MedicineToday 2012; 13(8): 39-47

PEER REVIEWED FEATURE
POINTS: 2 CPD/2 PDP

# Type 1 diabetes: insulin schedules for adults

**PAT PHILLIPS** MB BS, MA(Oxon), FRACP, MRACMA, GradDipHealthEcon(UNE); **JESSICA PHILLIPS** MB BS

**Basal–bolus, premix and self-mix are the common insulin schedules used in adults with type 1 diabetes, and offer varying degrees of flexibility and convenience. Episodes of hyper- and hypoglycaemia, periods of sickness and travel by air may require adjustments to insulin schedules.**

## Key points

- **Type 1 diabetes should not be confused with type 2 diabetes requiring insulin or late onset autoimmune diabetes of adults.**
- **Blood glucose swings in people with type 1 diabetes are considerable, with 10% of values lying from 30 to 60% above and below the 24-hour average blood glucose levels at $A_{1c}$ values between 5.0% and 9.1%.**
- **The three components of insulin schedules are the 'Bs' – 24-hour Basal, meal-time Bolus and corrective Boosts.**
- **The three common insulin schedules are basal–bolus (also known as multiple daily injection), premix and self-mix. These offer varying degrees of flexibility and convenience, with basal–bolus the most flexible but least convenient and premix the most convenient but least flexible.**
- **Common problems with insulin therapy include hypoglycaemia; glycaemic and fluid and electrolyte disturbances in sickness; and the need for adjustment after air travel.**



A general practice population of 1000 Australians includes 40 to 80 people with diabetes, of whom four to eight adults have type 1 diabetes. Although many of these people with diabetes consult endocrinologists when first diagnosed and regularly thereafter for specialised check-ups and fine-tuning of management, virtually all of them consult general practitioners for continuing care, including advice about insulin schedules and blood glucose management.

This article reviews the principles underlying the common insulin schedules used in adults with type 1 diabetes and highlights the important differences between these schedules. It also discusses common problems that occur in glycaemic management.

## ADULTS WITH TYPE 1 DIABETES

The three common patient populations with 'type 1 diabetes' are:

- those with type 2 diabetes (previously called non-insulin dependent diabetes) who have progressed to insulin therapy and are now considered 'insulin dependent' and therefore mistakenly thought to have type 1 diabetes
- those with classical type 1 diabetes (previously known as insulin dependent diabetes), with usual age of onset in childhood or adolescence
- those with late onset autoimmune diabetes of adults (LADA), with onset in adulthood.

It is important to distinguish between these groups because:

- those with type 2 diabetes have some ongoing insulin production even if they are treated with insulin, and also have some ongoing endogenous control of glycaemia
- those with true type 1 diabetes or LADA have different rates of development of insulin deficiency and of propensity to other autoimmune problems.

The question to ask to identify people with type 2 diabetes is: *'How long after diagnosis did you start insulin therapy?'*

Those people with type 1 diabetes or LADA will have started insulin within weeks or months of diagnosis, whereas those with type 2 diabetes usually start after several years of

Dr P. Phillips is a Consultant Endocrinologist at the QE Specialist Centre, Woodville. Dr J. Phillips is a Senior Registrar at the Women's and Children's Hospital, Adelaide, SA.

**TABLE 1. TYPE 1 DIABETES: THE COMMON FORMS[1]**

| Form | Age of onset | Time after diagnosis of insulin requirement | Associations |
|---|---|---|---|
| Type 2 requiring insulin therapy | Middle and later adulthood | Years | Metabolic syndrome and cardiovascular risk |
| True type 1 | Childhood and adolescence | Weeks or months | Some autoimmune propensity |
| Late onset autoimmune diabetes of adults (LADA) | Early adulthood | Months | Strong autoimmune propensity |

therapy with lifestyle and non-insulin hypoglycaemic agents.

Insulin therapy in patients with type 2 diabetes is relatively simple compared with in patients with type 1 diabetes, with fewer and less wide glycaemic swings. Screening for autoimmune problems is not indicated in patients with type 2 diabetes who are asymptomatic. Autoimmune problems are, however, more common in patients with true type 1 diabetes and LADA, and especially so in the latter. For example, postpartum autoimmune thyroiditis occurs in 25% of women with type 1 diabetes, and 10 to 20% of patients with LADA will have a second autoimmune problem (most commonly autoimmune thyroid disease or coeliac disease). The characteristics of these three groups are summarised in Table 1.[1]

## INSULIN THERAPY – THEORY

The ideal insulin schedule would mimic the normal beta cell insulin secretion. The normal physiological control of glycaemia has the following features:

- portal delivery of high levels of pancreatic and gut hormones relative to systemic levels (because the portal circulation is smaller than the systemic and because of hepatic hormonal clearance)
- bidirectional pancreatic glycaemic control, with glucagon 'pushing' blood glucose up by increasing hepatic glucose output and insulin 'pulling' blood glucose down by increasing hepatic and systemic glucose utilisation
- immediate and potentially large pancreatic hormonal responses to the ambient blood glucose level (BGL) and modification of these responses by gut hormone modulation related to nutrient absorption (the 'incretin effect'; Figure 1)[2]
- rapid blood clearance of pancreatic hormones so that hormone levels reflect recent pancreatic hormone release and minimise the lag between changes in ambient glucose level and the hormonally induced counter-regulatory changes in hepatic glucose output/uptake and systemic glucose uptake.

