Featured Cases: Strategies And Devices For Improving Self-Monitoring

Our guest author is Susan Porter, MSN, CRNP, CDE, Clinical Nurse Practitioner and Certified Diabetes Educator at the Johns Hopkins University School of Medicine in Baltimore, Maryland.

After participating in this activity, the participant will demonstrate the ability to:

- Describe how the use of SMBG data can guide the care of a T2DM patient.
- Identify how SMBG can be used to motivate the patient to improve his or her lifestyle.
- Recognize when anemia can interfere with SMBG data.

This discussion, offered as a downloadable audio file and companion transcript, covers the important topic of Strategies and Devices for Improving Self-Monitoring in the format of case-study scenarios for the clinical practice. This program is a follow up to the Volume 1, Issue 5 eDiabetes Review newsletter—Strategies and Devices for Improving Self-Monitoring.

Unlabeled/Unapproved Uses
Susan Porter has indicated that there will be no references to unlabeled/unapproved uses of drugs or products.

Faculty Disclosure
Susan Porter, MSN, CRNP, CDE discloses that she has no financial relationships with commercial supporters.

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Clinicians do not have a sufficiently current knowledge base to effectively consult patients about potential T2D therapeutic advances.

Clinicians do not adequately understand or treat to control CVD risk factors in patients with T2D.

Clinicians do not appropriately intensify therapy as necessary to maintain glycemic control.

Clinicians are not aware of and/or are not implementing strategies to maximize the value of SMBG readings to improve patient outcomes.

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MR. BOB BUSKER: Welcome to this eDiabetes Review Podcast.

Today’s program is a follow-up to our newsletter on Strategies and Devices for Improving Self-Monitoring of Blood Glucose, or SMBG. With us today is that issue’s author, Susan Porter, a Clinical Nurse Practitioner and Certified Diabetes Educator at the Johns Hopkins University School of Medicine in Baltimore.

eDiabetes Review is jointly presented by the Johns Hopkins University School of Medicine, and the Institute for Johns Hopkins Nursing. This program is supported by educational grants from Eli Lilly and Company, AstraZeneca, and Novo Nordisk, Inc.

Learning objectives for this audio program include:
- Describe how the use of self-monitoring data can guide the care of type 2 diabetes patients.
- Identify how SMBG can be used to motivate patients to improve their lifestyle.
- Recognize when anemia can interfere with the accuracy of SMBG data.

Susan Porter has indicated that she has no financial interests or relationships with any commercial entity whose products or services are relevant to the content of her presentation, and that her discussion today will not reference the unlabeled or unapproved uses of any drugs or products.

I’m Bob Busker, managing editor of eDiabetes Review. Susan Porter, thank you for joining us today.

SUSAN PORTER: Thank you for having me, Bob. It’s a pleasure being here.

MR. BUSKER: In your newsletter issue Susan, you reviewed recent publications on the benefits of structured SMBG, new recommendations for fasting and post-prandial glucose levels, and the new standards for glucometers. What I’d like to focus on today is how these data can be translated into actual practice. So to start things off, please describe a patient for us.

SUSAN PORTER: TS is a 51-year-old man referred by his primary care provider for evaluation and management of his recent-onset type 2 diabetes. His diabetes was diagnosed six months earlier on routine blood work, which was done at the time of his annual physical exam. His glucose at the time of diagnosis was 341 and his HbA1C was 10.2 percent. His weight at the time of diagnosis was about 183 pounds.

He had been inactive for the previous eight months after wrist surgery to repair a ligament, and during that time he had had about a 12-pound weight loss, which he attributed to muscle weight loss.

He also had a history of prediabetes, with an HbA1C of 6.3% and a fasting glucose in a diabetes range at 172. He is currently on Glipizide XL 10 mg daily and metformin 1000 mg bid. His most recent HbA1C was 7.8%.

