ONLINE LETTERS

OBSERVATIONS

Increasing the Low-Glucose Alarm of a Continuous Glucose Monitoring System Prevents Exercise-Induced Hypoglycemia Without Triggering Any False Alarms

he use of continuous glucose monitoring systems (CGMSs) with lowglucose alarms is advocated as a means to decrease the risk of hypoglycemia in type 1 diabetes. Unfortunately, marked mismatches between CGMS readings and actual blood glucose (BG) concentrations limit the usefulness of CGMS in preventing hypoglycemia (1). Although we showed recently that raising the alarm level to compensate for this mismatch decreases the incidence and duration of hypoglycemic episodes, this results in an unacceptably high rate of false alarms (1), defined as an alarm triggered when BG levels are greater than the alarm threshold. This is an important issue because repeated exposure to false alarms can discourage individuals from using their CGMSs (2). Given that CGMSs overestimate BG levels when they rapidly decline (3,4), we propose that raising the CGMS alarm in anticipation of a rapid fall in glycemia could be one condition where the incidence of hypoglycemia may be reduced without triggering any false alarms.

To test this hypothesis, four males and four females with uncomplicated type 1 diabetes and unimpaired awareness of hypoglycemia (age 31.5 ± 6.9 years; BMI 24.8 \pm 2.5 kg/m²; Vo_{2peak} 43.2 ± 4.9 mL/kg/min; diabetes duration 10.6 ± 8.3 years; HbA_{1c} 7.5 \pm 1.1%; mean \pm SD) wore a CGMS (abdomen; Paradigm 722 Real-Time; Medtronic, Northridge, CA) and attended the laboratory within 1.9 \pm 0.2 h of breakfast and their usual insulin bolus (8.4 \pm 5.1 units). When BG levels fell to between 8–10 mM,

participants exercised for 30 min (40% VO_{2peak}) on a cycle ergometer to induce a rapid fall in glycemia (5). During and for 2-h postexercise, the CGMS alarm was either switched off or set to 4.0 or 5.5 mM, with each treatment administered on consecutive mornings following a randomized counterbalanced design. Participants were treated with carbohydrates when an alarm was accompanied by a confirmed BG level \leq the alarm threshold or in response to the verbal expression of hypoglycemic symptoms. One-way repeatedmeasures ANOVA and Bonferroni post hoc tests compared differences in BG and CGMS levels. Hypoglycemic events were compared using a Fisher exact squared test.

In response to exercise, all participants in both the no alarm and 4.0 mM alarm conditions experienced an episode of hypoglycemia, defined as a confirmed BG level <3.8 mM (ABL 700 series; Radiometer, Copenhagen, Denmark), with no cases of false alarms in the 4.0 mM treatment. In comparison, the 5.5 mM alarm significantly reduced by half the proportion of hypoglycemic episodes (P = 0.048) with no cases of false alarms. When the 5.5 mM alarm was triggered, CGMS overestimated BG values by 1.6 ± 0.3 mM.

Our findings show for the first time that when glycemia is falling, the use of CGMS alarms can provide an effective means to reduce the risk of hypoglycemia without the inconvenience of false alarms. Although setting the CGMS alarm at 5.5 mM did not prevent all cases of hypoglycemia because of the large overestimation of BG levels by the CGMS, this mismatch had the benefit of contributing to the absence of false alarms. In conclusion, future diabetes management guidelines should highlight the benefits of using CGMS alarms for the prevention of hypoglycemia when a rapid fall in glycemia is anticipated.

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