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23 years of managing diabetic ketoacidosis at Auckland Hospital

Geoffrey Braatvedt, Amelia Tekiteki, Holly Britton, John Wallace, Manish Khanolkar

**ABSTRACT**

**AIMS:** To examine the length of stay and need for intensive care of people admitted with diabetic ketoacidosis (DKA) to a single centre between 1988 and 2011.

**METHODS:** Patients aged ≥15 years admitted for the first time with DKA (plasma glucose ≥ 10mmol/L and a bicarbonate concentration ≤15mmol/L and a pH <7.35, and raised plasma or urine ketones or anion gap) to Auckland City Hospital from 1988–2011 were identified retrospectively. The patients were divided into four cohorts (1988–1996; 1997–2001; 2002–2006; 2007–2011). Over this time period there was no significant change to the insulin infusion protocol.

**RESULTS:** There were 576 admissions with DKA in 388 people over the 23 years. The mean age of the patients and glucose concentration at presentation to hospital fell significantly over time. The admission pH and bicarbonate concentration was higher in more recent cohorts. The length of stay and need for intensive care admission fell significantly over time, but the number of patients subsequently readmitted with DKA remained high. In-hospital mortality remained low.

**CONCLUSIONS:** DKA remains an important reason for admission to this hospital, but the severity of DKA at presentation has reduced over time. The need for intensive care admission and length of stay has fallen dramatically.

Diabetic ketoacidosis (DKA) remains a life-threatening, complex metabolic disorder complicating diabetes, and is a common cause for admission to acute medical units. A previous study of 125 people admitted with DKA between 1988–1996 to Auckland Hospital,\(^1\) reported that the patients had a mean length of stay (LOS) of about a week, and nearly a third were admitted to the intensive care unit. In-hospital mortality was relatively low, with 2.4% of patients dying during their index hospital admission. However, 25% of these patients had a readmission with DKA during the audit period.

The protocol used to manage DKA in this hospital has not substantially changed since that time and remains “glucose-centric” (Appendix 1). It was previously available only as a hard copy sheet but available in all ward areas. In 2000, Auckland Hospital published its own Resident Medical Officer Handbook online and in hard copy (and more recently available to staff as a phone app) detailing the management guidelines for a large variety of medical conditions including DKA. Hospital policy encouraged the use of the DKA protocol wherever possible, and the protocols were made widely available through the hospital intranet and by hard copy in the Emergency Department and on all medical wards. The protocol has always been printed on green double-sided A4 paper, which also serves as a prescription for insulin and fluids, as well as records the insulin infusion delivered and the blood glucose results. If the patient’s hourly measured capillary glucose levels are not falling, the rate of insulin infused per hour is rapidly escalated by protocol and on a varying scale (with separate scales for patients with known severe renal impairment). For most patients, scale “B” or “C” is used initially, and for a patient with significant hyperglycaemia at presentation, the dose of insulin infused per hour is about 6–8 units—this approximates the new UK guidelines\(^2\) of 0.1 unit/kg/hour for...
a person of average weight. The insulin infusion continues to be infused alone until the capillary glucose falls to <15mmol/L, when insulin is continued and 10% dextrose is added at 80ml per hour (lower rates on renal protocol). Fluids containing varying concentrations of potassium are also infused by protocol according to the patient’s renal function and potassium concentrations, which are measured frequently. Once the patient is eating and drinking and glucose values are stable, subcutaneous insulin is commenced and the insulin infusion is weaned to stop. The protocol recommends frequent measurement of venous bicarbonate, potassium and pH, but does not stipulate repeat measures of plasma beta-hydroxybutyrate. This glucose-centric protocol has remained in use in this hospital over the past 23 years.

