LETTER

Preventing exercise-induced hypoglycaemia in insulin-dependent diabetes

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To the Editor: As a practising internist on insulin for type 2 diabetes, I found the article by Thabit and Leelarathna, on exercise-induced hypoglycaemia, of much professional and personal interest [1]. In the last four years, I have personally visited the emergency department twice for severe hypoglycaemia reactions, with glucose values as low as 0.78 mmol/l. One of these occurred when I did not eat for 8 h (14:00 to 22:00) and the other was 3 h after a 10 min bout of resistance activity before my daily aerobic exercise. No doubt, exercise-induced hypoglycaemia is a constant challenge for patients on insulin. Of the options for preventing exercise-induced hypoglycaemia listed in the article, the easiest is insulin dose reduction using an insulin pump. Continuous glucose monitoring is also helpful. Most people with type 1 diabetes, however, do not have these hi-tech options. Carbohydrate supplementation and provoking hepatic glucose output using a high energy/resistance exercise before aerobic activity may interfere with weight management and blood glucose regulation, unless the patient is extra vigilant. This letter points to a simpler exercise option for management of hyperglycaemia with minimal risk for hypoglycaemia. The key is in the timing.

In 1982 a team of Canadian scientists studied glycaemic response to moderate (55% maximum oxygen uptake [$\dot{V}O_{2\max}$]) aerobic exercise in healthy people and those with

type 1 diabetes [2]. The exercise started 30 min post-breakfast and lasted for 45 min. The activity reduced glucose levels and no hypoglycaemia ensued for the first 35 min of the exercise bout. When the exercise ended at 75 min post-meal, the glucose level slowly increased forming a small secondary peak, possibly signalling the continued arrival of exogenous glucose from the gut, offering some insurance against hypoglycaemia. Studies over the next 30 plus years have shown why the mid-postprandial period (30 min to 90 min post-meal) is the right time for people with diabetes to exercise [3, 4]: exogenous glucose is abundant in the blood, insulin:glucagon ratio is high, NEFA levels are low and hepatic glucose production is suppressed. With moderate aerobic activity, glucose transport to the muscles increases and glucose molecules fuel the physical activity, rather than contributing to the post-meal surge.

Transient hypoglycaemia may still occur if the intensity or duration of the physical activity is high [2]. Before starting an exercise regimen, it is a good idea to establish a predictable glucose profile by means of steady carbohydrate intake and medication dose. One way to coordinate meals and exercise is to designate the biggest meal of the day (preferably breakfast) as the ‘exercise meal’, with lunch, dinner and two to three snacks, which can be small. Eating every 2–4 h helps in guarding against hypoglycaemia. A brisk walk for 30 min starting 30 min post-breakfast would not be dangerous. Glucose should be monitored closely after exercise until the pattern in change has been determined. Here the exercise is timed to moderate the glucose surge in response to the biggest meal of the day; the other meals and associated surges are small by design.

If high intensity/resistance exercise comprises part of the exercise regimen, the risk for delayed hypoglycaemia increases [5, 6]. Frequent glucose checking and eating may become necessary. Moderate-intensity exercise during the

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pre-breakfast hours, when counterregulation acts to maintain normal blood glucose levels, may be a last-resort option for those who are prone to hypoglycaemia [7], but post-exertion hyperglycaemia may occur [3].

Short term (days) studies using continuous glucose monitoring are still needed to confirm the impact of moderate aerobic exercise during the mid-postprandial period, either alone or in combination with a short bout of high intensity/resistance activity, on hypoglycaemia risk. Long term (months) counterparts of such studies would help determine the effects on HbA_{1c}, lipids and markers of oxidative stress.

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