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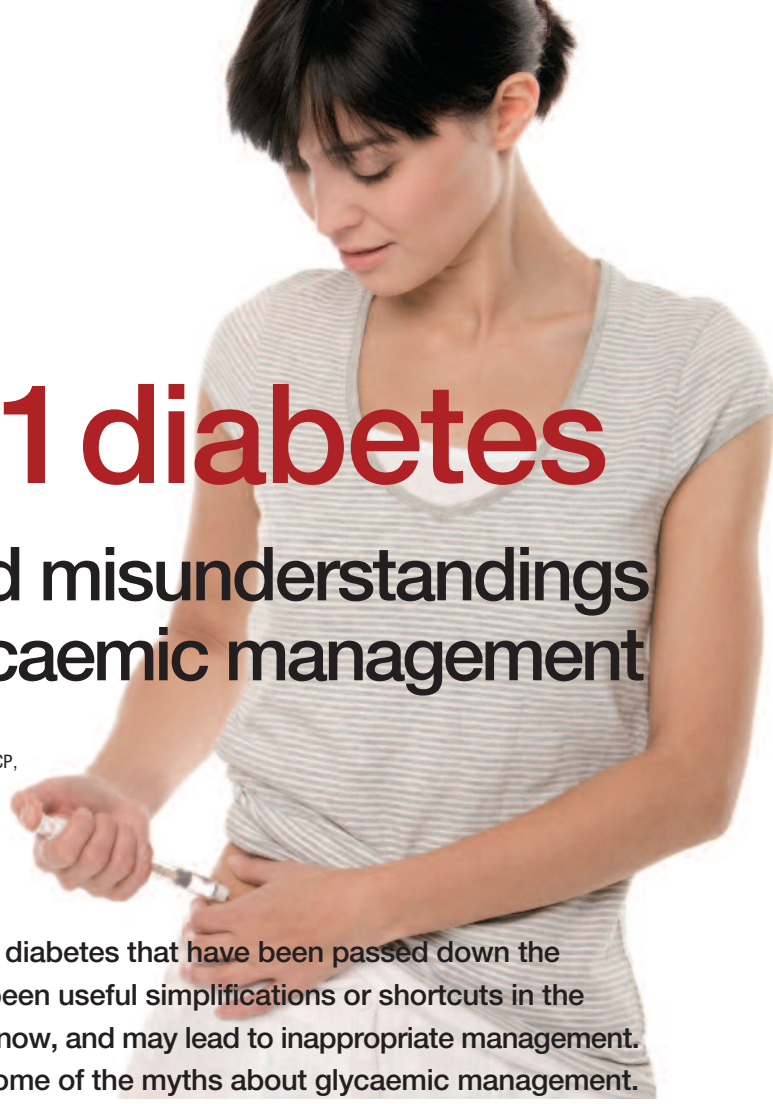

Type 1 diabetes

Myths and misunderstandings about glycaemic management

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The myths about type 1 diabetes that have been passed down the generations may have been useful simplifications or shortcuts in the past but are misleading now, and may lead to inappropriate management. This article discusses some of the myths about glycaemic management.

Key points

- People with type 1 diabetes can use basal–bolus, fixed premixed or self-mixed insulin schedules for glycaemic control.
- Insulin pumps are currently the best way to inject insulin.
- Analogue insulins are usually better than traditional insulins but traditional basal insulins can give more flexible day/night glycaemic control and traditional bolus insulins better interprandial control.
- Insulin is not the only effective hypoglycaemic medication for type 1 diabetes. Metformin and acarbose are of use for certain situations (off-label).
- In type 1 diabetes, the most important blood glucose value to monitor and control is the fasting value.
- The factor most predictive of severe hypoglycaemia in type 1 diabetes is a recent history of an episode.
- On sick days, a person with type 1 diabetes should continue with his or her usual insulin schedule, monitor BGL frequently and take extra bolus insulin if BGL exceeds 15 mmol/L. Basal insulin should not be stopped.