In 2011, the Joint British Society Guideline for the Management of DKA was published with a major shift away from a glucose-centric protocol to a ketone-centric one, with the recommendation to commence a weight-based fixed dose of insulin infusion until ketonaemia (measured at the bedside) is cleared. Most NHS trusts in the UK have now adopted this protocol, and a similar shift has occurred in some New Zealand Hospitals. A recent UK audit of the use of the new protocol in managing 50 people presenting with DKA reported a median LOS of 2 and mean 3.3 days.

The aim of the current study is to describe our experience in the management of DKA over a 23-year time frame, during which the management protocol has become more widely available and adopted, but has remained glucose-centric so as to help determine if adopting the new UK ketone-centric protocol should be considered.

Patients and methods
Auckland City Hospital serves the central Auckland population (currently approximately 400,000 people), and the Department of General Medicine has approximately 12,000 medical discharges per year. Children <15 years of age are admitted to a separate specialist children’s hospital. All patients discharged from Auckland Hospital are coded using standard internationally agreed codes. Patient records have been prospectively stored in electronic format only for many years, and as patients get admitted or readmitted, any past stored paper records are scanned to an electronic format, which enables review of patient records remotely. All laboratory results have been available electronically from the late 1990s. Every New Zealand citizen has a unique national patient identifying number, allowing for accurate searching of their health records.

Patients presenting with DKA are assessed either in the emergency department and then referred to general medicine or can be referred directly to general medicine if the referring Doctor has discussed the admission with the general medical registrar on call. The “team of the day” looks after the patients (there were a total of eight General Medicine teams consisting of House Officer, Registrar and Consultant Physician until 2000 when the number of teams was increased to 12), and the diabetes service is only involved in their inpatient care by referral. Unless the patients require intensive care, they are looked after in a general medical ward. A new hospital on the same site as the old one was opened in 2005 (1,200 beds) with a bigger emergency floor space, with an additional acute assessment area having 65 beds available for 24-hour care before transfer up to the medical wards. Both the emergency and acute assessment units are co-located on the same floor, with excellent lines of communication between the two departments. Patients admitted to the emergency department are expected to be referred to an appropriate service for ongoing inpatient care or be discharged within six hours. General medicine therefore is involved in DKA management early in the patient’s admission.

Data from the previously published 1988–1996 cohort has been compared with three subsequent five-year cohorts to 2011 (1997–2001, 2002–2006, 2007–2011). All people aged ≥15 years admitted to Auckland Hospital with DKA were identified using the hospital discharge codes. While all admissions with DKA during the time period were recorded, only the first admission of that person with DKA during the study period (the index admission) was analysed and used in comparative studies, but the number and
Timing of any subsequent readmissions with DKA was recorded. We continued to use the same definition of DKA as we had in the initial study—venous glucose ≥10mmol/L and arterial pH <7.35 and bicarbonate ≤15mmol/L and raised venous/urine ketones or raised anion gap. The electronic records of each person’s admission were reviewed and data extracted. One way ANOVA, t-tests and χ² tests were used to compare differences between the time periods. Local ethics committee approval for the study was granted.

Results

Over the 23-year period, a total of 576 DKA admissions in 388 patients met the study criteria. For the cohort 1997–2011, 516 patients were identified from coding as having had DKA, and 263 met our inclusion criteria.

Table 1 details the demography of the index admission of each patient. It can be seen that the mean age at presentation with DKA is lower in recent times and ethnic diversity is greater. The age of the patient, glucose and pH at admission with DKA, and LOS was no different between patients of New Zealand European origin compared with all other ethnicities (data not shown).

Table 2 details admission metabolic parameters and LOS. Patients admitted in the more recent cohorts have had lower admission blood glucose and a higher pH and bicarbonate concentration reflecting less severe ketoacidosis. The number of patients admitted to the intensive care unit (ICU) has fallen dramatically, and LOS has fallen by around 50%. The mortality rate in hospital has remained low, and in the most recent cohort was <1%. However, the readmission

Table 1: Details of the first admission of people admitted with DKA to Auckland City Hospital over 23 years by cohort (n=388 people).

