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How to Take This Course

Please take a look at the steps below; these will help you to progress through the course material, complete the course examination and receive your certificate of completion.

1. **REVIEW THE OBJECTIVES**

   The objectives provide an overview of the entire course and identify what information will be focused on. Objectives are stated in terms of what you, the learner, will know or be able to do upon successful completion of the course. They let you know what you should expect to learn by taking a particular course and can help focus your study.

2. **STUDY EACH SECTION IN ORDER**

   Keep your learning "programmed" by reviewing the materials in order. This will help you understand the sections that follow.

3. **COMPLETE THE COURSE EXAM**

   After studying the course, click on the "Course Exam" option located on the course navigation toolbar. Answer each question by clicking on the button corresponding to the correct answer. All questions must be answered before the test can be graded; there is only one correct answer per question. You may refer back to the course material by minimizing the course exam window.

4. **GRADE THE TEST**

   Next, click on "Submit Test." You will know immediately whether you passed or failed. If you do not successfully complete the exam on the first attempt, you may take the exam again. If you do not pass the exam on your second attempt, you will need to purchase the course again.

5. **FILL OUT THE EVALUATION FORM**

   Upon passing the course exam you will be prompted to complete a course evaluation. You will have access to the certificate of completion **after you complete the evaluation**. At this point, you should print the certificate and keep it for your records.

About The Author

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Jacquelin Deatcher NP, CDE is an Adult Nurse Practitioner and Certified Diabetes Educator, with over 20 years' experience in diabetes clinical management and self-management education. After 23 years with a large multi-specialty medical group in White Plains, New York, she now works in the rural setting of Attica, in Western New York.

Her goal is to offer diabetes services in an area where there is a significant need. She is currently in an independent and primarily fee-for-service practice, providing both individual consultation and group education. Ms. Deatcher believes that nurses have a very significant opportunity and responsibility to empower those with diabetes to attain and maintain maximum wellness.

This course was updated by Ms. Deatcher in October 2007.

Introduction

According to the Webster Encyclopedic Dictionary, a monster is defined as one "that is looked upon with horror on account of extraordinary crimes, deformity or power to do harm." By the end of this course, you will see that diabetes can, indeed, be looked upon as a monster, and that it's time the health care industry made more serious efforts at its taming.

It is currently estimated that there are approximately 21 million people in the United States with diabetes, and it is estimated that a third of this population do not know they have it (Centers for Disease Control and Prevention [CDC], 2006). A further breakdown reveals that 8.7% of all people age 20 or over have diabetes and 18.3% of all people 60 or over have this condition (CDC, 2003). In addition, it is estimated that there are another 41 million people in this country with "pre-diabetes;" in other words, they are at significant risk of developing this chronic condition (CDC, 2006). Some more recent estimates put this number as high as 54 million. The incidence of diabetes is rising rapidly, becoming a significant epidemic in this country. According to the CDC (2006), in the last 15 years, the number of people in the United States with diagnosed diabetes has more than doubled.

We are not the only country seeing a rapid rise in the incidence of diabetes. There are an estimated 240 million people worldwide with diabetes, and this number is expected to rise to 380 million by the year 2025. This is the first time that a non-infectious disease is recognized as posing as serious a threat to world health as infectious diseases like HIV/AIDS, Tuberculosis and Malaria. Diabetes kills as many people worldwide as does HIV/AIDS. In December 2006, the United Nations passed a resolution proclaiming November 14, 2007 (and every November 14 thereafter) as World Diabetes Day. This is to encourage all countries to put policies in place to better diagnose and care for those with diabetes (International Diabetes Federation, 2007). The official logo for World Diabetes Day is a blue circle that symbolizes life, health, and unity. The Unite for Diabetes campaign endeavors to draw the global diabetes community together to effectively combat this epidemic. Information regarding this campaign can be accessed at www.unitefordiabetes.org.

Clearly, diabetes has reached pandemic proportions worldwide. Why is this?

First let's review the main risk factors for Type 2 diabetes, which accounts for 90-95% of all cases of diabetes. These risk factors include aging, having a family history of diabetes, and being overweight (and having a sedentary lifestyle). In addition, certain ethnic populations, such as Native Americans, Latinos, Asian Americans, and African Americans, carry a higher risk of Type 2 diabetes. First, we are all at risk of developing diabetes as we age. If we live long enough, we age, and obviously there is no control for that risk factor. Second, Type 2 diabetes tends to run in families such that if an individual has a family history of diabetes, his or her risk is increased. Since we can't pick our parents, there is also no control for that risk factor. However, being overweight (and sedentary) is something we **can** control, and this will be discussed later. With these risk factors in mind, it is no wonder there is an alarming increase in the incidence of diabetes in this country. Consider the aging population, the rapid growth in obesity, and the growing ethnic populations of the United States, and one can see why diabetes has reached the epidemic it has in this country.

According to the Behavioral Risk Factor Surveillance System, the incidence of diabetes, initially reported at 4-6% for most states in the U.S. in 1990, was reported at greater than 6% by the year 2000 (CDC, 2003; Mokdad et al., 2000). There are some estimates that that number will reach 10% by the year 2010. According to a recent report, one out of every three Americans born in the year 2000 will develop diabetes at some point in their lives (Naravan et al., 2003). Why is this important and what can we do about it?

The purpose of this course is to review and update nurses' information and understanding of diabetes care and self-management education.

Objectives

- Distinguish Type 2 Diabetes from Type 1.
- Discuss the pathophysiology of Diabetes.
- Describe insulin resistance, known as the Metabolic Syndrome.
- Discuss the relationship between hypertension and hyperglycemia relative to heart disease.
- Identify dietary and exercise issues in the treatment of Diabetes.
- Discuss medications used in the treatment of Diabetes.

Don't Be Fooled By a Sweet Face: *The Impact of Diabetes*

If high blood sugar hurt, it's unlikely that diabetes would be the monster it is. Unfortunately, it is possible to have diabetes for years without symptoms; and worse yet; it is possible to have significant complications of diabetes without knowing it. Diabetes is the 6th leading cause of death and a major cause of disability in the U.S (CDC, 2005). The financial costs of diabetes are staggering. Direct and indirect costs (medical costs as well as disability, lost time on the job, etc.) were estimated at $132 billion in this country in 2002 (CDC, 2003). This means that more than one in ten U.S. healthcare dollars and one in four Medicare dollars is spent on diabetes (CDC, 2002).