His history is significant for having had pancreatitis in 2005 and 2008 that is apparently felt to be alcoholic in nature, and he continues to have two alcoholic drinks per day. He’s had been testing his glucose levels one to three times daily since his diagnosis. He reports a fasting glucose range typically 120 to 140. He noticed an improvement over the past few weeks since he increased his physical activity.

He had a few episodes of hypoglycemia, with the lowest glucose of 46 when his medication was initially increased from 5 mg to 10 mg. This episode did not require any outside assistance.

He did not bring his glucometer to the office visit today but did bring a handwritten log. His fasting glucose range is 46 to 140, most often between 110 and 130; midday readings, which were done sometimes before or after lunch, were 65 to 220, a typical range being 140 to 170; and bedtime, 70 to 158.

He has been eating two fruits and vegetables daily, two red meats servings per week, he moderates his carb intake, which is predominantly high in fiber. He is doing aerobic exercise, either running or working out with weights, for 60 to 70 minutes three to four times per week, and drinks two alcoholic drinks a day.

MR. BUSKER: To summarize: we have a 51-year-old male, on a sulfonylurea and metformin, with an A1c of 7.8. He’s had pancreatitis and a few episodes of hypoglycemia, and he’s come in without his
glucometer but he’s given you a written log. First question: what’s your A1C goal for this patient?

**SUSAN PORTER:** My A1C goal for this patient would be 6.5% to 7%. I determine that goal because he is young at 51, and he’s otherwise healthy. He has no history of heart disease or no history of a TIA or a stroke, and the lack of comorbidities decreases the risk associated with hypoglycemia. Although we want to avoid hypoglycemia as much as possible, the risks associated with hypoglycemia are much greater in patients with comorbidities such as heart disease and stroke.

**MR. BUSKER:** Now you don’t have his glucometer to work from, you’ve got to work from his handwritten log. How does that change the flow of his visit?

**SUSAN PORTER:** I really encourage my patients to bring their glucometer to each visit. At the diabetes center we have software that allows us to download most of the commonly used glucometers, and that way you can create a visual guide for what’s going on with the patient’s glycemic control.

When I have situations such as this where the patient did not bring their glucometer but brought a handwritten log, or if the patient is using the glucometer that we’re unable to download, what I like to do is spend the first few minutes of the office visit purging about two weeks of their glucose data onto an Excel spreadsheet and getting the average glucometers for fasting, midday, and bedtime or getting the averages for whatever time of day they’re taking their glucose readings. Usually this takes me a few minutes, and often I’m unable to ask other history questions while I’m downloading this information into an Excel spreadsheet.

Another thing you could do is ask your patient, depending on how comfortable they are with the computer, to do this for you before the visit or you could ask a family member to do it. Bringing a glucometer to the clinic really does make a big difference in the clinical momentum and clinical inertia, since the primary care provider is more likely to make a change in medication if he or she is able to visualize and easily interpret the blood glucose data to guide therapy.

In this situation, when I’m able to download all the information, I found that his average fasting glucose was 117, midday was 169, and bedtime was 125.

**MR. BUSKER:** So based on those numbers, what was your impression of his glycemic control?

**SUSAN PORTER:** Overall I thought he was doing well, but I was concerned about his hypoglycemic episodes. He had reported they occurred infrequently since his last visit, but he’s had a glucose as low as 46. For this reason I thought we could decrease his medication, particularly the Glipizide XL, and this would decrease his risk of hypoglycemia. A point-of-care HbA1c at the time of his visit was 5.7%. As his A1 goal was between 6.5% to 7%, the 5.7% was a little low.

According to the findings by Wei, et al., which were reviewed in the newsletter, his fasting glucose goal should be between 139 to 147 with the target being about 139. His post-meal glucose target should be 170 with the range 163 to 170, and bedtime 151 with the range of 139 to 162.

You may notice that these glucose targets are different than the current fasting glucose goals we commonly give in practice. Current glucose targets are anywhere from 80 to 120 or 90 to 130. The post-prandial glucose is in line with the current recommendation, a glucose target of 160 or less; sometimes we go up to 180 or less.