<table>
<thead>
<tr>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>125</td>
<td>62</td>
<td>81</td>
<td>120</td>
<td></td>
</tr>
<tr>
<td>Age (yr.)</td>
<td>42 ± 16</td>
<td>38 ± 18</td>
<td>35 ± 17</td>
<td>31 ± 11</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Duration known diabetes (yr.)</td>
<td>11.6 ± 11.4</td>
<td>11 ± 10</td>
<td>9.4 ± 11</td>
<td>10.6 ± 12</td>
<td></td>
</tr>
<tr>
<td>Female (%)</td>
<td>n/a</td>
<td>56</td>
<td>52</td>
<td>48</td>
<td></td>
</tr>
<tr>
<td>European (%)</td>
<td>81</td>
<td>56</td>
<td>57</td>
<td>64</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Māori (%)</td>
<td>15*</td>
<td>16</td>
<td>14</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Pacific (%)</td>
<td>18</td>
<td>14</td>
<td>7</td>
<td>10</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Other (%)</td>
<td>4</td>
<td>11</td>
<td>16</td>
<td>18</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

p<0.05 group over time. *This includes Māori and Pacific for the first cohort. Details of the cohort 1988–1996 have previously been published.1 Other = Asian/Indian. Data are mean ±SD.

Table 2: Metabolic parameters, length of stay (LOS) and number (%) cared for in the intensive care unit (ICU) for 388 people admitted with diabetic ketoacidosis to Auckland City Hospital.

<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma glucose (mmol/L)</td>
<td>43 ± 21</td>
<td>34 ± 15</td>
<td>33 ± 15</td>
<td>31 ± 11</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>pH</td>
<td>7.12 ± 0.12</td>
<td>7.16 ± 0.18</td>
<td>7.15 ± 0.15</td>
<td>7.18 ± 0.14</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Bicarbonate (mmol/L)</td>
<td>n/a</td>
<td>10.6 ± 7.2</td>
<td>9.1 ± 5.2</td>
<td>13.4 ± 6</td>
<td></td>
</tr>
<tr>
<td>LOS (days)</td>
<td>6.6 ± 4</td>
<td>5.94 ± 6.6</td>
<td>4.8 ± 5.0</td>
<td>3.4 ±3.6</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LOS median/ range(days)</td>
<td>4/0–35</td>
<td>4/0–35</td>
<td>2/0–29</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICU (%)</td>
<td>30.4</td>
<td>24</td>
<td>22</td>
<td>9</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Readmitted* with DKA (%)</td>
<td>25</td>
<td>28</td>
<td>23</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>In hospital mortality (%)</td>
<td>2.4</td>
<td>1.6</td>
<td>1.2</td>
<td>0.9</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

Details of the cohort 1988–1996 have been previously published.1 p<0.05 group over time. Data are mean ±SD. N/A = not available. *within five years.
rate with DKA has remained high at between 15–28% during the audit period following the index admission.

To investigate the influence of severity of DKA on LOS and ICU admission rates, two further analyses were done. Individual data from the 1988–1996 cohort was no longer available, and thus patient details from the three subsequent cohorts were examined. Table 3 presents LOS and ICU admission data of patients with severe DKA (pH≤7.1 and/or bicarbonate ≤5mmol/l) by cohort and demonstrates that patients with similar DKA severity had a (non-significant) shorter LOS and fewer requirements for ICU admission over time.

Table 4 presents data on all patients for 1997–2011 divided by tertile of admission pH, and demonstrates a shorter LOS and less need for ICU admission in those with highest pH on admission.

The number of patients admitted with DKA per 100,000 population aged >15 in the hospital catchment area did not change over the study period (1988–1996 8.4/100,000; 1997–2001 6.8/100,000; 2002–2006 8.1/100,000; 2007–2011 9.0/100,000).