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There are many myths about type 1 diabetes in general practice. Some may have been useful simplifications or shortcuts in the past, but all can now be misleading. A previous article (published in the May 2012 issue of *Medicine Today*) summarised seven of the most general myths about type 1 diabetes and this article reviews another seven applying to glycaemic management.[1]

MYTH 1

All people with type 1 diabetes need basal–bolus insulin therapy

In a multiple choice question, the word 'all' would remind us that rarely are statements universally applicable in medicine. In our practice though, we sometimes accept statements like this. We also assume we know what is meant. 'Basal–bolus' implies four or more insulin injections each day or insulin pump therapy. To apply basal–bolus insulin therapy properly, not only do you have the four-plus insulin injections each day, but also the four-plus blood glucose tests, three-plus carbohydrate counts, three-plus mealtime bolus insulin dose calculations and one to four occasions when you will need to think about corrective insulin doses and/or extra carbohydrate snacks. Basal–bolus insulin therapy is hard work. Many of those who are thought to be using it may be doing the four-plus injections but are not doing the monitoring, testing and calculating that can make basal–bolus therapy so effective.

The desirable characteristics of an insulin schedule and the relative rankings for features such as effectiveness and ease of use for basal–bolus, twice-daily self-mixed and twice-daily premixed insulin schedules are summarised in Table 1.[2] At the two extremes are the effectiveness and flexibility of basal–bolus schedules and the simplicity and lesser requirements of twice-daily premix schedules. Self-mixed insulin schedules have some of the advantages of both

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TABLE 1. RANKING OF 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 = a daily dose of basal insulin plus doses of bolus insulin with every meal.
† Twice-daily self-mix = twice-daily doses of basal (isophane) and bolus insulins.

basal–bolus and premix schedules: they are more effective and flexible than premix and simpler and less time-consuming than basal–bolus schedules. As usual in medicine, 'one size doesn't fit all' and you need to 'pick horses for courses'.[3]

Basal–bolus insulin schedules suit those people seeking tight glycaemic control and/or having a variable lifestyle who are willing and able to apply the basal–bolus approach. If they are less willing and/or less able, but still want tight glycaemic control and flexibility, a twice-daily self-mixed insulin schedule might suit. Those who want to minimise the hassle of diabetes care may be prepared to accept the limited glycaemic control and limited flexibility of twice-daily premixed schedules.

It is important for people with diabetes to choose the insulin schedule that fits with their glycaemic goals and their lifestyle as well as their capacity and willingness to apply the schedule.

MYTH 2

Insulin pumps are better than multiple dose insulin schedules

Insulin pumps are currently the best way we have to inject insulin. Basal and bolus doses are much more easily changed with pumps than with injections and also these dose changes become effective more rapidly. Pumps also make the injection of insulin simpler and quicker, and bypass the inconvenience and discomfort of injections.

However, pumps are expensive and are inconvenient to wear, and the rapidity with which a changed dose of the infused rapid-acting insulin takes effect can be a disadvantage if a mistake or malfunction occurs. If the insulin infusion stops, there is little insulin in reserve and ketoacidosis rapidly ensues, whereas with injection of basal insulin there is sufficient reserve for many hours. If hypoglycaemia occurs, the pump will continue to pump insulin and hypoglycaemia will continue, whereas with injections the insulin reserve will eventually run out and the blood glucose level (BGL) will rise.

The simplicity of dose adjustment may also become a disadvantage if the person 'fiddles' with insulin doses, trying to antic - ipate and respond to any high or low blood glucose values. Such 'fiddling' can result in: weight gain because extra corrective carbohydrate snacks are required; 'stacking', where repeated bolus doses accumulate and cause severe hypoglycaemia; and wide variations in blood glucose values as the 'fiddling' gets out of step with prevailing BGL and starts amplifying rather than minimising blood glucose swings.[4]

The medical disadvantages of the insulin pumps are an increased rate of hypoglycaemic episodes, and ketoacidosis, weight gain and infusion site infections. The main disadvantage for users, however, is the extra hassle of monitoring, calculating and thinking.

There are two situations where the use of an insulin pump has clear advantages over insulin injections.

- Hypoglycaemic unawareness – because the simplicity and rapid effectiveness of dose changes with pumps allows the person to adjust insulin doses according to prevailing blood glucose values and because the basal dose can be varied over the 24-hour period, rather than being fixed for 12 to 24 hours as occurs with basal injected insulin (see the box on page 47).
- 'Dawn phenomenon' – because the programmable variable basal dose can be tailored to mimic the physiological decrease in insulin secretion that occurs during the night and the surge of insulin that occurs in the early morning (see the box on page 47).[5] A bedtime basal insulin dose may provide too much insulin at the nadir of insulin requirements and too little as requirements increase in the morning.

The published clinical trials comparing insulin pumps and multiple injection therapy have not provided clear evidence for or against pump therapy. Some people think they are wonderful and use them enthusiastically, while others try them and stop or use them intermittently.

