DIABETIC KETOACIDOSIS (DKA)

PRACTICE GUIDELINE ©

DOCUMENT SUMMARY/KEY POINTS

Variations from this guideline may be required for individual patients but this should only occur under consultant supervision or in the intensive care setting.

- Refer to Algorithm for the management of DKA [next page].
- Appropriate aseptic technique and hand hygiene is applied for all procedures involving intravenous infusions or medication injection.
- This guideline for use within SCHN, including ward areas, emergency departments, intensive care units and other clinical areas. **Recommendations are based on the International Society for Paediatric and Adolescent Diabetes DKA Guideline**.
- Use standard concentrations for insulin preparation.

CHANGE SUMMARY

- Introduction of standard concentrations for insulin preparation across SCHN.
- Several other minor changes throughout the document.
- Recommend relevant clinical staff to re-read the entire document.

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:	SCHN Policy, Procedure and Guideline Committee	
Date Effective:	1st June 2018	**Review Period**: 3 years
Team Leader:	Endocrinologist	**Area/Dept**: Endocrine

Date of Publishing: 25 June 2018 1:57 PM Date of Printing: 25 June 2018

K:\CHW P&P\ePolicy\May 18\DKA.docx

Algorithm for the Management of DKA

IMMEDIATE ASSESSMENT

Clinical History
Polyuria
Polydipsia
Weight loss *(weigh patient)*
Abdominal pain
Tiredness
Vomiting
Confusion

Clinical Signs
Assess hydration
Deep sighing respiration (Kussmaul)
Smell of ketones
Lethargy/drowsiness ± vomiting

Biochemical features & Investigations
Ketones in urine (or blood ketone meter)
Elevated blood glucose
Acidemia
Pathology tests: UEC, BGL; VBG; CMP; Osmolality
Other investigations as indicated
Antibodies (Insulin, GAD & IA-2), Coeliac screen, TSH and thyroid antibodies,

Diagnosis Confirmed
DIABETIC KETOACIDOSIS
Contact Endocrinology Team on-call

Severe (≥8%) Dehydration
Shock (reduced peripheral pulses, tachycardia, delayed CR)
Reduced conscious level

Moderate (5-7%) Dehydration
but not in shock
Clinically acidotic (hyperventilation)
Vomiting

Mild (3%) Dehydration and mild acidosis [pH 7.25 to 7.3]
Clinically well
Tolerating fluids orally

Resuscitation
Airway ± NG tube
Breathing (100% Oxygen)
Circulation
Disability
Exposure
Fluids:
0.9% sodium chloride 10mL/kg as a bolus then reassess and repeat if signs of shock are not reversed.
If severely dehydrated but not overt shock, give 10mL.kg 0.9% sodium chloride over 1 hour.
Generally do not exceed 30mL/kg.
Reassess hydration status and continue fluids for the current hydration status (post bolus).

IV Therapy
Calculate fluid requirements:
Correct over 48 hours
0.9% sodium chloride
ECG for abnormal T-waves
Add potassium 40mmol/L once urine output confirmed

Therapy
Start with subcut insulin
Continue oral hydration

No improvement

Delay starting insulin until 1 hour of fluid administration has been given.
Check glucose prior to starting insulin
Commence at 0.05-0.1unit/kg/hour

Critical Observations
Hourly blood glucose level by bedside meter
Hourly fluid input & output
Neurological status at least hourly
UEC, VBG, glucose, osmolality (2nd hourly)
Monitor ECG for T-wave changes

Acidosis NOT improving

Re-evaluate
IV fluid calculations
Insulin delivery systems & dose
Need for additional resuscitation
Consider sepsis

Blood glucose ≤ 15mmol/L or falling >5mmol/L/hr after the initial fall associated with the first 1-2 hours of fluid therapy

IV Therapy
Change to IV fluids containing 5% glucose (either 0.9% or 0.45% sodium chloride or Plasma-Lyte 148 depending on corrected sodium and rate of change of this).
Maintain BGL >5mmol/L

Improvement
Clinically well, tolerating oral fluids

Transition to subcut Insulin
Start subcut insulin **then** stop IV insulin after an appropriate interval

Neurological
WARNING SIGNS: headache, slowing heart rate, irritability, decreased conscious level, incontinence, specific neurological signs

Exclude Hypoglycaemia

Is it Cerebral Oedema?