Discussion
This report details one hospital’s experience in the management of DKA over a 23-year time frame, during which time the management protocol has remained glucose-centric (and essentially unchanged, including no change in the hard copy) and under the care of the general medicine “team of the day”. The insulin infusion protocol has however become much more readily accessible since 2000, and the size and staffing of the emergency and acute assessment floor has improved significantly since the new hospital was opened in 2005. The study has shown that in-hospital mortality has remained low, and compares highly favourably with other reports, which showed a mortality of 3.9% for admissions between 1971–1991, and 1.8% for admissions between 2000–2009. The LOS has dramatically fallen from nearly a week to under four days, and the number of patients admitted to the intensive care unit has reduced two-fold. Similar trends in reduced LOS for other general medicine patient admissions have also been observed. The high readmission rate remains a concern.

Previous studies have shown that following protocols of care for complex disorders such as DKA improves outcome. Over the past 40 years many different protocols and guidelines for the management of DKA have been published. All agree that relatively low-dose IV insulin infusion with vigorous fluid resuscitation and close monitoring of patients is important. Recent studies from the UK have however shown that standard protocols for the management

<table>
<thead>
<tr>
<th>pH tertile</th>
<th>Number of patients</th>
<th>ICU (%)</th>
<th>LOS (days)</th>
<th>LOS range (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>6.7–6.92</td>
<td>34</td>
<td>67</td>
<td>9.4 ± 10.6</td>
<td>1–35</td>
</tr>
<tr>
<td>6.93–7.15</td>
<td>121</td>
<td>20</td>
<td>4.2 ± 3.1</td>
<td>1–18</td>
</tr>
<tr>
<td>7.16–7.35</td>
<td>233</td>
<td>6.3*</td>
<td>3.7 ± 3.7*</td>
<td>1–29</td>
</tr>
</tbody>
</table>

ICU (%) = % of patients admitted to intensive care unit *=p<0.01 across tertiles.
of DKA are in fact poorly followed. The recent UK Guidelines\(^2\) have recommended that emphasis should shift away from glucose concentration-driven protocols to ketone and pH-driven considerations using frequent bedside ketone and glucose testing to inform when insulin infusions can be safely changed to subcutaneous insulin. While the protocol may have merit, we are not aware of large and robust RCT evidence that this has advantages for patient outcomes that matter—mortality, LOS and early readmission with DKA due to a failed discharge. Our own experience has shown that the widespread use of a single protocol is probably the main contributor to better outcomes over time, although the patients in more recent years appear to have presented with less severe DKA than in the earlier years, perhaps reflecting the widespread use of modern analogue insulins in the community, better education of patients and other factors. Furthermore, patient’s ability to better self-monitor glucose and ketones could have led to an earlier discharge in more recent years without the need to ensure full metabolic clearance of ketones in a hospital setting.

Although the number of patients available for comparison has reduced statistical power, the LOS (and need for ICU admission) of patients with severe DKA (Table 3) has shown a progressive reduction over time suggesting that improvements in initial care, perhaps in the Emergency and Acute Assessment Departments, has led to the fall in length of stay observed in the whole cohort, as no changes to the protocol were made during that time. For the most recent three five-year cohorts, stratifying patients by tertiles of pH (Table 4) does show a reduction in LOS for those with the highest tertile of pH compared with patients admitted with the lowest pH tertile, suggesting this could be one of the reasons why the length of stay has reduced over time as patients in recent years had less severe DKA. We did not specifically analyse how well the protocol was followed over the study period, but note it has become more widely available since 2000 when it was more formally published in a handbook, and particularly in recent years with the availability of the protocol electronically, on smart phones, intranet and iPads.

