Abstracts from the Association of British Clinical Diabetologists (ABCD) meetings

Abstracts from the Autumn 2006 meeting

(P1): The effect of a 'Treat to Target' approach with insulin glargine in patients with persistent poor glycaemic control on traditional treatment with twice daily insulin mixtures

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Background and aims: In type 2 diabetes a 'Treat to Target' (T2T) approach, with once daily long acting insulin at bedtime and continuing oral hypoglycaemic agents, has been shown to be remarkably effective in clinical trials involving patients with inadequate glycaemic control despite maximum oral hypoglycaemic agents. The aim was to see if a similar T2T approach could be as successful in unselected patients in an inner city area who were already established on traditional twice daily insulin mixtures and yet remained poorly controlled (HbA1c >8% for at least 1 year).
Material and methods: This audit is of 85 such patients (mean [±SD] age 59 [±9] years; 54% female, 46% male; 53% South-Asian, 24% Caucasian, 23% Afro-Caribbean) were offered a T2T protocol involving a self-applied 2IU increase in the dose of insulin glargine every 3 days if the fasting glucose on self-testing was >5.4mmol/L on each of those days. Patients were on the maximum tolerated dose of metformin. If fasting glucose reached the target but HbA1c remained above 7%, a prandial oral glucose regulator was added and, if this failed, patients were offered prandial fast acting insulin analogue with a dose increasing algorithm. Patients reaching 200 units of insulin daily without glycaemic control were offered the addition of a glitazone. There were sufficient data to make an analysis based on 81/85 patients.
Results: A snapshot was taken at a median (range) of 2 (0.6–3.3) years into the ongoing rolling programme. Mean (±SD) HbA1c had fallen from 9.92 (±1.34)% to latest value of 8.86 (±1.77)% ($p<10^{-5}$). The lowest HbA1c achieved in response to the protocol was at 1.1 (0.2–2.7) years and was ≤6.5% in 16%, ≤7% in 30%, ≤7.5% in 32% and ≤7.9% in 46%. Only 28% failed to show significant improvement in HbA1c (HbA1c fall ≥1%). However, in many the improvement was not sustained. Latest HbA1c at the time of the snapshot was ≤6.5% in 4%, ≤7% in 12%, ≤7.5% in 17% and ≤7.9% in 26%. In 52% the HbA1c was not significantly better than baseline.
Conclusion: These data suggest that a T2T approach, based initially on once daily long acting insulin analogue, significantly improved glycaemic control in nearly three-quarters of patients who were poorly controlled on standard approaches using twice daily insulin mixtures, the improvement being sustained in nearly half. With 30% achieving HbA1c ≤7%, the T2T approach described above is worth considering in patients who are poorly controlled on twice daily insulin mixtures.

(P2): Application of a 'Treat to Target' approach with once daily long acting insulin at bedtime in the 'real world' of unselected patients in an inner city area

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Background and aims: In patients with type 2 diabetes and inadequate glycaemic control despite maximum oral hypoglycaemic agents, a 'Treat to Target' (T2T) approach, with once daily long acting insulin at bedtime and continuing oral hypoglycaemic agents, has been shown to be remarkably effective in clinical trials. The aim was to see to what extent this approach could be as successful in the 'real world' of unselected patients in an inner city area.
Material and methods: This audit is of 132 such patients (mean [±SD] age 57 [±11] years; 60% male, 40% female; 50% South-Asian, 36% Caucasian, 14% Afro-Caribbean) who between 2002 and 2004 were offered a T2T protocol involving a self-applied 2IU increase in the dose of insulin isophane every 3 days if the fasting glucose on self-testing was >5.4mmol/L on each of those days. Patients were on the maximum tolerated dose of metformin. Those experiencing nocturnal or fasting hypoglycaemia were switched to insulin glargine. If fasting glucose reached the target but HbA1c remained above 7%, a prandial oral glucose regulator was added and, if this failed, patients were offered prandial fast acting insulin analogue with a dose increasing algorithm. Patients reaching 200 units of insulin daily without glycaemic control were offered the addition of a glitazone. There were sufficient data to make an analysis based on 128/132 patients.
Results: A snapshot was taken at a median (range) of 2.2 (0.2–3.4) years into the ongoing rolling programme. Mean (±SD) HbA1c had fallen from 10.3 (±1.6)% to latest value of 8.2 (±1.5)% ($p<10^{-26}$). The lowest HbA1c achieved in response to the protocol was at 1.3 (0.2–3) years and was ≤6.5% in 20%, ≤7% in 41%, ≤7.5% in 55% and ≤7.9% in 60%. Only 5% failed to show significant improvement in HbA1c (HbA1c fall ≥1%). Latest HbA1c at the time of the snapshot was ≤6.5% in 9%, ≤7% in 23%, ≤7.5% in 38% and ≤7.9% in 48%. In only 27% was the HbA1c not significantly better than baseline.
Conclusion: Whilst the figures from this audit are less impressive than those from formal clinical trials, 41% achieved HbA1c ≤7%. This shows that even in the 'real world' of unselected patients in an inner city area, a T2T approach involving patient driven insulin dose adjustment can be considerably more effective than traditional methods involving twice daily insulin mixtures and insufficiently frequent dose adjustment by health professionals.

