, and coeliac screen
2. If patient looks well and pH greater than 7.3 then IV fluids may not be required and subcutaneous insulin can be given (consult endocrine team for insulin type/dose).
   - If patient looks unwell, site IV cannula, without waiting for pH result, and start 0.9% sodium chloride infusion (or Plasma-Lyte 148, if available):
     - only if shocked, give 10mL/kg bolus of 0.9% sodium chloride and oxygen by face mask (repeat boluses are rarely required in DKA)
     - ongoing fluid rate to give maintenance plus replacement of deficit over 48 hours (see Table in Appendix 2). Consider reducing rehydration rates if excessive fluid resuscitation has already been given (greater than 20mL/kg)
     - add potassium chloride (initially 4 – 5mmol/kg/day) if potassium less than 5.0mmol/L, unless patient oliguric or known to have renal failure

   The over-riding principle is to correct the metabolic derangements (acidosis, dehydration and hyperglycaemia) **slowly**. Rapid correction has been associated with cerebral oedema.
3. Insulin infusion (Actrapid or Humulin R) at 0.05 – 0.1units/kg/hr, as a sideline to the rehydration fluid. Delay starting insulin infusion until 1 hour of IV fluid administration has been given.
4. Site second IV cannula (22 gauge minimum) for venous sampling and send second blood sample for glucose, UEC, venous pH.
5. Consider nasogastric tube (to prevent aspiration in an obtunded patient) and urinary catheter (if needed to allow strict fluid balance).
6. Ongoing management
   - keep nil by mouth (except ice to suck)
   - indications for ICU
   - monitoring
   - adjustments to fluid and electrolyte replacement
   - add 5% dextrose to IV fluids when blood glucose falls below (or is rapidly approaching) 15mmol/L (don't decrease the insulin infusion below 0.05units/kg/hr in DKA, because the ketosis is likely to worsen)
7. Transition to subcutaneous insulin.

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Background

Definition of DKA

Diabetic ketoacidosis is a potentially life threatening disorder, defined as hyperglycaemia due to insulin deficiency with pH less than 7.3.

- It may be the initial presentation of Type I Diabetes or develop in an established patient, due to failure of insulin delivery or inadequate insulin in the context of intercurrent illness.
- Recurrent DKA in adolescence is almost always due to insulin omission.
- In patients on insulin pumps, DKA is often a result of an undetected infusion set failure, through insufficient blood glucose monitoring.

Pathophysiology

- Insulin deficiency causes hyperglycaemia and ketogenesis.
- The presence of ketones (beta-hydroxybutyrate and acetoacetate) causes acidosis. Fingerprick blood ketones by bedside meter will usually be greater than 3.0mmol/L in DKA.
- Osmotic diuresis causes dehydration and a total body deficit of all electrolytes
- Accumulation of lactate due to poor tissue perfusion may contribute to the acidosis.

Complications that may arise during therapy

1. Cerebral oedema[1,2]

- Usually occurs in the first 24 hour after therapy is started
- Usually manifests with decreased consciousness, headache and signs of raised intracranial pressure, but there may be minimal symptoms until sudden collapse
- May become life-threatening due to brain herniation.
- Water molecules leave the cells in a hyperosmolar state, but brain cells are protected from shrinkage by an active process that generates osmoprotective molecules (including the amino acids taurine and glutamate). If the serum tonicity is lowered too rapidly, the brain cells remains hypertonic and a disproportionate amount of water enters them. This is the rationale for slow correction of dehydration and hyperglycaemia in DKA.
- Subclinical brain oedema is detectable by CT in most patients with DKA[3].
- **Risk factors for cerebral oedema**:
  - severe acidosis and dehydration[4]
  - extended history of poor control (presumed increase in osmoprotective adaptation)
  - young age
  - hypernatraemia, hyponatraemia, or falling serum sodium during therapy[5]
  - excessive fluid replacement has been implicated[4,5]
- Despite these known risk factors, cerebral oedema may occur unpredictably[6]

2. Hypokalaemia

- total body deficit of potassium is present before initiation of therapy, irrespective of plasma concentration
- Potassium moves into cells as the acidosis is corrected with insulin administration