The human costs of diabetes are tremendous as well. According to the CDC, every single day, more than 2000 people in this country are diagnosed with diabetes. Every single day, 70 people in this country go blind because of diabetes. Every single day, more than 100 people with diabetes are diagnosed with end-stage renal disease. Every single day, more than 200 people undergo an amputation due to diabetes, and every single day, diabetes contributes to more than 500 deaths in this country (CDC, 2005).

The major complications of diabetes can be divided into those that are microvascular (small vessel) and macrovascular (large vessel). The common microvascular complications include vision loss, renal failure and lower extremity amputations. These tend to be the most feared by the general public. However, it's the macrovascular complication, cardiovascular disease, which poses the greatest threat to one's health.

People with diabetes have 2-4 times the risk of heart disease as those without diabetes (CDC, 2005). Heart disease is the leading cause of death in people with diabetes (CDC, 2005). The person with diabetes but with no known heart disease is at the same cardiovascular risk as the person without diabetes who has already had a heart attack. That point bears repeating: **The person who has diabetes, but no history of heart trouble is at the same risk as the person without diabetes who has already suffered a heart attack** (Malmberg et al., 2000). Diabetes is now considered a heart disease equivalent. In other words, it is time to become more aggressive with all cardiovascular risk factors in this high-risk population.

What's It All About: Pathophysiology Review

Diabetes means, essentially, that the body is having a hard time utilizing food. If I have diabetes, I am taking in my food, but I cannot use all of it, and the food I cannot use begins to build up in my bloodstream, in the form of sugar (glucose). You know the diagrams of blood vessels, cells, etc., but let's look at a simple analogy, one that may be useful in your patient teaching.

In a car, the fuel source is gasoline. The gasoline gets into the fuel tank, but it's not used there. The gas needs to get through the gas line to the motor; that's where the fuel is used. The body is similar, in that our fuel (food) gets to the gas tank (the stomach). The fuel needs to get from there through not **one** gas line, but **many** gas lines (the circulatory system), to not **one** motor, but **many** motors (the cells of the body). Think of the cells of the body like motors. The cells need fuel to enter the motors in order for the motors to function.

But just because the fuel has made it to the motor, will the engine automatically start? No. Another step must happen. In the car, the next step involves the key. One turns the key and ignition occurs and the engine starts, using the fuel. In the body, just because the glucose makes it to the cells, does not mean the cells can use the glucose for fuel. The body needs a key too, and that key is insulin. Insulin is a hormone that is made in the beta cells of the pancreas. Insulin in the blood acts like that key, to unlock the doors, so to speak, and enable the cells to utilize the glucose. One **must** have insulin in the cells to do that job. If I have no insulin, I could eat 5,000 calories a day, which might be fun, but I would starve. I will die if I do not have effective insulin action present to help me utilize my food (Travis, 1992).

So, in the normal order of things, I take in my food, I digest it (break most of it down into glucose), the glucose enters my blood stream and is carried all throughout my body. The right amount of insulin is there to enable the glucose to enter the cells to be used for energy. Now, if something goes wrong with that process, diabetes enters the picture. There are two main types of diabetes: Type 1 (the old juvenile onset diabetes) and Type 2 (the old adult onset diabetes). The other terms used, including insulin dependent and non-insulin dependent diabetes, were confusing, since many people with non-insulin dependent diabetes need insulin for adequate blood glucose (BG) control. So the nomenclature of choice today is Type 1 and Type 2 diabetes. Using these Arabic numerals rather than Roman numerals will prevent anyone from ever being confused by thinking they might have Type 11 diabetes.

In Type 1 diabetes, there is a lack of insulin. Thought to be due to a combination of genetic predisposition and environmental factors, the body makes antibodies against its own beta cells, destroying their ability to make insulin. This person must have daily insulin injections to survive. Our understanding of Type 2 diabetes, in recent years, has undergone some change. It was once thought that the body was just not making enough insulin. Therefore, we used a sulfonylurea type oral hypoglycemic to push the pancreas to make more insulin. Type 2 diabetes was thought of as a situation where there was a partial lack of insulin. The body was making some, but not all, of the normal amount of this hormone.

We now understand this differently. The person with Type 2 diabetes, at least during the first few years after diagnosis, usually has plenty of insulin, that is, perhaps several times the normal amount of insulin. And the insulin itself is just fine, it is not defective. However, the body becomes resistant to the effect of insulin (hence the term insulin resistance). It is as if the doors to the cells are closing. The cells are, in effect, saying: "I see you, insulin, but I won't let you do your job." This closing of the doors, so to speak, results in the body having to work harder to make more insulin to try to overcome this resistance. When the insulin resistance is greater than the amount of insulin the body is able to make to overcome this resistance, glucose begins to accumulate in the blood stream, and BG levels rise.

What's important to understand here is that the person with a new diagnosis of diabetes didn't just "get" diabetes. This process begins a decade or more before the possible diagnosis of diabetes can be made. The first step in the process is the body becoming a bit insulin resistant. However, the pancreas can compensate by making more insulin (banging the doors down, so to speak), keeping BG in the normal range. No one knows anything is wrong because BG levels are still normal. As the years go by, more and more doors are closing (increasing insulin resistance), but the pancreas can still overcome this resistance by making more insulin. Still, no one knows anything is wrong. Someday, again, a decade or more after the initiation of this process, enough doors are closing, and the pancreas can't make quite enough extra insulin anymore to overcome this resistance. This is the point at which BG levels begin to rise. Hopefully, this is when it's discovered, however, many people have diagnosable diabetes for quite some time before the diagnosis is actually made.

In addition, because this process has been going on for long periods before a diagnosis of diabetes can be made, approximately 50% of people, at the time of diagnosis, already have a diabetes complication (Harris et al., 1992). It has been said that by the time a diagnosis of diabetes is made, "the horse is out of the barn, and we're trying to catch it by the tail." It behooves us, then, to make the diagnosis of diabetes earlier, and to educate people at risk in the ways of prevention.

In an effort to intervene earlier, a new clinical entity has been described: prediabetes. Prediabetes is a time where BG levels are higher than normal, but not high enough to call diabetes. These elevations are either in the fasting BG (Impaired Fasting Glucose), postprandial BG (Impaired Glucose Tolerance), or both. The question was asked a few years ago as to whether clinical intervention during the period of prediabetes could prevent progression to a diagnosis of diabetes. A study by the Diabetes Prevention Program Research Group (2005), answered this question with a resounding "yes!" In addition, one's risk for complications such as cardiovascular disease and retinopathy are present before the actual diagnosis of diabetes can be made, that is, during this time of prediabetes. For a more in-depth discussion of prediabetes, refer to e-leaRN's online course: "Prediabetes: A Wake Up Call" (http://www.elearnonline.net/coursedesc.aspx?ClassID=185&s=52).