In terms of identifying suitable patients for insulin pump usage, the same principles apply as outlined for basal–bolus schedules: they suit those who desire the ease and more rapid effectiveness of dose changes and who are prepared for the extra cost, intrusion and hassle of their use.

MYTH 3

Analogue insulins are always better than human insulins

The ideal insulin schedule would mimic the normal beta cell insulin secretion profile, as shown in the box on page 47.[5]

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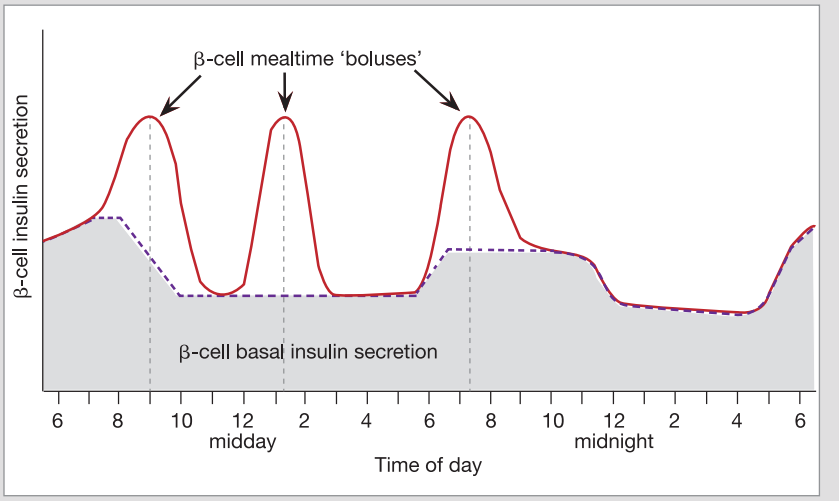
The features would be as follows:

- a variable basal secretion
- prompt mealtime bolus secretion
- continuously variable minute to minute secretion depending on the ambient BGL
- rapid clearance of circulated insulin so insulin levels reflect current secretion rates
- portal delivery so the liver is exposed to a higher insulin concentration than tissues and organs supplied by the systemic circulation.

Before the long-acting and very quick-acting analogue insulins became available our insulin preparations and delivery devices could not approach this ideal. The peaked profile of the intermediate-acting traditional basal insulin preparations (isophane insulin [human]) meant that a bedtime dose reached its peak at the time of the usual nadir of blood glucose and that insulin levels were declining at the time of the usual dawn insulin secretion surge. The duration of action was usually less than 24 hours, requiring two injections per day. The effect of the traditional so-called 'bolus' insulin preparations (neutral insulin [human]) started about 45 minutes after injection, peaked at about two hours and continued over six to eight hours. This meant that the insulin profile lagged behind nutrient input, often resulting in postprandial hyperglycaemia and interprandial hypoglycaemia (see later). Furthermore, the profiles of both basal and bolus human insulins are highly variable within and between individuals, making it hard to predict the glycaemic effects of doses.

The analogue insulins are much better, but still not perfect (Table 2).[6] The profiles of both the basal analogues, detemir and glargine, are flatter and more reproducible, with glargine having the longer duration (more than 24 hours) and usually requiring only one injection per day. The bolus analogues (aspart, glulisine and lispro) have a rapid onset and offset, and reach a higher peak in a shorter time than does bolus human insulin (Figure 1).[6]

NORMAL 24-HOUR INSULIN SECRETION



Total β-cell insulin secretion (the red line in the graph) is made up of a 'basal' secretion throughout the day (the blue dotted line) and periods of increased insulin secretion – the mealtime 'boluses'. The degree of the increased secretion depends on the amount and type of carbohydrate consumed.

The basal secretion varies during four phases:

- Dawn: Increases as cortisol and growth hormone levels increase
- Daytime: Decreases as activity increases
- Evening: Increases as activity decreases
- Nadir: Decreases as cortisol and growth hormone levels decrease.

The practical value of the theoretical advantages of analogue insulins is illustrated by two anecdotes of the experience of people taking human insulin:[4]

'I do the same thing each day. I eat the same, do the same activity, inject the same insulin. One day I'll wake up at 5 (mmol/L) – beautiful. The next day I'm 15. It drives me mad!'

The variability of the absorption profile of basal and bolus human insulins can make it impossible for people with type 1 diabetes to control wide blood glucose swings between hyper- and hypoglycaemia when insulin absorption is slower or faster than usual.