3. Intracerebral thrombosis[7-9]

Procedure

Step 1: Initial Assessment and Investigations

- **A**irway, **B**reathing, **C**irculation.
- Level of consciousness (Glasgow Coma Scale, see Appendix 1).
- Degree of dehydration. This may be greater than clinically apparent because water moves from the intracellular to extracellular space due to the hyperosmolar state, partially masking the severity of the dehydration. However it is better to be conservative in the assessment of dehydration and to review progress frequently. In one study, the median dehydration was 8.7% based on body weight before and after recovery[10].
- Measure blood glucose and blood ketones with bedside meter, and urinalysis for ketones and glucose.
  - Blood ketones are quantitative and measure beta-hydroxybutyrate (the major ketone in DKA). Urine testing detects acetoacetate but not beta-hydroxybutyrate, may give false negative results if the strips have been exposed to air for an extended period, and false positive results with certain drugs (e.g. captopril).
  - Blood ketone levels are usually over 3mmol/L in DKA[11], and up to 0.9 in healthy fasted individuals[12].
- Send baseline blood sample for:
  - Glucose, UEC, CMP, osmolality, venous pH, FBC.
  - If newly diagnosed diabetes (and sufficient blood available) add autoantibodies against insulin /GAD /IA-2, TSH, and coeliac screen.
- Consider a source of infection, which may have precipitated the onset of DKA, as clinically indicated.
- Obtain patient's weight.

Step 2: Start IV fluids

- **Start rehydration** with 0.9% sodium chloride (or Plasma-Lyte 148, if available) at a rate to give maintenance plus correction of fluid deficit over 48 hours (see table of IV fluid rates in Appendix 2 as a quick guide).
  - Fluid boluses should not be given routinely because of the danger of rapidly lowering plasma osmolality and precipitating cerebral oedema.
  - If shocked (hypotension, poor peripheral perfusion) give 10mL/kg bolus of 0.9% sodium chloride. Oxygen by face mask should be given if the patient is in shock.
  - More than 2 boluses are rarely required. Avoid repeated boluses, as this may increase the risk of cerebral oedema. Remember that a contribution to decreased peripheral perfusion comes from acidosis, which will only correct gradually as the acidosis is reversed.
  - Consider reducing rehydration rates if excessive fluid resuscitation has already been given (greater than 20mL/kg).

- **Add potassium chloride to the rehydration fluid** (unless oliguric or known to have renal failure, in which case wait for electrolyte results and indwelling catheter to be placed).
  - Initially 4 – 5mmol/kg/day (this usually equates to 30 - 40mmol per 1000mL bag of IV fluids).
  - If hyperkalaemic withhold until potassium less than 5.0mmol/L.
  - Reassess with electrolyte results initially every 1-2 hours, then every 2-4 hours.
  - Potassium dihydrogen phosphate may be warranted as an alternative to potassium chloride – consult with the Endocrinologist or Intensivist on-call. Check calcium level every 4 hours if phosphate is administered.

Step 3: Insulin infusion

- Delay starting insulin until 1 hour of fluid administration has been given[4]. The blood glucose level usually starts to fall with fluids alone, by increasing renal clearance.
- Commence infusion at 0.05 – 0.1units/kg/hr with a 50mL syringe pump (or flask and infusion pump), as a side-line to the rehydration fluid.

  Note: One randomised trial[14] and 2 non-randomised studies [15-17] found that the lower dose of 0.05units/kg/hour, compared with 0.1units/kg/hr, resulted in similar patient outcomes with respect to blood glucose decrease and resolution of acidosis, and a more gradual reduction in effective plasma osmolality.
- Prime the IV line with the prepared infusion (above) by flushing a small amount into a kidney dish.
- Make up a fresh insulin infusion every 24 hours.

  Note: Must clearly label insulin infusion in accordance with MoH PD2012_007 *Labelling of Injectable Medicines, Fluids and Lines*

Preparation of insulin infusion – Use one of the following 3 methods, depending on location of the patient.

At SCH and NETS* - *preparation of insulin

- Add enough short-acting insulin (Actrapid or Humulin R) to a 50mL syringe of 0.9% sodium chloride so that **0.05units/kg/hr corresponds to running the infusion at 1mL/hr**.

