The Management of Hospital In-patients with Diabetes Mellitus

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Consultant in Diabetes and Endocrinology, NNUH
Honorary Reader in Medicine, UEA
Before I Forget

• Thank you to
  – Prof Mike Sampson, Prof Gerry Rayman, Prof Jeremy Turner
  – Catherine Gooday and Rachel Murchison et al
  – Chris Jones, Nick Levy, Guillermo Umpierrez and the JBDS
  – Esther Walden and the nursing team in the diabetes clinic, NNUH
  – The multitude of medical students
    • Alec Beaney, Coral Stark, Harriet Daultrey, Thomas Murray, Zahra Essackajee, Elizabeth Swan, Edwin Li Ping Wah-Pun Sin, Francesca Li, Joyce Cheng, Anson Yue, Will Fry, Sean McCafferty, David Maxey, Nishchay Kakkar, Meera Patel, and Maithili Varadarajan, etc
Who is This Strange Man?

• I qualified in 1991
• I trained in Diabetes & Endocrinology and General (Internal) Medicine
• I worked in general practice for 2 years
• I worked in ITU / anaesthetics for a year
• I researched at the Mayo Clinic (DHEA anyone?)
• I have been in Norwich since 2004 – Hon SL since 2004, Hon Reader since 2017
• Current / former national roles are
  – Currently Honorary Secretary of the Diabetes and Endocrinology Section of the Royal Society of Medicine
  – Previously Executive Officer of the Association of British Clinical Diabetologists (meetings secretary)
  – Currently Chair of the Specialist Clinical Exam in Diabetes and Endocrinology (MRCP (D&E) – the UK ‘Board exam’)
  – Currently JBDS-IP group member (inpatient diabetes guidelines)
    • Peri-operative, diabetic ketoacidosis, hypoglycaemia, HHS, enteral feeding, self management, e-learning on safe use of IV insulin, renal unit, peri-partum management, steroid induced hyperglycaemia, etc,

DHEA, dehydroepiandrosterone; HHS, hyperosmolar hyperglycaemic state; ITU, intensive therapy unit; IV, intravenous;
JBDS – IP, Joint British Diabetes Societies for Inpatient Care.
Outline

• Objectives

• A brief history of diabetes and it’s treatment
  – Diabetes related emergencies
  – Inpatient diabetes care
  – Variations in care
Outline

• Admissions avoidance

• General management

• Outcomes of inpatient hyperglycaemia

• Diabetic ketoacidosis
What is Diabetes?

“A complex metabolic disorder characterised by chronic hyperglycaemia resulting from defects in insulin secretion or insulin action, or both”

First described in 1550 BC
Hypothesis

• That some of the work that I have done and contributed to have helped to improve the care of adult inpatients with diabetes
Admissions Avoidance
Inpatients With Diabetes

- Approximately 18% of all hospital inpatients have diabetes
- Most are in hospital with their diabetes rather than because of it
- The most common reason for a diabetes specific hospital admission is the ‘diabetic foot’ with £1Bn spent on this complication every year

Reasons for Acute Admission

Chart 8: Percentage of inpatients admitted for management of diabetes or a diabetes complication by diabetes type, England and Wales, 2015

Foot Disease

• A combination of infection, ischaemia and pressure on the wound

• Ulcers precede almost 80% of all lower limb amputations – most are infected
The Most Common Guideline

IDSA GUIDELINES

2012 Infectious Diseases Society of America Clinical Practice Guideline for the Diagnosis and Treatment of Diabetic Foot Infections

Benjamin A. Lipsky,1 Anthony R. Berendt,2 Paul B. Comia,3 James C. Pile,4 Edgar J. G. Peters,5 David G. Armstrong,6 H. Gunner Deery,7 John M. Embil,8 Warren S. Joseph,9 Adolf W. Karchmer,10 Michael S. Pinzur,11 and Eric Senneville12

Clinical Infectious Diseases 2012;54(12):e132-e173
## IDSA / IWGDF Classification