'After lunch my blood glucose goes so high I find it difficult to concentrate at work. I increase my quick insulin and the next thing I know I'm low at 5.00 pm and have to have something to eat before I drive home. I have tried low GI foods but it still happens. If I increase my insulin I go low, if I don't then I go high'.

The relatively slow onset and offset of bolus human insulin results in a longer, flatter profile with early postprandial hyperglycaemia because peak nutrient input precedes the slower insulin action, and then later hypoglycaemia because decreasing nutrient input is associated with continued insulin delivery.

The use of the basal analogue preparations has resulted in modest decreases in glycosylated haemoglobin level (A_{1c}) and significant decreases in hypoglycaemia (especially nocturnal) in clinical trials in type 1 diabetes.[4]

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TABLE 2. ANALOGUE INSULINS – PROS AND CONS COMPARED WITH HUMAN INSULINS

BASAL INSULIN ANALOGUES		BOLUS INSULIN ANALOGUES	
Pros compared with isophane insulin	**Cons compared with isophane insulin**	**Pros compared with neutral insulin**	**Cons compared with neutral insulin**
• Consistent profile • Often can be given as a single daily dose • Less hypoglycaemia • No mixing or resuspension needed for injection	• Slower response to dose changes • May be confused with bolus insulins as both are clear solutions • Cannot be mixed with bolus insulins* • Glargine may sting when injected • Some safety concerns†	• Inject when eating • Less hypoglycaemia • Better postprandial glycaemic control	• Need to eat promptly after injection • Possible insulin 'run out' before next meal • Need adequate carbohydrate in meal

* Little data on safety or efficacy.
† Including use in pregnancy and association with neoplasia.

In some situations, however, the theoretical advantages of analogue preparations become a problem in practice. Firstly, the long duration of basal analogue insulins may mean that a single night-time dose might control fasting glycaemia but cause evening hypoglycaemia. Switching the dose to the morning would only make things worse because the higher insulin activity in the first 12 hours (daytime) will worsen hypoglycaemia in the evening and the lower insulin activity in the second 12 hours (the night-time) will result in fasting hyperglycaemia. In this situation it is preferable to use twice-daily basal insulin (analogue or isophane), titrating the morning dose to control daytime glycaemia and the evening dose to control night-time glycaemia. Secondly, the rapid onset of bolus analogue insulins require the person to eat an appropriate amount of carbohydrate immediately after the injection and the rapid offset may result in low insulin activity and hyperglycaemia before the next meal.

There have also been some concerns about the safety of analogue insulins during pregnancy and their association with neoplasia. Experience in using insulin analogues safely during pregnancy is increasing, and a long-term surveillance program has been established to monitor the incidence of new neoplasia in insulin users.

The major advantages of the insulin analogues are the rapid onset and offset of the bolus analogues, the flatter insulin profile of the basal analogues and the reproducibility of glycaemic effect for both. The major disadvantage is the cost to the government (the PBS subsidies for analogue insulins are higher than those for human insulins).

MYTH 4

Insulin is the only effective hypoglycaemic medication to control glycaemia in type 1 diabetes

Insulin secretagogues (sulfonylureas and repaglinide), which act by stimulating the beta cell to release insulin, do not work in type 1 diabetes because there are no beta cells to stimulate. Similarly, the glucagon-like peptide (GLP)-related medications (the mimetics exenatide and liraglutide, and the enhancers the gliptins) will not have the desired augmentation and inhibition effect on insulin and glucagon secretion, respectively, since the beta and alpha cells are absent or dysfunctional in type 1 diabetes. However, other hypoglycaemic medications may be useful.

Although the insulin sensitisers (metformin and the glitazones) cannot replace insulin, they may reduce the doses used. Metformin in particular may be useful in people with type 1 diabetes who are significantly overweight/waist (BMI more than 30 kg/m^2; waist circumference more than 88 cm in women or more than 102 cm in men) because of its beneficial

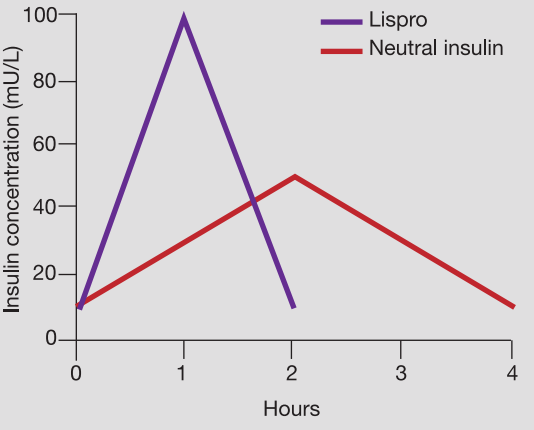

Figure 1. Bolus insulin time–action profiles: analogue (lispro) versus human (neutral). All bolus insulin analogues have a similar profile, reaching twice the maximum concentration in half the time and lasting half as long as neutral bolus insulin (2C-max, $^1/_2$T-max).