Understanding the concept of insulin resistance (described earlier), will help one understand the rationale behind the treatments for diabetes, as well as prevention strategies. Most methods of treatment for Type 2 diabetes (as well as its prevention) center on trying to "open the doors" to the cells. This is attempted through manipulation of medication (some of which attempt to bang the doors down and overcome the resistance, and some try to open the doors from the inside, helping the cells respond better to insulin), diet, exercise, coping with stress, etc. By coming at the task of decreasing insulin resistance in a variety of ways, the results of multiple efforts tend to work synergistically with each other, and it is very appropriate to see an individual with diabetes on several different medications in addition to attending to several different lifestyle interventions.

Another important key to understanding this process is that once the beta cell starts to fail, it will continue to fail. Type 2 diabetes is, by nature, a progressive disease (UKPDS, 1995). What happens initially is the loss of the first phase insulin response (the **initial** outpouring of insulin when a meal is consumed). This loss of the ability to cover a carbohydrate load to the body with enough effective insulin results in a quick rise of BG postprandially. In other words, it's the after meal BG level that rises first (Ramlo-Halsted & Edelman, 2000). Interestingly, we screen people for diabetes with a **fasting** BG. However, as we know now, this is not the first to rise. There are, therefore, times when the oral glucose tolerance test (OGTT) is still an appropriate diagnostic test. Or, what has been called the "poor man's glucose tolerance test," that is, a BG measured 1-2 hours postprandially. Because most of us spend a significant amount of time throughout the day in the postprandial state (especially those of us who consume large meals plus snacks), it becomes necessary to look at postprandial BG levels in the overall management of diabetes.

So why is all this important? What does it matter if one's body is insulin resistant and the BG is elevated? Well, we only fairly recently have the answer to that question. We have already looked at the impact of diabetes in terms of diabetes complications, but let's take a closer look at the connection between BG levels and the magnitude of this impact. We finally have the proof that BG control, over time, matters. The DCCT, or Diabetes Control and Complications Trial (reported on in June of 1993), finally told us what many have suspected for some time. In this study, the subjects with lower BG levels (an average of 70 mg/dl lower than the control group) dropped their risk of long-term complications by an average of 60% (American Diabetes Association [ADA], 2003).

In the DCCT, the control group maintained an average A1c of approximately 9%, where the intensively treated subjects were able to achieve an average A1c of 7% for a mean duration of six years. Those with the lower A1c's did see a decreased risk of complications by about 60%, as mentioned above, but these were mostly microvascular complications. There was a suggestion of cardiovascular (macrovascular) benefit, but the results were not highly statistically significant. Since completion of the study, 93% of the original cohort were continued to be followed (without intervention). Over the next ten years, an interesting pattern emerged. The control group, starting with an A1c of 9%, gradually dropped their A1c's to about 8% ten years later (perhaps the news that metabolic control matters may have influenced their self-care behaviors). The A1c's of the intensively treated group (starting at 7%), gradually began to rise to an average of about 8% as well, by the end of those same ten years (perhaps they "relaxed" a bit after their intensive work during the study). So both groups, ten years after the completion of the DCCT, were at the same A1c level (8%). However, the group which had maintained the lower A1c's during the DCCT **now** saw a 42% decreased risk of all cardiovascular events, and a decreased risk of severe events (e.g., stroke, MI, and death) by 57% (Herman, 2007)! This effect is being called "metabolic memory" by some, in that early intensive control of BG levels leads to cardiovascular protection, years down the road, Others see this as simply less time under the "influence" of elevated BG levels, resulting in fewer complications over time. Either way, it is imperative that our patients know the benefit of tighter BG control.

Because the DCCT only included people with Type 1 diabetes, the question arose as to whether it would be appropriate to apply its conclusions to the much larger population of people with Type 2 diabetes. That question was answered and reported on in September of 1998, when the United Kingdom Prospective Diabetes (UKPDS) Study essentially concluded the same thing. This study included people from the point of diagnosis of Type 2 diabetes and followed them for an average of 10 years. An interesting statistic from this study is that for every one percentage point drop in Hemoglobin A1c (an average drop of 35 mg/dl BG), one's risk for small vessel disease (retinopathy and renal disease, for example) drops by approximately 35% (ADA, 2003a). The bottom line is that **metabolic control matters.**

An interesting observation in relation to insulin resistance is that one doesn't need to have a diagnosis of diabetes to have an elevated risk of cardiovascular disease, as noted earlier. Insulin resistance itself is associated with an increased risk for cardiovascular disease, underscoring the importance of prevention efforts (De Simone, G., Devereux, R. B., Chinali, M., Best, L. G., Lee, E. T., Galloway. J. M. et al., 2007). The insulin resistance syndrome, previously called Syndrome X, then the Dysmetabolic Syndrome, and now the Metabolic Syndrome, encompasses several conditions: obesity, hyperglycemia, dyslipidemia, hypertension, and hypercoagulability. According to the National Cholesterol Education Program Adult Treatment Panel (NCEP ATP III), the formal criteria for the diagnosis of the Metabolic Syndrome are three of the following (National Institute of Health, 2001; De Simone et al., 2007):

> Fasting BG $\geq$ 110 mg/dl (Note: This is likely to change based on 2004 ADA Clinical Practice Recommendations; see the next section of this course for more information on new guidelines.)
>
> Waist circumference > 40" (men), > 35" (women)

BP $\geq$ 130/85 mm/Hg

Triglycerides $\geq$ 150 mg/dl

HDL < 40 mm/dl (male), < 50 mm/dl (female)

It's a Numbers Game: Home Blood Glucose Monitoring and Other Targets

So if we know that numbers are important, what are the numbers and what are the targets?

One may remember the diagnostic criteria for diabetes having been a fasting of 140 or above a few years ago. As we learn more about diabetes and understand the significance of the risk involved before significant elevations in blood glucose occur, it is no surprise that the diagnostic criteria have tightened some over the past few years. The American Diabetes Association Clinical Practice Recommendations for 2004 reflect this change: normal blood sugar is now defined as 70-99 (not 70-109 anymore). This means Impaired Fasting Glucose is now defined as 100-125 (not 110-125 as previously). Let's review normal blood glucose levels and the current diagnostic criteria for diabetes (ADA, 2007):

	Random	**Fasting**	**2 Hours Post Glucose Load on Oral Glucose Tolerance Test (OGTT)**
Normal Blood Glucose level		< 100 mg/dl	< 140 mg/dl
Diabetes Mellitus (on two occasions)	≥ 200 mg/dl (with symptoms)	≥ 126 mg/dl	≥ 200 mg/dl on OGT
Impaired Glucose Tolerance (Prediabetes)			≥ 140 and < 200 mg/dl on OGT
Impaired Fasting Glucose (Prediabetes)		≥ 100 and < 126 mg/dl	

Once the diagnosis of diabetes is made, how do we attain and maintain blood glucose control over time? Home Blood Glucose Monitoring has opened the doors to more effective self-management of diabetes. By checking BG values at varied times of the day, one can get a more complete picture of one's BG control. If a patient were to present with a log of once a day testing, for example, with only fasting readings, one would have a pretty good idea of that individual's BG pattern upon arising in the morning. However, it tells one nothing about the rest of the day. BG levels are constantly changing, being affected by such things as food, physical activity, medication and stress. For example, eating a meal starts one's blood glucose level to rise within minutes. A peak is reached approximately one hour after eating is finished and then returns to preprandial levels within approximately three to four hours. This pattern is repeated throughout the day, with each meal and snack.