<table>
<thead>
<tr>
<th>Clinical Description</th>
<th>IDSA</th>
<th>IWGDF</th>
</tr>
</thead>
<tbody>
<tr>
<td>No symptoms or signs of infection</td>
<td>Uninfected</td>
<td>1</td>
</tr>
<tr>
<td>Local infection involving only the skin and the subcutaneous tissue (without involvement of deeper tissues and without systemic signs as described below). If erythema, must be &gt;0.5 cm to ≤2 cm around the ulcer.</td>
<td>Mild</td>
<td>2</td>
</tr>
<tr>
<td>Local infection (as described above) with erythema &gt; 2 cm, or involving structures deeper than skin and subcutaneous tissues (e.g., abscess, osteomyelitis, septic arthritis, fasciitis), and no systemic inflammatory response signs (as described below)</td>
<td>Moderate</td>
<td>3</td>
</tr>
<tr>
<td>Local infection (as described above) with the signs of SIRS, as manifested by ≥2 of the following:</td>
<td>Severe</td>
<td>4</td>
</tr>
<tr>
<td>• Temperature &gt;38°C or &lt;36°C</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Heart rate &gt;90 beats/min</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Respiratory rate &gt;20 breaths/min or PaCO2 &lt;32 mm Hg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• White blood cell count &gt;12 000 or &lt;4000 cells/μL or ≥10% immature (band) forms</td>
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</table>

IWGDF – International Working Group for the Diabetic Foot

### Admissions Avoidance

<table>
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<tr>
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<td>Moderate</td>
<td>3</td>
</tr>
<tr>
<td>Cellulitis &gt; 2 cm around the ulcer associated with lymphangitis or foot failing to respond to oral antibiotics alone and not systemically unwell</td>
<td>Moderate infection - borderline admission</td>
<td></td>
</tr>
<tr>
<td>Local infection (as described above) with the signs of SIRS, as manifested by ≥2 of the following:</td>
<td>Severe</td>
<td>4</td>
</tr>
<tr>
<td>- Temperature &gt;38°C or &lt;36°C</td>
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<td>- White blood cell count &gt;12 000 or &lt;4000 cells/μL or ≥10% immature (band) forms</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

## Quick Reference Guideline: Table 2: Antibiotic Management of Diabetes Related Foot Infections in Adults

<table>
<thead>
<tr>
<th></th>
<th><strong>FIRST CHOICE</strong></th>
<th><strong>PENICILLIN ALLERGY</strong></th>
<th><strong>DURATION</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PARTIAL OR FULL THICKNESS</strong></td>
<td><strong>EXTENDING TO UNDERLYING SOFT TISSUE/BONE</strong></td>
<td><strong>PARTIAL OR FULL THICKNESS</strong></td>
<td><strong>EXTENDING TO UNDERLYING SOFT TISSUE/BONE</strong></td>
</tr>
<tr>
<td><strong>MILD</strong></td>
<td>Co-amofoxclav 625mg tds PO</td>
<td>Co-amofoxclav 625mg tds PO</td>
<td>Clarithromycin 500mg bd PO</td>
</tr>
<tr>
<td><strong>MODERATE</strong></td>
<td>Co-amofoxclav 625mg tds PO + Ciprofloxacin 500mg bd PO</td>
<td>Co-amofoxclav 625mg tds PO + Ciprofloxacin 500mg bd PO</td>
<td>Cindamycin 150mg - 300mg qds PO</td>
</tr>
<tr>
<td><strong>SEVERE BORDERLINE ADMISSION</strong></td>
<td>Cotrimoxazole 1.2g od IM* (see notes below re IM administration)</td>
<td>Cotrimoxazole 1.2g od IM* (see notes below re IM administration)</td>
<td>Ciprofloxacin 500mg bd PO + Metronidazole 400mg tds PO</td>
</tr>
<tr>
<td><strong>SEVERE NEEDS ADMISSION</strong></td>
<td>Tazocin 4.5g tds IV</td>
<td>Tazocin 4.5g tds IV</td>
<td>Clarithromycin 500mg bd IV + Metronidazole 400mg tds IV</td>
</tr>
</tbody>
</table>

*Antibiotics should only be given where there are appropriate facilities available to test sensitivity. Cotrimoxazole 1.2g IM should be given as two separate 1g injections in different sites.