## INSULIN THERAPY – PRACTICE

Compared with the ideal physiological system for controlling glycaemia, existing schedules for administering insulin in people with type 1 diabetes leaves a lot to be desired. Insulin therapy involves:

- systemic insulin delivery, which reduces the relatively more intense insulin signalling to the liver compared with that in systemic tissues
- abnormalities of endogenous glucagon response
- disruption of the feedback loop between ambient BGL and insulin delivery, and loss of any modification of the loop by gut hormones
- longer lags in the feedback loop between the glycaemic changes and the changes in insulin delivery affecting hepatic and systemic glucose flux.

The disruption in the feedback loop between ambient BGLs and the counter-regulatory response in insulin delivery and the subsequent changes in hepatic glucose output and hepatic and systemic glucose uptake results in wide swings in BGLs in people with type 1 diabetes. This variability occurs even when overall glycaemia is well controlled (e.g. glycosylated haemoglobin [$A_{1c}$] level is 5%), as shown in Figure 2.[3]

Since the 1980s, self-blood glucose monitoring and, more recently, interstitial fluid continuous glucose monitoring have attempted to restore the feedback between glucose levels and insulin delivery. Also, better formulations of insulin and the use of insulin pumps have shortened the lag between changes in insulin delivery and the resultant changes in hepatic and systemic glucose flux. These improvements have made it possible for people to improve overall glycaemic control and to reduce the swings between hypo- and hyperglycaemia. However, the first two limitations listed above (systemic insulin delivery and abnormalities of endogenous glucagon response) continue to apply, and the feedback loop remains imperfect.

Further improvements in blood glucose monitoring and insulin delivery can be expected. There is also the promise in the future of an artificial pancreas that will come close to restoring the normal feedback loop between blood glucose concentration and insulin delivery.

## INSULIN SCHEDULES – COMMON PATTERNS

Current schedules combine various components of the three 'Bs' – basal, bolus and booster:

- 24-hour Basal insulin
- mealtime Bolus insulin
- corrective Booster insulin (quick- or very quick-acting insulin).

Basal insulin (the intermediate-acting human insulin isophane and the long-acting analogue insulins detemir and glargine) attempts to mimic the endogenous basal insulin delivery, which has a 24-hour pattern of night-time nadir,

### THE INCRETIN EFFECT

The stimulating effect of gut hormones (incretins) secreted in response to nutrient ingestion on insulin secretion has been known a long time, but only recently has the physiology been understood.

In the 1960s, the so-called 'incretin' effect was demonstrated by comparing the insulin response to identical blood glucose profiles produced by oral or intravenous glucose loads. Both the insulin peak and area under the curve were greatly augmented by an oral glucose load compared with an intravenous load, despite BGLs being identical (Figure 1).

As well as increasing insulin secretion, the incretins decrease glucagon secretion, thus decreasing the 'push' up as well as increasing the 'pull' down of blood glucose.

The incretin effect is diminished in people with type 2 diabetes.[2]

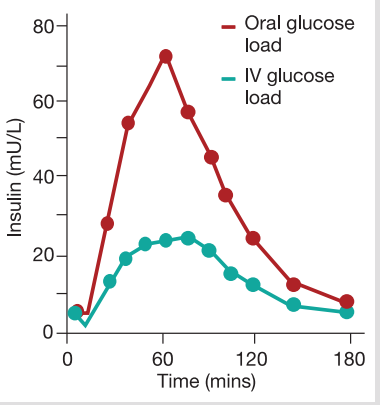



Figure 1. Physiological response to hypo - glycaemia: insulin secretion in response to oral and intravenous glucose loads in a person without diabetes.

### BLOOD GLUCOSE SWINGS

It is now possible, using continuous glucose monitoring, to review 24-hour blood glucose patterns. Variability within an individual is surprising, even when overall glycaemic control is reasonable, and this variability further increases with increasing glycosylating haemoglobin ($A_{1c}$) values. In the past, the frequency in amplitude of blood glucose swings was less obvious because of the infrequency of self-blood glucose monitoring and the variability of pre- and postprandial values.