A first step in assessing BG control may be to encourage your patient to simply vary the time of day of testing. It is possible for the fasting BG to be the lowest, the average or the highest of the day. Have you ever had a frustrated patient complain that he can go to bed at night with a BG of say, 110, eat nothing, sleep eight hours and wake up the next morning with a BG of 150 or higher? This is actually quite common, and here's why. When one goes to sleep at night (presuming daytime alertness and nighttime sleep), the BG level drops a bit, getting to its normal

low point at about 1-2 am. By 4-5 am (during the dawn hours); however, changes in hormone levels begin to effect a rise in BG (e.g., growth hormone and cortisol). In addition, the liver releases some glucose into the bloodstream in those early morning hours. A common problem with Type 2 diabetes is that the liver sometimes doesn't know when to stop, and there can be a significant release of glucose into the circulation in the early morning hours, resulting in hyperglycemia (hence the "dawn phenomenon").

Because BG values vary throughout the day, it becomes important to check BG readings at varied times of the day to understand the person's individual pattern. The person with high fasting readings but reasonable bedtime readings the night before may be experiencing the dawn phenomenon described above. The person with high fasting levels, however, who goes to bed the night before with significantly elevated BG levels due to late evening snacking, is another story, and the intervention would be quite different. Looking at one's individual pattern of BG levels throughout the day is a starting point that will enable the individual to learn what is effective for him or her in order to attain better BG control. Let's say I test before each meal and at bedtime, and see that most of my readings during the day are okay, but bedtime is consistently high. Now come the "What If's:" What if I eat half a bag of double stuff Oreo cookies instead of a whole bag? What if I have an earlier snack? What if I have a good dinner and no snack afterward? What if I go for a walk after dinner? As I "play," I will learn what I can and cannot get away with. Patients will come back to you saying "You know what I learned? If I do X, my blood sugar goes way up. If I do Y, my blood sugar stays more normal. So I won't do X anymore, or at least I have a choice and understand the consequences, in terms of sugar control." Home blood glucose monitoring is a very powerful tool that can truly put the patient in control of his or her diabetes.

How about testing before or after meals? For quite a long time, it was felt that before meal testing was the most appropriate. However, it is now better understood that postprandial readings are important too. If we consider that many of our patients eat large meals, and perhaps large snacks in addition to those large meals, there is a significant portion of the day spent in the postprandial state. The larger the meal, the longer the BG may remain elevated after that meal. Some of our patients may spend very significant portions of the 24-hour period in the postprandial state. If we are only looking at premeal readings, we will miss these elevations, and the necessity of addressing them.

So how does one go about doing postprandial testing effectively? If I test once tomorrow, after lunch, and my reading is high, I don't know if what I ate for lunch was a poor choice or if the meal itself was fine and I was high going **into** lunch. If I test before the meal and then 1-2 hours after the meal, I then have a much better picture of the glucose excursion and the appropriateness of the meal. So it is important to link the before and after meal readings. Some people who test infrequently may test before and after breakfast one day, before and after lunch the next, etc.

As for frequency of testing, it has been suggested that the person who is insulin treated test his or her BG levels three or more times per day (ADA, 2007a). The person taking oral hypoglycemics, perhaps twice a day, and the person on diet alone, perhaps once a day. The need is obviously variable depending on the individual situation, but this may be a place to start. Current American Diabetes Association targets for premeal BG values are 90-130 mg/dl, postprandial readings under 180 (ADA, 2007b). Some other organizations aim a bit lower (e.g., fastings 80-110, postprandial <140 mg/dl). Individual goals should be agreed upon between patient and provider.

The Hemoglobin A1c (or glycohemoglobin) test represents quite an advancement in diabetes care. While the fingerstick blood glucose measurement indicates that moment's BG value (a "snapshot"), the Hemoglobin A1c (A1c) represents a 24-hour average BG over a period of about 6-8 weeks preceding the test. It fills in the blanks, so to speak, between all the individual's home readings. Because it gives one the "big picture," it is the bottom line in BG management. If the person with diabetes checks his or her BG several times per day, and those results are very different from the A1c reading, it challenges him or her to test at other times of the day to get a more complete picture of BG control. Let's look at the numbers:

A1c		**Glucose**
12%	=	345 mg/dl
11%	=	310 mg/dl
10%	=	275 mg/dl
9%	=	240 mg/dl
8%	=	205 mg/dl
7%	=	170 mg/dl
6%	=	135 mg/dl
5%	=	100 mg/dl
4%	=	65 mg/dl

As you can see, the higher the A1c, the higher the average BG level. It is interesting to note that these values do not stop at 12%. The highest A1c value this author has personally seen was 19.4%, reflecting an average BG in the 600's. This was in a person who felt reasonably well, underscoring the fact that one cannot rely on how one feels to tell one what is going on in the body.

Another important point is that there is no threshold over which the A1c reading indicates danger, or under which one is "safe." We now know that every decrease in the A1c value decreases the individual's risk for long term complications. As mentioned earlier, for example, with every 1-point drop in the A1c, there is a decrease in a person's risk for small vessel disease of approximately 35% (ADA, 2003a). Patients with diabetes should be encouraged to understand this test, and to ask their Primary Care Providers for their test results, and to be sure the A1c is being done on a regular basis. Generally, the A1c is done every three months, although in the very stable, well-controlled patient it can be argued that every 6 months may be appropriate.

Interpreting the A1c result does not need to be difficult, or necessitate having the above chart available. If one simply remembers that 6% = 135 mg/dl, and knows that for every 1 percentage point rise in A1c, there is a corresponding 35 point rise in average BG, one can recreate this chart when necessary. When a patient bemoans the fact that his or her recent A1c was high (let's say 8.6%), ask what the previous reading was. If the previous reading was 11.2%, then pat the patient on the back, he or she is making progress and needs the encouragement to continue those efforts.

