BSPED Guideline for the Management of Children and Young People under the age of 18 years with Diabetic Ketoacidosis - 2021

This guideline for the management of DKA replaces the BSPED interim guideline published in 2020 and has been updated in light of the NICE Guidance NG18 which was updated in December 2020 and UK Resuscitation Council recommendations published in May 2021. It has been revised by the BSPED special interest group in diabetic ketoacidosis following a series of meetings. The relatively limited evidence regarding the management of DKA has been reviewed. Where there is appropriate evidence these guidelines have been based on such evidence. For many aspects of the management of DKA the evidence base is limited and where there is limited evidence, consensus recommendations have been consolidated. The guideline is broadly similar to the International Society for Paediatric and Adolescent Diabetes (ISPAD) and takes account of the updated NICE NG18 guidance.

These BSPED guidelines are believed to be as safe as possible in the light of current evidence. However, no guidelines can be considered entirely safe as complications may still arise. In particular the pathophysiology of cerebral oedema is still poorly understood.

The following changes have been made since the last BSPED guideline was published in 2015 and the interim guideline in 2020:

1) NICE guidance NG18 applies to all individuals <18 years and does not make explicit recommendation's for the group aged 16-18 years who may be managed by either Adult or Paediatric medical teams. The BSPED special interest group remained of the opinion that where young people aged 16-18 years are managed by adult medical teams because of local arrangements, it is appropriate for them to be managed using local adult guidelines that the teams are familiar with rather than using potentially unfamiliar paediatric guidelines. Where individuals aged 16-18 are managed by Paediatric teams the Paediatric guidelines should be followed.

2) The ISPAD definition for DKA with acidosis and a bicarbonate of <15 mmol/l **or** a pH <7.3, **and** ketones of >3.0 mmol per litre has been adopted.

3) This guideline uses pH to categorise the severity of DKA and to determine the degree of dehydration. It has been revised since the interim guideline, in light of the revised NICE guidance, and recommends assuming 5% dehydration for mild and moderate DKA and 10% dehydration for severe DKA. The recommendation that patients with moderate DKA be assumed to be 7% dehydrated has been withdrawn.
    - Mild DKA – venous pH 7.2- 7.29 or bicarbonate < 15 mmol/l. Assume 5% dehydration
    - Moderate DKA – venous pH 7.1-7.19 or bicarbonate < 10 mmol/l. Assume 5% dehydration
    - Severe DKA – venous pH less than 7.1 or serum bicarbonate < 5 mmol/l. Assume 10% dehydration

4) There is increased emphasis within this guideline on ensuring adequate restoration of the circulation and treatment of shock. The use of inotropes in preference to fluid volume particularly early in resuscitation has been de-emphasised. Careful management of fluid administration remains an important part of the management of diabetic ketoacidosis because of the risk of cerebral oedema but there is increased emphasis on the importance of treating shock and restoring appropriate circulatory volume.
    - The 2020 update to NICE NG18 guidance recommends that patients presenting with shock should receive a 20 ml/kg bolus of 0.9% saline over 15 minutes. However the UK Resuscitation Council revised its guidance for the treatment of shock in children and young people in May 2021 to suggest that fluid be given as 10ml/kg boluses repeated as required rather than a single initial 20ml/kg bolus. It also recommended isotonic crystalloids to treat shock and if not available 0.9% Saline. The aetiology of septic shock differs from that of DKA, however this guideline has adopted the UK Resuscitation Council guidance that boluses be given in 10ml/kg aliquots repeated as required, to correct shock with reassessment between boluses rather than a single 20ml/kg bolus. The PECARN FLUID

trial investigating the management of DKA whose evidence was considered in revising the guideline suggested that a 20ml/kg bolus in patients presenting with DKA (either with or without shock) did not increase the incidence of cerebral oedema. Shock is defined as the APLS definition of tachycardia, prolonged central capillary refill etc – it is not just poor peripheral perfusion. Acidosis and hypocapnia cause peripheral vasoconstriction. NICE NG18 emphasises weak thready peripheral pulses and hypotension as a feature of shock. Following the initial 10 ml/kg bolus patients with shock should be reassessed and further boluses of 10 ml/kg may be given if required to restore adequate circulation up to a total of 40 ml/kg at which stage ionotropes should be considered. Boluses given to treat shock SHOULD NOT be subtracted from the calculated fluid deficit.

- All patients with DKA (mild, moderate or severe) in whom intravenous fluids are felt to be indicated AND WHO ARE NOT IN SHOCK should receive an initial 10 ml/kg bolus over 30 minutes. This recommendation has been revised from the interim guideline to suggest that the bolus is given over 30 minutes rather than 60 minutes. Shocked patients do NOT need this extra bolus as they will already have received appropriate fluid boluses. This 10ml/kg bolus SHOULD be subtracted from the calculated fluid deficit

5) The calculation of maintenance fluids should be based on the traditional formula used in paediatrics in the UK. – 100 ml/kg/day for the first 10 kg body weight, plus 50 ml/kg/day for 10 to 20 kg and 20 ml/kg/day for each additional kilogram above 20 kg. This is a more permissive maintenance fluid rate than in the previous DKA guideline and is a significant change.

6) A maximum weight of 75kg should be used for the calculation of fluid replacement and deficit as this ensures that excessive volumes of fluids are not given. The maximum weight has been revised to 75kg from the 80kg recommended in the interim guidance.

7) The option to consider either 0.05 units/kg/hr or 0.1 units/kg/hr for Insulin infusion is maintained but the working group felt that 0.05 units/kg/hour would be sufficient in most cases except perhaps in severe DKA. In children younger than 5 years 0.05 Units/kg/hr was suggested (consensus recommendation) to reduce the incidence of subsequent hypoglycaemia

8) Where Potassium is above the upper limit of the normal range at presentation it is recommended that Potassium is only added to Intravenous fluids after the patient has passed urine **or** until after the Potassium has fallen to within the upper limit of the normal range. NICE guidance specifies a Potassium of 5.5 mmol/l but the working group preferred to maintain the recommendation in its interim guideline of using the upper limit of the local normal range as the threshold. If potassium is low on admission (Potassium < 3.0 mmol) it is recommended that starting insulin is deferred until Potassium is >3.0 mmol. Oral Potassium supplements can be considered where access to a central line to give high concentration parenteral Potassium is likely to be delayed.