(P3): Successful treatment of severe hypoglycaemia in type I diabetes with human islet transplantation

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Background: Treating type 1 diabetic patients with severe hypoglycaemia (SH) by islet transplantation has become a realistic option but experience is lacking in the UK and limited by insufficient donors. We sought to improve the quantity and quality of human islets from conventional (heart-beating) donors and controlled non-heart beating donors (NHBD).
Methods: We modified techniques of isolation of islets and assessed islets *in vitro* for viability, morphology, insulin response to glucose challenge and the ability to reverse hyperglycaemia in a diabetic animal model. Since June 2002, 76 isolations have been performed from conventional donors, and 10 from NHBD. Five patients with type 1 diabetes and recurrent SH have been identified, characterised and transplanted.
Results: Mean islet IEQ from conventional donors was 410 970±1724 30IEQ/pancreas *vs* 505 000±84 230/pancreas from NHBD (p=0.011). Islets from both groups were highly viable with 88.56±7.02 and 91.3±3.2% viability (p=NS). Islets from both

groups showed normal morphology, and were fully functional. Two patients achieved insulin independence and 1 remains insulin independent 8 months after a single donation; 4 have resolution of hypoglycaemia experience 1–4 years post-transplant. The 5th patient is awaiting completion of transplantation.
Conclusions: The modification of the standard islet isolation procedure has allowed us to establish human islet isolation at clinical grade from both groups of donors. Both the clinical and research programmes will be developed further.

(P4): Hyperthyroidism presenting with persistent headache and vomiting

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Background: The majority of patients with hyperthyroidism are readily diagnosed clinically. Vomiting, headache and/or abnormal liver function have rarely been described as the main presenting features of hyperthyroidism.
Case: A 58-year-old lady with no significant past medical history was admitted with 3 weeks' history of severe headache and persistent vomiting. Physical examination was unremarkable. Initial investigations including FBC, U/E, CRP and CXR were normal. CT head and CSF pressure and analysis were also normal. She was treated symptomatically and discharged home 8 days post-admission. Two weeks later, she was re-admitted with the same symptoms. She reported weight loss of 2 stones. Physical examination was unremarkable except for sinus tachycardia of 134beats/min. Her liver function tests (LFTs) were abnormal: ALP 109, ALT 207, bilirubin 41 and GGT 139. Autoimmune and viral hepatitis screen was normal. Ultrasound abdomen showed no abnormalities. Upper gastrointestinal endoscopy revealed reflux oesophagitis secondary to the vomiting. Barium meal and small bowel studies were negative. She was treated with omeprazole, anti-emetics and analgesia with no improvement. While she was in the hospital, she was noted to have a heat intolerance which, in combination with weight loss and tachycardia, led to consideration of hyperthyroidism. Thyroid function tests confirmed the diagnosis of hyperthyroidism, TSH <0.05mU/L (0.4–4), FT4 68.8pmol/L (10–25). Thyroid U/S revealed a small multinodular goitre. On treatment with carbimazole and propranolol, her symptoms resolved completely within 4 days (after 5 weeks of persistent vomiting and headache). On 6 weeks' follow up at the outpatient clinic, she was asymptomatic and biochemically euthyroid on carbimazole 20mg/day with normalisation of her LFTs.
Comment: Hyperthyroidism should be considered in patients with persistent vomiting, headache and/or abnormal LFTs. Awareness of these atypical presentations will assist physicians to make a timely and cost-effective clinical decision.