**MR. BUSKER:** So your thought was to reduce his risk of hypoglycemia by decreasing his dose of the sulfonylurea. Did you actually make medication adjustment recommendations?

**SUSAN PORTER:** I recommended that he decrease his dose of glipizide XL to 5 mg daily. This reduction will decrease his risk of hypoglycemia. He reported the hypoglycemia was occurring infrequently but have been as low as 46. With his recent increase in physical activity and improvement in diet, I thought he would be able to maintain his HbA1C goal of less than 7 with the metformin 1000 mg bid and the glipizide XL 5 mg daily. Glipizide is a sulfonylurea, and as you probably know, sulfonylurea, that class has an increased risk of hypoglycemia.

**MR. BUSKER:** What other recommendations did you have for this patient?

**SUSAN PORTER:** The other recommendations I had for him was to continue his self-monitor blood glucose checks, continue his healthy diet and exercise. We discussed his glucose log and reviewed the averages
for fasting, midday, and bedtime, and discussed how exercise improves his glycemic control.

I find that when I can show the patients their averages at the different times of the day, it also allows them to visualize what’s going on and how food and exercise can play a part in helping to control their glucose after meals. He should continue to check his glucose occasionally after his largest meal. If he notices his postprandial glucose levels start to increase, he should try to increase his physical activity either before or after a meal. He should use the self-monitor glucose data to keep himself motivated to continue the healthy diet and exercise.

And as you might recall from the newsletter, the STEP study found that the use of structured self-monitor blood glucose improves glycemic control, and then the findings by Friedman, et al, which found that two years after the STEP study, clinicians who were using the self-monitored blood glucose tool in clinical practice felt it improved the quality of time they spent counseling their patient.

MR. BUSKER: So I think that what you’ve been talking about clearly illustrates the value of SMBG data. Not only does it provide information that helps guide the clinician about making medication changes, but it also directly helps the patient. It’s a feedback mechanism. They see their numbers and that’s going to give them a better grip on what their lifestyle choices, particularly diet and exercise, what those choices are doing to help them maintain control.

So let me ask you, again, about this patient: you’ve changed his regimen, what would your recommendation be should his control start to deteriorate?

SUSAN PORTER: Since he has a history of pancreatitis, he, unfortunately, is not a candidate for GLP-1 agonists or DPP-4 inhibitors, although this has been an ongoing controversy. Pancreatitis risk is not higher in patients with diabetes patients who take incretins. Patients with type 2 diabetes who took incretin-based drugs had a risk profile for acute pancreatitis similar to that of patients who were on sulfonylurea treatment. According to the study in the BMJ April 15, 2014, researchers said the findings, though consistent with those of other studies, warrant further investigation.¹ However, pancreatitis is still listed as a drug side effect and most clinicians would not be comfortable having a patient continue it, especially if he has other risk factors for pancreatitis such as his alcohol use. I would hope his self-monitored blood glucose data would keep him motivated, which was one of the findings by Chanel, et al in the review of the recent studies on self-monitoring of blood glucose in type 2 diabetes. If needed, he could increase his glipizide XL back to 10 mg should his glucose levels start to increase.

MR. BUSKER: A link to that recent BMJ study can be found in the transcript version of this podcast. Susan, I want to thank you for that case and discussion, and let our listeners know that we’ll return, with Susan Porter from Johns Hopkins, in just a moment.

BOB BUSKER: This is Bob Busker, managing editor of eDiabetes Review.

If you found today’s program on iTunes or on the web, please be sure to subscribe. This podcast is part of Johns Hopkins eDiabetes Review, an educational program providing monthly activities certified for CME credit and nursing contact hours. eDiabetes Review provides expert commentary and useful practice information for clinicians treating patients with type 2 diabetes.