Patient's weight [kg] x 2.5[#] = number of units of insulin (Actrapid or Humulin R) in 50mL 0.9% sodium chloride

Example: For a 30kg child:

- Calculate amount of insulin (Actrapid or Humulin R) required [30kg x 2.5 = 75units]

- make up to 50mL with 0.9% sodium chloride for a final concentration of 75units/50mL

[#]*Formula has been simplified: in full = weight x 0.05 x 50*

At CHW* - *preparation of insulin

In PICU [administered via syringe driver]

Add 50units of insulin (Actrapid or Humulin R) to a 50mL syringe containing 49.5mL 0.9% sodium chloride (so that concentration in syringe 1mL = 1unit)

OR

All other CHW areas [administered via a 1000mL IV fluid flask and pump]

Add 100units of insulin (Actrapid or Humulin R) to 1000mL bag of 0.9% sodium chloride to make an insulin concentration of 0.1unit per mL

Step 4: Repeat biochemistry

Site second IV in the other arm for venous sampling (22 gauge cannula minimum) and send a second blood sample for glucose, UEC, venous pH and osmolality.

Step 5: Consider a urinary catheter and nasogastric tube if conscious level depressed

- Accurate measurement of urine output is required.
- Ketoacidosis is often accompanied by ileus and a nasogastric tube may be needed if the level of consciousness is depressed. Nil by mouth, except for ice to suck, until metabolically stable (pH greater than 7.3) and bowel sounds are present, at which point low calorie fluids can be offered.

Step 6: Ongoing management

Any of the following criteria usually require admission to ICU, however these are not absolute criteria and any child causing concern should be discussed with the Intensivist:

- severe acidosis with initial pH less than 7.1.
- severe electrolyte disturbance (corrected sodium greater than 150 or less than 130mmol/L, or potassium greater than 5.5 or less than 3.0mmol/L)
- blood glucose greater than 50mmol/L
- neurologic or haemodynamic compromise
- DKA in a child aged less than 2 years

- Monitor with:
  - **i.** hourly pulse, respiratory rate, BP, neurology observations and 2-4 hourly temp
  - **ii.** blood glucose with beside glucose meter, **hourly while on IV insulin infusion**
  - **iii.** hourly blood ketones with bedside meter if blood ketone strips are not available, test all urine for ketones (until negative)
  - **iv.** accurate fluid balance chart
  - **v.** 2-4 hourly (initially 2 hourly) venous pH, electrolytes (calculate the corrected sodium, see below), glucose, osmolality
  - **vi.** reassess state of hydration every few hours
- If the pH is not improving over the first 3 hours of therapy:
  - Check for problems with delivery of the insulin infusion.
  - Increase the insulin infusion rate if needed to 0.075 or 0.1units/kg/hr. Some patients are quite insulin resistant due to the effects of critical illness and an extended period of hyperglycaemia and ketosis.
  - Consider changing the rehydration fluid from sodium chloride to Plasma-Lyte 148, if available. Plasma-Lyte 148 has a significantly lower chloride concentration than 0.9% sodium chloride (98 versus 154 mEq/L) and higher pH (7.4 versus 5.5), preventing the tendency to hyperchloraemic acidosis with 0.9% sodium chloride.

    Note: In a randomised trial Plasma-Lyte versus 0.9% sodium chloride resulted in lower serum chloride and higher bicarbonate levels[18].
  - Consider sending a urine drug screen in teenagers with existing type 1 diabetes who present in DKA without explanation. Ketamine abuse has been reported to cause severe acidosis out of proportion to the degree of ketosis[19].
  - In parallel with pH improvement, the blood ketone level should fall and the anion gap [(Sodium + Potassium) – (Bicarbonate + Chloride)] should fall back to normal (less than 16).
- Aim to produce a fall in blood glucose of ~4mmol/L per hour (*exception:* over the first few hours rehydration alone usually results in a larger fall, especially if a fluid bolus has been given).
- Add 5% dextrose to the rehydration fluid when blood glucose <15mmol/L (or rapidly approaching 15mmol/L), or if the rate of fall in blood glucose exceeds 4mmol/L after the

first few hours. (See Appendix 3 on how to increase the concentration of glucose in commonly used IV fluids).

- Make sure potassium supplementation is added to this fluid.
- In the first 4 – 6 hours use 0.9% sodium chloride + 5% dextrose. After the first 4 – 6 hours, switch to 0.45% sodium chloride (N/2) + 5% dextrose, depending on changes in the serum sodium concentration (see below, under corrected sodium)
- If necessary, in order to maintain the blood glucose between 5 and 10mmol/L, add a sideline of 10% dextrose and run this at a variable rate, reducing the rate of the sodium chloride + 5% dextrose so as to maintain the same overall hourly fluid rate. The rates of the 10% dextrose and sodium chloride + 5% dextrose lines will be adjusted each hour depending on the hourly blood glucose monitoring. (Add the same concentration of potassium chloride to the 10% dextrose so that potassium replacement will not be affected.)
- **At CHW only**, the infusion rate may be varied (see Appendix 4 [CHW Wards & ED] and Appendix 5 [CHW PICU]). The algorithm is **only to be used** when acidosis improved (bicarbonate 12-15mmol/l and **ONLY** after 4-6 hours of resuscitation.