(P5): Self-poisoning with pet medications

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Introduction: Many pet medications are pharmacologically similar to those prescribed for human use. An intentional overdose with pet medications is uncommon. We describe a case in which a pet's anti-epileptic drug was deliberately used for self-poisoning.
Case: A 55-year-old woman with no significant past medical history apart from depression on no medications was found unresponsive at home by her husband. On arrival at the hospital, her vital signs were normal and physical examination was unremarkable except for reduced conscious level with Glasgow coma scale of 9/15. Initial investigations – including full blood count, renal and liver functions, arterial blood gas, ECG and CXR – were normal. CT head was also normal. On the second day, an empty bottle of phenobarbitone, prescribed for her epileptic dog, was found at home by the husband. Toxicology screen revealed serum phenobarbitone level of 76mg/L, confirming the diagnosis of phenobarbitone overdose. Four days later, she made a full recovery and admitted taking 60 tablets (1.8g) of her dog's phenobarbitone. She was discharged to an outpatient psychiatric service 6 days post-admission.
Comment: Obtaining a pet medical history may provide critical information in the assessment of a patient with a suspected overdose.

(P6): Montelukast and Churg-Strauss syndrome

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Introduction: Churg-Strauss syndrome (CSS) is a rare systemic vasculitis associated with late-onset asthma, eosinophilia and late neurological manifestations such as peripheral neuropathy. Several case reports have described the occurrence of CSS in patients who were treated with leukotriene receptor antagonists (LARs). The pathogenic role of LARs in the development of CSS remains controversial.
Case: We describe a case of CSS developed while the patient was receiving an LAR (montelukast). A 79-year-old man with a history of bronchial asthma for 2 years was admitted with 2 weeks' history of progressive generalised weakness and sensory disturbance. His bronchial asthma had been treated with low doses of inhaled steroids, beta-2-agonists and intermittent courses of oral corticosteroid. Montelukast 10mg/day was added 6 months before admission to control the asthma symptoms. On admission, neurological examination revealed marked global muscle weakness and reduced pinprick and joint position sensations in all 4 limbs. Chest X-ray, cervical spine MRI and CT head were normal. CSF analysis was also within normal limits. Nerve conduction study showed diffuse axonal polyneuropathy. Further investigations revealed a markedly raised blood eosinophil count and a positive serum P-ANCA. A diagnosis of CSS was made and the patient was treated with high dose of methylprednisolone, which led to a marked clinical improvement with reduction of the eosinophil count.
Comment: Our case suggests a direct aetiological role for montelukast in the development of CSS. Clinicians should be aware of this rare but potentially severe complication of an LRA.

(P7): Thyrotoxic Graves' Disease following T-cell depleted autologous bone marrow transplantation

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In September 2000, a 47-year-old woman with a 15-month history of rapidly progressive scleroderma, unresponsive to oral, pulsed intravenous corticosteroids, cyclophosphamide and prostaglandin infusions underwent T-cell depleted autologous bone marrow transplantation (ABMT). The protocol consisted of combined peripheral blood stem cell and bone marrow harvesting using granulocyte-colony stimulating factor (G-CSF), followed by autograft T-cell depletion by Milteyi processing. Patient conditioning was through intravenous cyclophosphamide 50mg/kg/day from days -5 to -2 of transplantation. Engraftment was successful and both cutaneous and visceral signs of scleroderma improved.

In September 2003, she presented with a year-long history of dyspnoea, palpitations and weight loss. Clinical examination revealed thyrotoxicosis, large goitre with accompanying bruit and with Grade 1 thyroid eye disease; TSH <0.05mU/L (0.1–5.5mU/L); T3 >20pmol/L (3.5–6.5pmol/L); antithyroid peroxidase antibody titre 849IU/ml (positive >100IU/ml). She received an 18-month course of carbimazole but remained thyrotoxic on cessation of therapy. A total thyroidectomy was performed. Histology showed patchy lymphocytic inflammation and occasional germinal centres, consistent with a diagnosis of Graves' Disease. She currently feels well on thyroid replacement therapy. Her scleroderma remains in remission.

We postulate that her thyrotoxic Grave's Disease was a complication of T-cell depleted ABMT. Possible explanations include disruption of the T lymphocyte population as a result of bone marrow transplantation favouring early return of effector over regula-

tory T cells, as well as a shift from a Th1 to a Th2 immune profile. Emergence of Graves' Disease is described following Campath-1H therapy in multiple sclerosis, and immune reconstitution in HIV infected individuals. This is, however, the first report of thyrotoxic Graves' Disease complicating ABMT. Physicians should be aware of this possible complication as popularity of ABMT grows.