Management
Manage ABCDEF
Urgent consultation with ICU regarding management & in anticipation transfer to ICU
Consider Mannitol 0.5 – 1.0 g/kg or 3% sodium chloride [3-5 mL/kg]
Consider CT brain - **only** after patient stabilised

Algorithm for the Management of diabetic ketoacidosis. Adapted from ISPAD Clinical Practice Consensus Guidelines 2014 Compendium[1]

TABLE OF CONTENTS

1 Background

1.1 Diagnosis of DKA

Diagnosis
Hyperglycaemia: blood glucose greater than 11mmol/L
Venous pH less than 7.3 or bicarbonate less than 15mmol/L
Presence of ketonaemia or ketonuria

- 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.

1.2 Clinical Features of DKA

- Dehydration
- Polydipsia and polyuria continuing despite the dehydration
- Weight loss due to fluid loss and loss of muscle and fat
- Hyperventilation of DKA (Kussmaul respiration), characterised by high respiratory rate and large tidal volume giving a sighing quality.
- Acetone smell on the breath and flushed cheeks due to ketosis
- Shock (rapid pulse rate, low blood pressure, poor peripheral circulation, mottling and peripheral cyanosis, lactic acidosis)
- Nausea, vomiting (may be mistaken for gastroenteritis)
- Abdominal pain (may mimic an acute abdominal condition)
- Disordered sensorium (disoriented, drowsy, or comatose)

1.3 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.

1.4 Complications of DKA

Cerebral oedema	*Hypokalaemia*	*Thrombosis*	*Hyperglycamic Hyperosmolar State [HHS] and mixed HHS DKA*
Usually occurs in the first 24 hour after therapy is started. Usually presents 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. **Risk factors** for cerebral oedema: - severe acidosis and dehydration - extended history of poor control (presumed increase in osmoprotective adaptation) - young age - hypernatraemia, hyponatraemia, or falling serum sodium during therapy - excessive fluid replacement	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	There is an increased thrombotic tendency. Prophylactic low dose heparin is not routinely recommended, but should be considered and discussed with intensivists for patients who have additional risk factors for thrombosis e.g. central venous catheter, young age, severe dehydration or coexisting HHS.	**Criteria for the diagnosis of HHS are**: - Plasma glucose greater than 33mmol/L - pH greater than 7.3 - Serum bicarbonate greater than 15mmol/L - Absent or mild ketonaemia - Calculated serum osmolality greater than 320mOsm/kg: [(2 x corrected plasma sodium) + plasma glucose] - Altered consciousness or seizures When the patient meets criteria for DKA with acidosis and ketosis, and the calculated serum osmolality greater than 320mOsm/kg then “mixed HHS DKA” is present. DKA treatment should be modified after discussion with Intensive Care Consultant and Endocrinologist on-call. (see Step 5: ongoing management)