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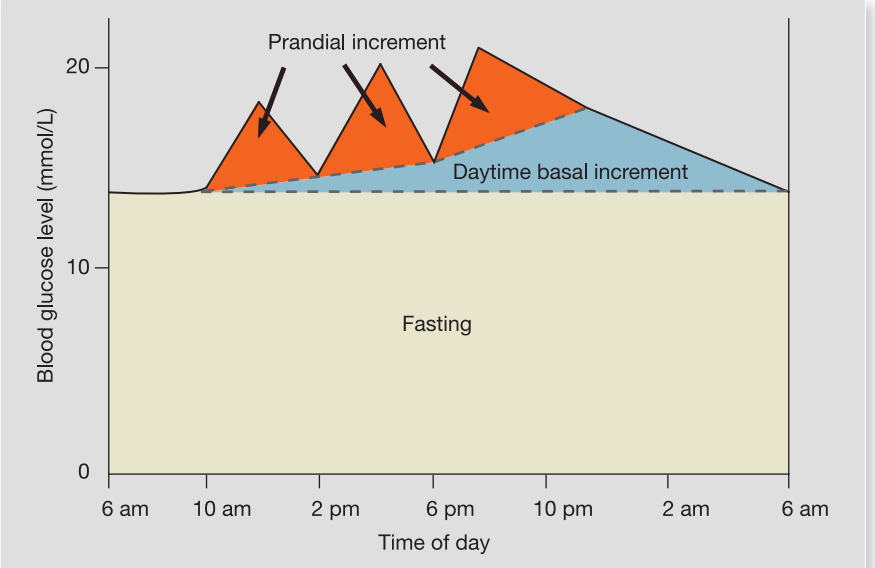

Figure 2. Blood glucose profile in a patient with diabetes (average BGL, 16.9 mmol/L), showing the three components of total blood glucose. This is the common blood glucose profile. Most of the excess glycaemic exposure (the area under the curve) is the high fasting/basal component (75%). The second largest component of excess glycaemia is the daytime basal increment (15%). The excess prandial increment is the smallest component (10%).[9]

effect on both glycaemia and weight. The use of glitazones in type 1 diabetes is contraindicated by the TGA; theoretically they might be useful but they are associated with weight gain and also with fluid retention that can be dangerous in those with, or who develop, heart failure.

The alphaglucosidase inhibitor acarbose slows carbohydrate digestion so the mealtime glycaemic load is absorbed over a longer period, resulting in lower levels of postprandial glycaemia. Once again, this medication may not replace mealtime bolus insulin but it may reduce the dose, improve control of postprandial hyperglycaemia and decrease the risk of postprandial hypoglycaemia. Acarbose is useful to improve postprandial glycaemia for meals with a significant glycaemic load (the amount of carbohydrate multiplied by its glycaemic index) and may be used at only one or two meals a day (e.g. the largest – usually the evening meal). To avoid gastrointestinal side effects such as bloating or flatulence, start with a low dose (such as 25 mg – half a 50 mg tablet) and increase the dose gradually (e.g. in 25 mg increments) at weekly intervals if there are no troublesome gastrointestinal side effects. Make sure patients know that acarbose will slow the absorption of sucrose and that they understand that they should use glucose tablets, powder or drinks to treat hypoglycaemia, rather than food or drink containing sucrose (listed on ingredient labels as sugar). Use of metformin or acarbose in type 1 diabetes is not indicated by the TGA so use would be off-label.

MYTH 5

In type 1 diabetes, the most important blood glucose values to monitor and control are the fasting and evening postprandial (two hours after the evening meal)

This myth is commonly stated but greatly mistaken. Postprandial glycaemia has three components:[7]

- the basal preprandial value (the fasting value) resulting from hepatic glucose output
- any increment in the basal glycaemia during postprandial nutrient absorption
- the prandial increase.