**If patient is MRSA positive then prescribe according to sensitivities (combination of 2 of the following oral antibiotics, doxycycline, trimethoprim, rifampicin, fusidic acid but do not use fusidic acid in combination with rifampicin). Discuss with a Medical Microbiologist or 45881 if sensitivities not available.**

Co-amofoxclav may cause cholestasis jaundice if use is prolonged, especially in patients over 65 years. If treatment continues over 2 weeks renal function tests (LFTs) should be carried out. Cholestasis jaundice may occur up to 6 weeks after treatment is stopped.
Results

• We rationalised antibiotic prescribing
• We avoided or delayed acute hospital admissions
• The use of outpatient once daily intramuscular antibiotics
  – avoided admission in more than 50% of cases
  – saved almost £6500 per patient compared to those who did not receive them

Admission avoidance using intramuscular antibiotics for the treatment of borderline foot infections in people with diabetes in a tertiary care foot clinic

Ketan Dhatariya
2012 QIC DIABETES WINNER

Best admissions avoidance and/or safe discharge initiative

An analysis of the impact of intramuscular antibiotics for the treatment of severe-borderline foot infections in diabetes: an admission avoidance strategy

Dr Ketan Dhatariya and colleagues,
Norfolk & Norwich University Hospitals NHS Trust
Proposed Update to IDSA Guideline

A Proposed New Classification of Skin and Soft Tissue Infections Modeled on the Subset of Diabetic Foot Infection

Benjamin A. Lipsky, Michael H. Silverman, and Warren S. Joseph

1University of Oxford, United Kingdom; 2BioStrategies Consulting Ltd, Marblehead, Massachusetts; 3Roxborough Memorial Hospital, Philadelphia, Pennsylvania

Open Forum Infectious Disease 2017;4(1):ofw255
General Management
Glucocorticoids and Diabetes

- Is it a problem?
- How to control hyperglycaemia associated with glucocorticoid use?
How do Glucocorticoids Affect Carbohydrate Metabolism?

Glucocorticoids

Brain

- Modulates insulin sensitivity
- Hypothalamus:
  - ↑Neuropeptide Y (sympathetic neurons) which leads to
    - Hyperinsulinemia
    - Hyperphagia
    - Weight gain

Liver

- ↑Gluconeogenesis
- ↑Lipogenesis
- ↑TG, Hepatic Steatosis
- Insulin sensitization
- (?) Insulin sensitization

Skeletal Muscle

- Glucose uptake
- Proteolysis
- β-oxidation and lipolysis
- TG, intramyocellular fat deposit
- (+) Glucose uptake
- (+) Lipolysis (VAT>SAT)
- (+) Preadipocyte differentiation
- (+) Adipogenesis (VAT>SAT)
- ↑Visceral Fat Depot
- (+) HSL and ATGL

White Adipose Tissue

- (+) Glucose uptake
- (+) Lipolysis (VAT>SAT)
- Insulin Resistance
- ↑↑ Free FA

Hyperglycemia

A Bit Of Background

• At any one time, ~0.75% of the UK population is on oral glucocorticoids (0.2% in 20-29 year olds, 2.5% in 70-79 year olds)

• 40% of glucocorticoid use is for respiratory disease, with most of the rest being musculoskeletal and cutaneous diseases and conditions requiring immunosuppression

• Most use is for <5 days, but 22% is for > 6 months and 4.3% for > 5 years
NNUH Prevalence Data

- All adult wards (excluding A+E, CCU, ITU/HDU)
- 120 out of 940 (12.8%) patients were receiving glucocorticoids – of whom 16 had pre-existing diabetes
- Only 25 (13 with diabetes) had their BG checked regularly
- 3 people with diabetes on glucocorticoids had no BG checked
- 95 patients had no evidence of BG checking

Swafe L et al Clinical Medicine 2014;14(3):327-328
Surgical Considerations

Does dexamethasone-induced hyperglycaemia contribute to postoperative morbidity and mortality?