Figure 2 shows for individuals without or with diabetes (normal to high $A_{1c}$ values) the 24-hour average BGL and the levels beyond which 10% of values for that person will lie (i.e. 80% of the blood glucose values for each individual lie between these lines).[3] Looking at the two extremes, 80% of BGL values range between 3.8 and 6.2 mmol/L in a person without diabetes and with an $A_{1c}$ of 5% (far left, A) compared with values ranging between 4.6 and 16.6 mmol/L in a person with diabetes and an $A_{1c}$ of 9.1% (far right, D). In people with diabetes (B, C, D), 10% of BGL values lie from 30 to 60% above and below the average BGL values.

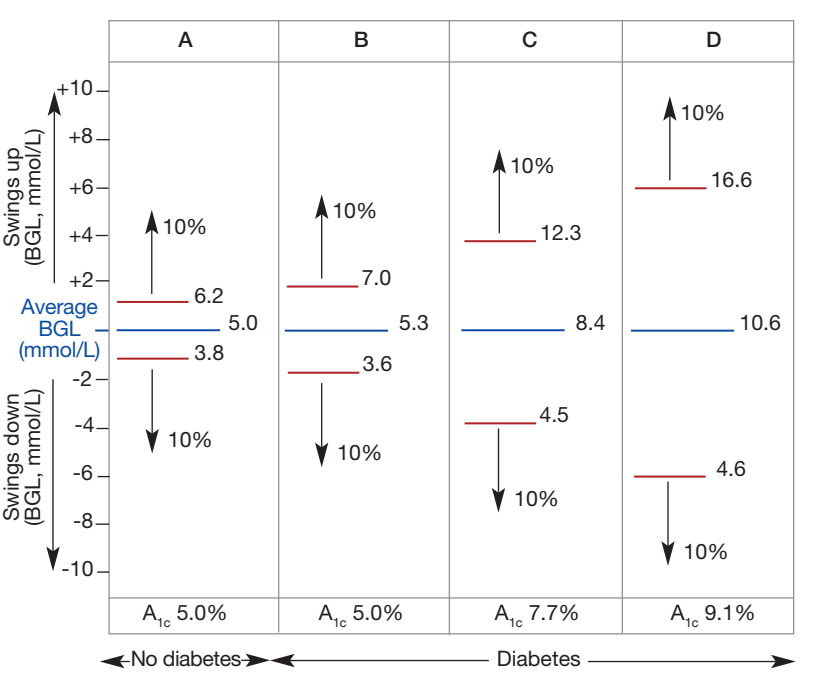


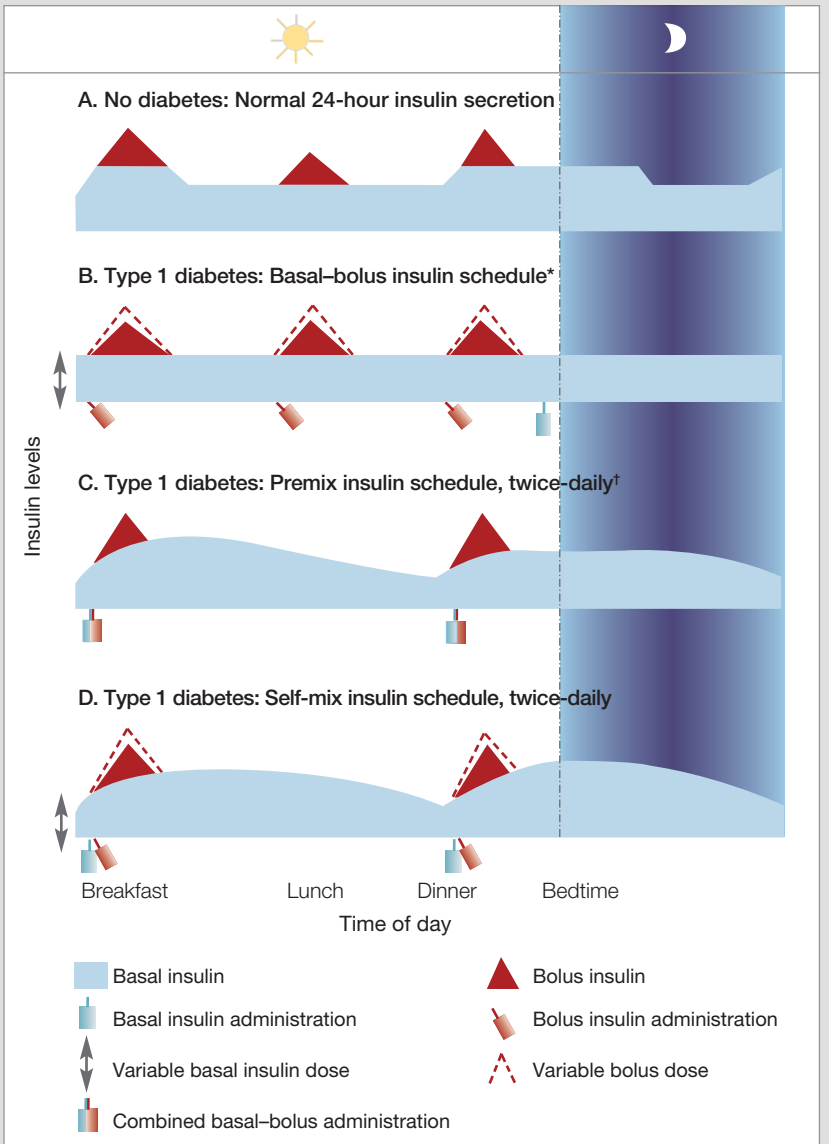

Figure 2. Variability in BGLs in an individual, showing the 24-hour average BGL and the levels beyond which 10% of values for that person will lie. (Adapted from Mazze RS, Strock E, Wesley D, et al. *Diabetes Technol Ther* 2008; 10: 149-159.[3])

## COMMON INSULIN SCHEDULES IN ADULTS WITH TYPE 1 DIABETES: ADMINISTRATION AND DELIVERY

Insulin schedules commonly used in adults with type 1 diabetes include the following:

- basal–bolus (also known as multiple daily injection): once- or twice-daily basal doses with fixed or varied bolus mealtime doses and in some cases extra corrective bolus doses
- premix: twice-daily or rarely thrice-daily mealtime premixed insulin including bolus and basal insulin in fixed proportions (bolus:basal, 25:75, 30:70 and 50:50)
- self-mix: twice-daily or sometimes thrice-daily mealtime fixed or variable proportions of bolus and basal insulin, usually drawn up in the same syringe.

These schedules are represented diagrammatically in Figure 3.




* In some people on basal–bolus schedules, basal insulin is given in the morning instead of or as well as at bedtime.
† In some people on premix schedules, the dose of the premixed insulin is variable, which varies both bolus and basal insulins in fixed proportions (25:75, 30:70 or 50:50).

Figure 3. Insulin levels over a 24-hour period in people without diabetes (A) and in people with type 1 diabetes using the common insulin schedules (B, C and D).