For additional information, or to subscribe to receive our newsletters and podcasts without charge, please visit www.ediabetesreview.org. Thank you.

MR. BUSKER: Welcome back to this eDiabetes Review podcast. Our guest today is Susan Porter, a Clinical Nurse Practitioner and Certified Diabetes Educator at the Johns Hopkins University School of Medicine. And our topic is Strategies and Devices for Improving SMBG. We’ve been looking at how the information in her newsletter issue can impact clinical practice. So to continue, Susan, bring us another patient, if you would please.

SUSAN PORTER: DD is a 67-year-old, morbidly obese woman with a height of 5’4” and weight of 314 pounds. She presents to the diabetes clinic for the management of her type 2 diabetes. The diagnosis was initially made in 2007 while she was in the hospital for a knee replacement.
She was initially treated with diet and exercise, but then two years after her diagnosis, her glycemic control started to deteriorate, and she was started on metformin and glipizide by her primary care provider.

At her first visit to me, her HbA1C was 8.8, and she was started on sitagliptin 100 mg daily. She checks her glucose level two times daily fasting and in the evening, two to three hours after dinner, which is also her largest meal. Her fasting glucose average is 130 and postprandial range is 180 to 200. A review of her diet reveals she eats three meals a day. A typical breakfast is a protein shake with fruits and vegetables; snack is usually a Glucerna shake; lunch is sandwich or salads; and dinner consists of meat and two vegetables. She tries to avoid rice, pasta, and potatoes, and she described her diet as low carb.

Her physical activity includes walking daily for ten minutes. She’s been trying to lose weight with improving her diet and exercise, but her exercise is limited due to her history of COPD.

MR. BUSKER: A 67-year-old obese female with COPD that limits her ability to exercise. She’s on metformin and a sulfonylurea and sitagliptin. What’s your impression of her control on this regimen?

SUSAN PORTER: My impression of her control is that her A1C is too high. I would like her A1C to be between 6.5% and 7%. Currently, her fasting glucose is good at 130. Her fasting glucose goal should be about 139 with a range of 139 to 147, and this is based on the findings by the article that was reviewed by Wei, et al. Her postprandial range currently is 180 to 200, and that is a little high according to that same article, which found that with an HbA1C goal of 6.5 to 6.99%, the postprandial goal should be 170 with a range of 163 to 177.

MR. BUSKER: So what were your treatment recommendations for this patient?

SUSAN PORTER: Her weight is an obvious concern, with a BMI of 54. We discussed the need for her to lose weight to control her diabetes. She was motivated to lose weight but really limited by her physical activity because of her history of COPD. We discussed the option of bariatric surgery, but she was not willing to consider that at this time.

I recommended switching from her DPP-4 inhibitor to GLP-1 agonist. We discussed that often modest weight loss can be seen with the GLP-1 agonist. She agreed to make the switch and returned for a follow-up three months later.

MR. BUSKER: So three-months after she switched from the DPP-4 inhibitor to the GLP-1 agonist, had she improved?

SUSAN PORTER: At her follow-up she had lost 16 pounds. She brought her glucometer, which showed much improvement in her glycemic control. She now had a fasting glucose ranging typically 85 to 90; prelunch and predinner, 70 to 90; and after meals was 100 to 120. Her HbA1C had dropped to 6.2%.

Her glucose readings were actually too low at this point. She mentioned she was happy to see the lower numbers with her self-monitored blood glucose data, and it gave her instant feedback, especially about her diet.

MR. BUSKER: So her numbers have gone from too high to too low. What did you recommend?

SUSAN PORTER: DD was very excited about her weight loss and improved A1c. At this point, I recommended that she discontinue her glipizide XL because of the hypoglycemia, which would hopefully allow her to lose even more weight. You might know that sulfonylureas are often associated with weight gain. I recommended that she continue to check her glucose levels fasting and two hours after her largest meals. I had given her instructions that if her fasting glucose readings rose above 140 or postprandial above 160, she should restart her glipizide at 5 mg, which was a lower dose than she had previously been taking.