Despite these known risk factors, cerebral oedema may occur unpredictably			

2 Procedure

Step 1: Initial Assessment and Investigations

- **Resuscitation**: **A**irway, **B**reathing, **C**irculation, **D**isability (neurologicial assessment), **E**xposure, **F**luids.
- Level of consciousness (Glasgow Coma Scale).
- 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.
- Measure blood glucose and blood ketones with bedside meter, (or urinalysis for ketones and glucose if blood ketone meter is not available).
  - Blood ketones are quantitative and measure beta-hydroxybutyrate (the major ketone in DKA).
- Send baseline blood sample for:
  - Blood glucose level (BGL), urea, electrolytes and creatinine (UEC), calcium, magnesium phosphate (CMP), osmolality, venous blood gas (VBG) and full blood count (FBC). Calculate the corrected (i.e. actual) sodium = measured sodium + 0.3 (BGL - 5.5) mmol/L. If corrected sodium is greater than 160 mmol/L, discuss with the on-call Intensivist.
  - Calculate the anion gap ([sodium + potassium] – [bicarbonate+ chloride]).
  - If newly diagnosed diabetes (and sufficient blood available) add insulin autoantibodies (IAA), glutamic acid decarboxylase (GAD), tyrosine phosphatase antibodies (IA-2), thyroid stimulating hormone (TSH), thyroid autoantibodies and coeliac screen.
- Consider a source of infection, which may have precipitated the onset of DKA, as clinically indicated.
- Obtain patient's weight, and most recent prior weight if available.

Step 2: Start IV fluids

- **Start rehydration** with 0.9% sodium chloride (Plasma-Lyte 148 is a suitable alternative) 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 (tachycardia, thready pulses, delayed capillary refill time (CRT) +/- hypotension) 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**
  - Initially 4 – 5mmol/kg/day (this usually equates to 30 - 40mmol per 1000mL bag of IV fluids).
  - If hyperkalaemic, oliguric or known to have renal failure, withhold potassium until urine output is documented and potassium less than 5.0mmol/L.
  - If normokalaemic start potassium replacement after the initial volume expansion and concurrent with starting insulin infusion.
  - DO NOT use potassium containing fluids to give a fluid bolus. If the patient is hypokalaemic at presentation discuss with the Endocrinologist or Intensivist on call as the patient may require earlier initiation of potassium replacement.
  - Reassess with electrolyte results initially every 1-2 hours, then every 2-4 hours.
  - Consider potassium dihydrogen phosphate if patient is hyperchloraemic. Consult with the Endocrinologist or Intensivist on-call. Monitor calcium levels if phosphate is administered.
  - Note: Plasma-Lyte-148 contains 5mmol/L of potassium – this must be taken into consideration when adding extra potassium.

Step 3: Insulin infusion

- Delay starting insulin until 1 hour of fluid administration has been given. 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 volumetric infusion pump), as a sideline to the rehydration fluid. Consider using the lower insulin dose if hypokalaemic, if risk factors for cerebral oedema exist or if HSS is present.
- Prime the IV line with the prepared infusion and discard a small amount.
- A new insulin infusion must be prepared every 24 hours as detailed below:

If using a "smart" syringe pump with error reduction software*	If no smart pump is available
Patient >10kg: Add 50units of insulin (Actrapid or Humulin R) to a 50mL syringe containing 49.5mL 0.9% sodium chloride *(so that concentration is 1unit per mL)*	Add 100units of insulin (Actrapid or Humulin R) to 1000mL bag of 0.9% sodium chloride *(so that concentration is 0.1unit per mL)*
Patient ≤10kg Add 25units of insulin (Actrapid or Humulin R) to a 50mL syringe containing 49.75mL 0.9% sodium chloride *(so that concentration is 0.5unit per mL)*	

* A smart syringe pump has error reduction software which enables the user to select the drug that they are administering, enter the concentration of the solution, weight of the patient and the intended dose per hour to be delivered. Smart pumps are currently in use at SCH and in PICU at CHW.

- Insulin infusion must be clearly labelled in accordance with MoH PD2016_058 Labelling of Injectable Medicines, Fluids and Lines.

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 BGL, UEC, VBG and osmolality. Monitor glucose, anion gap and corrected sodium.

