An Improved PID Switching Control Strategy for Type 1 Diabetes

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***Abstract*—In order for an "artificial pancreas" to become a reality for ambulatory use, a practical closed-loop control strategy must be developed and validated. In this paper, an improved PID control strategy for blood glucose control is proposed and critically evaluated *in silico* using a physiologic model of Hovorka *et al.* [1]. The key features of the proposed control strategy are: 1) a switching strategy for initiating PID control after a meal and insulin bolus; 2) a novel time-varying setpoint trajectory; 3) noise and derivative filters to reduce sensitivity to sensor noise; and 4) a practical controller tuning strategy. Simulation results demonstrate that proposed control strategy compares favorably to alternatives for realistic conditions that include meal challenges, incorrect carbohydrate meal estimates, changes in insulin sensitivity, and measurement noise.**

***Index Terms*—Control set-point trajectory, glucose control, insulin sensitivity, PID controller, switching, type 1 diabetes.**

I. Introduction

DIABETES mellitus is a metabolic disease characterized by insufficient production of insulin by the pancreas and elevated concentrations of blood glucose for prolonged periods of time (i.e., hyperglycemia). Chronic, untreated hyperglycemia can lead to serious complications that include cardiovascular diseases, blindness, kidney failure, and stroke. Furthermore, very low values of blood glucose (hypoglycemia) for even a few hours can time can result in loss of consciousness and coma. Thus, the maintenance of normal glucose concentrations (euglycemia) is of critical importance for both diabetic and nondiabetic individuals [2], [3].

People with type 1 diabetes must rely on exogenous insulin for survival. The current treatment method requires either multiple daily insulin injections or continuous subcutaneous insulin infusion (CSII) delivered via a pump. For people with type 1 diabetes, euglycemia has been difficult to achieve due to the infrequent blood glucose measurements, patient variability, and changes in insulin sensitivity secondary to exercise, stress, illness, etc. Consequently, there has been considerable interest in developing an "artificial pancreas," a portable (or implantable) automated insulin delivery system that consists of three components: a glucose sensor which provides frequent measurements (e.g., every five minutes), an insulin infusion pump, and a control algorithm which calculates the appropriate insulin dosage for the current conditions.

During the past 30 years, a variety of glucose control strategies based on continuous glucose sensing has been reported. Since this literature has been reviewed in recent survey articles [4]–[7], we will not present a detailed review here. In summary, the control strategies include variations of the proportional-integral-derivative (PID) control algorithm that is widely used in industrial control applications [8]. A PID control strategy is attractive for glucose control because it mimics the first and second phase responses that the pancreas beta cells use to secrete insulin in response to the continuously sensed glucose [6].

This paper is concerned with developing an improved glucose controller that is based on a novel PID controller, bolus injections for meals, and a strategy for switching between them. Controller performance is evaluated for realistic conditions such as inacurrate (meal) carbohydrate estimates, changes in insulin sensitivity, and measurement noise.

II. Physiological Model

Many physiological models have been proposed that describe glucose and/or insulin dynamics [1], [9]–[12]. In this paper, the simulation studies are based on the model developed by Hovorka *et al.* [1] and the modifications reported by Wilinska *et al.* [12]. This model will be referred to as the "Hovorka model." It represents the relationship between input variables, subcutaneous insulin infusion rates (basal and bolus), and the output variable, intravenous glucose concentration. This model also includes a submodel for meal ingestion. The Hovorka model is a nonlinear compartmental model with three subsystems for glucose, insulin, and insulin action. Both insulin and glucose compartments are divided into two more subsystems. The insulin action describes the physiological effect of insulin on glucose transport, removal and endogenous production. The values of the model parameters were determined from experimental data for both normal and diabetic subjects [13]. Model constants were defined to be those quantities difficult to identify, while model parameters were *a priori* identifiable.

Although this model has been effectively used in several studies [1], [14], it can result in nonphysiological responses for some conditions. For example, large (but reasonable) basal

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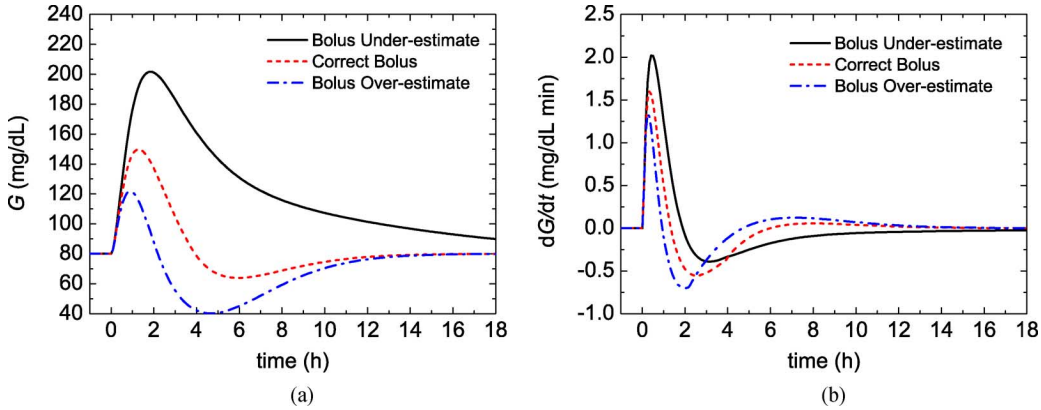