Now let's look at some other targets, starting with blood pressure. BP control becomes critically important in the face of diabetes. As diabetes itself raises one's risk of both heart disease and renal disease, so does hypertension. The individual with both uncontrolled hypertension and uncontrolled diabetes is at much greater risk of renal failure, for example, than the individual with either problem alone. There was a time when the upper limit of normal BP was considered to be 140/90. That number has changed, according to The JNC 7 Report (National Heart, Lung and Blood Institute, 2003). Normal BP is now defined as below 120/80. BP values between 120-139 systolic or 80-89 are now classified as Pre-Hypertension. There are some providers that feel "as low as you can go and still be conscious is good." The goal BP for the individual with hypertension is now under 130/80 (ADA, 2007c). This raises a new challenge, as many patients with diabetes and hypertension are not successfully controlled even by the old standards. Patients with diabetes need to understand these new targets and be encouraged to work aggressively with their primary care providers to get to target, despite the effort required (and possibly multiple medications).

Also relating to renal function is the urine test for microalbumin. The kidneys are not supposed to leak protein into the urine. If they do, it can be an indication of nephropathy, or renal damage due to diabetes. The standard urinalysis, however, doesn't identify protein until the level is above 300 mg/gm creatinine. At this point, nephropathy is present, and will progress, at some rate, toward end stage renal disease. The urine for microalbumin, however, shows **microscopic** protein, at a point where preventive intervention is possible. A reading of less than 30 mg/gm creatinine is considered normal. A reading between 30 and 300 mg/gm creatinine is considered **microalbuminuria**, and above 300 mg/gm creatinine is considered proteinuria (ADA, 2007d). A positive test for microalbumin (usually a first morning urine sample, if possible, but a random urine can be used) is usually confirmed with a second test.

When the patient has two positive urines for microalbumin, the conversation one would have with him or her would go something like this: "You have a change in your kidneys, but it is nowhere near a problem. We have the opportunity **now** to put some things in place to **prevent** the progression to the problem. Four things become important. First and foremost, blood pressure control is critical (as described above). Next is blood glucose control. The third is the use of a class of medications called ACE inhibitors (angiotensin converting enzyme inhibitors, such as lisinopril or captopril), or ARB's (angiotensin receptor blockers, such as losartan or valsartan). These medications are used for hypertension, but have a specific protective effect on the kidneys, so much so that these meds are even used in the person with microalbuminuria and no hypertension (average doses will not unduly lower blood pressure). Think of this medication as your 'kidney medicine.' If you do have hypertension, then this medicine is doing double duty for you (antihypertensive effect as well as renal protection). The fourth item centers on the amount of protein in the diet. The typical American diet is very high in protein. Studies have shown that decreasing this extra protein intake helps slow the progression of proteinura, GFR (glomerular filtration rate) decline, and occurrence of ESRD (end stage renal disease) (ADA, 2007d). Of course, one cannot just tell the patient to watch their protein intake. They're already watching sugar, fat, and salt intake (if they have hypertension), and may feel a bit overwhelmed at this new dietary consideration. Instead, this is a good time for a referral to a Nutritionist, who can assess the patient's diet to determine if an adjustment is necessary.

You Are What You Eat: Diet Basics

Diet is one of the cornerstones of therapy (along with exercise and education), regardless of the pharmacological therapies used. Variations in food intake can have a profound effect on blood glucose levels in many people. The goals of Medical Nutrition Therapy are (ADA, 2007e):

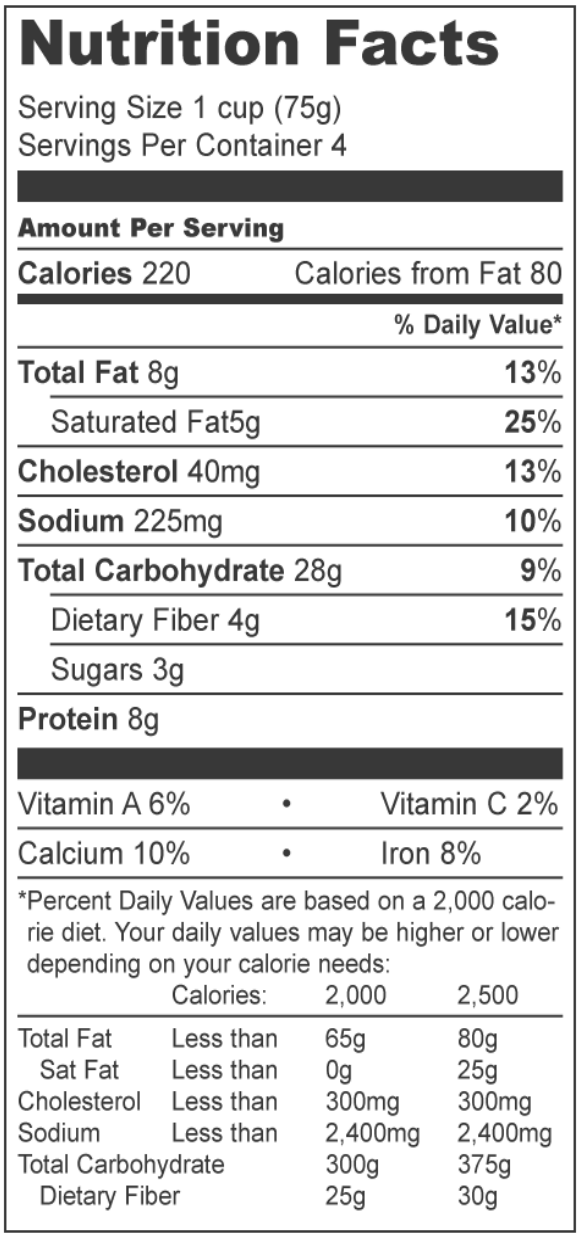
1. Achieve and Maintain
    - blood glucose levels in the normal range or as close to normal as is safely possible
    - a lipid and lipoprotein profile that reduces the risk for vascular disease
    - blood pressure levels in the normal range or as close to normal as is safely possible
2. To prevent, or at least slow, the rate of development of the chronic complications of diabetes by modifying nutrient intake and lifestyle
3. To address individual nutrition needs, taking into account personal and cultural preference and willingness to change
4. To maintain the pleasure of eating by only limiting food choices when indicated by scientific evidence.