The reduction in admissions to intensive care over the study period is striking. This could reflect less severe presenting acidosis in recent times, better immediate care in the emergency room and acute assessment area and an ability to manage acutely unwell patients in these well-staffed and fully monitored areas independent of whatever insulin infusion protocol is in place. Recent UK guidelines recommend HDU–ICU admission for patients meeting a number of criteria including a bicarbonate <5 or pH <7.1.\(^2\) The number of patients fulfilling these criteria over the three recent five-year cohorts was 31%, 34% and 22% respectively. However, the number of patients admitted to ICU in each time frame has reduced from 45% to 33% to 23% respectively (Table 3). Interestingly, a recent\(^1\) large study of DKA admissions to New Zealand and Australian ICUs between 2000–2013 showed that the incidence of DKA admission has increased as a proportion of other reasons for admission to ICU, but the pH and bicarbonate has increased and glucose decreased consistent with our study, showing that patients are presenting to hospital with less severe DKA. The LOS in the ICU rose over time, but the overall LOS in hospital fell. Twenty-seven percent of patients were not on established insulin on admission to ICU, suggesting a large number of these patients had DKA in the context of type 2 diabetes. Our own study has also shown a rising prevalence of DKA in non-New Zealand Europeans, also suggesting that DKA is not an uncommon complication of type 2 diabetes. Although we did not record the presence of co-morbidities in our cohorts with DKA, the age, metabolic parameters and LOS of patients of New Zealand ethnicity was no different from those patients of other ethnicities, confirming the increasing evidence of an earlier age of presentation with type 2 diabetes in non-New Zealand Europeans.

The number of patients readmitted with DKA over the subsequent five years is of concern. Although the apparent reduction in readmission rate in the most recent cohort to 15% could reflect a real reduction in readmission rates, it could also reflect the fact that by definition this cohort has only been followed for a maximum of five years, with some patients near the close-out time point of the audit having been followed only for a
few weeks to months. Potential reasons for 
the high readmission rate in these cohorts 
have been reported in a separate study.16 

Study limitations include that this was a 
retrospective review and based on discharge 
codes to identify patients. It is likely that 
a number of episodes of DKA were incor-
rectly coded, and thus the number of 
patients admitted with DKA is not accurate. 
However, the discharge codes have not 
changed over the years, and thus case 
ascertainment is likely to have been equal 
(good or poor) across the time periods. The 
number of patients admitted with DKA per 
100,000 of population did not change over 
time, despite the known rising incidence 
of type 1 diabetes in New Zealand. This 
could suggest that case ascertainment was 
worse in recent cohorts, but equally that 
the incidence of DKA in patients with type 
1 diabetes has fallen. Most patient details 
were available through the electronic 
record, which also allowed the opportunity 
to double audit patients with any queries 
identified from a first review. We also did 
ot review whether over time the protocol 
was followed more rigorously or not, nor 
how much fluid was infused, as our main 
focus was to examine trends in patient 
outcomes over time. The LOS was recorded 
in “days” and not hours, and thus patients 
with a stay of <24 hours could have a LOS of 
“0” days. A recent study17 from Christchurch 
has shown that many patients with DKA 
only need an overnight admission. Finally 
a new hospital was opened in 2005 with 
much better emergency room and acute 
assessment area integration and commu-
nication, which may well have contributed 
to better patient outcomes independent of 
what protocol was in place. We do however 
have a unique opportunity to investigate 
this further, as North Shore City Hospital in 
Auckland, which serves a similar number of 
patients with the same acuity as Auckland 
Hospital, uses the same pool of medical 
registrars in training rotating through both 
hospitals and opened a new emergency 
department in 2010, did change its protocol 
of care for DKA to the UK guidelines in 2012. 
We are thus currently comparing outcomes 
in LOS, need for intensive care, rates of 
hypoglycaemia and time to resolution of 
acidosis in the cohorts admitted with DKA in 
2013 to each hospital.