9) In patients already on long acting insulin this should be continued and in new patients, consideration should be given to starting long acting subcutaneous insulin alongside intravenous insulin. NICE NG18 update recommends continuing long acting insulin in existing patients but doesn't make any recommendations regarding new patients. The working group maintained its consensus recommendation that consideration be given to starting long acting insulin in newly diagnosed patients with DKA where appropriate. Although there is no clear evidence in newly diagnosed patients it is recommended by ISPAD

Remember: children can die from DKA.

They can die from -

- Cerebral oedema This is unpredictable, occurs more frequently in younger children and newly diagnosed diabetes and has a mortality of around 25%. The causes are not known and evolution of cerebral oedema can be unpredictable. The management of cerebral oedema is covered within the guideline.
- Hypokalaemia This is preventable with careful monitoring and management
- Aspiration pneumonia Use a naso-gastric tube in semi-conscious or unconscious children
- Inadequate resuscitation It is important to ensure that children with DKA receive adequate resuscitation if they are shocked. Inadequate resuscitation is likely to increase the risk of brain injury.Cerebral perfusion is influenced both by the circulatory perfusion pressure (blood pressure) and the intracranial pressure in incipient cerebral oedema.

Guidelines for the Management of Diabetic Ketoacidosis

CONTENTS

A. DIAGNOSIS:

These are general guidelines for management. Treatment may need modification to suit the individual patient and these guidelines do not remove the need for frequent detailed reassessments of the individual child's requirements and specific treatment tailored to those requirements.

Diagnose DKA in children and young people who have

- acidosis (indicated by blood pH below 7.3 or plasma bicarbonate below 15 mmol/litre) and
- ketonaemia (indicated by blood beta-hydroxybutyrate above 3 mmol/litre)

Blood glucose levels are generally high (above 11 mmol/l) but children and young people with known diabetes may develop DKA with normal blood glucose levels.

Children and young people with a pH 7.2- 7.29 &/or bicarb < 15 have MILD DKA

Children and young people with a pH less than 7.1-7.19 &/or bicarb < 10 have MODERATE DKA

Children and young people with a pH less than 7.1 &/or bicarb < 5 have SEVERE DKA

Use a near-patient testing method for blood ketone (beta-hydroxybutyrate) level for the diagnosis and monitoring of the treatment of DKA. If a near-patient testing method is not available, and it is recommended that they should be, use urinary ketonelevels to make the diagnosis, but they are not useful for monitoring. *Urinary ketones of more than ketones++ on standard dipsticks are typically equivalent to near patient blood ketones of >3.0 mmol/l. Urinary ketones must be read **15 seconds** after stick is dipped.*

These guidelines are intended for the management of **children and young people** who have, in addition to the biochemical features above -

- clinical dehydration

They may also have the following clinical features –

- acidotic respiration
- drowsiness
- abdominal pain/nausea/vomiting

Always consult with the consultant paediatrician on call as soon as you suspect DKA even if you feel confident of your management.

IMPORTANT NOTES – PLEASE READ

1. Children who are alert, not clinically dehydrated, not nauseated or vomiting, do not always require IV fluids, even if their ketone levels are high. They usually tolerate oral rehydration and subcutaneous insulin but do require monitoring regularly to ensure that they are improving and their ketone levels are falling.

2. If a child is hyperosmolar with a very high BG level (>33 mmol/l), with little or no acidosis or ketones, this is a **Hyperosmolar Hyperglycaemic State** and requires DIFFERENT treatment. Discuss this with the senior doctor– these children can be very difficult to manage.
There are guidance and starting instructions in Appendix 5. A Hyperosmolar Hyperglycaemic state is commoner in type 2 diabetes but can occur in type 1 diabetes

Discuss both groups of children and young people with the responsible senior paediatrician.

B. EMERGENCY MANAGEMENT IN A & E:

1. General Resuscitation: A, B, C.

Airway Ensure that the airway is patent and if the child is comatose, insert an airway. If consciousness reduced or child has recurrent vomiting, consider inserting N/G tube, aspirate and leave on open drainage.

Seek urgent anaesthetic review and discuss with a paediatric critical care specialist if the child or young person has a reduced level of consciousness and is unable to protect their airway.

Breathing Give 100% oxygen by face-mask.

Circulation Insert IV cannula and take blood samples (see below).
Cardiac monitor for T waves (peaked in hyperkalaemia)
Measure blood pressure and heart rate

Shocked patients require adequate fluid volume resuscitation. A fluid bolus of 10ml/kg should be given if shocked, in line with recent UK Resuscitation Council guidance. Repeated 10ml/kg boluses should be considered if patient remains shocked until they are adequately resuscitated.

2. Initial fluid bolus:

- **All** children and young people with mild, moderate or severe DKA **who are not shocked and are felt to require IV fluids** should receive a **10 ml/kg 0.9% sodium chloride** bolus over 30 minutes. (PlasmaLyte 148 is used by some teams in the UK for initial resuscitation in place of 0.9% sodium chloride but its use was not recommended by NICE due to insufficient evidence)
- Patients with shock require appropriate restoration of their circulation and circulatory volume. **SHOCKED patients** should receive a 10 ml/kg bolus over 15 minutes. Shock is defined by the APLS definition of tachycardia, prolonged central capillary refill, poor peripheral pulses and hypotension (though this is a late sign of shock). It is not just poor peripheral perfusion. Acidosis and hyocapnia can both cause peripheral vasoconstriction. NICE emphasised weak thread pulses and hypotension as an appropriate clinical indicator of shock. The UK Resuscitation Council suggests isotonic crystalloids for the initial treatment of shock (eg Plasmalyte 148 or Ringers lactate) and 0.9% Saline if these are not available.
- Following the initial 10 ml/kg bolus **shocked** patients should be reassessed and further boluses of 10 ml/kg may be given if required to restore adequate circulation up to a total of 40 ml/kg at which stage inotropes should be considered.
- **Shocked patients** will require high dependency care and should be discussed with the most senior paediatrician or intensivist available at the earliest opportunity.
- Whilst excessive fluid should be avoided because of the risk of cerebral oedema it is important to ensure that the circulation is adequate and fluid should be given to support this. Cerebral perfusion is dependant on both perfusion pressure and intracranial pressure and hypotension will exacerbate the risk of brain injury.