early morning surge and daytime plateau (see Figure 3, part A). Bolus mealtime insulin (the quick-acting human insulin neutral insulin and the very quick-acting analogue insulins aspart, glulisine and lispro) attempts to mimic the endogenous response but lacks its important modification by gut hormones (as discussed in the box on page 41) and the immediacy and amplitude of insulin delivery and rapidity of clearance. Booster corrective doses with quick- or very quick-acting insulin attempt to reduce the amplitude and duration of hyperglycaemic swings.

For all three of the 'Bs', the newer analogue insulin preparations offer significant advantages over the human insulin preparations in terms of their pharmacodynamic profiles and their consistency of absorption. The advantages are apparent in people with type 1 diabetes, with both improved glycaemic control ($A_{1c}$ decreasing by approximately 0.5%) and reduced hypoglycaemia (particularly at night). These advantages of the basal and bolus analogue insulins usually make them the preferred insulins in type 1 diabetes, but their profile may not suit certain patients (Table 2).

Common insulin schedules include the following (see Figure 3):

- basal–bolus (also known as multiple daily injection), with once- or twice-daily basal doses and fixed or varied mealtime bolus doses, and in some cases extra corrective bolus doses
- premix, with twice-daily (rarely three) mealtime injections of premixed insulin in fixed proportions of bolus and basal insulins (bolus:basal, 25:75, 30:70 and 50:50)
- self-mix, with twice-daily (rarely three) mealtime injections of fixed or variable proportions of bolus and basal insulins, usually drawn up in the same syringe.

Varying the mealtime bolus insulin dose in basal–bolus and self-mix schedules addresses any deviation from the

desired preprandial BGL, varied mealtime carbohydrate intake and variable postprandial physical activity. Varying the basal insulin dose in the schedules can address deviations in previous overall fasting and preprandial blood glucose control and variable physical activity.

The basal insulin component in basal–bolus schedules can be either isophane or an analogue (detemir or glargine) and in the self-mix and the premix schedules it is isophane or its equivalent.

The bolus component in all schedules can be human (i.e. neutral insulin) or analogue (aspart, lispro or glulisine, except that there is no glulisine premix).

These three insulin schedules offer varying degrees of flexibility and convenience. Basal–bolus is the most flexible but least convenient, and premix is the most convenient but least flexible (Table 3).