Do not reduce the insulin infusion rate below 0.05units/kg/hr until the acidosis is corrected and ketones are cleared.

- **Management of hypoglycaemia**
  - *Blood glucose less than 4mmol/L*: temporarily cease insulin infusion, check that glucose-containing fluids are running, and recheck blood glucose after 30 minutes.
  - *Blood glucose less than 3mmol/L or symptomatic*: as above plus administer 2mL/kg IV bolus of 10% dextrose and recheck blood glucose in 15 minutes.

- **Monitor the corrected Sodium = measured Sodium + 0.3 (glucose - 5.5)** i.e., 0.3mmol/L is added to the measured sodium for every 1mmol/L of glucose above 5.5mmol/L.
- An increase in the concentration of glucose in the extracellular fluid causes water to move out of cells with dilution of extracellular solute, including sodium[13]. It is useful to estimate the expected fall in measured sodium for a given elevation of glucose because a deviation from this allows the diagnosis of a hyponatraemic or hypernatraemic state that may require alteration of the IV fluid therapy. Hyperlipidaemia (another consequence of insulin deficiency) may also falsely lower the measured sodium. In this situation the laboratory will usually comment that the serum is macroscopically lipaemic.
- The measured sodium concentration should rise as that of glucose falls. Failure of the measured sodium to rise, associated with falling corrected sodium, usually indicates excess free water administration and is associated with an increased risk of cerebral oedema[20].
- **If the corrected Sodium falls less than 140mmol/L, continue 0.9% sodium chloride (rather than 0.45% sodium chloride) as the rehydration fluid, then slow the rate of fluid administration by 30% if the corrected sodium continues to fall**.

- If the corrected sodium is greater than 145mmol/L, then hypernatraemia may aggravate the hyperosmolar state produced by hyperglycaemia. **If corrected sodium is greater than 150mmol/L, consider changing IV fluid to 0.45% sodium chloride (N/2) and slowing the rate of rehydration after discussion with consultant.**
- **Cerebral oedema:**
  - Headache, irritability, depressed consciousness, unstable body temperature, bradycardia and hypertension (late signs) may indicate increased intracranial pressure. Signs may be subtle and a high index of suspicion is needed:
  - Raised intracranial pressure due to cerebral oedema is an emergency requiring:
    - mannitol 0.5grams/kg by IV infusion over 20 minutes;
    - stop insulin infusion and reduce IV fluid to 5mL/hr temporarily;
    - transfer to ICU, ventilator support as needed;
    - CT scan and urgent neurosurgical consultation;
    - Consider 3mL/kg of 3% sodium chloride after consultation with on-call Endocrinologist or Intensivist.
- **Bicarbonate is very rarely used**. Bicarbonate administration is associated with paradoxical worsening of cerebral acidosis[21,22] and hypokalaemia (due to correcting acidosis too quickly) and was of no benefit in a retrospective case series[23]. Consider bicarbonate therapy only in patients with cardiogenic shock due to acidosis or with symptomatic hyperkalaemia. The calculated dose (in mmol) to correct deficit = 0.3 x weight (kg) x base deficit, but give only ¼ of this infused over 1 hour, then reassess.

Step 7: Transition to subcutaneous insulin

- Adjustment of the insulin infusion once the patient is eating:
  - **For meals:** double the infusion rate when the patient starts eating, continuing for the duration of the meal and one hour thereafter, before returning to the basal rate.
  - **For snacks:** double the infusion rate when the patient starts eating, continuing for the duration of the snack and 30 minutes thereafter, before returning to the basal rate.
- The infusion can be stopped when the patient is alert, stable [blood glucose less than 12, pH greater than 7.3, bicarbonate greater than 15] and ready to eat a meal:
  - just before the meal, give subcutaneous insulin (dose and type determined by the endocrine team);
  - keep the infusion running during the meal at the same rate (the half-life of intravenous insulin is only 4.5 minutes);
  - stop the infusion 90 minutes after the subcutaneous insulin has been given.
- Once established on subcutaneous insulin, the frequency of blood glucose monitoring can be reduced to pre-prandial (including meals and snacks), plus midnight and 3am.
- Before review by the dietician, the patient can initially commence their normal diet with no more than 3.5 hours between meals and snacks, except overnight.