What is the first thing that people with diabetes are told about the "diabetes diet?" It's usually to stay away from **sugar**, as if it were a poison. But if we remember that most all of the food we eat is broken down into sugar (glucose), we'll realize that sugar in itself is not a bad thing. Rather, let's take a look at the different types of food available to us:

- **Protein** (meat, chicken, fish, eggs, cheese, etc.)
- **Fat** (oil, butter, margarine, mayonnaise, etc.)
- **Carbohydrates** (these are divided further, below)
    - **Simple:** (**sugars**, such as cakes, candy, cookies, ice cream, jams, jellies, syrup, honey, etc.)
    - **Complex:** (**starches**, such as bread, pasta, potatoes, rice, cereal, grains, etc.)

First, it is important to realize that it is carbohydrates that affect BG levels to any significant degree. For the most part, proteins and fats do not affect blood glucose immediately. In other words, if I take a premeal medication to lower my blood glucose, it is to balance the **carbohydrates** in that meal. Let's say I take a consistent dose of glucose-lowering medication at dinner from day to day, and usually have a balanced meal consisting of protein, starch, and a vegetable. If, the next day, I have a one pound steak and nothing else for dinner (protein and fat only), I may suffer a hypoglycemic reaction afterward. This is because there was no carbohydrate in this meal. With no carbohydrate to balance the effect of the medication, the medication can push the BG too low. So, for those individuals taking a consistent dose of medication to lower the blood glucose, it becomes important to maintain a consistent amount of carbohydrate at a given meal from day to day. In addition, to avoid high peaks of blood glucose after a meal, one learns to avoid large amounts of carbohydrate at one time.

Getting back to the sugar question, the reason we focus so much attention (initially) on sugar is that sugar-rich foods take a very large amount of carbohydrate and pack it into a small package. They are **concentrated** forms of carbohydrates. Therefore, we get the most "bang for the buck" in decreasing the carbohydrate load to the body by decreasing the individual's intake of these simple carbohydrates. First steps in this process include using some simple substitutions, such as switching from regular soda to diet soda, from regular Jello to sugar-free Jello, and using Nutrasweet or Sucralose, for example, instead of sugar in one's coffee. It is important to tell our patients not to believe anything they read on the front of the label. A label can say "No Sugar Added," however, that doesn't tell one how much sugar is present to begin with. The only way to reliably know what is in a product is to look at the nutrition label on the back.



Nutrition Facts

Serving Size 1 cup (75g)
Servings Per Container 4

Amount Per Serving

Calories 220	Calories from Fat 80
	% Daily Value*
Total Fat 8g	**13%**
Saturated Fat5g	**25%**
Cholesterol 40mg	**13%**
Sodium 225mg	**10%**
Total Carbohydrate 28g	**9%**
Dietary Fiber 4g	**15%**
Sugars 3g	
Protein 8g	

Vitamin A 6%	•	Vitamin C 2%	
Calcium 10%	•	Iron 8%	

*Percent Daily Values are based on a 2,000 calorie diet. Your daily values may be higher or lower depending on your calorie needs:

	Calories:	2,000	2,500
Total Fat	Less than	65g	80g
Sat Fat	Less than	0g	25g
Cholesterol	Less than	300mg	300mg
Sodium	Less than	2,400mg	2,400mg
Total Carbohydrate		300g	375g
Dietary Fiber		25g	30g

The information listed is per serving. This example was from a package of tortellini. The serving size is 1 cup, and there are 3 grams of sugar per serving. What does this mean? Let's take a closer look at the sugar content of some common foods. A can of regular cola drink has about 40 grams of sugar. A diet version of that cola drink, if it is truly sugar free, will have 0 grams of sugar. This is how one knows if a product is truly sugar-free. A common recommendation regarding breakfast cereals is to choose those with 5 gm or less of sugar per serving. Therefore, some good choices include Cheerios, Rice Krispies, Special K, Wheaties, Total, Product 19, Crispix, Kix, to name just a few. Now, I would intuitively know that a cereal like Honey Smacks would have perhaps 13 or 14 gm of sugar per serving. That makes sense to me. However, some may not be so obvious. Raisin Bran, for example, may have 8-12 grams of sugar, or more, depending on the brand. It's not the raisins that are the issue here, it's the sugar that is put on the raisins to help preserve them. How about choosing a cereal like Corn Flakes (2 gm sugar per serving) and adding my own raisins to them? I then know I'm not consuming extra sugar. This is a simple way to teach our patients to make initial healthy choices.

Next, let's look at milk. One cup of milk (regardless of the percentage of fat) will have approximately 11-12 gm of sugar per serving. This is why milk is recommended as a treatment for hypoglycemia. In the past, fruit juice (with extra, added sugar) was the usual recommendation, before the realization that this could result in significant hyperglycemia. Milk has a more reasonable amount of sugar for this purpose. A cup of milk a few times a day is fine for most people, but the person who consumes very large quantities of milk will often notice a significant rise in BG levels, which can be controlled by limiting one's milk intake.

Fruit juices are, potentially, a very significant source of sugar in one's diet. The reason is this: a typical label for orange juice, for example, will indicate that one cup (8 oz) of "all natural, no sugar added" OJ has approximately 26 or 27 gm of sugar. If I compare that cup of orange juice to an orange, the orange has the amount of sugar in one orange. The juice has the amount of sugar in three or four oranges, squeezed to make that much juice. In addition, eating the orange means I'm also consuming the pulp, or fiber. This fiber does help to blunt (slightly) the rise in BG as I consume the orange. In drinking the juice, not only do I have the amount of sugar from several oranges at one time, but I don't have that fiber intake (the "pulp" on the label is not the same thing). So in general, it's healthier to choose a piece of fruit over the juice, or at least limit its serving size. There are also alternatives to fruit juices (drink mixes with sugar substitutes as sweeteners) that give one yet more choices. In an office setting, if you find your patient is having a hard time getting BG levels down, it might be useful to ask the question regarding fruit juice intake. Hearing something like "I drink the all natural, no sugar added, Brand X orange juice, a quart of it a day," one has the opportunity to respond with something like: "Let's change this one thing in your diet, and we may see a significant change in your BG control."

It's also important to realize that serving sizes become very important. If a patient comes to you with the statement that he or she used to consume large quantities of regular soda, but has now made the switch to orange juice, he may be thinking that he's made a significant change. He's at least "cutting down" from 40 gm to 26 or 27 gm of sugar per serving. That's at least an improvement, isn't it? But is it? The can of soda is 12 ounces. The serving of juice is 8 oz. If you do the math, ounce per ounce, orange juice has about the same amount of sugar per ounce as a regular cola drink!