K. Dhatariya*

Limitations of Significance Testing in Clinical Research: A Review of Multiple Comparison Corrections and Effect Size Calculations with Correlated Measures

Terrie Vasilopoulos, PhD,* Timothy E. Morey, MD,* Ketan Dhatariya, MD, FRCP† and Mark J. Rice, MD‡
Joint British Diabetes Societies

Management of Hyperglycaemia and Steroid (Glucocorticoid) Therapy

October 2014
Outcomes of Inpatient Hyperglycaemia
Acute Admissions

• To investigate the relationship between a single glucose concentration at the time of acute hospital admission and outcomes
  – length of stay
  – 28 day readmission rates
  – mortality
Acute Admissions

• We analysed data from all 1502 patients admitted through the Acute Medical Unit at NNUH in February 2010

• 893 had a glucose concentration measured
LOS vs Admission Glucose

Trend $R^2 = 0.5556$

P = 0.002

Those above 20mmol/L excluded (most under the diabetes team)

Evan NR, Dhatariya KK Clinical Medicine 2012;12(2):137-139
28 Day Readmission vs Admission Glucose

Trend $R^2 = 0.7918$

Of the 1,502 admissions in February 2010, 71 (4.73%) were readmitted within 28 days.
28 Day Mortality vs Admission Glucose

Trend $R^2 = 0.7874$

$P < 0.0001$

Of the 1,502 admissions in February 2010, 63 (4.19%) died within 28 days

Evan NR, Dhatariya KK Clinical Medicine 2012;12(2):137-139
But What About Longer Term Outcomes?

- We looked at 1 and 2 year outcomes in this same cohort to see if that index glucose concentration could predict mortality

<table>
<thead>
<tr>
<th>Blood glucose (mmol/l)</th>
<th>For death within 28 days</th>
<th>For death within 1 year</th>
<th>For death within 2 years</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Crude odds ratio (95% CI)</td>
<td>Adjusted odds ratio (95% CI)</td>
<td>p-value</td>
</tr>
<tr>
<td>&lt; 6.5</td>
<td>1.52 (0.78–2.99) 0.22</td>
<td>1.61 (0.81–3.19) 0.174</td>
<td>1.43 (0.9–2.28) 0.129</td>
</tr>
<tr>
<td>6.5–7</td>
<td>1.71 (0.79–3.68) 0.171</td>
<td>1.53 (0.7–3.33) 0.281</td>
<td>1.5 (0.87–2.79) 0.143</td>
</tr>
<tr>
<td>7.1–9</td>
<td>1.37 (1.2–6.66) 0.023</td>
<td>2.01 (1.04–3.89) 0.037</td>
<td>2.04 (1.01–4.11) 0.047</td>
</tr>
<tr>
<td>9.1–11</td>
<td>2.83 (2.83–6.66) 0.011</td>
<td>3.23 (1.4–7.45) 0.006</td>
<td>2.07 (1.11–3.87) 0.023</td>
</tr>
<tr>
<td>11.1–20</td>
<td>2.91 (1.28–6.61) 0.011</td>
<td>3.23 (1.4–7.45) 0.006</td>
<td>2.07 (1.11–3.87) 0.023</td>
</tr>
<tr>
<td>&gt; 20</td>
<td>1.09 (0.33–3.63) 0.887</td>
<td>1.41 (0.41–4.82) 0.585</td>
<td>1.39 (0.63–3.07) 0.417</td>
</tr>
</tbody>
</table>

But - Where is the Evidence?

**BMJ**

*BMJ* 2013;346:f134 doi: 10.1136/bmj.f134 (Published 17 January 2013)

**PRACTICE**

**UNCERTAINTIES**

**Should inpatient hyperglycaemia be treated?**

Ketan Dhatariya *consultant in diabetes and endocrinology*

Elsie Bertram Diabetes Centre, Norfolk and Norwich University Hospitals NHS Foundation Trust, Norwich NR4 7UY, UK
Diabetic Ketoacidosis
In 1922, Joslin reports that 31 out of 33 patients with DKA survive – with gentle fluid replacement. RD Lawrence advocates very aggressive fluid management. In 1945, Howard Root in Boston reports a reduction in mortality from 12% to 1.6% between 1940 and 1944 – using up to 1770 units of insulin in the first 24h after admission. RD Lawrence advocates very aggressive fluid management. In 1948, Micks in Dublin used 100 units for those in ‘pre-coma’ and 100 units every 15 minutes – between 500 and 2000 units, depending on severity of coma. Malins and Black in Birmingham used between 140 and 1400 units of insulin in the first 24h depending on severity in 170 consecutive cases. The first UK national guideline for managing DKA published in 2010. Three consecutive papers in the BMJ showed that low-dose insulin infusions (5–6 units/h) work just as well as high-dose in lowering glucose and ketones. Updated in 2013. Survey of current management in 2014.