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Appendix 1: Glasgow Coma Scale

Age 4 years and over		Age under 4 years	
Eye Opening: Best Response		**Eye Opening: Best Response**	
Spontaneously	**4**	Spontaneously	**4**
To verbal stimuli	**3**	To verbal stimuli	**3**
To painful stimuli (not applied to face)	**2**	To painful stimuli (not applied to face)	**2**
No response to pain	**1**	No response to pain	**1**
Best Verbal Response		**Best Verbal Response**	
Orientated	**5**	Appropriate words OR social smile, fixes, follows	5
Confused conversation	**4**	Cries, but consolable; less words than usual	**4**
Inappropriate words	**3**	Persistently irritable	**3**
Incomprehensible sounds	**2**	Moans to pain	**2**
No response to pain	**1**	No response to pain	**1**
Best Motor Response		**Best Motor Response**	
Obeys verbal commands	**6**	Spontaneous movements Or Obeys commands	**6**
Localises to stimuli	**5**	Localises to stimuli	**5**
Withdraws to stimuli	**4**	Withdraws to stimuli	**4**
Abnormal flexion to pain (decorticate)	**3**	Abnormal flexion to pain (decorticate)	**3**
Abnormal extension to pain (decerebrate)	**2**	Abnormal extension to pain (decerebrate)	**2**
No response to pain	**1**	No response to pain	**1**
Table 1: Coma severity on a scale from 3 to 15, based on eye, verbal and motor criteria.			

Appendix 2: IV Fluid Rates

Table 2: IV fluid rates (mL/hr) to give maintenance fluids plus replacement of the deficit over 48 hrs

Weight (kg)		Dehydration			
		3%	5%	7%	10%
5		24	26	28	31
7		34	36	39	44
8		38	42	45	50
10		48	52	56	63
12		53	58	63	71
14		59	65	70	79
16		64	71	78	88
18		70	77	85	96
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

Appendix 3: How to increase the concentration of glucose in commonly used IV fluids

Fluid Type (1000mL bag)	How much 50% Glucose to add	Total Glucose Concentration
Sodium Chloride 0.9% + Glucose 5%	Nil	5%
Sodium Chloride 0.9% + Glucose 5%	50mL	7.5%
Sodium Chloride 0.9% + Glucose 5%	100mL	10%
Sodium Chloride 0.9% + Glucose 5%	150mL	12.5%
Sodium Chloride 0.45% + Glucose 5%	Nil	5%
Sodium Chloride 0.45% + Glucose 5%	50mL	7.5%
Sodium Chloride 0.45% + Glucose 5%	100mL	10%
Sodium Chloride 0.45% + Glucose 5%	150mL	12.5%

Appendix 4: IV Insulin infusion for Diabetic Ketoacidosis – Adjustment Algorithm (for use at CHW in Wards and ED)

Infusion Concentration 0.1units/mL

Only to be used when acidosis improved (bicarbonate 12 – 15mmol/L) and ONLY after 4 – 6 hours of stabilisation

(E.g. for a 20kg child starting at 0.1unit/kg/hr = 2 units per hour = 20mL/hr). Document on the CHW Insulin Infusion Chart [M45]

The table indicates the change in insulin infusion rate from the current hourly rate according to the current BGL and rate of change of BGL in the previous hour.

Current BGL (mmol/L)	Change in BGL from last hour	No change (within 0.5mmol/L of last hour)	Falling slowly Fall of 0.6-2mmol//L/hr	Falling moderately Fall of 2-4mmol/hr	Falling quickly Fall of > 4mmol/L/hr	Rising slowly Rise of 0.6-2mmol/L/hr	Rising moderately Rise of 2-4mmol/L/hr	Rising quickly Rise of > 4mmol/L/hr
> 15mmol/L		Increase by 10%	Increase by 10%	No change	Decrease by 20%	Increase by 20%	Increase by 20%	Increase by 20%
10.1 –15mmol/L (when BGL first falls to <15 mmol/L, first step is to add glucose to IV fluids before adjusting insulin infusion)		Increase by 10%	No change	No change	Decrease by 20%	Increase by 20%	Increase by 20%	Increase by 20%
5.1 – 10mmol/L		No change	Decrease by 10%	Decrease by 20%	Decrease by 20%	No change	No change	Increase by 20%
4.1 – 5mmol/L		Decrease by 10%	Decrease by 20%	Decrease by 20%	Decrease by 50%*	No change	No change	Increase by 10%
3.1 – 4mmol/L		Cease temporarily. Recheck BGL in 30 mins & recommence infusion when BGL >5mmol/L at half the previous rate				Cease temporarily. Give IV glucose bolus of 2mL/kg of 10% Glucose. Recheck BGL in 30 mins & when BGL >5mmol/L recommence infusion at half the previous rate.	Recheck BGL in 30 minutes	
< 3mmol/L or symptomatic hypoglycemia		Cease temporarily Give IV glucose bolus 2mL/kg of 10% Glucose. Recheck BGL in 15 mins & when BGL >5mmol/L recommence infusion at half the previous rate						