In addition, when one looks at the containers of drinks available at the deli or convenience store, it's important to look at the servings per container. A 20 oz. container of pink lemonade may have 28 gm sugar per serving, but if the serving size is 8 oz, and the container states that it holds 2.5 servings, the total amount of sugar in that container rises to 70 gm. And who's going to share that container with another 1½ people?

Let's expand our focus now to the rest of the carbohydrates. Once your patient has a working knowledge of label reading for sugar content, and has an understanding of how to avoid taking in large amounts of sugar, it's time to move on to the bigger picture. You'll notice that sugars are

listed (indented) under the heading of carbohydrates on a food label. This is because sugar is a form of carbohydrate. If one is taking the next step to look at carbohydrates on the label, one can essentially let go of the focus on sugar, and focus instead on the carbohydrates, or "carbs."

A very useful piece of information is that one piece of bread has approximately 15 gm of carbohydrate. If I know that, I can take any package off the shelf in a grocery store and check the label to determine how much of that product is equivalent (carb-wise) to that piece of bread. For example, if you look at a box of saltine type crackers, you will notice that 6 saltines will have about 15 gm of carbohydrate. Let's say that I'm used to having a tuna sandwich on two pieces of bread each day for lunch. If I still feel like having tuna today, but feel like having it on saltine crackers instead of bread, how do I know how many saltines to have? If I know that 6 saltines equals one piece of bread, I can have 12 saltine crackers and keep my carbohydrate intake consistent for that meal. This is the basis of carb counting.

As we said earlier, those patients taking a consistent amount of medication to cover a particular meal from day to day need to maintain a consistent amount of carbohydrate at that meal from day to day. Helping our patients understand the basics of carb counting can enable them to make good food choices and help maintain more consistent BG control. For some, it may mean being able to identify carbohydrate foods in order to avoid having large amounts at a time without realizing it. For others it may mean picking and choosing foods to more precisely maintain a consistent amount from day to day, and for others it may mean learning to vary the amount of medication to cover the particular amount of carbohydrate one **chooses** to have at that particular meal. Below is a simple list of some of the most common carbohydrate choices:

15 gm Carbohydrate Equivalents

1 piece of bread

1 small baked potato

½ cup mashed potatoes

½ cup cooked pasta

1/3 cup cooked rice

½ cup corn, peas, and lima beans

1 cup milk

1 medium piece of fruit

You'll notice that milk and fruit are included on this list. Introducing this concept to our patients slowly, at a pace they can handle, can empower them to become masters of their diet in a way they may never have been able to before.

Pasta is an example of a food that is often misunderstood. Many people with diabetes have assumed they could not eat pasta, because they might see extreme elevations in BG levels after a pasta meal. However, it's usually not the pasta itself, but rather, the amount of pasta we, as a culture, are accustomed to. We may be used to a large bowl of pasta, not realizing that it may reflect a measurement of several cups. Most people don't know that that big bowl of pasta is the same to one's body as perhaps 6-8 pieces of bread at one time. We don't do that with bread, culturally, but we do that with pasta all the time. And then what do we do? We add garlic bread to it! If we look at the healthier alternative, how about having the smaller bowl of pasta, as a side dish, and then having the meatballs, vegetables, salad, fruit for dessert, and the diet soda, the

meal is no longer all (or mostly) carbohydrate. This alternative is healthier for all of us, whether or not we have diabetes.

The next important point is that when we encourage our patients to make these changes, we need to encourage **small** changes. Going from 4 cups of pasta to 1 or 1½ cups will only make the person feel deprived and unsatisfied. The better approach may be this: encourage your patient, the next time he plans to have pasta, to measure his usual portion size. If he discovers he is used to 4 cups of pasta, first realize that this is just information, and not an excuse for self-battering. Then suggest he cut down to 3 cups, for example, while adding a salad, etc. Hold to this portion size for several weeks to a month, and then take the next small step toward a smaller serving. It is important to encourage small changes in our patients, that is, changes that are easily doable. Small changes which give one a measure of success, are more likely to become learned habits over time. The simple decreasing of portion sizes, gradually but consistently, is much more likely to make significant persistent differences in BG control over time than the overhauling of one's entire diet overnight.

Now let's turn to the fat content of the diet. Once again, it's important to go to the food label, and not rely on information on the front of the label. A product may be labeled "95% fat free" on the front, for example, such as turkey cold cuts. However, this can be very misleading. The 95% fat free refers to weight. In other words, I can take a large glass of water, mix two teaspoons of butter into it, and say it's 95% fat free (it's mostly water). However, 100% of the calories in that drink (if you could drink it) would be from fat. Go to the nutrition label and look at the grams of fat. A useful reference here is that 5 gm of fat is one teaspoon of butter. Knowing that, if you look at the nutrition label on a package of hot dogs, you will notice that a serving is one hot dog, and the fat content is approximately 15 gm of fat. That means eating that hot dog would be like taking a spoon, going to the refrigerator, taking three teaspoons of butter and licking it right off the spoon. It may not stop someone from eating that hot dog, but it may make one stop and think about it, or perhaps limit how many are consumed.

It can be eye-opening to start looking at the fat content of nutrition labels. A salad choice at a fast food restaurant may be a good choice, but if the salad dressing contains 20 something grams of fat, that "healthy" choice begins looking like a cheeseburger. By simply comparing the fat content of similar products, one can often decrease significantly the fat content (and therefore calories) without very much effort.