Step 5: 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

- hyperosmolar state (corrected serum osmolality greater than 320mOsm/kg)
- abnormal or falling GCS
- other neurologic or haemodynamic compromise
- DKA in a child aged less than 2 years

- **Monitor with**:
  - **i.** hourly pulse, respiratory rate, blood pressure, neurology observations and 2-4 hourly temperature.
  - **ii.** blood glucose with beside glucose meter, hourly while on IV insulin infusion
  - **iii.** hourly accurate fluid balance chart
  - **iv.** 2 hourly blood ketones with bedside meter. If blood ketone strips are not available, test all urine for ketones (until negative)
  - **v.** 2-4 hourly (initially 2 hourly) VBG, UEC, serum osmolality, BGL.
  - **vi.** Calculate the corrected sodium, see below), anion gap and osmolality, (if serum osmolality is unavailable)
  - **vii.** reassess state of hydration every few hours
- Urinary catheter and nasogastric tube are not commonly required but should be considered for the following reasons.
  - Ketoacidosis is often accompanied by ileus.
  - A nasogastric tube may be needed if the level of consciousness is depressed

Monitor the anion gap (normal anion gap = 8-16mmol/L)

- In parallel with pH improvement, the blood ketone level should fall and the anion gap [(sodium + potassium) – (bicarbonate + chloride)] should return to normal.
- **If the anion gap is not falling**, check for problems with administration of the insulin infusion, and consider increasing the insulin infusion rate.
- **If anion gap is falling**, but pH remains low due to hyperchloraemia, consider changing the rehydration fluid from sodium chloride to Plasma-Lyte 148, which has lower chloride concentration than 0.9% sodium chloride (98 versus 154 mmol/L).

Monitor BGL and rate of fall of BGL

- Aim to produce a fall in BGL of approximately 4mmol/L per hour (exception: over the first 2 hours rehydration alone usually results in a larger fall, especially if a fluid bolus has been given).
- Change fluids to those containing 5% glucose when BGL less than 15mmol/L (or rapidly approaching 15mmol/L), or if the rate of fall in BGL exceeds 5mmol/L/hr after the first 2 hours. IV fluids should be either 0.9% or 0.45% sodium chloride or Plasma-Lyte 148 depending on corrected sodium and rate of change of this.
- Make sure potassium supplementation is added to this fluid.

Modification of the glucose content of the IV fluids may be required to maintain the BGL between 5 and 10mmol/L. This can be achieved by increasing the glucose concentration of the IV fluids as detailed in Appendix 3.

An alternative method to modify the glucose content is to run two IV fluid lines, with the same sodium and potassium content, but with different glucose concentrations to allow titration. This method may be particularly useful during transportation when it is difficult to make up a new glucose concentration.

When acidosis is improved (bicarbonate 12-15mmol/L) consider reducing the the insulin infusion rate to 0.05 Units/kg/hr and then further adjustments should be made as directed in Appendix 4: IV Insulin Infusion for Diabetic Ketoacidosis – Adjustment Algorithm. Alternatively the infusion rate can be reduced to 0.05 Units/kg/hr and then adjusted in 10-20% increments to keep BGL 5 -10mmol/L.

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

Management of hypoglycaemia

- BGL 3.1 - 4mmol/L:
  - Temporarily cease insulin infusion, check for problems with the administration of the glucose-containing fluids, and recheck blood glucose after 30 minutes Recommence insulin infusion at half the rate once the BGL is greater than 5mmol/L.
- BGL less than 3.1 mmol/L or symptomatic hypoglycaemia:
  - As above plus administer 2mL/kg IV bolus of 10% glucose and recheck blood glucose in 15 minutes. Recommence insulin infusion at a reduced rate, usually half the previous rate (in consultation with Endocrinology team) once the BGL is greater than 5mmol/L.

Monitor the corrected Sodium

- **Corrected Sodium = measured Sodium + 0.3 (glucose - 5.5 mmol/L)**
- The measured sodium concentration should rise as the glucose falls (corrected sodium should stay the same). Failure of the measured sodium to rise, associated with falling corrected sodium, indicates excess free water administration and is associated with an increased risk of cerebral oedema.
- **If the corrected Sodium falls less than 140mmol/L, continue 0.9% sodium chloride or Plasma-Lyte 148 (rather than 0.45% sodium chloride) as the rehydration fluid, and slow the rate of fluid administration by 30%**.
- If the corrected sodium increases by greater than 5mmol/L/hr to greater than 150mmol/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 and slowing the rate of rehydration after discussion with an Intensivist or Endocrinologist.