(P8): Iopanoic acid – an underutilised treatment for severe thyrotoxicosis in the UK

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Despite being the most potent known 5'-deiodinase inhibitors, all oral cholecystographic agents have been withdrawn from the market in the US. In the UK, iopanoic acid (IA) is currently the only oral cholecystographic agent to remain in clinical use for the treatment of thyrotoxicosis. We report our experience with the use of IA in a series of 10 subjects with thyrotoxicosis.
Method: Retrospective case review of 10 consecutive subjects to receive IA at the Southmead Medical Hospital, Bristol. Clinical course and thyroid function tests were analysed.
Findings: All patients received a loading dose of 1g, then 500mg BD of IA; mean age of patient 39.6 years (range 18–66); mean duration of use 28.9 days (range 7–68); mean fall in serum FT3, FT4 level after 24 hours from baseline of IA use 46.2%, 5.5%; after 78 hours 63.3%, 7%; after 7 days 70.3%, 34.0%. No side effect from IA use was noted. One patient had relapse of thyrotoxicosis on stopping accompanying thionamide, but improved on restarting thionamide. One patient died from exacerbation of pre-existing cardiac failure despite biochemical amelioration of thyrotoxicosis.
Conclusions: IA is a safe and effective adjunct in the treatment of severe thyrotoxicosis. In keeping with published data, the drop in Free T3 observed in our small cohort is remarkable, especially when IA is used in combination with a thionamide. We believe it is under-utilised in the UK and fear its future availability is threatened by ever-declining clinical use. IA use should be considered in all patients with severe thyrotoxicosis, especially those awaiting urgent thyroidectomy.

(P9): Faster resolution of ketosis with higher insulin and glucose concentrations in the management of diabetic ketoacidosis

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Aim: To assess the response of ketone concentrations to the treatment of diabetic ketoacidosis (DKA).
Methods: A retrospective analysis of the first 24 hours of treatment of 17 patients admitted with DKA. We divided patients into 2 groups (Gp1, Gp2) being those with blood glucose (BG) >10mmol/L (n=10, Gp1) and those with BG <10mmol/L (n=7, Gp2) during the first 16 hours of treatment.
Results: The difference in baseline BG and ketones was not statistically significant. During the first 8h, average BGs were 15.8 and 13.9mmol/L, ketones were 4 and 4.4mmol/L, and insulin infusion rates were 4.3 and 3.3 units/h respectively (all NS). In the second 8h BGs were 12.1 and 7.8mmol/L ($p<0.0001$), ketones were 1.4 and 2.7mmol/L ($p<0.001$), and insulin rates were 3.3 and 1.9 units/h ($p<0.0001$) respectively. In the third 8h, BGs were 10.4 and 8.6mmol/L ($p=0.07$), ketones were 0.5 and 3mmol/L ($p<0.0001$), and insulin rates were 2.7 and 2 units/h ($p=0.03$) respectively. In Gp1 the ketones fell faster than in Gp2 ($p<0.001$).
Conclusion: These data support the hypothesis that the use of higher insulin concentrations in the management of DKA facilitated suppression of ketogenesis. We suggest that ketones should be measured in the management of DKA and that insulin rate should be titrated against both ketone concentration and glucose.

(P10): Glucagonoma: a rare cause of diabetes

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A 55-year-old lady had presented with pain in her abdomen and dyspeptic symptoms 6 years ago. She was known to have depression, treated by psychiatrists, as well as osteoarthritis, on NSAID. An oesophagogastroduodenoscopy was normal. Ultrasound abdomen was unremarkable except haemangioma of the right lobe of the liver. She then developed diarrhoea with a history suggestive of steatorrhoea. Clinical examination, blood investigations as well as repeat gastroscopy, barium follow-through and flexible sigmoidoscopy were normal. A diagnosis of irritable bowel syndrome was made. She then developed diet controlled diabetes with some history of weight loss. Two years ago she was admitted with uncontrolled diabetes and started on insulin. An urgent CT scan of her abdomen revealed a heterogeneous mass head of pancreas with atrophic body and tail with multiple liver metastases. Subsequent liver biopsy and histopathology confirmed a metastatic neuroendocrine tumour of the pancreas; 24-hour urinary 5HIAA was normal. Fasting gut hormone profile showed an elevated glucagon level of 214(<50) confirming glucagonoma. She was started on alpha interferon as well as Creon with much benefit. Soon after, her diabetic control worsened. She had low mood, complained of profound lethargy and worsening arthralgia and myalgia. Interferon was stopped. Subcutaneous octreotide was started, followed later by Sandostatin LAR which helped with reducing the insulin requirement as well as better control of her diabetes. Unfortunately, her scans suggest progressive disease and an MIBG uptake scan followed by possible MIBG treatment is planned.