MR. BUSKER: Thank you for that case and discussion, Susan. I think we’ve got time for one more patient — so if you would please.

SUSAN PORTER: KI is a 67-year-old man with a history of renal transplant-related diabetes, anemia, hypertension, and hyperlipidemia. He had advancing chronic kidney disease and received a living-donor kidney from his daughter three months before his visit to the diabetes center.

He was doing well after his transplant, but his blood glucose did go up with the addition of medications for
MR. BUSKER: A kidney transplant patient — he’s an older male, and besides his diabetes, he’s got anemia, hypertension, and hyperlipidemia. He’s on a mixed insulin regimen, and you did not have an A1c for him. What did you do?

SUSAN PORTER: I was unable to get a HbA1C at the time of his visit because we were just a few days shy of the three-month interval for the test. Often insurance companies will not pay for HbA1C to be done before that three-month point. So since I did not have an HbA1C, I had to rely on his self-monitoring blood glucose data to predict his HbA1C.

We were able to print out his glucose data and get an overall average glucose of 153. Now, this number, along with the averages at specific times of day, a standard deviation, the highest and lowest glucose readings, and the number of tests the patient actually performs during a specific period of time, is all provided from a glucometer printout. It’s very useful information. From this information I was able to determine that his average glucose was likely a very good predictor of his overall control. And using his average glucose of 153, I predicted his HbA1C would be around 7%.

The other important observation noted with his glycemic control was that with the averages throughout the day, his control was very consistent throughout the day, and that was noted visually as a line drawn from one average to the next, which showed a pretty flat line throughout the day.

So based on his self-monitoring blood glucose data, I felt comfortable making recommendations on insulin adjustment, even without his HbA1C results. I recommended that he increase his glargine from 12 units to 13 units, continue his current dose of apart 4 units before each meal, and continue to check his glucose levels three times a day, fasting and before each meal. I told him that if he had any fasting glucose readings less than 90, to decrease his glargine back to 12 units.

I also referred him to the lab to get the HbA1c done once it was beyond that three-month period, and I got that back about a week later, and he did have a HbA1c of 7.1 which was very close to what I had predicted of 7.0%.

MR. BUSKER: As you mentioned, this patient has a history of anemia, which can sometimes alter HbA1C values. Talk to us a little bit about why that happens, if you would, please.

SUSAN PORTER: Because of the way the HbA1c is measured, HbA1C levels are affected by the presence of variant hemoglobins, hemolytic anemias, and nutritional anemias. HbA1C levels in anemia patients are often affected. Their values are influenced by red blood cell survival; thus, falsely high levels in relation to a mean blood glucose value can be obtained when red blood cell turnover is low, resulting in disproportionate number of older red blood cells. This problem can occur in patients with iron, vitamin B12, or folic acid deficiency.

On the other hand, rapid red cell turnover leads to greater proportion of younger red blood cells and falsely low A1c values. Examples include patients with thalassemia, or hemolysis or patients treated with erythropoietin.

MR. BUSKER: In patients with conditions that might contribute to falsely high or falsely low A1c values, what can be done to get the most accurate readings?

SUSAN PORTER: Depending on the methodology, the values may be high or low in patients with abnormal hemoglobins. However, many methods for measuring A1C are no longer affected by hemoglobin variance. The National Glucohemoglobin Standardization
Program contains current information about substances that interfere with A1C test results.

MR. BUSKER: A link to the National Glucomhemoglobin Standardization Program website can be found in the transcript version of this podcast. Now Susan, that information from that website is about substances that interfere with A1C test results. What about other conditions, aside from anemia?

SUSAN PORTER: HbA1C values may be falsely elevated or decreased in those with chronic kidney disease. False elevations may be due in part to the analytical interference from carbamylated hemoglobin formed in the presence of elevated concentrations of urea, leading to false elevations in A1C levels with some assays. False decreases in measured A1C may occur with hemodialysis and altered red blood cell turnover, especially in the setting of erythropoietin treatment.