3. Initial Investigations:

- Blood glucose
- FBC, Urea and electrolytes (electrolytes on blood gas machine give a guide until accurate results available) and CRP
- Blood gases (venous or capillary)
- Ketones - Near patient blood ketones (beta-hydroxybutyrate) testing should be used.
- If able to obtain sufficient blood, send new diagnosis investigations (HbA1c,TFT, Coeliac screen)

Other investigations should be done only if indicated e.g. CXR, CSF, throat swab, blood cultures, urinalysis, culture and sensitivity etc. (A raised white blood cell count is common in DKA and does not necessarily indicate sepsis).

DKA may be precipitated by sepsis or intercurrent infection, and fever is not part of DKA. Infection may co-exist with DKA. Suspect sepsis if there is fever or hypothermia, hypotension, refractory acidosis or lactic acidosis. A high lactate should increase concern about possible infection or sepsis.

C. FULL CLINICAL ASSESSMENT:

Assess and record in the notes, so that comparisons can be made by others later, the following -

1. Conscious Level -

Institute hourly neurological observations including Glasgow Coma Score (see Appendix 1) whether or not drowsy on admission.

If **reduced conscious level on admission**, or there is any subsequent deterioration,

- seek urgent anaesthetic review if the airway cannot be protected
- discuss with the responsible senior paediatrician
- discuss with a paediatric critical care specialist to decide the appropriate care setting (paediatric HDU or PICU)
- conscious level is directly related to degree of acidosis, but signs of raised intracranial pressure suggest cerebral oedema
- if cerebral oedema is suspected, go to page 8 for details on urgent management.

2. Full Examination - looking particularly for evidence of -

- **cerebral oedema** headache, irritability, slowing pulse, rising blood pressure, reducing conscious level **N.B.** papilloedema is a late sign.
- **infection**
- **ileus** (which is common in DKA)

3. WEIGH THE CHILD. If this is not possible because of the clinical condition, use the most recent clinic weight as a guideline, or an estimated weight from centile charts.

Consider where the child or young person should be nursed –

Children and young people with DKA should be cared for with **one-to-one nursing**, either on a high-dependency unit, (preferably a paediatric unit) or on a general paediatric ward if:

- they are younger than 2 years or
- they have severe DKA (blood pH below 7.1).

If one-to-one nursing cannot be provided on HDU/general paediatric ward, consider transfer to PICU

N.B. Where PICU or HDU do not exist within the admitting hospital, transfer to another hospital for such care (unless ventilatory support becomes necessary) may not be appropriate.

However, ALL children with DKA are high-dependency patients and require a high level of nursing care, even if on general paediatric wards.

D. MANAGEMENT:

1. FLUIDS:

N.B. It is essential that all fluids given are documented carefully, particularly the fluid which is given in the accident and emergency department and on the way to the ward, as this is where most mistakes occur.

The **DKA fluid calculator is available here.** Please note that if you use Internet Explorer (IE), it will take slightly longer to generate the PDF than if used in other web browsers.

a) Volume of fluid -

By this stage, the circulating volume should have been restored. Children with shock should have been adequately resuscitated with appropriate fluid volume replacement. Other patients with DKA will have received an initial fluid bolus of 10ml/kg as part of the routine management of DKA.

Once circulating blood volume has been restored and the child adequately resuscitated, calculate fluid requirements as follows:

Requirement = Deficit + Maintenance

Fluid Deficit

It is not possible to accurately clinically assess the degree of dehydration to work out the deficit. Clinical methods are unreliable. Estimation of the fluid deficit should be based on the **initial** blood pH. The fluid deficit should be replaced over 48 hours alongside maintenance fluids.

Therefore –

Assume a 5% fluid deficit in children and young people in mild DKA (indicated by a blood pH 7.2-7.29 &/or bicarbonate <15)

Assume a 5% fluid deficit in children and young people in moderate DKA (indicated by a blood pH of 7.1- 7.19 &/or bicarbonate <10)

Assume a 10% fluid deficit in children and young people in severe DKA (indicated by a blood pH <7.1 &/or bicarbonate <5)

Resuscitation fluid – The volume of any **fluid boluses** given for **resuscitation** in **children with shock** should NOT be subtracted from the estimated fluid deficit.

The initial **10ml/kg bolus** given to **all non-shocked patients** requiring IV fluids **SHOULD be subtracted** from total calculated fluid deficit.

The **deficit should be replaced over 48 hours** alongside appropriate maintenance fluids

Maintenance fluid

Maintenance fluid volumes should be calculated using the Holliday – Segar formula (the traditional method of calculating fluid volume in children in the UK) – 100 ml/kg/day for the first 10 kg body weight, plus 50 ml/kg/day for 10 to 20 kg and 20 ml/kg/day for each additional kilogram above 20 kg.

Maintenance Fluid:

- 100 ml/kg/day for the first 10 kg of body weight
- 50 ml/kg/day for the next 10 to 20 kg
- 20 ml/kg/day for each additional kilogram above 20 kg

N.B. Neonatal DKA will require special consideration and larger volumes of fluid than those quoted may be required, usually 100-150 ml/kg/24 hours

Fluid Calculation -

Calculate the fluid deficit (either 5% or 10% dehydration depending on whether the patient has mild, moderate or severe DKA), subtract the initial 10ml/kg bolus (unless given for Shock) then divide this over 48 hours and add to the hourly rate of maintenance fluid volume , giving the total volume **evenly** over the next 48 hours. i.e.

Hourly rate = ({Deficit – initial bolus} / 48hr) + Maintenance per hour

Weight

Wherever possible the patient's actual weight on admission should be used rather than an estimated weight or approximation. Maintenance fluids should be based on the actual weight not an estimate of the likely weight following rehydration.

To avoid excessive amounts of fluid in overweight and obese children it is recommended that consideration be given to using a **maximum weight of 75kg or 97th centile weight for age (whichever is lower)** when calculating both deficit and maintenance requirements. Whilst clinical judgement should be used regarding the height and size of the patient, in a 75kg patient with severe DKA the recommendations would suggest a fluid volume over 24 hours in excess of 6.0 litres. This is approaching the fluid volumes recommended for the treatment of adults with DKA so it is suggested that unless the clinical situation indicates otherwise that a maximum weight of 75kg is used in the majority of cases.