Glucagonomas are rare tumours and fewer than 250 cases have been described in the literature. In patients with a diagnosis of diabetes and irritable bowel syndrome a careful evaluation is necessary to rule out neuroendocrine tumours.

(P11): Prospective study to determine acute effect of central laser photocoagulation on driving ability in patients with diabetic maculopathy

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DVLA guidelines (based on panretinal laser data) advise central laser photocoagulation patients not to drive until visual acuity (VA) and visual fields (VF) have been formally tested. The acute effects of central laser photocoagulation for diabetic maculopathy on VA and VF have not been previously examined. There are also no data on the acute effects of tropicamide eye drops on VA in older people with diabetes.
Methods: We prospectively studied 9 patients requiring laser treatment for diabetic maculopathy (laser group) and 10 controls (diabetic people not requiring laser treatment); (all age >50 years, VA >6/12). VA and VF were tested at: t=1 (baseline), t=2 (20min post-1% tropicamide), t=3 (30min post-laser), t=4 (2 hours post-laser). Controls were assessed at time matched intervals. VA was measured using a LogMar chart (a change of 0.02 = 1 letter on the chart). Binocular VF were tested using the Esterman Fields Test according to DVLA criteria.
Results: There was no difference in VA between the laser and control groups at baseline. Tropicamide reduced VA (t=2: change LogMar score, mean ± SD; [controls] -0.08±0.09, $p<0.001$). There was no significant difference from baseline in mean LogMar score at t=3 and 4. VA decreased following laser; (change in VA, laser *v* controls, t=3: -0.17±0.12[8 letters] *v* -0.03±0.08[1 letter], $p<0.02$, t=4: -0.13±0.2[6 letters] *v* -0.02± 0.08[1 letter], $p<0.05$). Neither tropicamide nor laser reduced VF. Controls fulfilled DVLA vision requirements for VA and VF at all timepoints. Two laser patients failed DVLA VA requirements at t=3 and 1 patient at t=4. Only 33% of the laser group returned to their baseline binocular VA compared to 60% of controls.

Conclusion: VA is reduced by tropicamide but remains within DVLA requirements in patients over 50. Central laser treatment significantly decreases VA acuity but not VF for at least 2 hours to the extent that patients may fail DVLA requirements. Our data suggest that the DVLA guidelines on post-laser VF testing should be reviewed.

(P12): Re-audit outcome of diabetic foot osteomyelitis in large district general hospital setting

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There are around 1.3 million people in the UK currently diagnosed with diabetes and another million may have diabetes without knowing it. Diabetic patients have over 60% a greater risk of leg amputations and approximately 20–40% develop peripheral vascular disease and neuropathy.

It has been argued for long since 1989 (St Vincent Declaration) that the incidence of diabetic osteomyelitis should be reduced by 50% over a 5-year period.

We designed a foot proforma looking at referral source, diagnostic criteria, site of lesion, risk factors, route and duration of antibiotic given, follow up and outcome. We then carried out a retrospective analysis of 30 patients admitted to KGH with osteomyelitis identified by PAS system over a 3-year period (2000–2003).

Results: Most of the patients were in their 40s. Around 70% of patients had type 1 diabetes and were white Caucasian. In all, 50% of patients were admitted from A&E, 33% following a non-urgent referral to the vascular clinic and the rest from the podiatry department. Around 70% of patients were male and had a previous history of foot ulcers. Around 20% had HbA_{1c} >11%. Investigations and outcome treatment of osteomyelitis vary significantly depending on the admitting team and speciality.

Recommendations: These are: development of a foot care protocol, referral form and dedicated multidisciplinary foot team (diabetologist, vascular surgeon, microbiologist, podiatrist and diabetes nurse specialist); and educating health care professionals and encouraging early referral. Following implementation of the recommendations, we re-audited the service by looking at all diabetic patients admitted with osteomyelitis in the year 2004 at KGH and the outcome showed significant improvement, with amputation of 19% compared to 56%, and fully healed ulcers/osteomyelitis at 56% compared to 17% in the year 2004 *vs* 2003.