Fig. 1. Open loop G and dG/dt responses for correct bolus (3.16 U), bolus under-estimate (1.58 U) and over-estimate (4.74 U).

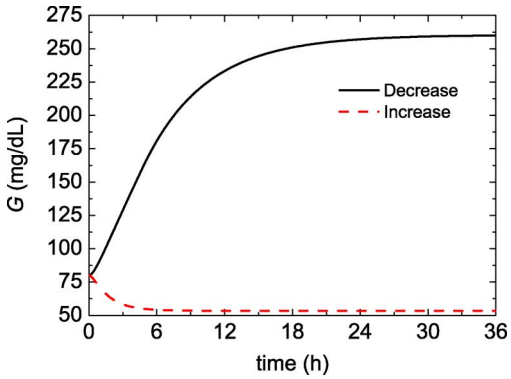

Fig. 2. Open loop G response in case of insulin sensitivities decrease (-50%) and increase ($+50\%$). The insulin infusion rate is 10.35 mU/min.

insulin infusion rates can produce extended postprandial phase and meaningless negative G values [15].

In order to simulate the measurement noise of the glucose sensor [16], [17], a normally distributed random error, ϵ, with mean zero and standard deviation equal to 0.0333 was introduced

$$G_m = G(1+\epsilon) \tag{1}$$

where G_m (mg/dL) is the measured value.

The open-loop response characteristics of the Hovorka model for a 75 kg person are illustrated by postprandial responses (Fig. 1) and responses to insulin sensitivity changes during nonmeal conditions (Fig. 2). For Fig. 1 the basal insulin was maintained constant at the nominal value of 10.35 mU/min and an insulin bolus of 3.16 U was administered at the meal time ($t = 0$). This bolus was based on the actual carbohydrate content (CHO) of the assumed meal, 60 g. The appropriate bolus insulin/CHO ratio, 1 U/19 g, was determined by trial and error and used throughout the study. Fig. 1 demonstrates that boluses based on poor CHO estimates can have a significant and adverse effect on glucose control.

Fig. 2 demonstrates that 50% changes in insulin sensitivity (all three insulin sensitivity parameters were simultaneously changed at $t = 0$) have major effects on both the transient glucose response and the new steady-state value. Some insulin sensitivity changes can be anticipated (e.g., the "dawn effect," a relatively low value in the early morning). But other changes that result from the effects of stress, illness or exercise, are more difficult to anticipate. Thus, a practical glucose control strategy must be able to accommodate both known and unknown intrapersonal changes, including insulin sensitivity changes.

III. PID Controller Design

A variety of PID control strategies have been developed for diabetes control and described in survey papers and review articles [4]–[7]. Most of the related evaluations have been based on simulation studies but experimental applications to dogs or humans have also been reported. Several authors have considered PD controllers with heavy weighting on the derivative action [18], [19]. Integral action is often omitted in order to reduce potential overdosing and hypoglycemia. However, integral action is also desirable to accommodate unanticipated patient changes (e.g., insulin sensitivity). PID controllers have also been used in conjunction with "sliding scales" that partition blood glucose levels into several ranges [20].

The PID controller calculates the insulin infusion rate that is released by the pump into subcutaneous tissues. The velocity form of the PID controller with a derivative filter was used [8]. Furthermore, in order to reduce the effect of noise, the measured glucose concentration G_m was filtered using a standard first-order filter [8].

Integral action is required, but needs to be limited to avoid postprandial hypoglycemia. One solution to this problem is to limit the integral term by introducing upper and lower limits. With this constraint, the effect of the integral action for meal challenges is negligible compared to the effects of proportional and derivative action, but integral action will eliminate steady-state error after insulin sensitivities changes. Details on the PID algorithm and its implementation can be found in [21].

Based on these considerations, a novel controller tuning procedure was employed. First, K_c (controller gain) and τ_D (controller derivative time) were tuned for meal challenges using a nominal value of τ_I; then τ_I (controller integral time) was tuned for insulin sensitivity changes. Finally, the resulting PID controller was re-evaluated for the three meal challenges to ensure that postprandial hypoglycemia did not occur, even when the insulin bolus was over-estimated by 50%. In each case, the resulting value of τ_I was satisfactory.