**TABLE 2. DISADVANTAGES OF ANALOGUE INSULINS COMPARED WITH HUMAN INSULINS**

| Basal analogues | Bolus analogues |
|---|---|
| • Slower response to dose changes<br>• May be confused with bolus insulins as both are clear solutions<br>• Cannot be mixed with bolus insulins<br>• Glargine may sting when injected | • Need adequate carbohydrate in meal<br>• Possible insulin 'run out' before next meal<br>• Need to eat promptly after injection |

## INSULIN SCHEDULES – COMMON PROBLEMS

### Hypoglycaemia

Symptomatic hypoglycaemia is common in people with type 1 diabetes (occurring, for example, several times per week) but severe hypoglycaemia, where another person is needed to help in recovery, is fortunately uncommon (occurring, for example, less than once per year). There is, however, a group of people who are especially prone to hypoglycaemia, as identified by the presence of the 'red flags' for hypoglycaemia listed, in order of importance, in the box on page 45.

In individuals without diabetes, the normal physiological responses activated at different levels of glycaemia (the 5, 4, 3, 2, 1 sequence) protect against severe hypoglycaemia (Table 4). The asymptomatic first responses (decreased insulin secretion and increased glucagon secretion) usually maintain blood glucose above levels where the symptomatic sympathoadrenal response is triggered. In type 1 diabetes, however, both the earlier responses (changes in insulin and glucagon secretion) are lost because of beta cell destruction and alpha cell dysfunction. The sympathoadrenal response may also be lost later because of autonomic neuropathy. This leaves individuals defenceless against hypoglycaemia unless they or an observer becomes aware of the cognitive dysfunction that precedes loss of consciousness.

There are also several 'vicious cycles' that predispose to recurrent hypoglycaemia (see the box on page 45). A key driver in these cycles is decreased hypoglycaemic awareness because of the hypoglycaemic event itself, exercise, sleep or autonomic neuropathy. If a person has decreased awareness, an event that might otherwise cause only mild symptoms before successful treatment may cause severe hypoglycaemia. The response to any hypoglycaemic event should include deliberate increases in ambient BGLs for some days so the hypoglycaemic awareness threshold is reset to a higher level. This may avoid a series of hypoglycaemic events that successively reduce hypoglycaemic awareness, predisposing to yet another hypoglycaemic event.

Treatment of hypoglycaemia occurs in three stages:

- firstly, acutely increasing BGLs with oral quick-acting carbohydrate if the person is able to swallow and, if not, with intramuscular glucagon or intravenous glucose
- then, increasing BGLs in the longer term by ingestion of long-acting carbohydrate and occasionally ongoing intravenous glucose infusion
- finally, determining the reason for the hypoglycaemic episode, making plans to avoid any recurrence and discussing with the patient an action plan and a 'hypo' kit to improve early detection and appropriate response to hypoglycaemia.

**TABLE 3. PROS AND CONS OF DIFFERENT INSULIN SCHEDULES**

| Characteristic | Insulin schedule | | |
|---|---|---|---|
| | Basal–bolus* | Twice-daily self-mix† | Twice-daily premix‡ |
| Effective glycaemic control | +++ | ++ | + |
| Flexible | +++ | ++ | + |
| Ease of application | + | ++ | +++ |
| Less time consuming | + | ++ | +++ |

* Basal–bolus = once- or twice-daily (evening and/or morning) dose of basal insulin plus mealtime doses of bolus insulin.
† Twice-daily self-mix = twice-daily doses of basal (isophane) and bolus insulins mixed in syringe in fixed or variable proportions.
‡ Premix = twice-daily mealtime injections of premixed insulin in fixed proportions of bolus and basal insulins.

**HYPOGLYCAEMIA RED FLAGS, IN ORDER OF IMPORTANCE**

- Past history of hypoglycaemic episode
- Hypoglycaemic unawareness (autonomic neuropathy)
- Erratic lifestyle
- Tight glycaemic targets
- Living or sleeping alone

**TABLE 4. PHYSIOLOGICAL RESPONSE TO HYPOGLYCAEMIA – 5, 4, 3, 2, 1**

| Blood glucose level | Physiological response and symptoms |
|---|---|
| 5 mmol/L | Normoglycaemia |
| 4 mmol/L | Lower physiological barrier |
| 3 to 4 mmol/L | Insulin secretion ↓; glucagon secretion ↑; no symptoms |
| 3 mmol/L | Sympathoadrenal responses |
| 2 to 3 mmol/L | Adrenergic and cholinergic symptoms |
| 2 mmol/L | Severe – neuroglycopenic symptoms |
| 1 mmol/L | Severe – life-threatening |

## Sick days

The disturbances of concern when people with type 1 diabetes are sick are glycaemia and fluid and electrolyte imbalance.

If the person is systemically unwell (e.g. febrile), hyperglycaemia is likely and an increased insulin dose may be needed. However, if the sickness is confined to gastrointestinal disturbance (such as following ingestion of staphylococcal toxin in food), the vomiting and straining at stool may not only be distressing but may predispose the person to hypoglycaemia because of lack of carbohydrate intake and the muscular work involved in vomiting.