NB: Call the Endocrinologist/Intensivist on call if acidosis is not improving

Appendix 5: IV Insulin infusion for Diabetic Ketoacidosis –Adjustment Algorithm (for use at CHW in PICU)

Infusion Concentration 1unit/mL

Only to be used when acidosis improved (bicarbonate 12 – 15mmol/L) and ONLY after 4 – 6 hours of stabilisation

(E.g. for a 20kg child starting at 0.1unit/kg/hr = 2mL/hr via a syringe driver). Document on the CHW Insulin Infusion Chart [M45]

The table indicates the change in insulin infusion rate from the current hourly rate according to the current BGL and rate of change of BGL in the previous hour.

Current BGL (mmol/L)	Change in BGL from last hour	No change (within 0.5mmol/L of last hour)	Falling slowly Fall of 0.6-2mmol//L/hr	Falling moderately Fall of 2-4mmol/hr	Falling quickly Fall of > 4mmol/L/hr	Rising slowly Rise of 0.6-2mmol/L/hr	Rising moderately Rise of 2-4mmol/L/hr	Rising quickly Rise of > 4mmol/L/hr
> 15mmol/L		Increase by 0.01 units/kg/hr	Increase by 0.01 units/kg/hr	No change	Decrease by 0.02 units/kg/hr	Increase by 0.02 units/kg/hr	Increase by 0.02 units/kg/hr	Increase by 0.02 units/kg/hr
10.1 –15mmol/L (when BGL first falls to <15 mmol/L, first step is to add glucose to IV fluids before adjusting insulin infusion)		Increase by 0.01 units/kg/hr	No change	No change	Decrease by 0.02 units/kg/hr	Increase by 0.02 units/kg/hr	Increase by 0.02 units/kg/hr	Increase by 0.02 units/kg/hr
5.1 – 10mmol/L		No change	Decrease by 0.01 units/kg/hr	Decrease by 0.02 units/kg/hr*	Decrease by 0.02 units/kg/hr*	No change	No change	Increase by 0.02 units/kg/hr
4.1 – 5mmol/L		Decrease by 0.01 units/kg/hr	Decrease by 0.02 units/kg/hr	Decrease by 0.02 units/kg/hr*	Decrease by half*	No change	No change	Increase by 0.01 units/kg/hr
3.1 – 4mmol/L		Cease temporarily. Recheck BGL in 30 mins & recommence infusion when BGL >5mmol/L at half the previous rate				Cease temporarily. Give IV glucose bolus of 2mL/kg of 10% Glucose. Recheck BGL in 30 mins & when BGL >5mmol/L recommence infusion at half the previous rate.	Recheck BGL in 30 minutes	
< 3mmol/L or symptomatic hypoglycemia		Cease temporarily Give IV glucose bolus 2mL/kg of 10% Glucose. Recheck BGL in 15 mins & when BGL >5mmol/L recommence infusion at half the previous rate						

NB: Call the Endocrinologist/Intensivist on call if acidosis is not improving

Appendix 6: Clinical Features of DKA

- Polydipsia
- Polyuria continuing despite the dehydration
- Weight loss due to fluid loss and loss of muschloridee and fat
- Flushed cheeks due to the ketosis
- Acetone detected on the breath moderate - severe dehydration
- Frequent vomiting
- Abdominal pain
- Decreased perfusion
- Hyperventilation of DKA (Kussmaul respiration), characterised by high respiratory rate and large tidal volume giving a sighing quality. This is due to attempted respiratory compensation for the underlying metabolic acidosis.
- Disordered sensorium (disoriented, drowsy, or, rarely, comatose)
- Shock (rapid pulse rate, low blood pressure, poor peripheral circulation, mottling and peripheral cyanosis, lactic acidosis)