In the next section, alternative glucose control strategies are proposed and evaluated. In order to avoid biased comparisons, it

is important that the controllers be tuned using the same criteria. Some of the control strategies consist of combinations of PID control plus meal boluses. Thus, K_c and τ_D were tuned based on meal challenge results for three cases: the correct insulin bolus was injected at mealtime, the bolus was 50% too large, and the bolus was 50% too small. The incorrect values were considered to result from poor CHO estimates for the 60 g meal. For these meal challenges, a preliminary value of $\tau_I = 1.67\,\mathrm{h}$ was used. In the second step of the controller tuning, τ_I was tuned for insulin sensitivity changes.

For each PID controller, K_c and τ_D were tuned to minimize the following objective function:

$$J = \sum_i \left(\mathrm{IAE}_i + w_i\right) + c_1 + c_2 \tag{2}$$

with

$$\mathrm{IAE} = \sum_k^{t_\infty} |e(k)|\Delta t \tag{3}$$

$$w_i = \exp\left(-100\left(G_{\min,i} - 60\right)\right) \tag{4}$$

$$c_1 = \exp\left(-10000\left(-0.01 - K_c\right)\right) \tag{5}$$

$$c_2 = \exp\left(-10000\left(1500 - \tau_D\right)\right) \tag{6}$$

where index $i = c, u, o$ refers to the response for a correct, under-estimated and over-estimated CHO content of the meal; IAE (mg min/dL) is the discrete version of the integral absolute error, w_i is the soft constraint to avoid low G values (hypoglycemia); c_1 is the soft constraint to avoid K_c values larger than -0.01 mU dL/mg min; c_2 is the soft constraint to avoid τ_D values larger than 1500 min; t_∞ is duration of the simulation; Δt is the sampling period (5 min), and $G_{\min,i}$ is the minimum value of G_f for the ith run.

Note that when the constraints are not active ($K_c > -0.01\,\mathrm{mU\ dL/mg\ min}, \tau_D > 1500\ \mathrm{min}, G_{\min} > 60\,\mathrm{mg/dL}$), the objective function is simply the sum of the IAE values for the three cases.

IV. Control Strategies

In this section a new switching PID control strategy is proposed that consists of meal boluses and a novel PID control algorithm with a time-varying setpoint. The PID controller is switched off immediately prior to a meal bolus. A switching strategy is proposed for postprandial restarting of the PID controller. The proposed strategy (Strategy E, below) will then be compared to four alternative strategies (A-D) in Section V. These five control strategies will now be described.

A. Bolus Only

This strategy consists of a constant basal insulin infusion rate plus a meal bolus that is proportional to the estimated CHO for the meal. This traditional approach is used by diabetics when only intermittent (fingerstick) glucose measurements are available.

B. PID Control Only

The PID control algorithm of Section III provides a basis for comparison because many previous studies have considered conventional PID control.

C. Bolus Plus PID Control

In this approach, the insulin bolus is introduced at meal time and the PID controller operates continuously.

D. Bolus Plus PID Control With Switching Criteria

The insulin bolus occurs at meal time but the PID controller is not initiated until a switching criterion is satisfied.

E. Bolus Plus PID Control With Switching Criteria and a Time-Varying Glucose Setpoint

The novel features of Strategies D and E will now be described in more detail.

First, we consider why a switching strategy is desirable. Preliminary simulations for Strategy C indicated that it is detrimental to have the PID controller active during and immediately after an insulin bolus. For this situation, the PID controller senses the increasing glucose level of the postprandial response and consequently makes the insulin infusion rate greater than its basal value. This additional insulin release leads to hypoglycemia unless the controller is tuned very conservatively. Consequently, the optimal controller settings for Strategy C were the minimum allowable values of K_c and τ_D. Thus, Strategy C was essentially identical to Strategy A, the bolus-only approach, for these conditions. This experience motivated the development of the switching technique that is a key element of Strategies D and E.

Improved glucose control for meal challenges and inaccurate CHO estimates can be achieved by starting the PID controller after the meal and bolus occur. However, the specification of the switching time is important. If the PID controller is started too early, hypoglycemia can occur. On the other hand, if the PID controller is switched on too late, the postprandial glucose peak may be very large and slowly decrease to the setpoint value. Our simulation studies have indicated that an effective switching strategy is obtained if the PID controller is started when one of two criteria is satisfied: 1) G reaches its peak value and 2) $G > 150$ mg/dL and $dG/dt > 1.5$ mg/dLmin. The 150 mg/dL threshold was selected because it is the peak value of G for a correct bolus and Strategy A [see Fig. 1(a)]. The rate-of-change limit of 1.5 mg/dL min was chosen to be greater than the maximum rate of change for this same situation [see Fig. 1(b)].