Hyperglycaemia may be associated with significant glycosuria and osmotic diuresis, which may compound any gastrointestinal fluid and electrolyte loss caused by vomiting and/or diarrhoea. Extra quick- or very quick-acting insulin should be given if the BGL exceeds 15 mmol/L (administer 10% of the basal dose if BGL is 15 to 20 mmol/L and 20% if more than 20 mmol/L).[4] Further doses should be given at four-hourly intervals.

Ketonaemia/uria should be checked if BGL exceeds 15 mmol/L and strongly positive tests for ketones suggest a more severe metabolic disturbance, which might prompt earlier referral.[4] Further details about sick day management can be found in the NHMRC approved *National Evidence-Based Clinical Care Guidelines for Type 1 Diabetes in Children, Adolescents and Adults*.[5]

Fluid intake and losses should be monitored, with intake being maintained at 5 to 10 mL/kg/h in addition to any major gastrointestinal losses. Ideally an oral fluid and electrolyte correction solution (possibly as ice blocks) should be used to replace any gastrointestinal losses; otherwise, water and diet cordial should be used.

Approached to managing sick day disturbances secondary to diabetes in people with type 1 diabetes are given in the flowcharts on page 46.

'Red flags' that should trigger referral of sick people with diabetes include the following:

- concern by the GP, the person or carer that ongoing, competent, confident support is not possible
- worsening of the underlying sickness, particularly if associated with confusion, sleepiness or increasing pyrexia

**THE HYPOGLYCAEMIA VICIOUS CYCLE**

Hypoglycaemic risk depends on the balance between hypoglycaemic tendency and hypoglycaemic protection. Both sleep and exercise increase hypoglycaemic tendency and decrease hypoglycaemic protection. Insufficient intake of carbohydrate or excess hypoglycaemic medication or exercise may further increase hypoglycaemic tendency. Any hypoglycaemic episode reduces hypoglycaemic protection and further increases overall hypoglycaemic risk.

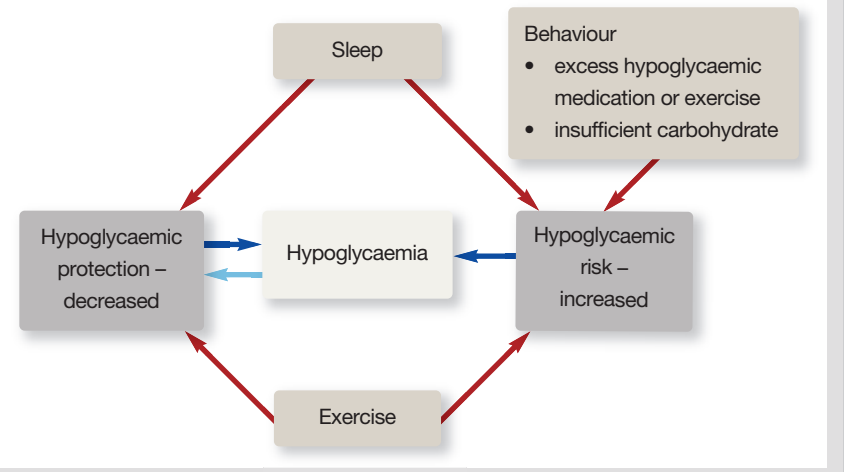
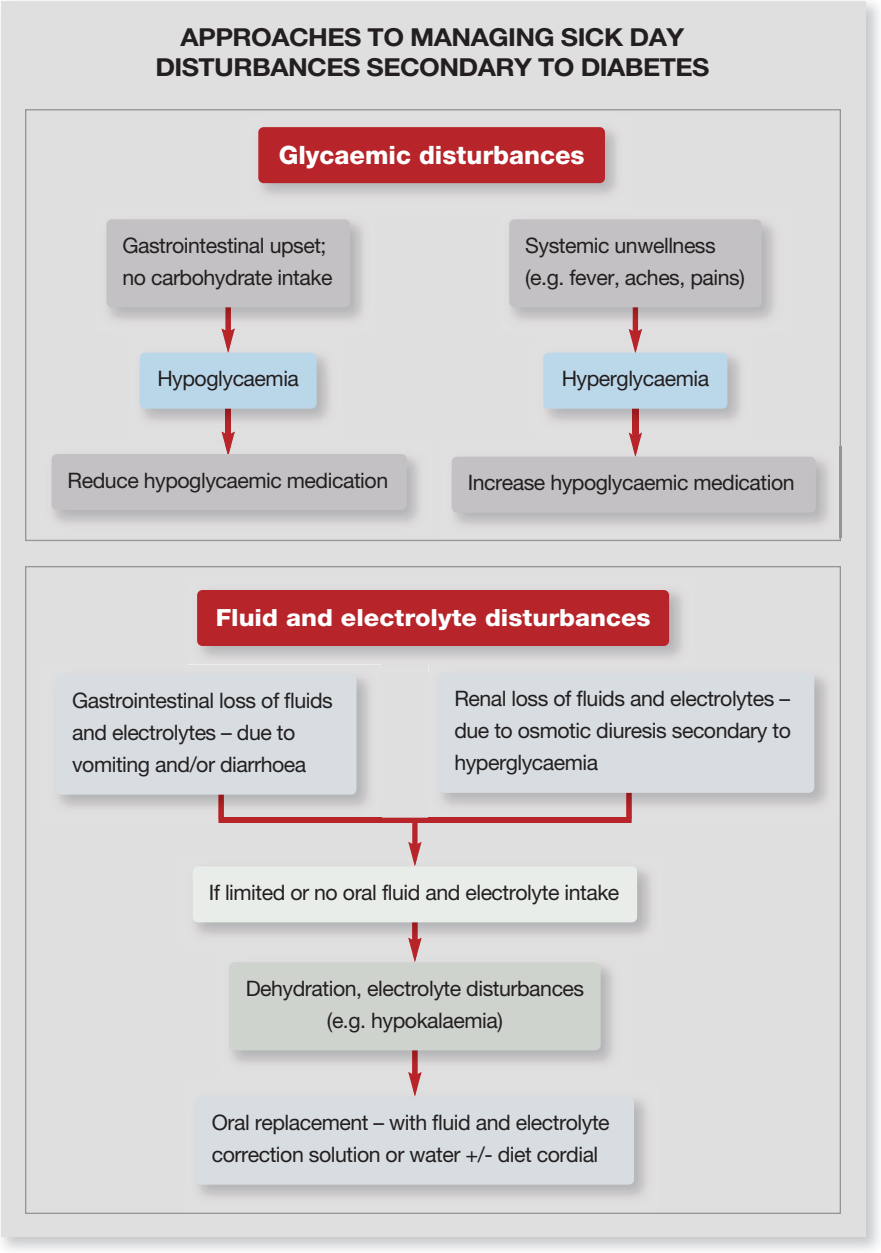



