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# Sliding Scale Insulin—Time to Stop Sliding

Irl B. Hirsch, MD

**I**N MOST TEACHING HOSPITALS IN THE UNITED STATES, PRIMARY care first-year residents and medical students learn about sliding scale insulin (SSI), usually from a senior resident. The more experienced resident explains how to prescribe regular insulin every 4 to 6 hours without any scheduled basal or mealtime (prandial) insulin. For the typical patient who is too sick to eat, this results in a roller coaster effect on blood glucose variability due to poor matching of insulin with individual blood glucose patterns. Unfortunately, for the patient who is able to eat, insulin scheduled to be administered based on a bedside capillary glucose measurement is actually administered long after the meal is consumed. Although there are often challenges with hospital logistics in terms of timing of insulin administration in relation to actual food intake, the SSI orders typically do not mention the relationship of the insulin injection as it pertains to a meal, even though at one time, the resident was taught that regular insulin is mealtime insulin. Even worse, SSI, as used here, does not account for the basic principles of insulin therapy.<sup>1-3</sup>

Certainly, SSI has evolved considerably since first described by Joslin more than 70 years ago.<sup>4</sup> For decades, insulin doses were based on the amount of glycosuria (fractional urine glucose testing) for both inpatient and outpatient settings.<sup>5</sup> Although glycosuria is no longer used for this indication, there is still the fundamental problem of clinicians and even health care systems having nonstandardized definitions of SSI. Usually, SSI involves use of regular insulin or a rapid-acting insulin analogue provided without any other scheduled short-acting or long-acting insulin,<sup>6,7</sup> but there are variations. For example, in one recent study, SSI was defined as regular insulin provided prior to meals and at bedtime based on bedside blood glucose testing or every 6 hours for the patient who is not eating.<sup>8</sup> Both clinicians and patients often refer to the use of SSI as the addition of insulin to previously scheduled prandial insulin. So how can the merits or inadequacies of SSI be discussed when they cannot even be defined?

Strategies that do not mimic normal insulin secretion (nonphysiologic regimens such as basal insulin at bedtime only)

can also result in different patient outcomes based on the type of diabetes. With the more common type 2 diabetes mellitus manifested by insulin resistance and relative insulin deficiency, long periods of insulin deficiency or even insulin stacking (when insulin doses are injected at times too close together, resulting in an overlap of action of the insulin) will usually not result in any metabolic crisis. However, in patients with type 1 diabetes mellitus, who generally are not resistant to insulin and have complete insulin deficiency, SSI is more apt to result in clinically significant hyperglycemia, ketosis, ketoacidosis, or hypoglycemia. No data are available for how often this happens but experienced clinicians have observed this repeatedly over the years. Perhaps the lack of major clinical crises when SSI is used in type 2 diabetes mellitus helps to fuel its acceptance in our medical culture.

Surprisingly, there are not more clinical trials assessing the efficacy and safety of SSI. One randomized controlled trial<sup>8</sup> reported superior glycemic control of a more physiologic basal-bolus (replacing both background and mealtime) insulin therapy compared with SSI, but the SSI group received a much lower dose of daily insulin (12.5 U/d vs 42 U/d for basal-bolus), potentially biasing the results. Nevertheless, inpatient diabetes therapy has gained interest this decade due to both interventional and observational studies, suggesting that hyperglycemia in hospitalized patients can result in deleterious outcomes.<sup>9</sup> There is also increasing literature to support that hypoglycemia in the inpatient setting, especially for critically ill patients, is more dangerous than originally appreciated.<sup>10,11</sup>

Appropriate insulin nomenclature includes 3 components: basal insulin (inhibits hepatic glucose production overnight and between meals), prandial (also called bolus or mealtime) insulin (promotes glucose disposal into muscles from food consumption), and correction-dose insulin.<sup>1</sup> This latter component is often confused with SSI, yet it is quite different. Correction-dose insulin is usually administered as an addition (or supplement) to the usual dose of mealtime insulin as a specific algorithm based on total daily dose of insulin or patient weight. SSI is defined (for the purpose of this Commentary) as it is used in the traditional sense: an

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amalgamation of all 3 insulin components that has neither theoretical basis nor evidence for effectiveness. SSI traditionally would include regular insulin or a rapid-acting analogue provided to treat hyperglycemia after it had already occurred. Thus, SSI is a reactive as opposed to a proactive strategy.

Often with SSI, no insulin is provided (or a minimal amount) for premeal glucose levels within target range, resulting in a subsequent blood glucose level greater than 300 mg/dL (16.7 mmol/L).<sup>6</sup> The other fundamental problem is that the traditional SSI regimen assumes that all patients have similar insulin sensitivities or no change in insulin sensitivity during different stages of acute illness. Clearly, this assumption is incorrect. Therefore, the differences between SSI and correction-dose insulin are not subtle ones as they encompass an entire culture of insulin philosophy.

Perhaps most importantly SSI, as defined in the traditional sense, has no track record of effectiveness. A MEDLINE search of 52 trials from 1966 to 2003 did not reveal even 1 report of benefit from SSI.<sup>12</sup> In fact, the concerns of this nonsensical practice were noted 45 years ago.<sup>13</sup>

So why does the practice of SSI continue to flourish in academic medical centers and community hospitals? Is it simply tradition or because it is too difficult to change a culture of mediocre hyperglycemia management? SSI is clearly convenient and straightforward to administer, but it has not been shown to provide benefit and it may induce harm. SSI is a relic of past generations of ineffective and potentially dangerous glucose management that is not evidence-based and does not attempt to mimic normal physiology. Moreover, the use of scheduled basal, prandial, and correction-dose insulin in the hospital is poorly studied, constituting the primary reason for the continued popularity of SSI. Clearly, more rigorous clinical trials are needed that compare SSI with more physiologically based insulin regimens—not only in terms of glycemic control but also with respect to safety and effectiveness in the hospital.

A hospital-wide protocol has the advantage of standardizing hyperglycemia management in addition to encouraging all clinicians to think about appropriate strategies for physiologic insulin replacement. Correction-dose algorithms based on total daily dose or body weight should be implemented.<sup>14,15</sup> Moreover, given the longer pharmacodynamics of regular insulin (as compared with the rapid-acting analogues) and the attendant risk for both hyperglycemic exposure immediately after the injection and then hypoglycemia later due to insulin stacking,<sup>1</sup> one of the rapid-acting analogues (aspart, glulisine, or lispro) is preferable for use as the correction dose.

As nonsensical as SSI is, even more interesting is the unusual practice of sliding scale basal insulin. Although there are no comments about this in the literature, this practice has occurred using neutral protamine Hagedorn insulin and

insulin glargine. Why would a clinician try to correct acute hyperglycemia with an insulin preparation with duration of activity as long as 24 hours? This use of sliding scale basal insulin is a sad commentary about how poorly as a group clinicians understand basic principles of insulin therapy.

At the University of Washington, a system-wide protocol has been followed that has worked well and perhaps as importantly, taught physicians and physicians in training the basic principles of insulin therapy.<sup>14</sup>

Medical professionals do not use sliding scale penicillin for fever or sliding scale oxygen for pulmonary edema. It is time to discontinue amusement park diabetes therapy so that decades from now clinicians are still not trying to abolish an illogical treatment. Perhaps next July or the following summer, when the senior resident is explaining to the intern hyperglycemia management for a newly admitted patient with pneumonia, the discussion will revolve around basal insulin, prandial insulin, and correction-dose insulin based on a protocol that all hyperglycemic patients receive throughout the entire health care system.

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