The rationale for these switching criteria is as follows. When the CHO estimate is either correct or too large, the PID controller switches on when criterion 1) is satisfied. Then the insulin infusion rate starts to decrease because $dG/dt < 0$. When the CHO estimate is too small, the bolus is also too small and thus when G reaches the threshold of 150 mg/dL, dG/dt is large. Consequently, after the PID controller is switched on, it immediately increases the insulin infusion rate, which is the correct action. Fig. 3 shows that the switching strategy is not sensitive to the specified threshold for criterion 2) and a 50% under-bolus.

Next, we consider the rationale for using a time-varying glucose setpoint. During the postprandial period, the blood glucose concentration is expected to increase, and then decrease. Consequently, it is appropriate to have a time-varying setpoint G_{sp} that reflects this expected behavior [1], [7]. The following strategy has been devised. When the PID controller is initiated, G_{sp} should be set equal to the current filtered measurement G_f

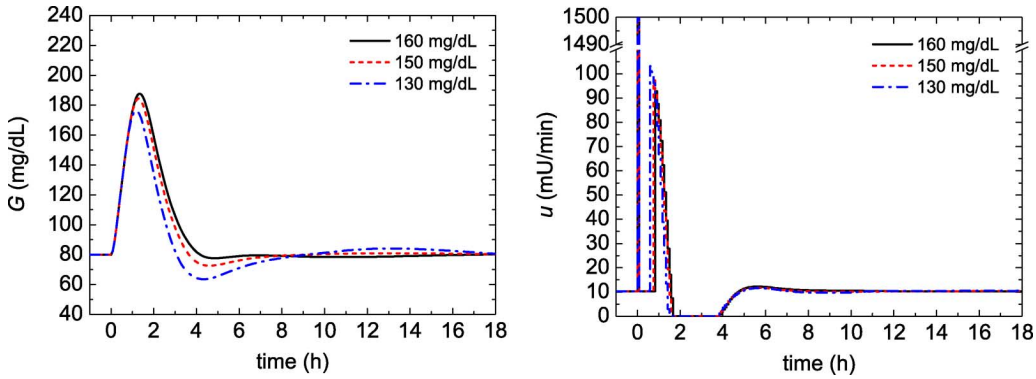

Fig. 3. Responses to meal with bolus under-estimate for control strategy D with different values of the switching threshold.

and then eventually decrease to the desired value of G, 80 mg/dL for this study. However, for the case of a CHO under-estimate, G is still increasing when the PID controller is switched on. Thus, it would be inappropriate to force $G_{\rm sp}$ to decrease right away. Extensive simulations have demonstrated that good glucose control is obtained when the setpoint trajectory is specified as follows:

$$G_{\rm sp}(k^*) = \begin{cases} 80\ \dfrac{\rm mg}{\rm dL}, & \text{if } G_f(k^*) \le 80\ \dfrac{\rm mg}{\rm dL} \\ \left(G_f\left(k^*\right) - 80\right)\exp\left(-\frac{k^*}{\tau_{\rm sp}}\right) + 80, & \text{else} \end{cases} \tag{7}$$

where $k^* = k - k_{sw}$, k (min) is the current sampling instant, k_{sw} (min) is the switching instant, and $\tau_{\rm sp}$ (min) is a design parameter. For $k^* < 0$, $G_{\rm sp} = G_f$. In order to avoid unexpected hypoglycemia, a lower limit of 80 mg/dL was used. The time-varying setpoint trajectory defined in (7) has the following properties.

1) It is affected by the actual value of G_f for every k^*;
2) It varies from $G_f(k^* = 0)$ to 80 mg/dL;
3) As $\tau_{\rm sp} \to 0$, $G_{\rm sp}(k^*) \to 80$ mg/dL;
4) As $\tau_{\rm sp} \to \infty$, $G_{\rm sp}(k^*) \to G_f(k^*)$.

Therefore, if the controller is switched on at the peak in G (switching criterion 1 is satisfied), G starts decreasing and the setpoint trajectory in (7) gradually causes G_f to decrease to 80 mg/dL. In the case of a CHO under-estimate, the PID controller is switched on when G_f reaches 150 mg/dL (switching criterion 2 is satisfied). But since G is still increasing, the setpoint trajectory will initially increase and then eventually decreases to 80 mg/dL.

This novel feedback control strategy has been extended recently to include feedforward control action [22].