Examples:

A 20 kg 6 year old boy who has a pH of 7.15 (Moderate DKA => 5% Dehydrated) will receive a 10ml/kg bolus (200mls fluid) over 30 minutes as part of his initial management. His ongoing fluids will comprise:

Deficit 5 % x 20 kg	=	1000 ml
Subtract initial bolus		1000-200 bolus = 800ml to be replaced over 48 hours
	=	17 ml/hr
Maintenance		10 x 100 = 1000 ml per day for 1st 10 kg
		10 x 50 = 500ml per day for next 10 kg (weighs 20kg)
	=	1500 ml per day total (over 24 hours)
	=	62 ml/hour
Total fluid	=	17ml/hour - Deficit of 5 % (minus bolus) over 48 hours
	+	62 ml/hr – Maintenance fluids
	=	**79 ml/hour**

A 60 kg 15 year old girl with a pH of 6.9 who was shocked at presentation has received 30ml/kg of 0.9% Saline for **resuscitation**. These boluses are **not** subtracted from ongoing maintenance fluids. Her ongoing fluids will comprise:

Deficit 10 % x 60 kg	=	6000 ml to be replaced over 48 hours
	=	125 ml/hr
Maintenance		10 x 100 = 1000 ml per day for 1st 10 kg
		10 x 50 = 500ml per day for next 10 kg (10-20kg)
		40 x 20 = 800ml per day for next 40kg
	=	2300 ml per day total (over 24 hours)
	=	96 ml/hour
Total fluid	=	125 ml/hour - Deficit of 10 % over 48 hours
	+	96 ml/hr – Maintenance fluids
	=	**221 ml/hour**

Do not give additional intravenous fluid to replace urinary losses. Urinary catheterisation should be avoided but may be useful in the child with impaired consciousness.

b) Type of fluid

Use 0.9% sodium chloride with 20 mmol potassium chloride in 500 ml (40 mmol per litre) until blood glucose levels are less than 14 mmol/l (see below section F Continuing Management). PlasmaLyte 148 has been suggested as an alternative as it has a lower chloride content and hyperchloraemic acidosis is therefore less likely. However its use was not supported by the NICE NG18 2020 update due to lack of evidence. NOTE: Additional potassium would need to be added to PlasmaLyte 148 as it only contains 5mmol/l Potassium.

c) Oral Fluids:

- Do not give oral fluids to a child or young person who is receiving intravenous fluids for DKA until ketosis is resolving and there is no nausea of vomiting.
- A nasogastric tube may be necessary in the case of gastric paresis.
- If oral fluids are given before the 48hr rehydration period is completed, the IV infusion needs to be reduced to take account of the oral intake.

d) Fluid Losses:

If a massive diuresis continues for several hours fluid input may need to be increased; this should be isotonic to the urine. However urinary losses should not be routinely replaced. If large volumes of gastric aspirate continue, these will need to be replaced with 0.45% saline with Potassium Chloride.

2. POTASSIUM:

Ensure that all fluids (except any initial boluses given) contain 40 mmol/l potassium chloride, unless there is evidence of renal failure. Hypokalaemia can occur up to 48 hours after starting DKA treatment.

Potassium is mainly an intracellular ion, and there is always depletion of total body potassium although initial plasma levels may be low, normal or even high. Levels in the blood will **fall** once insulin is commenced.

Therefore ensure that **every** 500 ml bag of fluid contains **20 mmol** potassium chloride (**40 mmol per litre**).

Where Potassium is above the upper limit of the normal range at presentation it is recommended that Potassium is only added to Intravenous fluids after the patient has passed urine (to confirm they are not becoming anuric), gives a history of having recently passed urine, **or** after the Potassium has fallen to within the upper limit of the normal range (which it typically will have done after the initial 10ml/kg bolus has been given). NICE recommends that the Potassium should be less than 5.5mmol/l in such circumstances.

If Potassium is low at presentation (<3.0 mmol/l) then insulin administration should be deferred until Potassium is >3.0mmol/l. This may require high concentrations of intravenous Potassium in fluids which would require a central line. Where obtaining central access is likely to result in significant delays to starting insulin then oral Potassium can be considered following discussion with an intensivist to correct hypokalaemia.

If the child or young person with DKA develops hypokalaemia (potassium below 3.0 mmol/litre):

- think about temporarily stopping the insulin infusion
- discuss urgently with a critical care specialist, because a central venous catheter is needed for intravenous administration of potassium solutions above 40 mmol/litre. Oral Potassium can be considered in certain circumstances where there is not ready access to a central line

3. INSULIN:

Once rehydration fluids and potassium are running, blood glucose levels will start to fall. There is some evidence that cerebral oedema is more likely if insulin is started early. Do not give bolus doses of intravenous insulin.

Therefore start an intravenous insulin infusion 1-2 hours after beginning intravenous fluid therapy.

Use pre-filled syringes containing 50 Units of soluble insulin in 50 ml 0.9% sodium chloride where available. If pre-filled syringes are not available, add 50 units of soluble insulin (e.g. Actrapid) to 49.5ml 0.9% sodium chloride.

Insulin rates of 0.05 Units/kg/hr and 0.1 Units/kg/hr are typically suggested. Your local policy may have a particular preference for the dose, but there is no evidence that one dose is superior to the other. An infusion rate of 0.05 Units/kg/hr is likely to be sufficient in most cases, and may have a lower incidence of subsequent hypoglycaemia, though in severe DKA an infusion rate of 0.1 Units/kg/hr may be needed. The recommendation of the BSPED working group was that a starting dose of 0.05 Units/kg/hr should be used unless severe DKA or in adolescents.

Use a soluble insulin infusion at a dosage between 0.05 and 0.1 units/kg/hour.

Other insulin management -

- For children and young people on **continuous subcutaneous insulin infusion (CSII) pump therapy,** stop the pump when starting intravenous insulin.

- For **children who are already on long-acting insulin**, you may wish to continue this at the usual dose and time throughout the DKA treatment, in addition to the IV insulin infusion, in order to

shorten length of stay after recovery from DKA.

- ISPAD guidelines suggest that starting an appropriate dose of long acting background insulin in newly diagnosed patients alongside the intravenous infusion should be considered. The BSPED working group felt this was an issue to be agreed locally and did not feel there was strong evidence or consensus either way.

4. BICARBONATE:

Do not give intravenous sodium bicarbonate to children and young people with DKA. Only consider bicarbonate if there is life threatening hyperkalaemia or in severe acidosis with impaired myocardial contractility. It is anticipated that this would only ever be done following discussion with an Intensivist.

5. RISK OF VENOUS THROMBOSIS:

Be aware that there is a significant risk of femoral vein thrombosis in young and very sick children with DKA who have femoral lines inserted. Line should be in situ as short a time as possible. Thromboembolic prophylaxis should be considered in young people >16 years (in line with NICE guidance), in young women taking the combined oral contraceptive pill and sick patients with femoral lines, following discussion with an Intensive Care Specialist.