The ethnic origin of the people admitted 
with DKA has changed substantially over 
the study period, with many more patients 
of Māori, Pacific and other ethnic origin 
admitted in later cohorts (Table 1). Type 1 
diabetes in these communities is relatively 
rare,18 and thus many of these patients are 
likely to have had type 2 diabetes. The inci-
dence of type 2 diabetes in Māori and Pacific 
people in Auckland is increasing, and the 
age at presentation is much younger than in 
people of New Zealand European origin.19, 20

Conclusion

In conclusion, our retrospective cohort 
study has described the outcome of patients 
with DKA over a long period of time, and 
has shown that DKA remains a common 
reason for presentation to our medical unit, 
but that the in-hospital mortality is low, and 
the length of stay and number of patients 
admitted to intensive care has fallen dramat-
ically over time. The readmission rate for 
DKA, however, remains high. During this 
time the insulin infusion protocol has not 
changed, and suggests therefore that the 
improvements shown are likely systems 
of care improvements independent of 
what protocol is used. Whether a different 
protocol of care such as the UK ketone-
centric one, results in even better patient 
outcomes is the subject of another study 
comparing patients admitted with DKA in 
two neighbouring hospitals in Auckland that 
use different insulin infusion protocols.
REFERENCES:


17. Yong KW, Moore MP, Lunt H. Medically facilitated discharge of adult...
Appendix

Suggested medical management DKA

1. Admit under the team of the day.
2. Consider resuscitation status.
3. Look for underlying illness, eg Sepsis, MI.
4. Nil by mouth if vomiting or reduced level of consciousness for the first 12h at least.
5. Vital signs q2h (may need q1h initially) for 8h then q4h until stable.
6. Capillary blood glucose q1h until off insulin infusion.
7. Urine output q1h initially; be concerned if less than 30 mL/h.
8. Consider intensive care referral if reduced level of consciousness, BP <90 systolic, pH <7.2 or renal impairment.
9. Contact nursing supervisor early: these patients often require one-on-one nursing for the first 8–12h.

Investigations/management:

- Electrolytes, creatinine, FBC.
- ABG at presentation plus beta hydroxybutyrate. Thereafter venous bicarbonate is usually adequate to monitor progress.
- Culture of blood, urine and other clinically indicated sites.
- CXR.
- Nasogastric tube if vomiting (ileus is common in DKA).
- ECG.
- Troponin T if >30y and no obvious alternative precipitant or ECG abnormal.
- Repeat electrolytes at least q2h until stable especially watching for hypokalaemia.
- Use a flowchart to record fluid balance, pH or bicarbonate and electrolytes.

Fluids and insulin:

- The first litre of hydrating solution should be sodium chloride 0.9% given as quickly as possible in the first hour and followed by 500–1000mL/h of sodium chloride 0.45% of 0.9% (depending on the state of hydration and serum sodium) during the next 2h.
- The type and rate of continued fluid replacement will depend on assessment of clinical and biochemical factors.
- If hypernatraemic (>146mmol/L) consider sodium chloride 0.45%.
- Repeat electrolytes regularly as above. K+ may be required in large amounts (often >20mmol/L). Do not begin to replace until K+ <5.0mmol/L and urine output >30mL/h. When K+ <5.0mmol/L begin replacement at 20mmol/hour—don’t wait until K+ is low.
- Insulin: do not strive for rapid control as glucose will often fall significantly with rehydration alone. Commence an IV insulin infusion according to Table 1.
- Start with Scale B as it is the most commonly appropriate and should be the initial default scale.
If blood glucose persistently below 5mmol/L, ie on two or more tests one hour apart, move one scale to the left and/or ask for advice.

If blood glucose persistently high, ie on two or more tests one hour apart, check pump for correct rate and line for patency, then move one scale to the right and/or ask for advice.

Once glucose is <15mmol/L on two consecutive tests one hour apart, introduce 10% dextrose at 80mL/hr.

GIK Infusions
GIK is an insulin infusion run in conjunction with an infusion of 500ml of glucose 10% with 10mmol of potassium chloride. This combination is suitable for most patients (unless the patient is sodium depleted or is a renal patient).