V. Simulation Results

The five glucose control strategies of Section IV were evaluated for three situations:

1) meal challenges and either a correct insulin bolus, a 50% under-bolus, or a 50% over-bolus;
2) changes in insulin sensitivity during basal conditions: a 50% increase or a 50% decrease;
3) random changes in the daily CHO meal content and in the daily insulin sensitivity profile, over a 30-day period.

The incorrect boluses represent situations where the diabetic subject incorrectly estimates the CHO content of the meal. The changes in insulin sensitivity were simulated by making the indicated change in all three of the insulin sensitivities for the Hovorka model. The simulation conditions were the same as for Figs. 1–3. In order to provide a fair comparison of Strategies A–E, each PID controller was tuned using the method described in Section III.

A. Results for Meal Challenges

The simulation results are shown in Fig. 4 (left) and in Table I. Results for Strategy C are not included in Fig. 4 because they are identical to those for Strategy A, as indicated in Table I and discussed in Section III. Insulin profiles for the simulations of Fig. 4 can be found elsewhere [21]. Table I includes several metrics: the maximum and minimum glucose concentrations for each response, $G_{\rm max}$ and $G_{\rm min}$, and the settling time t_s. The settling time was defined to be the time at which the glucose concentration entered and remained with the desired range, 75–85 mg/dL.

Fig. 4 and Table I indicate that the new switching control strategies (D and E) are superior to the standard control strategies (A, B, and C) for these meal challenges due to their smaller IAE values, shorter settling times, and smaller $G_{\rm max}$ value for the under-estimated bolus. Strategy E provides the best control based on these criteria. Table I indicates that the $G_{\rm min}$ values for Strategies D and E include small violations of the lower constraint of 60 mg/dL that was used in the controller tuning of Section III. However, these violations can be eliminated by increasing the weight in the soft constraint for $G_{\rm min}$ in (4) (e.g., use a value of 10 000 instead of 100).

B. Results for Insulin Sensitivities Changes

The simulation results for the two insulin sensitivity changes during basal conditions and no meal are shown in Fig. 4 (right). The corresponding insulin profiles are reported in [21]. As described in Section III, the tuning procedure for each controller determined the optimal value of τ_I for insulin sensitivity changes. The simulation results in Fig. 4 are for the average value of τ_I for both insulin sensitivity changes.