E. MONITORING:

a) Nursing Observations –

Ensure full instructions are given to the **senior** nursing staff emphasising the need for:

- strict fluid balance including oral fluids and urine output, using fluid balance charts (urinary catheterisation may be required in young/sick children)
- hourly **capillary blood glucose** measurements (these may be inaccurate with severe dehydration/acidosis but are useful in documenting the trends. Do not rely on any sudden changes but check with a venous laboratory glucose measurement)
- **capillary blood ketone** levels every 1-2 hours
- hourly BP and basic observations
- hourly level of consciousness initially, using the modified Glasgow coma score
- **half-hourly** neurological observations, including level of consciousness (using the modified Glasgow coma score) and heart rate, in children under the age of 2, or in children and young people with a pH less than 7.1, because they are at increased risk of cerebral oedema
- reporting **immediately** to the medical staff, even at night, symptoms of **headache**, or slowing of pulse rate, or any change in either conscious level or behaviour
- reporting any changes in the ECG trace, especially signs of hypokalaemia, including ST-segment depression and prominent U-waves
- twice daily weight; can be helpful in assessing fluid balance

Start recording all results and clinical signs on a flow chart. The new **BSPED DKA Calculator** and the **Integrated Care Pathway** is available on the BSPED website.

b) Medical reviews

At 2 hours after starting treatment, and then at least every 4 hours, carry out and record the results of the following blood tests -

- glucose (laboratory measurement)
- blood gas (for pH and pCO2)
- plasma U&E – **ensure samples are sent URGENTLY to lab**
- finger-prick (near patient) blood ketones

A doctor should carry out a face-to-face review at the start of treatment and then at least every 4 hours, and more frequently if:

- children are aged under 2 years
- they have severe DKA (blood pH below 7.1)
- there are any other reasons for special concern.

At each face-to-face review assess the following:

- clinical status, including vital signs and neurological status
- results of blood investigations
- ECG trace
- cumulative fluid balance record.

Sodium and Corrected Sodium (Na_{corr}) and Effective Osmolality

If the child is becoming hypernatraemic, this is not generally a problem, and is probably protective against cerebral oedema. Hyponatraemia occurs in DKA as with hyperglycaemia the extracellular osmolality rises resulting in water movement from the intracellular space into extracellular space causing dilution of extracellular sodium and a low serum sodium. However when glucose begins to fall through hydration and insulin, and the plasma glucose concentration is reduced, water leaves the extracellular space entering intracellular space raising the extracellular sodium concentration again and the serum sodium typically rises.

It is recommended that the corrected sodium levels are monitored during the management of DKA. The corrected sodium (Na_{corr}) represents the expected serum sodium in the absence of hyperglycaemia.

$$\textbf{Corrected sodium (mmol/L)} = \text{measured } \textbf{sodium} + \frac{(\textbf{glucose} - 5.6)}{3.5}$$

Corrected sodium levels should be calculated on laboratory sodium results not on blood gas results and would typically be monitored every 4 hours when U&Es are checked (see worked example in Appendix 3). Corrected sodium levels should typically rise as blood glucose levels fall during treatment. Some have suggested that corrected sodium levels give an indication of the risk of cerebral oedema with a falling corrected sodium indicating an excess of free water and an increased risk of cerebral oedema.
If corrected sodium levels fall during treatment, discuss with the consultant on call.

Consider adjusting the total fluid rate using corrected Sodium (Na_{corr}) taking into account the circulation and patient's general condition and state of hydration:

- If the rise in Na_{corr} is >5mmol/L in 4-8 hrs it suggests too much fluid loss or insufficient replacement. Consider increasing the fluid rate
- If there is a fall in Na_{corr} by more than 5mmol/L in 4-8 hrs it suggests too much fluid gain or too rapid replacement. Consider reducing the fluid rate

Some authorities suggest that the effective osmolality is a more appropriate marker to monitor than corrected sodium and that management should aim to ensure that the effective osmolality remains stable during treatment of DKA.

$$\textbf{Effective osmolality} = 2 \times \text{Sodium} + \text{Glucose}$$

Please discuss with the consultant on call

Anion gap

If the clinical picture is not improving consideration should be given to calculating the anion gap
The anion gap is typically 20-30 mmol/l in a patient with ketoacidosis. However an anion gap >35 mmol/l may suggest concomitant lactic acidosis due to sepsis or poor perfusion and should prompt a review of the overall clinical picture. It is not required for routine monitoring but may be helpful if the clinical picture or biochemistry is not improving (See Appendix 3)

Hyperchloraemic metabolic acidosis

Hyperchloraemic metabolic acidosis may occur following the administration of large amounts of chloride containing fluids given during the management of DKA. The preferential renal excretion of ketones instead of chloride can result in hyperchloraemia. The acidifying effect of chloride can mask the resolution of ketoacidosis if base deficit alone is used to monitor progress as there may appear to be a continuing base deficit with a continued low bicarbonate due to the chloride component rather than due to ketosis. Direct monitoring of ketones and calculation of the component of the base deficit due to chloride will help differentiate whether persisting acidosis is due to ongoing ketosis that may need additional treatment (adjustment to insulin infusion or fluids) or due to hyperchloraemia. Acidosis due to hyperchloraemia will correct spontaneously and doesn't need specific treatment. Acidosis due to hyperchloraemia need not delay the transition to oral fluids and subcutaneous insulin. It needs differentiating from ongoing ketosis (See Appendix 3)

Phosphate and Hypophosphataemia

Phosphate is lost during DKA due to the osmotic diuresis and serum phosphate is often low in the recovery phase of severe DKA. Supplements or replacement, for example potassium acid phosphate, are not required unless there is severe hypophosphataemia associated with metabolic encephalopathy, reduced myocardial contractility, myopathy, dysphagia or ileus. Clinicians should be aware that administration of phosphate can precipitate hypocalcaemia.

F. CONTINUING MANAGEMENT:

Continue with 0.9% sodium chloride containing 20 mmol potassium chloride in 500ml until blood glucose levels have fallen to 14 mmol/l.

If the blood glucose rises out of control, or the pH level is not improving after 4-6 hours consult senior medical staff and re-evaluate (possible sepsis, insulin dosage errors, blocked or leaking lines, excessive urine loss, fluid calculation error or other conditions), and consider starting the whole protocol again.