Diabetic ketoacidosis
Saline should be used for fluid replacement rather than Hartmann’s solution

Diabetic ketoacidosis is a life threatening condition caused by insulin deprivation or inadequate use of insulin in people with type 1 (or occasionally type 2) diabetes mellitus. Precipitants include deliberate insulin omission, intercurrent illness, surgery, trauma, alcohol, late presentation of previously undetected type 1 diabetes, and the use of drugs that alter carbohydrate metabolism. People with diabetic ketoacidosis need swift intervention by specialists because of the substantial morbidity and mortality arising from the acid-base imbalance, profound fluid loss, and electrolyte disturbances.

Current guidelines written by diabetes specialists from the United States and the United Kingdom recommend initial replacement of fluids and electrolytes and intravenous insulin. The fluid advocated in these guidelines is 0.9% saline. However, people may be treated by emergency and intensive care doctors as well as diabetes specialists, and the type of fluid used can vary.

During the first few hours of hospital admission many people with diabetic ketoacidosis are treated by emergency or intensive care doctors who commonly prefer to use Hartmann’s solution (sodium lactate intravenous infusion). Subsequent care is usually delivered by the diabetes team, who prefer to use 0.9% saline. The conflict arises because guidelines for fluid replacement in the acute setting are written by diabetes specialists, whereas no widely accepted guidelines have been written by emergency or intensive care doctors for fluid replacement in diabetic ketoacidosis.

For decades, 0.9% saline has been the fluid of choice for diabetic ketoacidosis, and its use continues to be advocated in modern textbooks on diabetes. Early studies on diabetic ketoacidosis in the 1970s used 0.9% saline, and this approach was reinforced a decade later. However, giving patients large amounts of chloride can cause a hyperchloremic metabolic acidosis, so administration of 0.9% saline for diabetic ketoacidosis could potentially worsen the metabolic acidosis. Thus, 0.9% saline may be the fluid of choice simply because evidence for the efficacy of other fluids is lacking. The question of which fluid replacement is optimal in patients with acute diabetic ketoacidosis is, therefore, still unanswered.
What is a Guideline?

• ‘A principle put forward to set standards or determine a course of action’

List of Published JBDS Guidelines (so far)

- Hospital management of hypoglycaemia in adults with diabetes
- The management of DKA in adults
- Management of adult patients with diabetes undergoing surgery
- Glycaemic management during enteral feeding in stroke
- Management of HHS
- Self-management of diabetes in hospital
- Admissions avoidance in diabetes
- Variable rate insulin infusion (VRII) for medical inpatients with diabetes
- Steroid use for inpatients with diabetes
- Management of adults with diabetes on the haemodialysis unit
- Managing diabetes during and after delivery
- New diagnosis of diabetes in inpatients
- Diabetes in inpatients with mental health issues

Why Are They Needed?

• To standardise and improve the quality of care people receive and outcomes

• A bit of history.....

• It used to be the incoming registrar’s job to ‘rewrite the DKA guideline’

• Why? Because every hospital did something slightly different, which led to variations in care

DKA, diabetic ketoacidosis.

Woolf SH et al BMJ 1999;318(7182):527-530
February 2013

“Commissioners.......must insist on quality and challenge the inefficiencies of providers, particularly unevidenced variations in clinical practice”
Joint British Diabetes Societies
Inpatient Care Group
The Management of Diabetic Ketoacidosis in Adults
March 2010

Joint British Diabetes Societies
Inpatient Care Group
The Management of Diabetic Ketoacidosis in Adults
Second Edition
Update: September 2013
Research: Care Delivery

National survey of the management of Diabetic Ketoacidosis (DKA) in the UK in 2014

K. K. Dhatariya¹, I. Nunney², K. Higgins³, M. J. Sampson¹ and G. Iceton⁴

¹Elise Bertram Diabetes Centre, Norfolk and Norwich University Hospitals NHS Foundation Trust, Norwich, ²Norwich Medical School, University of East Anglia, Norwich, ³University Hospitals of Leicester NHS Trust, Leicester and ⁴Clinical Audit and Improvement Department, Norfolk and Norwich University Hospitals NHS Foundation Trust, Norwich, UK