In people without diabetes, mealtime glucose absorption stimulates beta cells to secrete insulin and inhibits alpha cells from secreting glucagon. Both reduce hepatic glucose output and insulin increases tissue glucose uptake. In both type 1 and type 2 diabetes, but especially type 1 diabetes, both beta cell insulin secretion and alpha cell glucagon inhibition are reduced because of absence or dysfunction of these cells. The absence of beta cell stimulation and alpha cell inhibition results in ongoing prandial and postprandial hepatic glucose output, which contributes to basal glycaemia, and in lower tissue glucose uptake, which increases the prandial increment. The typical blood glucose profile of a patient with diabetes is shown in Figure 2.[8]

If the preprandial blood glucose level is high then the postprandial value will also be high unless the prandial increment is very small.[7,9] The preprandial value should, therefore, be controlled before assessing and intervening to control postprandial glycaemia (such as by changing the mealtime glycaemic load and/or adjusting bolus insulin and/or adding or increasing the dose of acarbose).

It is not just the fasting and evening postprandial BGL that should be controlled but all pre- and postprandial glycaemia and nocturnal glycaemia. All of these contribute to overall glycaemic control, which is assessed by measuring the A_{1c}. For both type 1 and type 2 diabetes, the first step in achieving overall glycaemic control is achieving control of all preprandial BGLs; only then should the need to modify postprandial glycaemia be assessed.

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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

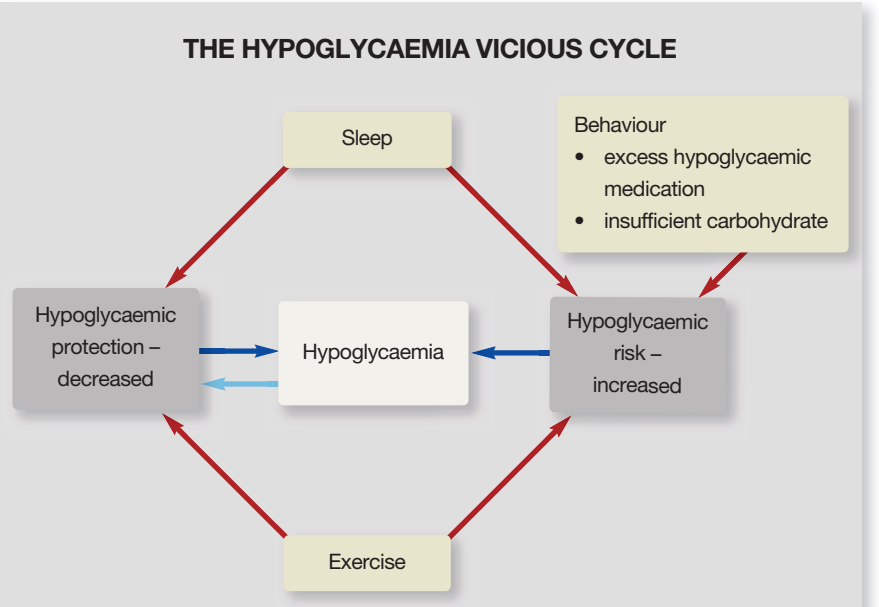

Figure 3. The hypoglycaemia vicious cycle. Hypoglycaemic risk depends on the balance between hypoglycaemic tendency and hypoglycaemic protection. Both sleep and exercise increase hypoglycaemic tendency and decrease hypoglycaemic protection. Insufficient carbohydrate or behaviour such as excess hypoglycaemic medication or exercise may further increase hypoglycaemic tendency. Any hypoglycaemic episode reduces hypoglycaemic protection and further increases overall hypoglycaemic risk.

MYTH 6

The most important factor causing severe hypoglycaemia (where help is required from another person) in type 1 diabetes is striving for tight glycaemic control (A_{1c} <7%)

The frequency of severe hypoglycaemia (number of episodes per 1000-person years) increases as the achieved A_{1c} decreases (e.g. from no episodes to 28 episodes as the A_{1c} decreases from 12% to 6%).[10] However, the level of A_{1c} is not the only, nor the most important, risk factor for severe hypoglycaemia (see the box on this page).

The factor most predictive of a future hypo is a history of a previous one, especially a recent one One severe hypoglycaemic episode identifies that this can occur in that particular individual, and a recent severe episode can reduce hypoglycaemic protection for several days, often initiating a vicious cycle of recurrent hypoglycaemic episodes (Figure 3).[11] This is why it is important that after a severe hypoglycaemic episode the person deliberately aims for higher blood glucose values for the next week so that hypoglycaemic awareness can return.