If the blood ketone level is not falling within 6–8 hours then get senior help and advice and consider increasing the insulin dosage to 0.1 units/kg/hour or greater.

Once the **blood glucose** has **fallen to 14 mmol/l** add glucose to the fluid and think about the insulin infusion rate, as follows -

- Change the fluid to contain 5% glucose; use 500 ml bags of 0.9% sodium chloride with 5% glucose and 20 mmol potassium chloride in 500ml which are available from Pharmacy (or see Appendix 2)
- Reduce insulin infusion rate to 0.05 units/kg/hr from 0.1 Units/kg/hour (or maintain at that rate if patient initiated on 0.05 units/kg/hr)
- If local policy is to maintain 0.1 units/kg/hour insulin infusion or if a higher dose of insulin is thought necessary then change the fluid to contain 10% glucose rather than 5% glucose, in order to prevent hypoglycaemia when the higher dose of insulin is continued (use 500 ml bags of 0.9% sodium chloride with 10% glucose and 20 mmol potassium chloride in 500mol)
- Once ketones are < 1.0 mmol/l, consider switching from intravenous to subcutaneous insulin

DO NOT stop the insulin infusion while glucose is being infused, as insulin is required to switch off ketone production.

If the blood glucose falls below 6 mmol/l -

- increase the glucose concentration of the intravenous fluid infusion, and
- if there is persisting ketosis, continue to give insulin at a dosage of least 0.05 units/kg/hour

If the blood glucose falls below 4 mmol/l, give a bolus of 2 ml/kg of 10% glucose and increase the glucose concentration of the infusion. Insulin can temporarily be reduced for 1 hour.

If acidosis is not correcting, consider the following

- insufficient insulin to switch off ketones (including incorrectly made insulin infusion)
- inadequate resuscitation
- fluid calculation error
- sepsis
- hyperchloraemic acidosis
- salicylate or other prescription or recreational drugs

Use **near-patient ketone testing** to confirm that ketone levels are falling adequately. If blood ketones are not falling, then check infusion lines, the calculation and dose of insulin and consider giving more insulin.

Consider sepsis, inadequate fluid input and other causes if sufficient insulin is being given.
Once all these causes of acidosis have been excluded, and if ketones are falling gradually, then residual acidosis is likely to be due to hyperchloraemia; this can be left to resolve on its own and does not require any treatment.

G. INSULIN MANAGEMENT ONCE KETOACIDOSIS RESOLVED -

Think about stopping intravenous fluid therapy when ketosis is resolving and oral fluids are tolerated without nausea or vomiting.

Do not change from intravenous insulin to subcutaneous insulin until ketosis is resolving (for example, blood beta-hydroxybutyrate level below 1.0 mmol/litre and the child or young person with DKA is alert and is tolerating fluids without nausea or vomiting.

Start subcutaneous insulin at least 30 minutes before stopping intravenous insulin.

For a child or young person with DKA who is using insulin pump therapy, restart the pump at least 60 minutes before stopping intravenous insulin. Change the insulin cartridge and infusions set, and insert the cannula into a new subcutaneous site.

Subcutaneous insulin should be started according to local protocols for the child with newly diagnosed diabetes, or the child should be started back onto their usual insulin regimen at an appropriate time (discuss with senior staff).

H. CEREBRAL OEDEMA:

Immediately assess a child or young person with DKA for suspected cerebral oedema if they have any of these early manifestations:

- headache
- agitation or irritability
- unexpected fall in heart rate
- increased blood pressure.

If cerebral oedema is suspected in these children or young people, treat immediately with the most readily available of

- hypertonic saline (2.7% or 3% 2.5-5 ml/kg over 10-15 minutes) or
- mannitol (20% 0.5-1 g/kg over 10-15 minutes)

Many intensivists prefer hypertonic saline to mannitol but the key point is that treatment should not be delayed sourcing either – so give what is available.

If a child or young person develops any of these signs –

- deterioration in level of consciousness
- abnormalities of breathing pattern, for example respiratory pauses &/or drop in SaO2.
- oculomotor palsies
- abnormal posturing
- pupillary inequality or dilatation.

treat them **Immediately** for cerebral oedema using the most readily available of

- hypertonic saline (2.7% or 3% 2.5-5 ml/kg over 10-15 minutes) or
- mannitol (20% 0.5-1 g/kg over 10-15 minutes)

In addition fluids should be restricted to ½ maintenance rates and **inform senior staff immediately.**

After starting treatment for cerebral oedema with mannitol or hypertonic saline immediately seek specialist advice on further management, including which care setting would be best for the child or young person.

- do not intubate and ventilate until an experienced doctor is available
- once the child is stable, exclude other diagnoses by CT scan - other intracerebral events may occur (thrombosis, haemorrhage or infarction) and present similarly. Treatment of suspected cerebral oedema should not be delayed through pending imaging.
- The effect of mannitol should be apparent within 15 minutes and typically lasts for 120 minutes. If there is no improvement with mannitol within 30 minutes a repeated dose of mannitol may be given (or hypertonic saline may be preferred). Mannitol may promote a brisk diuresis due to its osmotic effect and renal excretion.
- If mannitol was given initially and there is no response to mannitol treatment within 15-30 minutes then hypertonic saline may be also be given and there is some suggestion that the effect of mannitol and hypertonic saline may be additive.

I. OTHER COMPLICATIONS:

- **Hypoglycaemia and hypokalaemia** – avoid by careful monitoring and adjustment of infusion rates. Consideration should be given to adding more glucose if BG falling quickly even if still above 4 mmol/l.
- **Systemic Infections** – Antibiotics are not given as a routine unless a severe bacterial infection is suspected. Fever, raised lactate and raised inflammatory markers may all indicate possible concomitant infection.
- **Aspiration pneumonia** – avoid by nasogastric tube in vomiting child with impaired consciousness

Other associations with DKA require specific management:

Continuing abdominal pain is common and may be due to liver swelling, gastritis, bladder retention, ileus. However, beware of appendicitis and ask for a surgical opinion once DKA is stable. A raised amylase is common in DKA.

Other problems are pneumothorax ± pneumo-mediastinum, interstitial pulmonary oedema, unusual infections (eg TB, fungal infections), hyperosmolar hyperglycaemic non–ketotic coma, ketosis in type 2 diabetes.

Discuss these with the consultant on-call.

J. EDUCATION AND FOLLOW-UP

After a child or young person with known diabetes has recovered from an episode of DKA, discuss with them and their family members or carers (if appropriate) the factors that may have led to the episode.