As I noted earlier, this patient had a normal hemoglobin and hematocrit with his most recent CBC, and, fortunately, he was using a glucometer that was shown to be accurate in patients with anemia. If this was not the case, it would be important to take all that information into consideration when analyzing his control and making recommendations.

It was noted in the study by Brang, et al, which was reviewed in the newsletter, that hematocrit effect was seen with many of the glucometers evaluated, and bias was greatest in the lower and upper ranges of the hematocrit, the lower range being less than 35 percent and the upper range being greater than 55 percent.

MR. BUSKER: Getting back to the patient — you recommended that he increase his glargine from 12 units to 13, but to cut it back to 12 if he had any fasting glucose readings less than 90. At his follow-up three months later, what did you find?

SUSAN PORTER: He returned three months later without any new complaints and reported he was feeling good. A review of his glucometer printout showed a fasting glucose average of 112; prelunch 131; predinner 180; and bedtime 135. We reviewed the glucometer data together and explored what might have caused the spike between lunch and dinner. He had admitted missing a few of the doses of his aspart insulin at lunchtime because he was away from his house around that time and forgot to travel with his aspart. He assured me that when he takes his lunchtime aspart at the current prescribed dose of 4 units, this does control his glucose.

His HbA1C at the time of his visit was 6.2. He denied any episodes of hypoglycemia, and none were noted on his self-monitor glucose review. For this reason I did not make any changes in his medication at the time of his visit. I saw him again recently, and he continues to do well with a very stable glycemic control. His most recent HbA1C was 6.7 percent and his pre-dinner glucose average was down from 180 to 140.

MR. BUSKER: Very good. Thank you, Susan, for bringing us these patients and sharing your insights about their management. I’d like to wrap things up by reviewing the key points of our discussion today in light of our learning objectives. So to begin: how the use of SMBG readings can guide the care of a diabetes patient.

SUSAN PORTER: In the first case, the patient was having hypoglycemia with glucose levels above 46 and his glipizide was decreased. In the last case we discussed, I noticed that his predinner glucose readings were elevated, and we discussed specifically what was happening at that time of the day, and it turns out he was missing some of those doses of aspart. In the last case it was also determined that, as predicted, HbA1C was based solely on his self-monitoring blood glucose data.

We found that SMBG data was very useful for guiding the clinician in medication and dosage adjustment during a patient’s visit.

MR. BUSKER: Our second learning objective: how SMBG readings can motivate the patient for lifestyle improvement.

SUSAN PORTER: Reviewing the SMBG data with the last patient, he was able to see how his missed doses of aspart were affecting his control predinner. And in the second case, the patient noticed the improved control with her self-monitoring blood glucose data, and that really helped to keep her motivated and, in turn, helped with her weight loss.

The first case, the patient made a commitment to exercise and improve his diet, and his diabetes improved which was noted not only in his HbA1C
but also in his SMBG data and will hopefully continue to serve as a constant reminder of the benefits of exercise and diet.

MR. BUSKER: And finally: how anemia can interfere with SMBG data.

SUSAN PORTER: In the last case the patient had a history of anemia and kidney disease. We discussed how anemia can affect the SMBG result, which is going to be an issue with some glucometers on the market. Anemia can also affect the HbA1C result.

It’s important for clinicians to know that certain glucometers will be more accurate with the presence of anemia than others, and this was reviewed in the newsletter.

MR. BUSKER: Susan Porter, from the Johns Hopkins University School of Medicine, — thank you for participating in this eDiabetes Review podcast.

SUSAN PORTER: Thank you, and I’ve enjoyed presenting this information today.

MR. BUSKER: To receive CME credit for this activity, please take the post-test at www.ediabetesreview.org/test.

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2. National Glucohemoglobin Standardization Program