Institutional factors in the management of adults with diabetic ketoacidosis in the UK: results of a national survey

Research: Care Delivery

Diabetic ketoacidosis in an adolescent and young adult population in the UK in 2014: a national survey comparison of management in paediatric and adult settings

J. A. Edge¹, I. Nunney² and K. K. Dhatariya³

¹Oxford Children’s Hospital, Headington, Oxford, ²Norwich Medical School, University of East Anglia and ³Elise Bertram Diabetes Centre, Norfolk and Norwich University Hospitals NHS Foundation Trust, Norwich, UK

Ketan Dhatariya

Elsie Bertram Diabetes Centre, Norfolk and Norwich University Hospitals NHS Foundation Trust, Colney Lane, Norwich, Norfolk NR4 7UY, UK, e-mail: ketan.dhatariya@nnuh.nhs.uk

Manuscript submitted July 8, 2016; resubmitted September 6, 2016; accepted September 25, 2016
Treatment of Diabetic Ketoacidosis (DKA)/Hyperglycemic Hyperosmolar State (HHS): Novel Advances in the Management of Hyperglycemic Crises (UK Versus USA)

Ketan K. Dhatariya¹,³ • Priyathama Vellanki²
Guidelines for management of diabetic ketoacidosis: time to revise?

Guidelines and position statements from medical organisations are widely used by clinicians to guide the care of their patients. The 2009 American Diabetes Association (ADA) position statement for diagnosis should be changed to a blood glucose concentration of 11.1 mmol/L (200 mg/dl) or higher. The key diagnostic laboratory feature of DKA is the increase in circulating ketone concentrations.
## Uptake of JBDS Guidance

<table>
<thead>
<tr>
<th>Initiative type</th>
<th>Initiative name</th>
<th>Percentage of sites</th>
</tr>
</thead>
<tbody>
<tr>
<td>JBDS guidelines</td>
<td>DKA and hypoglycaemia guidance (2013)</td>
<td>65.5</td>
</tr>
<tr>
<td></td>
<td>Hypoglycaemia management in hospital (2013)</td>
<td>57.7</td>
</tr>
<tr>
<td></td>
<td>Management of adults with diabetes undergoing surgery (2011)</td>
<td>46.3</td>
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<td></td>
<td>Self-management of diabetes in hospital (2012)</td>
<td>25.9</td>
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<tr>
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<td>Hyperosmolar Hyperglycaemia State (2012)</td>
<td>44.6</td>
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<tr>
<td></td>
<td>Glycaemic management of enteral-fed stroke patients (2012)</td>
<td>25.7</td>
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<tr>
<td></td>
<td>Admission Avoidance (front door/AMU protocols) (2013)</td>
<td>9.3</td>
</tr>
<tr>
<td></td>
<td>Steroid use for inpatients with diabetes (2014)</td>
<td>20.6</td>
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<tr>
<td></td>
<td>Discharge planning (2014)</td>
<td>11.5</td>
</tr>
<tr>
<td></td>
<td>Variable rate insulin infusion (VRIII) for medical inpatients (2014)</td>
<td>44.1</td>
</tr>
</tbody>
</table>
Back to the Hypothesis

• The National Diabetes Inpatient Audit (NaDIA) shows that several aspects of care have improved (statistically and clinically) between 2011 and 2016
  – More people are being seen by a member of the diabetes specialist team
  – Rates of mild and severe hypoglycaemia have fallen
  – Fewer people are on inappropriate intravenous insulin infusions
  – Fewer medication errors

But as Always

• There is a lot more to do
  – What are the costs of DKA
    – 1\textsuperscript{st} paper accepted last week
  – How can the peri-operative care of patients with diabetes be improved (NCEPOD)
  – What to do with those previously undiagnosed patients who present with co-incidental hyperglycaemia
  – How can Trusts be ‘encouraged’ to make changes (CQC)

Watch this space!
The Management of Hospital In-patients with Diabetes Mellitus

www.norfolkdiabetes.com
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@ketandhatariya