The next most important risk factor for hypoglycaemia is hypoglycaemic unawareness secondary to the autonomic neuropathy that occurs early in the course of diabetic neuropathy. The unawareness does not cause the hypoglycaemia but it does mean that what might have been a minor symptomatic episode that would have been promptly dealt with by the person becomes a severe hypoglycaemic episode because it was not recognised. Autonomic neuropathy probably also explains why hypoglycaemia becomes progressively more common with longer diabetes duration (e.g. increasing from 110 episodes per 100-person years in those having had type 1 diabetes for less than 5 years to 320 episodes per 100-person years in those with type 1 diabetes for longer than 15 years).[12]

Erratic lifestyle in the young, the old and those with very busy lives is also a major risk factor for severe hypoglycaemia. Sleeping alone, and worse still, living alone, means that a person with severe hypoglycaemia may not be found the next day, unconsciousness continues and severe brain damage or death occurs.

MYTH 7

When people with type 1 diabetes are sick enough to need rest in bed, they should stop their usual insulin schedule and switch to a quick-acting insulin using a sliding scale based on the prevailing blood glucose values.

It would be very dangerous to take the action described in this myth. The intermediate- or long-acting insulin being taken as part of the person's usual multidose insulin schedule helps stabilise glycaemic control and reduces the swings that would otherwise occur if quick-acting insulin were used alone.

The blood glucose record shown in the box on page 53 demonstrates what can go wrong when a person with type 1 diabetes is managed using only a four-hourly quick-acting insulin on a sliding scale. Blood glucose values swing from

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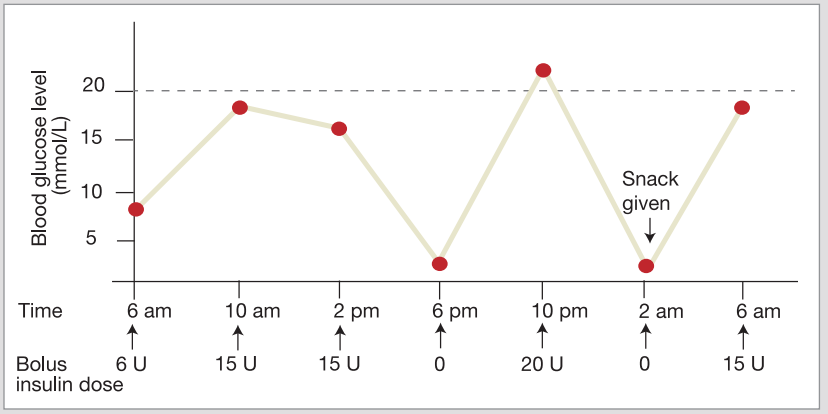
high to low without the stabilising basal insulin. In this case, the quick-acting insulin should have been given at meal-times (rather than four-hourly) and basal insulin should also have been given so that blood glucose values did not swing so high and prompt the large doses of quick-acting insulin that caused the low values.

The general rules for sick day management for a person with type 1 diabetes are for him or her to:[13,14]

- maintain the basal insulin (injected or infused by a pump)
- monitor BGL frequently (at least four-hourly) and monitor blood or urine ketone levels if blood glucose values consistently exceed 15 mmol/L and/or if nausea occurs
- take extra bolus insulin if BGL greater than 15 mmol/L, four-hourly if fasting and before meals if eating (10% of the daily basal dose)
- maintain fluid intake
- contact a resource person (experienced doctor or diabetes educator) for advice
- present to hospital if blood glucose or ketone levels are increasing or if vomiting occurs.

SICK DAY MANAGEMENT IN TYPE 1 DIABETES: WHAT CAN GO WRONG WITH USE OF FOUR-HOURLY INSULIN ON A SLIDING SCALE



In this blood glucose profile, the person had breakfast at 6.30 am, which caused the blood glucose level (BGL) at 10.00 am to be high. Even though 15 U of quick-acting insulin (dose determined from the sliding scale in the table below) was administered at 10.00 am, eating lunch at 12.00 midday caused the 2.00 pm BGL to still be high. The 15 U of insulin given at 2.00 pm caused the 6.00 pm BGL to be low, and so no insulin was given at 6.00 pm. Giving no insulin between 2.00 pm and 10.00 pm, combined with the evening meal at 6.00 pm, caused the high BGL at 10.00 pm. The high insulin dose of 20 U at 10.00 pm caused the hypo at 2.00 am. Giving no insulin between 10.00 pm and 6.00 am and the snack at 2.00 am caused the high BGL at 6.00 am.