The insulin infusion should be prescribed on the Insulin Chart. Scale B (see table above) is most commonly appropriate and should be in the initial default scale. The insulin infusion should be prepared in a 50ml syringe by mixing 50 units Actrapid® in 49.6ml sodium chloride 0.9% (to make a 1unit/ml solution). This should be administered using a syringe pump.

The glucose 10% infusion with 10mmol of potassium chloride should be prescribed on the fluid balance chart and given at a rate of 80ml/h via a volumetric pump. This regimen is designed to control diabetes. Other problems such as fluid depletion, other electrolyte imbalance etc. should be managed through separate infusions.

Discontinuing a GIK
An insulin infusion is best discontinued 2–4 hours after a meal. The usual pre-meal dose of insulin is given, and the infusion should be continued for around a further 2 hours if Humalog® or Novorapid® is used or 4 hours if Actrapid® or Humulin R® is used. This overlap allows time for the subcutaneous insulin to reach peak concentration and prevent hyperglycaemia.

Patients with type 1 diabetes should also have their intermediate/long-acting subcutaneous insulin restarted or initiated. (Protaphane® or Humulin NPH® or Glargine). This is especially important if a rapidly acting analogue is used. The patient's usual dose of insulin should be prescribed.

Patients with DKA not previously known to have diabetes should be started initially on a twice daily dose of intermediate acting insulin (eg Protaphane® or Humulin NPH®) at a dose of 0.2–0.4 units/kg/day and specialist referral made. Short-acting insulin will be added when they are stable and ambulant.

Diabetologist/diabetic education referral (early). DKA is usually a failure of education/self-care.

### Table 1:

<table>
<thead>
<tr>
<th>Scale</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Thin small individuals, especially women: some very sensitive normal (total daily dose &lt;30units/day); athletes; hypopituitary, hypoadrenal and hypothyroid patients; post-pancreatectomy patients (surgical or functional); some well controlled non-obese diet controlled type 2 diabetics.</td>
</tr>
<tr>
<td>B</td>
<td>Individuals (type 1 or type 2) with no special circumstances; use if normal daily insulin requirements approximately 30–80units/day.</td>
</tr>
<tr>
<td>C</td>
<td>Seriously ill (high fever etc.); moderate steroid doses (up to 20mg/daily); very obese or insulin resistant patients (&gt;80units/day); uncomplicated post-infarct patients.</td>
</tr>
<tr>
<td>D</td>
<td>High steroid doses (&gt;20mg daily); very stressed individuals; complicated post-infarct patients.</td>
</tr>
<tr>
<td>E</td>
<td>Major cardiac surgery (never use this scale without specialist physician/anaesthetic advice).</td>
</tr>
</tbody>
</table>

### Table 2:

<table>
<thead>
<tr>
<th>Blood glucose</th>
<th>Scale A</th>
<th>Scale B</th>
<th>Scale C</th>
<th>Scale D</th>
<th>Scale E</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;5.0mmol/L</td>
<td>Stop/consult</td>
<td>0.5–1 unit/h or Scale A</td>
<td>2 units/h or Scale B</td>
<td>4 units/h or Scale C</td>
<td>8 units/h or Scale D</td>
</tr>
<tr>
<td>5.0–11.0mmol/L</td>
<td>1 unit/h</td>
<td>3 units/h</td>
<td>4 units/h</td>
<td>8 units/h</td>
<td>16 units/h</td>
</tr>
<tr>
<td>11.1–17mmol/L</td>
<td>3 units/h</td>
<td>6 units/h</td>
<td>8 units/h</td>
<td>16 units/h</td>
<td>32 units/h</td>
</tr>
<tr>
<td>&gt;17mmol/L</td>
<td>6 units/h or Scale B</td>
<td>12 units/h or Scale C</td>
<td>16 units/h or Scale D</td>
<td>32 units/h or Scale E</td>
<td>64 units/h</td>
</tr>
</tbody>
</table>