Figure 4. The hypoglycaemia vicious cycle.




- inability to tolerate oral intake of food or fluid, despite the need or likely need for replacement of gastrointestinal losses or need for foods/fluids with a high glycaemic index (because of existing or threatened hypoglycaemia)
- hyperglycaemia without the availability of quick- or very quick-acting insulin
- hyperglycaemia that is not responding to four-hourly corrective doses of bolus insulin, is worsening or is associated with ketonaemia/uria.

## Air travel

When travelling by air, the place of departure and the destination may be in different time zones. If they are, and depending on how many time zones are crossed, a person with diabetes may need, to adjust the insulin dose on the day of travel to control glycaemia before adopting the 'local' time of the destination for meals and insulin injections.

If the person is travelling across several time zones, it is advisable to continue following the local time of the place of departure until arrival at the destination to keep track of insulin injections and meals (i.e. wristwatches are not adjusted until the destination is reached). The length of time the flight takes has no effect on the time difference experienced between the start and end of the journey because that time passes at both the place of departure and the destination.

The general rules regarding adjustment, if needed, of insulin dosages with air travel are:

- if travelling to a time zone ahead of local time (i.e. travelling east), the day of travel will be shorter than 24 hours and less insulin is needed
- if travelling to a time zone behind local time (i.e. travelling west), the day of travel will be longer than 24 hours and more insulin is needed.

More specific guidelines for changes in insulin doses are:

- if the day of travel is made shorter:
  - by four hours or less (four or fewer time zones crossed), there should be no need to make any changes to the insulin dose or food intake
  - by more than four hours (more than four time zones crossed), the insulin dose should be reduced on the day of departure by 20 to 30%, depending on the degree of shortening
- if the day of travel is made longer:
  - by four hours or less (four or fewer time zones crossed), there should be no need to make any changes to the insulin dose, but extra carbohydrate (about 20 to 30 g, which is equivalent to one or two slices of bread) may need to be

eaten to avoid low blood glucose before the next insulin injection at the destination (following the local time at destination)
  - by more than four hours (more than four time zones crossed), extra insulin and food may be needed. The usual insulin dose(s) are taken on the day of departure up to the departure time and then quick- or very quick-acting insulin is given before the extra meals. The usual daily mealtime dose is used, and can be adjusted up or down, depending on the BGL at the time.