- If the calculated serum osmolality is greater than 320mOsm/kg, calculated as [(2 x corrected plasma sodium) + plasma glucose], management should be adjusted after discussion with the Intensive Care Consultant and Endocrinologist on-call. The principles of management for **HHS and "mixed HHS DKA"** to be considered are:
  - Fluid deficits are greater than in straightforward DKA, so an initial fluid bolus of greater than or equal to 20mL/kg 0.9% sodium chloride will be required to restore peripheral perfusion
  - Thereafter 0.45% sodium chloride should be used to replace the deficit. 0.9% sodium chloride can be restarted if perfusion and haemodynamic status appears inadequate. Serum sodium concentrations should be measured frequently and the sodium concentration in fluids may need to be adjusted to promote a gradual decline in corrected sodium.
  - Aim for a gradual reduction of sodium (less than 0.5mmol/L per hour) and glucose (less than 5mmol/L per hour)
  - Given that fluid deficits are greater and osmotic diuresis often persists, consider replacement or partial replacement of urine loss with 0.45% sodium chloride.
  - As with straightforward DKA, defer starting the insulin infusion until after the initial fluid bolus in mixed HSS DKA. However, in isolated HHS start an insulin infusion only when BGL no longer decreases by at least 3mmol/hour with fluid administration alone.
  - Consider using a lower insulin infusion dose e.g. 0.05 units/kg/hr in mixed HHS DKA and 0.025-0.05 units/kg/hr in isolated HHS.
  - Frequent circulatory and fluid reassessment.
  - Monitoring of mental state, continuous cardiac monitoring, CK levels and temperature are required. Rhabdomyolysis, malignant hyperthermia, arrhythmias and venous thrombosis are known complications.
  - Heparin may be considered if prolonged immobility or central venous catheter required.

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 urgent treatment by:
  - Elevating the head of the bed to 30 degrees
  - Immediately reducing the rate of IV fluids by at least one third
  - Discuss with ICU consultant and consider further fluid restriction by temporarily stopping all IV fluids that reduce plasma tonicity, including insulin infusion

- Give mannitol 0.5-1.0g/kg by IV infusion over 10-15 minutes and repeat if there is no initial response in 30mins to 2 hours
- Hypertonic saline (3%), 3 – 5mL/kg over 10 -15 min, may be used as an alternative to mannitol. If no response or deterioration after mannitol the addition of hypertonic saline may be indicated after expert consultation.
- Transfer to ICU, intubation and ventilatory support as required
- After treatment for cerebral oedema has been started consider cranial imaging and neurosurgical consult.

Bicarbonate

- **Bicarbonate is very rarely used**.
- Bicarbonate administration is associated with paradoxical worsening of cerebral acidosis and hypokalaemia (due to correcting acidosis too quickly) and was of no benefit in a retrospective case series. Consider bicarbonate therapy only in patients with cardiogenic shock due to acidosis or with symptomatic hyperkalaemia, under the direction of the Intensivist on-call.

Step 6: 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 [BGL 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:
    - 30 minutes after ***rapid acting*** subcutaneous insulin has been given OR
    - 90 minutes after regular or ***long acting*** subcutaneous insulin has been given.
- Once established on subcutaneous insulin, the frequency of BGL monitoring can be reduced to pre-prandial (including meals and snacks), plus midnight and 3am.