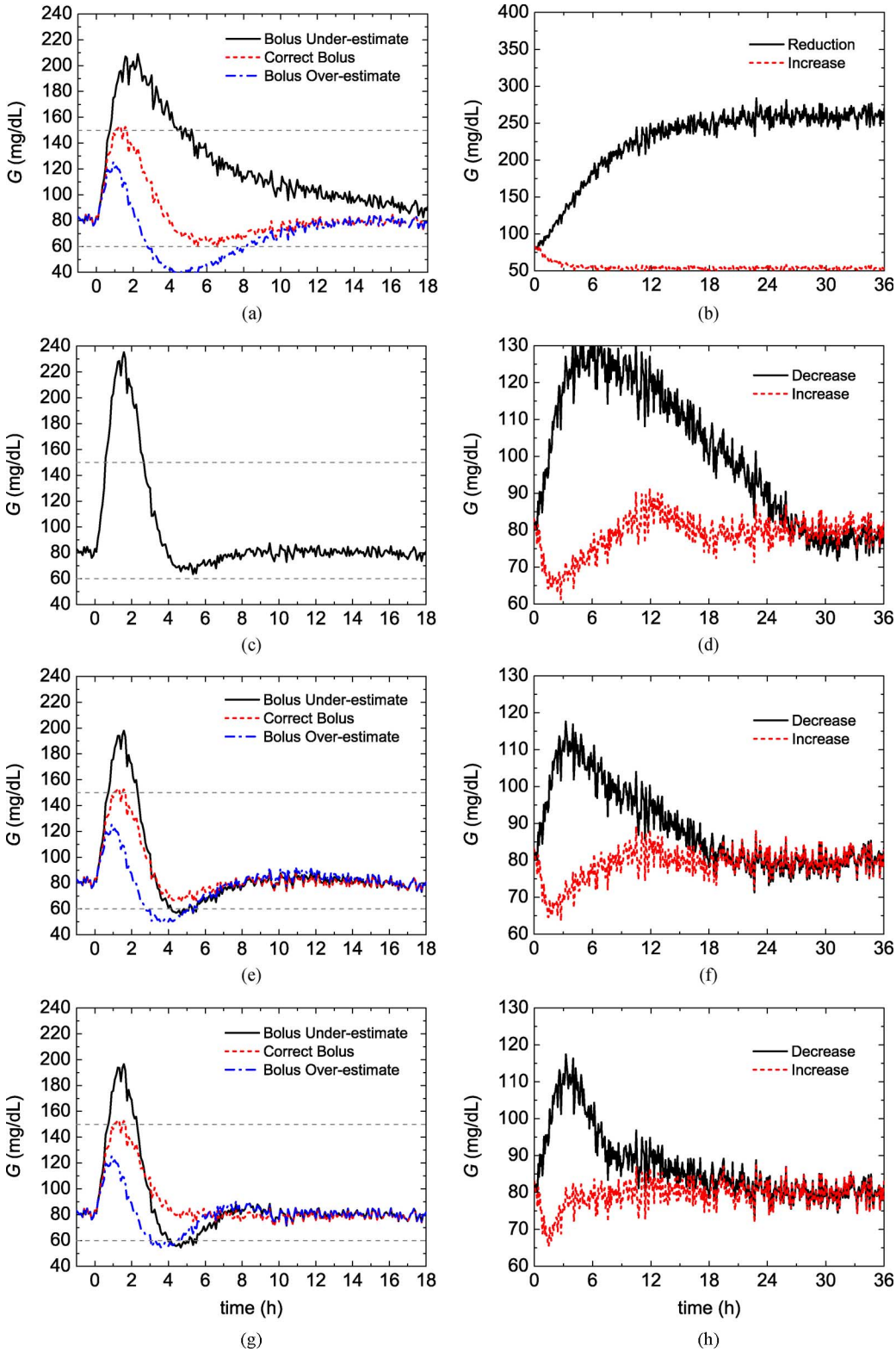

Fig. 4. Simulation results for (left) meal challenges rejection and (right) insulin sensitivities changes ($\mathrm{correct\ bolus} = 3.16\ \mathrm{U}$, $\mathrm{bolus\ under\text{-}estimate} = 1.58\ \mathrm{U}$, $\mathrm{bolus\ over\text{-}estimate} = 4.74\ \mathrm{U}$). Bolus and PID controller strategy turned out to be equivalent to bolus without PID controller. (a)–(b) Strategy A: bolus only. (c)–(d) Strategy B: PID control only. (e)–(f) Strategy D: Bolus plus PID control with switching criteria. (g)–(h) Strategy E: Bolus plus PID control with switching criteria and time-varying glucose setpoint. ($\tau_{\mathrm{sp}} = 25$ min)

The results in Fig. 4 demonstrate that Strategies A and C are not able to cope with these sensitivity changes and result in undesirable glucose excursions. For Strategy B and the 50% decrease in insulin sensitivity, G eventually returns to the desired value of 80 mg/dL but only after a large, slow response. Strategy E provides the best response to the insulin sensitivity changes based on its small IAE values and short settling times. The total insulin utilization is essentially the same for all five control strategies.

TABLE I
RESULTS FOR MEAL CHALLENGES REJECTION

Control Strategy	Bolus Estimation	IAE (10^{-4}) mg min/dL	G_{max} mg/dL	G_{min} mg/dL	t_s h
A	correct	1.40	150	64	10.2
	under	4.69	202	80	>18
	over	1.68	122	**40**	>18
B	-	1.97	228	67	6.7
C	correct	1.40	150	64	10.2
	under	4.69	202	80	>18
	over	1.68	122	**40**	>18
D	correct	1.08	150	68	6.2
	under	1.71	192	**58**	7.1
	over	1.10	122	**51**	12.8
E	correct	0.99	150	78	3.9
	under	1.61	192	**56**	8.5
	over	0.79	122	**56**	8.4

*Glucose values under 60 mg/dL are reported in boldface.

For strategies D and E, the optimal τ_I values were similar for both insulin sensitivity changes. For example, the optimal values of τ_I for Strategy E were 3.7 h for the increase and 2.6 h for the decrease.

C. Robustness Results for a 30-Day Simulation

In order to evaluate the proposed Control Strategy E for realistic conditions for patients with type 1 diabetes, a 30-day simulation study was performed. It included random changes in CHO meal contents and both daily and random variability in insulin sensitivity. The simulation study was based on the Hovorka model of Section II and a 75 kg person. The main features of this long term simulation study were as follows.

1) The *nominal* CHO meal contents were 30 g for breakfast (at 8 AM) and 60 g for both lunch (at 1 PM) and dinner (at 7 PM).
2) For Control Strategy A, the estimated *nominal* CHO content of the meals of 1) was used to calculate the insulin bolus at meal time.
3) The *actual* CHO meal contents varied randomly around the nominal values. A uniform distribution over the range of $\pm 50\%$ of the nominal values was used.
4) The *nominal* insulin sensitivity profile in Fig. 5 varied over a 24 hour period to account for normal variability (e.g., the "dawn phenomenon") as well as the effects of stress, exercise, illness, etc. (This type of daily profile was employed for each of the three insulin sensitivity parameters in the Hovorka model.) The actual maximum and minimum values, $IS_{\max}$ and $IS_{\min}$ respectively, varied randomly on a daily basis following uniform distributions. Thus, $IS_{\max}$ for each day was between the nominal insulin sensitivity value of the Hovorka model, IS_{nom}, and 1.5 IS_{nom} while $IS_{\min}$ for each day varied between 0.5 IS_{nom} and IS_{nom}.
5) The transitions from one *IS* level to the next were modeled as exponential change with a time constant that varied randomly between 15 and 30 min based on a uniform probability distribution.