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APPENDIX 1 Glasgow Coma Scale

Best Motor Response	1 = none 2 = extensor response to pain 3 = abnormal flexion to pain 4 = withdraws from pain 5 = localises pain 6 = responds to commands
Eye Opening	1 = none 2 = to pain 3 = to speech 4 = spontaneous
Best Verbal Response	1 = none 2 = incomprehensible sounds 3 = inappropriate words 4 = appropriate words but confused 5 = fully orientated

Maximum score 15, minimum score 3

Modification of verbal response score for younger children:

2-5 years	< 2 years
1 = none	1 = none
2 = grunts	2 = grunts
3 = cries or screams	3 = inappropriate crying or unstimulated screaming
4 = monosyllables	4 = cries only
5 = words of any sort	5 = appropriate non-verbal responses (coos, smiles, cries)

APPENDIX 2

How to make up special Intravenous Fluids

The following fluid is generally available from Pharmacy

500ml bag of 0.9% sodium chloride / 5% glucose containing 20 mmol potassium chloride

But this may not be available on every ward. If you need to make it up, please do so as below, rather than waiting for pharmacy.

Glucose 5% & Sodium Chloride 0.9% with 20mmol K in 500ml (if this bag is unavailable in the clinical area) Remove 50ml from a bag of Sodium Chloride 0.9% with 20mmol K in 500ml
Draw up 50ml of Glucose 50% using a syringe and add to the above bag which will make the Glucose concentration 5%
Mix well before administration

The following fluid may not easily available and MUST be made up if required rather than waiting for pharmacy.

Glucose 10% & Sodium Chloride 0.9% with 20mmol K in 500ml
Remove 50ml from a bag of Glucose 5% & Sodium Chloride 0.9% with 20mmol K in 500ml Draw up 50ml of Glucose 50% using a syringe and add to the above bag which will increase the Glucose concentration to 10%
Mix well before administration

Plasmalyte does not contain enough potassium to be used on its own; discuss with pharmacy/PICU before using as maintenance fluid to ensure adequate potassium replacement is provided.

APPENDIX 3
Corrected Sodium, Anion gap, Hyperchloraemic acidosis and partitioning Albumin

Sodium and Corrected Sodium (Na_{corr})

Hyponatraemia occurs in DKA as with hyperglycaemia the extracellular osmolality rises resulting in water movement from the intracellular space into extracellular space causing dilution of extracellular sodium and a low serum sodium. However when glucose begins to fall through hydration and insulin, and the plasma glucose concentration is reduced, water leaves the extracellular space entering intracellular space raising the extracellular sodium concentration again and the serum sodium typically rises. Corrected sodium levels give an indication of the amount of free water in the circulation.

Corrected sodium levels should typically rise as blood glucose levels fall during treatment. It has been suggested that Corrected Sodium levels give an indication of the risk of cerebral oedema with a falling corrected sodium indicating an excess of free water and an increased risk of cerebral oedema.
If corrected sodium levels fall during treatment, discuss with the consultant on call.

The formula for Corrected sodium is:

$$\textbf{Corrected sodium (mmol/L)} = \text{measured } \textbf{sodium} + \frac{(\textbf{glucose} - 5.6)}{3.5}$$

For example:

On admission Serum sodium = 135 mmol/l and blood glucose = 37mmol/l

$$\text{Corrected sodium (mmol/L)} = \text{measured sodium} + \frac{(\text{glucose} - 5.6)}{3.5}$$

$$= 135 + \frac{(37 - 5.6)}{3.5}$$

$$= 135 + \frac{(31.4)}{3.5}$$

$$= 135 + 8.9$$

$$= 143.9$$

On review 4 hours later Serum sodium = 141 mmol/l and blood glucose = 24 mmol/l

$$\text{Corrected sodium (mmol/L)} = \text{measured sodium} + \frac{(\text{glucose} - 5.6)}{3.5}$$

$$= 141 + \frac{(24 - 5.6)}{3.5}$$

$$= 141 + \frac{(18.4)}{3.5}$$

$$= 141 + 5.3$$

$$= 146.3$$

Corrected sodium is rising as expected so no change in management is required

Anion gap

If the clinical picture is not improving consideration should be given to calculating the anion gap
The anion gap is typically 20-30 mmol/l in a patient with ketoacidosis. However an anion gap >35 mmol/l may suggest concomitant lactic acidosis due to sepsis or poor perfusion and should prompt a review of the overall clinical picture. It is not required for routine monitoring but may be helpful if the clinical picture or biochemistry is not improving

Anion gap = Sodium – (Chloride + Bicarbonate)

Hyperchloraemic metabolic acidosis

Hyperchloraemic metabolic acidosis may occur following the administration of large amounts of chloride containing fluids given during the management of DKA. The preferential renal excretion of ketones instead of chloride can result in hyperchloraemia. The acidifying effect of chloride can mask the resolution of ketoacidosis if base deficit alone is used to monitor progress as there may appear to be a continuing base deficit with a continued low bicarbonate due to the chloride component rather than due to ketosis. Direct monitoring of ketones and calculation of the component of the base deficit due to chloride will help differentiate whether persisting acidosis is due to ongoing ketosis that may need additional treatment (adjustment to insulin infusion or fluids) or due to hyperchloraemia. Acidosis due to hyperchloraemia will correct spontaneously and doesn't need specific treatment. Acidosis due to hyperchloraemia need not delay the transition to oral fluids and subcutaneous insulin. It needs differentiating from ongoing ketosis.

Base excess due to Chloride = (Sodium – Chloride) – 32	(ISPAD formula)

For example:

If following intravenous fluids the patient remains acidotic with Sodium = 142 and Chloride = 126 then the component of the apparent base excess attributable to the chloride is calculated as:

Base excess due to Chloride = (Sodium – Chloride) – 32

= (142 - 126) – 32

= (16) – 32

= - 16

Albumin

A low serum albumin can also contribute to a persisting acidosis which may be erroneously attributed to

Base excess component due to **Albumin** = 0.25 x (42 - Albumin)

persisting ketosis. Some intensivists also recommend partitioning the component of apparent acidosis due to the reduced albumin to avoid it being inappropriately attributed to persisting ketosis.