Sliding scale insulin schedule

Blood glucose (mmol/L)	4-hourly bolus insulin dose (units)
0 to 5	0
5 to 10	6
10 to 15	10
15 to 20	15
Above 20	20

The problem with sliding scale insulin is that it deals with the current blood glucose level but does not address the preceding causative factors.

SUMMARY

Myths and misunderstandings about glycaemia demystified

- **Myth 1:** All people with type 1 diabetes need basal–bolus insulin therapy.
  Reality: 'One size doesn't fit all'. Basal–bolus insulin schedules offer more effective glycaemic control and more flexibility but are less simple and more time-consuming than fixed premixed insulin schedules. Self-mixed insulin schedules offer a compromise that is more effective and flexible than premixed insulin and more simple and less time-consuming than basal–bolus insulin.
- **Myth 2:** Insulin pumps are better than multiple dose insulin schedules.
  Reality: Insulin pumps are currently the best way we have to inject insulin but are expensive and inconvenient to wear. Medical disadvantages are an increased rate of hypoglycaemia, and ketoacidosis, weight gain and infusion site infections. The disadvantage to users is the extra hassle of monitoring/calculating and thinking. However, insulin pumps have clear advantages for those with hypoglycaemic unawareness and those with the 'dawn phenomenon' because it is simple to adjust the pump's basal and bolus doses.
- **Myth 3:** Analogue insulins are always better than human insulins.
  Reality: Analogue basal insulins have a flatter, longer and more consistent profile than traditional basal insulin (isophane insulin [human]). In some

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people, the shorter duration of isophane insulin can be useful in the titration of basal insulin during the day and night. Analogue bolus insulins reach a higher peak in a shorter time and have a shorter duration than traditional bolus insulin (neutral insulin [human]). In some patients, the longer duration of neutral insulin provides better interprandial glycaemic control.

- **Myth 4:** Insulin is the only effective hypoglycaemic medication to control glycaemia in type 1 diabetes.
  Reality: Insulin secretagogues (sulfonylureas and repaglinide) and GLP agents (exenatide, liraglutide and the gliptins) do not work in type 1 diabetes because the beta and alpha cells responsible for insulin and glucagon secretion are either destroyed or dysfunctional. Although theoretically advantageous as insulin sensitisers, the use of glitazones in type 1 diabetes is contraindicated by the TGA. Metformin can sometimes be useful in people who are overweight/waist and acarbose can reduce postprandial hyperglycaemia; although neither is approved by the TGA for use in type 1 diabetes, they can be used off-label.
- **Myth 5:** In type 1 diabetes, the most important blood glucose values to monitor and control are the fasting and postprandial (two hours after the evening meal).
  Reality: If the preprandial BGL is high, the postprandial BGL will usually be high as well. It is important to check that the preprandial BGL is on target before testing for postprandial hyperglycaemia. If all three preprandial BGLs are on target but the A_{1c} is above target, check for postprandial hyperglycaemia.
- **Myth 6:** The most important factor causing severe hypoglycaemia (where help is required from another person) in type 1 diabetes is striving for tight glycaemic control (A_{1c} <7%).
  Reality: Severe hypoglycaemia does increase as the achieved A_{1c} decreases but the major risk factors for severe hypoglycaemia are (in order of importance): recent history of severe hypoglycaemia, hypoglycaemic unawareness, erratic lifestyle, tight glycaemic targets and living or sleeping alone. After an episode of severe hypoglycaemia it is important that the person deliberately aims for higher blood glucose values for the next week so that hypoglycaemic awareness can return.
- **Myth 7:** When people with type 1 diabetes are sick enough to need rest in bed, they should stop their usual insulin schedule and switch to a quick-acting insulin using a sliding scale based on prevailing blood glucose values.
  Reality: This action would be very dangerous. The intermediate- or long- acting insulin helps stabilise glycaemic control and reduces the glycaemic swings that would otherwise occur if quick-acting insulin were used

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alone. On sick days, the basal insulin should be maintained, BGL should be monitored frequently (with blood or urine ketone testing if BGL exceeds 15 mmol/L and/or nausea occurs) and extra bolus insulin should be administered if BGL exceeds 15 mmol/L. Fluid intake should be maintained and a resource person should be available for advice. If blood glucose or ketone levels are increasing or vomiting occurs, the person should present to hospital. MT

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