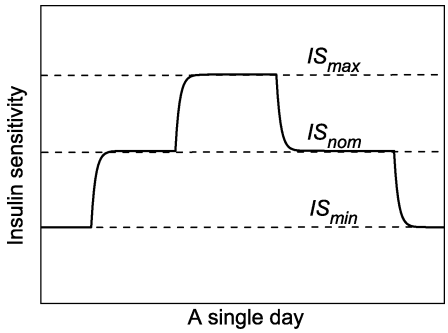

Fig. 5. Nominal daily insulin sensitivity profile for the simulations of Figs. 6 and 7.

6) At the end of Day 14, for all three insulin sensitivities the IS_{nom} values were reduced by 50%, in order to simulate a major change in the general physiology of the patient, for example due to illness or stress. The random variations of 4) and 5) were also employed.

A 30-day simulation of Control Strategy A (bolus only) and recommended Control Strategy E is shown in Figs. 6 and 7, respectively, with statistics for glucose concentration summarized in Table II. The dashed lines in Figs. 6 and 7 denote the glucose setpoint, 80 mg/dL, and the range of acceptable values, 60–150 mg/dL. These results demonstrate that the proposed control strategy significantly outperforms the traditional bolus-only approach (i.e., Control Strategy A) for the wide variety of conditions in the 30 day simulation. In particular, the mean glucose value is much closer to the set point of 80 mg/dL and the standard deviation is much smaller.

VI. DISCUSSION

The simulation results demonstrate that an insulin bolus for a meal challenge is required in order to avoid the large glucose peak that occurs when a bolus is not used (Strategy B). However, the bolus-only approach (Strategy A) results in poor glucose control for incorrect boluses and for insulin insensitivity changes. Thus, a combination of insulin boluses for meals and PID control is desirable. But for meal challenges, the PID controller should be switched on at an appropriate time after the bolus is introduced (Strategies D and E); otherwise, it will result in insulin overdosing and hypoglycemia unless it is tuned very conservatively, as was necessary for Strategy B. Furthermore, for the successful application of PID control, it is important to limit the integral control action in order to avoid insulin overdosing. In this study, the integral control action was limited by imposing constraints. Alternatively, classical "anti-reset-windup" approaches [8] could be considered. For this simulation study, the proposed Strategy E gave the best performance for both meal challenges and changes in insulin sensitivity.

The reason that Strategy E outperforms the other four strategies is that the combination of the switching criterion and the time-varying glucose setpoint allows more aggressive controller settings to be used without sacrificing robustness. In particular, larger controller gains can be used when the controller's task is to reduce deviations from a nominal setpoint trajectory, rather than deviations from a constant value (e.g., 80 mg/dL). The optimal controller settings for the five control strategies are shown

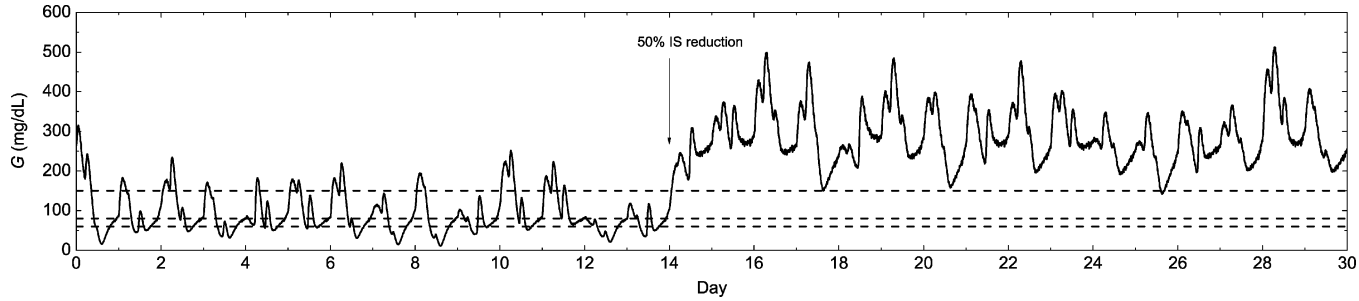

Fig. 6. Performance of Control Strategy A (bolus only) for a 30-day period with meals and random variations in the insulin sensitivity profile. (The nominal IS profile is reduced by 50% at the end of Day 14.).