British Society for Paediatric Endocrinology and Diabetes

Overview Algorithm for the Management of Children and Young People under the age of 18 years with Diabetic Ketoacidosis

Version 1 - 2022-01-24

Appendix 4

Clinical History:
- Polyuria/polydipsia
- Weight loss
- Abdominal pain
- Weakness
- Vomiting
- Confusion

Clinical Signs:
- Dehydration
- Kussmaul breathing
- Ketotic smell
- Lethargy, drowsiness

Biochemistry:
- Hyperglycaemia (>11mmol/L)
- Acidaemia (pH<7.3)
- Ketosis (blood ketones >3mmol/L or urine ketones ++)

Confirm diagnosis
DIABETIC KETOACIDOSIS
Call senior staff

pH <7.1 = Severe DKA (10% dehydration
pH <7.2 = Moderate DKA (5% dehydration)
pH <7.3 = Mild DKA (5% dehydration

- Tachycardia
- Prolonged central capillary refill
- Poor peripheral pulses
- Hypotension (late sign)

Is the patient shocked?

Yes

Resuscitation

Airway +/- NG tube
Breathing 100% O_2
Circulation
- 10mL/kg fluid*
- Repeat until circulation restored
- By 40mL/kg discuss with senior doctor and consider inotropes

No

Slow Bolus
- 10mL/kg fluid* bolus over 30 min

Intravenous therapy
- Calculate fluid requirements: **dka-calculator.co.uk**
- Use fluid* with 40 mmol/L potassium (check serum K^+ in normal range and urine output first)
- Start insulin at 0.05 or 0.1 Units/kg/hour 1-2 hours after starting fluids

Signs of cerebral oedema:
- Headache, irritability
- Slowing HR
- Reduced GCS / coma
- Signs of raised ICP
- Others as show on care pathway

Observations
- Hourly blood glucose - 1-2 hourly blood ketones
- Hourly neuro obs and fluid balance
- Check electrolytes at 2 hours, then 4 hourly

Acidosis failing to improve?

Features of cerebral oedema?

Management of Persisting Acidosis
- Re-evalutate fluid balance - may require further resus fluid
- Check insulin rate and running properly
- Consider sepsis and other differentials as per care pathway
- Consider restarting protocol

Management of Cerebral Oedema
- Give 5mL/kg 2.7% Sodium Chloride **OR** 20% Mannitol 2.5 - 5 mL/kg
- Call senior staff
- Restrict IV fluids by 50%
- Refer to care pathway for further actions

Blood glucose <14mmol/L

- Change fluids* to contain 5% glucose
- Continue monitoring as above

* 0.9% Sodium Chloride or Plasmalyte 148

Blood glucose <6mmol/L

Management of Falling Blood Glucose
- Change fluids* to contain 10% glucose
- Do not reduce insulin below 0.05 Units/kg/hour if ketones >1 mmol/L
- If glucose falls below 4mmol/L refer to care pathway for management of hypoglycaemia

Resolution of DKA
- Clinically well, tolerating oral fluids, blood ketones <1mmol/L or pH normal
- Start S/C insulin **THEN** stop IV insulin 1 hour later

This algorithm is a summary of the main care pathway and should not be considered as a complete guide to the management of paediatric DKA.

Refer to the main care pathway at the earliest opportunity by visiting **dka-calculator.co.uk** or the BSPED guidelines page.

Appendix 5

Initial management of Hyperosmolar Hyperglycaemic State (HHS)

Features which differentiate it from other hyperglycaemic states such as DKA are:

- Hypovolaemia
- Marked hyperglycaemia (Glucose >33.0 mmol/L or more)
- No significant hyperketonaemia (<3.0 mmol/L) or acidosis (Arterial pH>7.3, Venous pH . 7.25 bicarbonate >15 mmol/L)
- Osmolality usually 320 mosmol/kg or more
- Often altered consciousness

This picture usually occurs in Type 2 diabetes, especially where there are learning difficulties or other factors preventing proper hydration. It has a high mortality rate. It can occur in type 1 diabetes. Where DKA and HHS co-exist treatment of DKA takes priority and treatment should be initiated as for DKA.

Goals of treatment

The goals of treatment of HHS are to treat the underlying cause and to gradually and safely:

- Normalise the osmolality
- Replace fluid and electrolyte losses
- Normalise blood glucose

Other goals include prevention of arterial or venous thrombosis and other potential complications e.g. cerebral oedema/ central pontine myelinolysis

Fluid therapy

The goal of initial fluid therapy is to expand the intra and extravascular volume and restore normal renal perfusion. The rate of fluid replacement should be **more rapid** than is recommended for DKA.

- Give an initial bolus should be of 20 mL/kg of isotonic saline (0.9% NaCl)
- Assume a fluid deficit of approximately 12–15% of body weight.
- Additional fluid boluses should be given, if necessary, to restore peripheral perfusion.
- Thereafter, 0.45–0.75% NaCl with potassium should be administered to replace the deficit over 24–48 hours.
- The goal is to promote a gradual decline in serum sodium concentration and osmolality.
- As isotonic fluids are more effective in maintaining circulatory volume, isotonic saline should be restarted if perfusion and hemodynamic status appear inadequate as serum osmolality declines.
- Serum sodium concentrations should be measured frequently and the sodium concentration in fluids adjusted to promote a gradual decline in corrected serum sodium concentration.
- Mortality has been associated with failure of the corrected serum sodium concentration to decline with treatment, which may be an indication for haemodialysis.
- Although there are no data to indicate an optimal rate of decline in serum sodium, 0.5 mmol/L per hour has been recommended for hypernatraemic dehydration.

If there is a continued rapid fall in serum glucose (>5 mmol/l per hour) after the first few hours, consider adding 2.5 or 5% glucose to the rehydration fluid. Failure of the expected decrease of plasma glucose concentration should prompt reassessment and evaluation of renal function.

Unlike treatment of DKA, replacement of urinary losses is recommended. The typical urine sodium concentration during an osmotic diuresis approximates 0.45% saline; however, when there is concern about the adequacy of circulatory volume, urinary losses may be replaced with a fluid containing a higher sodium concentration.

Insulin therapy

- Blood glucose levels will fall with fluid alone and insulin is NOT required early in treatment.
- Insulin administration should be initiated when serum glucose concentration is no longer declining at a rate of at least 3 mmol/l per hour with fluid administration alone.

Potassium

Patients with HHS also have extreme potassium deficits; a rapid insulin-induced shift of potassium to the intracellular space can trigger an arrhythmia. Therefore Potassium MUST be included in all fluids.

For further information see ISPAD Guidelines