Conclusion: Update of foot management guidelines and protocols, and also further resource and early referral to the multidisciplinary foot team are required.

(P13): The use of glargine insulin in pregnancy

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Aims: A review of the outcomes in pregnant diabetic women taking glargine insulin as part of their therapeutic regimen.

Methods: Glargine insulin was used as the basal insulin with a soluble insulin in 12 pregnancies (11 mothers). Average maternal age 29 years (range 15–35). There were 8 type 1, 1 gestational and 1 type 2 on insulin. The reasons for using or continuing glargine insulin were: 5 mothers were already on glargine at the time of conception, and poor control of diabetes in 7 pregnancies (2 of whom had hypoglycaemic fits).

Results: The mean HbA_{1c} at booking was 7.3% (range 5.6–9.6), at delivery was 6.3% (range 5.6–8). The hypoglycaemic fits ceased in the 2 patients. The average gestational age at delivery was 38.5 weeks (range 36–40). Delivery was normal vertex 5, ventouse 2, elective section 1, emergency section 4. Apgar score was 9 or 10 in all babies at 5 minutes. The mean weight of babies (6 girls and 6 boys) was 3464g (range 2230–4507). There were no fetal abnormalities; 1 baby had significant hypoglycaemia.

Conclusion: The use of glargine insulin in 12 pregnancies was safe to administer, and helped improve the control of the diabetes; in 2 mothers hypoglycaemic fits no longer occurred.

(P14): Audit of glycaemic control from insulin sliding scale regimens with and without the continuation of glargine

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Aim: To evaluate the glycaemic control obtained on standard insulin sliding scale regimens and on regimens modified for patients whose glargine treatment was continued whilst on the sliding scale.

Method: Retrospective analysis of the case notes of 72 patients (45 standard and 27 modified for glargine) given sliding scale insulin in a district general hospital. Fifty-one of the episodes were surgical, 11 obstetric and 10 medical.

Results: On standard sliding scales, 88% of random blood glucose measurements were in the 4–14mmol/L range with 9% >14mmol/L and 3% <4mmol/L. This compared with 83% 4–11mmol/L, 10% <4mmol/L and 7% >14mmol/L on the glargine-modified regimens. (P=0.04 for increase in hypoglycaemia.) Junior medical staff compliance with and understanding of the glargine-modified regimen was poor and in contravention of local guidelines. Six patients on glargine received intravenous insulin when their glucose was <11mmol/L. Furthermore, 4 (15%) patients on glargine and 5 (11%) on standard regimens received 0.9% saline instead of a dextrose-containing intravenous fluid to accompany the insulin infusion.

Conclusion: The regimens themselves probably lead to adequate glycaemic control. However, errors in execution of the glargine-modified regimens resulted in an increase in hypoglycaemia. A new chart of the administration of sliding scale insulin for patients in whom glargine was continued was needed and has been developed.

(P15): Case report. Relapsing and remitting insulin requiring diabetes: type 1 or type 2?

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A 28-year-old Barbadian man presented acutely in 1998 with osmotic symptoms, dehydration and a random plasma glucose of 21mmol/L with ketonuria and arterial pH 7.24. He was treated uneventfully for diabetic ketoacidosis, and was discharged on twice-daily mixed insulin. Anti-islet cell antibodies were negative.

He was troubled by frequent hypoglycaemia, and subsequently stopped his insulin altogether, with no recurrence of his ketoacidosis. He remained well off all hypoglycaemia treatment for 6 years, and a glucose tolerance test as part of an employment medical was normal. He was referred acutely with symptomatic hyperglycaemia in 2004, and ketonuria was present. Insulin was again commenced, but he rapidly stopped this due to recurrent hypoglycaemia. Relapse occurred in 2005, with marked hyperglycaemia and ketonuria, when he was treated with repaglinide, with good glycaemic control achieved.

He is likely to have 'ketosis prone type 2 diabetes', also called idiopathic type 1, J-type, type $1\frac{1}{2}$, or type 3 diabetes. The mechanism of this condition is thought to arise from acute glucose toxicity leading to beta-cell dysfunction and insulin deficiency, and it is hypothesised that a predisposition to oxidative stress is a risk factor for this type of diabetes, as evidenced by that fact that these patients often have G-6PD deficiency. The epidemiology and pathogenesis of this type of diabetes are discussed.