## SUMMARY

- Type 1 diabetes should not be confused with type 2 diabetes treated with insulin or LADA. Type 1 diabetes usually presents in childhood and adolescence, type 2 in middle to late adult life and LADA in early adulthood. Both type 1 and LADA are often associated with other autoimmune disease (especially thyroid or coeliac disease) whereas type 2 diabetes is often associated with characteristics of the metabolic syndrome and cardiovascular disease.
- The ideal insulin schedule would reproduce the normal insulin 24-hour secretion profile with portal delivery, bidirectional pancreatic glycaemic control and rapidly changing insulin delivery in response to the prevailing BGL.
- In practice, insulin delivery is subcutaneous, bidirectional glycaemic control with insulin and glucagon is lost or impaired and insulin delivery is slowly responsive to BGLs through self-blood glucose monitoring. More - over, there is considerable within and between individual variability of insulin absorption after injection.
- As a result of these limitations, blood glucose swings in people with type 1 diabetes are considerable, with 10% of values lying from 30 to 60% above and below the 24-hour average BGL at $A_{1c}$ values of 5.0% and 9.1%.
- The three components of insulin schedules are the 'Bs' – 24-hour Basal, mealtime Bolus and corrective Boosts of insulin. The analogue insulins offer significant advantages over human preparations for all three 'Bs'.
- The three insulin schedules commonly used by individuals with type 1 diabetes are:
  - basal–bolus, with once- or twice-daily basal doses, mealtime bolus doses and intermittent booster bolus doses (also known as multiple daily injection)
  - premix, with twice-daily (rarely three) mealtime injections of premixed insulin in fixed proportions of bolus and basal insulins
  - self-mix, with twice-daily (rarely three) mealtime injections of fixed or variable proportions of bolus and basal insulins, usually drawn up in the same syringe.
- These three schedules offer varying degrees of flexibility and convenience, with basal–bolus being the most flexible but least convenient and premix being the most convenient but least flexible.
- Common problems with insulin therapy include:
  - hypoglycaemia, especially in people with a past history, hypoglycaemic unawareness/autonomic neuro-pathy, an erratic lifestyle and tight glycaemic targets or who sleep alone
  - sick days, where the priorities are to maintain basal insulin dosing and fluid intake, to have contact with a health professional for advice and to attend the emergency department if the situation worsens
  - air travel, where insulin adjustment will be necessary if the time zones of the place of departure and the destination differ by more than four hours (more than four time zones are crossed). More insulin is needed if travelling to the west and less if travelling east. MT

## REFERENCES

References are included in the pdf version of this article available at www.medicinetoday.com.au.

## FURTHER READING


Phillips P. KISS: 'keep insulin safe and simple'. Part 2: Living with insulin and type 2 diabetes. Med Today 2007; 8(4): 43-52.

Phillips P. Insulin analogues. What do they offer to the insulin KISS ('keep insulin safe and simple')? Med Today 2008; 9(11): 22-34.

Phillips P. Hypoglycaemia: a major barrier to glycaemic control. Med Today 2011; 12(8): 55-60.

Phillips P. Sick day management of diabetes. Med Today 2011; 12(9): 64-70.

Phillips P, Phillips J. Type 1 diabetes: myths and misunderstandings about glycaemic management. Med Today 2012; 13(6): 45-55.



COMPETING INTERESTS: Dr P. Phillips has received research and travel grants, acted on advisory boards and been involved with clinical trials and seminars sponsored by a range of pharmaceutical companies. He does not think these associations have influenced the content of this article. Dr J. Phillips: None.

Medicine Today 2012; 13(8): 39-47

# Type 1 diabetes: insulin schedules for adults

**PAT PHILLIPS** MB BS, MA(Oxon), FRACP, MRACMA, GradDipHealthEcon(UNE)
**JESSICA PHILLIPS** MB BS

## REFERENCES

1. American Diabetes Association. Diagnosis and classification of diabetes mellitus. [Position statement.] Diab Care 2011; 34 (Suppl 1): S62-S69.
2. Nauck M, Stöckmann F, Ebert R, Creutzfeldt W. Reduced incretin effect in type 2 (non-insulin-dependent) diabetes. Diabetologia 1986; 29: 46-52.
3. Mazze RS, Strock E, Wesley D, et al. Characterizing glucose exposure for individuals with normal glucose tolerance using continuous glucose monitoring and ambulatory glucose profile analysis. Diabetes Technol Ther 2008; 10: 149-159.
4. Harris P, Mann L, Phillips PJ, Bolger-Harris H, Webster C. Diabetes management in general practice. Guidelines for type 2 diabetes. 17th ed. 2011/12. Canberra: Diabetes Australia; 2011. Available online at: http://www.racgp.org.au/guidelines (accessed July 2012).
5. Craig ME, Twigg SM, Donaghue KC, et al; for the Australian Type 1 Diabetes Guidelines Expert Advisory Group. National evidence-based clinical care guidelines for type 1 diabetes in children, adolescents and adults. Canberra: Australian Government Department of Health and Ageing; 2011.