3 References

1. Wolfsdorf JI et al. A Consensus Statement from the International Society for Pediatric and Adolescent Diabetes: Diabetic ketoacidosis and hyperglycemic hyperosmolar state. Pediatric Diabetes 2014: 15 (Suppl. 20): 154–179.
2. Dunger DB et al. European Society for Paediatric Endocrinology/Lawson Wilkins Pediatric Endocrine Society Consensus Statement on Diabetic Ketoacidosis in Children and Adolescents. Pediatrics. 2004;113(2):e133-e40.

Appendix 1: Abbreviations

BGL	Blood Glucose Level
CMP	Calcium, Magnesium, Phosphate
CK	Creatine kinase
CRT	Capillary Refill Time
DKA	Diabetic Ketoacidosis
ECG	Electrocardiogram
FBC	Full Blood Count
GAD	Glutamic Acid Decarboxylase
GCS	Glasgow Coma Score
HSS	Hyperglycaemic Hyperosmolar State
IAA	Insulin autoantibodies
IA-2	Tyrosine Phosphatase Antibodies
NG	Nasogastric tube
TSH	Thyroid Stimulating Hormone
UEC	Urea, Electrolytes, Creatinine
VBG	Venous Blood Gas

Appendix 2: IV Fluid Rates

Table 1: 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

Starting Fluid	Volume to be removed from 1000mL bag#	Volume of 50% glucose to be added to starting fluid	Final glucose concentration*
0.9% NaCl	50mL	50mL	0.9% NaCl + 2.5% glucose
0.9% NaCl + 5% glucose	55mL	55mL	0.9% NaCl + 7.5% glucose
0.9% NaCl + 5% glucose	110mL	110mL	0.9% NaCl + 10% glucose
0.9% NaCl + 5% glucose	167mL	167mL	0.9% NaCl + 12.5% glucose
0.45% NaCl + 5% glucose	55mL	55mL	0.45% NaCl + 7.5% glucose
0.45% NaCl + 5% glucose	110mL	110mL	0.45% NaCl + 10% glucose
0.45% NaCl + 5% glucose	167mL	167mL	0.45% NaCl + 12.5% glucose
5% glucose	55mL	55mL	7.5% glucose

* When adding glucose to solutions, dilution of base solution will occur, therefore all final concentrations of sodium chloride and glucose are *approximate.*

The formula below can be adapted to all volumes simply by using the correct ratio. It can also be used to obtain other concentrations of glucose solutions.
Please ensure that all calculations are double checked at all times.

If: a = concentration of glucose in base solution,
b = concentration of glucose required in final solution
and,
x = number of mL of 50% glucose required
y = number of mL of base solution (a% glucose) required
Values for x and y in order to make a solution of 1000 mL of b% glucose are given by:

$$y = \left(\frac{5000 - 100b}{50 - a} \right) \times 10$$

and

$$x = 1000 - y$$

Appendix 4: IV Insulin infusion for Diabetic Ketoacidosis – Adjustment Algorithm

Only to be used when acidosis improved (bicarbonate 12 – 15mmol/L)

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.

	Change in BGL from last hour						
Current BGL (mmol/L)	**Falling quickly Fall of > 4mmol/L/hr**	**Falling moderately Fall of 2-4mmol/hr**	**Falling slowly Fall of 0.6-2mmol//L/hr**	**No change (within 0.5mmol/L of last hour)**	**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	Decrease by 20%	No change	Increase by 10%	Increase by 10%	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)	Decrease by 20%	No change	No change	Increase by 10%	Increase by 20%	Increase by 20%	Increase by 20%
5.1 – 10mmol/L	Decrease by 20%	Decrease by 20%	Decrease by 10%	No change	No change	No change	Increase by 20%
4.1 – 5mmol/L	Decrease by 50%*	Decrease by 20%	Decrease by 20%	Decrease by 10%	No change	No change	
3.1 – 4mmol/L	Cease temporarily. Recheck BGL in 30 mins & recommence infusion when BGL >5mmol/L at 50% lower than the previous rate Give IV glucose bolus 2mL/kg of 10% glucose only if symptomatic						
< 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 50% lower than the previous rate						

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