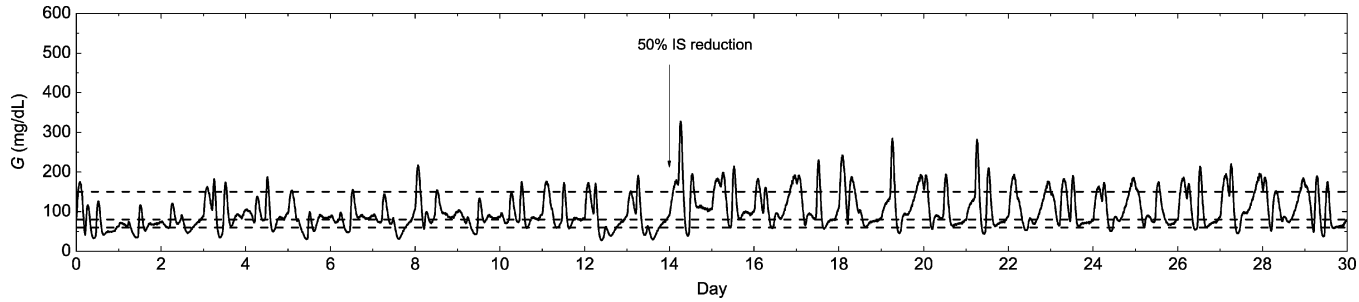

Fig. 7. Performance of Control Strategy E (proposed strategy) for a 30-day period with meals and random variations in the insulin sensitivity profile. (The nominal IS profile is reduced by 50% at the end of Day 14.).

TABLE II
GLUCOSE CONCENTRATIONS (MG/DL) FOR THE 30 DAY SIMULATION

	Mean	**Std. Dev.**	**Min.**	**Max.**
Control Strategy A	196	115	11	513
Control Strategy E	101	44	27	328

TABLE III
FINAL CONTROLLER SETTINGS

Control Strategy	K_c (mU dL/min mg)	τ_D (h)	τ_I^{**} (h)
A	-	-	-
B	-0.096	3.14	1.14
C	-1.02 10^{-7}	2.56 10^3	-
D	-0.177	2.07	1.60
E	-0.496	1.10	3.14

**Average value for insulin sensitivities increase and decrease.

in Table III. Note that Strategy E has the largest absolute value of K_c.

The results for the 30-day simulation in Figs. 6 and 7 and Table II demonstrate that the proposed control strategy is very robust for a wide range of conditions. Additional simulation results demonstrate that Control Strategy E also performs well for a wide variety of conditions that include: different patient weights, meal sizes, initial glucose values; measurement time delays; and different values of tuning parameter $\tau_{\rm sp}$ [21].

VII. PRACTICAL CONTROLLER TUNING METHOD

An important characteristic of a practical glucose control strategy is that it can be easily tuned (or re-tuned) based on a minimal amount of information and/or experimental tests. In this section, a controller tuning method is proposed that is analogous to the approach that is typically used for industrial control systems [8]: first, preliminary controller settings are determined from simple experimental tests. Then they are fine tuned by trial and error, if necessary. However, the simulation results for our glucose control strategy did not require subsequent fine tuning because the preliminary settings were satisfactory.

The proposed controller tuning strategy is based on two types of experimental results that are readily available for subjects who wear continuous glucose sensors: 1) a postprandial response after a meal and insulin bolus (without feedback control) and 2) the response to a step change in the basal insulin infusion rate (for no meal and no feedback control). Test 1) is used to determine a suitable value for $\tau_{\rm sp}$, the time constant of the desired setpoint trajectory in (7). A simple transfer-function model can be obtained from the step response for test 2). Then a model-based controller design procedure is used to determine the PID controller settings based on the transfer-function model. Numerical results and an evaluation of this practical tuning procedure are reported elsewhere [21].

VIII. CONCLUSION

A new glucose control strategy has been proposed based on a novel combination of insulin boluses for meals and an improved PID control algorithm. The key features of the control strategy are 1) a switching strategy for determining when to initiate PID control action after a meal; 2) a novel time-varying trajectory for the glucose setpoint; and 3) a limit on the integral control action that greatly reduces the possibility of insulin overdosing. The new control strategy has been compared to four alternatives in a simulation study based on Hovorka's physiological model [1], [12], and was shown to be superior for both insulin sensitivity changes and meal challenges with poor CHO estimates.

A practical controller tuning procedure has been proposed. The new control strategy was demonstrated to be very robust for a variety of conditions for a simulated, average diabetic subject. It has also been extended recently to include feedforward control action [22].

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