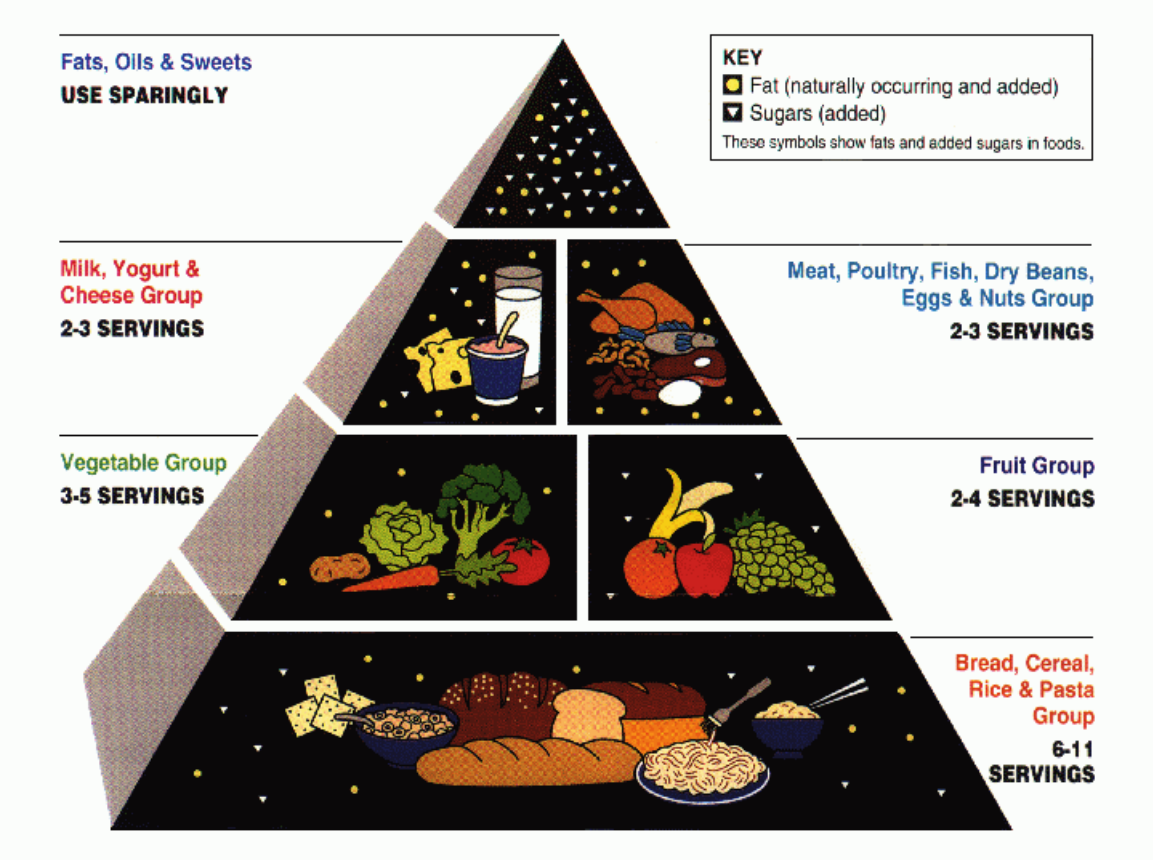
Speaking of calories, high fat foods have many more calories than carbohydrate and protein foods. One gm of protein has 4 calories. One gm of carbohydrate has 4 calories. One gm of fat has 9 calories, more than twice that of carbohydrate and protein. This means that if weight loss is a goal, one gets "more bang for the buck," so to speak, by decreasing the fat content than by decreasing the carbohydrate or protein content of foods. Again, however, it is important to encourage our patients to make these changes slowly, such as decreasing portions sizes a little at a time, or even mixing 2% and 1% milk for a while before making the full switch to 1%, for example.

Here is a summary of the Medical Nutrition Therapy recommendations, according to the American Diabetes Association (ADA, 2007f):

Protein:	15-20% of daily calories
Fat:	Saturated < 10% total calories Polyunsaturated ~ 10% of daily calories Cholesterol < 300 mg/day Intake of trans-unsaturated fatty acids should be minimized
Carbohydrate:	Individualized, including carbs from whole grains, fruits, vegetables, legumes and lowfat milk. Low carb diets (<130g/day) are discouraged.

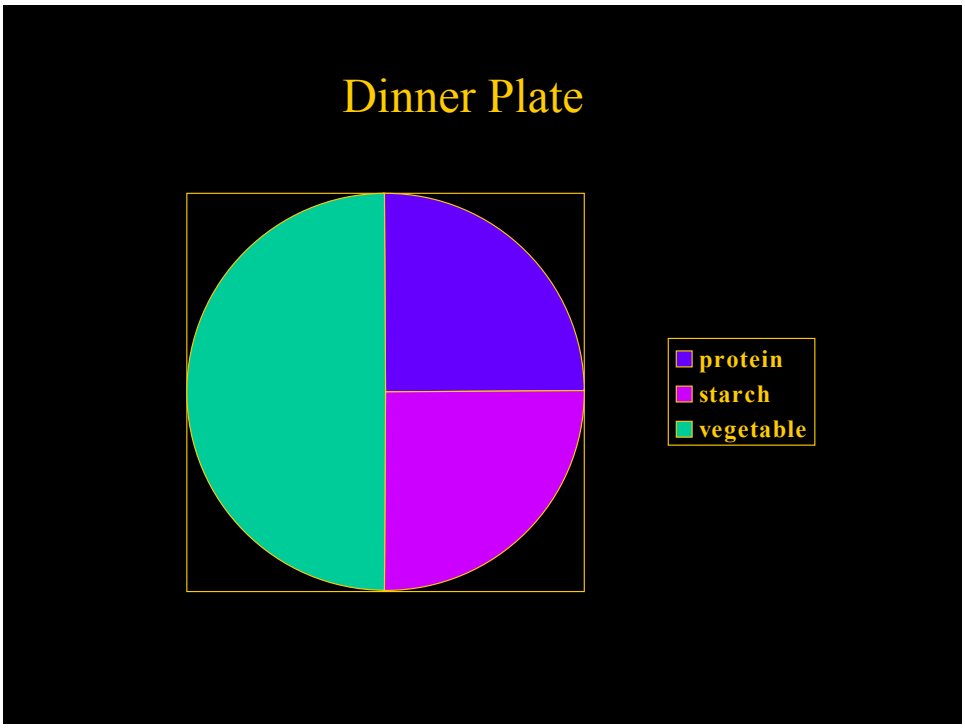
Fiber: Dietary fiber encouraged, no greater amounts than for general public

So how does one interpret this for our patients? One available visual tool has been the food pyramid:



The general idea behind this diagram is that the majority of the calories we take in should come from carbohydrates, fruits and vegetables. This is the opposite of what we have done, culturally, for years. Most adults in this country may remember middle school home economics classes that taught us that the meat item was focus of the meal. The potato and vegetables were side dishes. The food pyramid turns this pattern upside down. A representative meal may look like this: a large salad, with greens, red peppers, broccoli, cauliflower, etc, etc, with a 3 ounce chicken breast, grilled, in strips on top of the salad, and a small dinner roll on the side. Is that how we eat in this country today? No. However, the point is, as we approach this type of meal, we approach healthier eating.

How do we translate this information to one's lunch or dinner plate? Here's a simple drawing to illustrate how to do that.



Approximately 25% of what's on our lunch or dinner plate should be protein foods, 25% should be carbohydrate, and roughly 50% should be vegetables. That's not what most of us do, of course. However, the way to use this tool is to first start out with where our patient's "lines" are. The person who consumes a large bowl of pasta and very little else may have a drawing showing perhaps 90% of the pie as carbohydrate. What if that person adds a meatball or two and a little salad? The lines begin to shift toward a healthier alternative. Allow the lines to shift slowly, as discussed earlier, so that the individual can make small changes that are more likely to be long term successes.

There have been modifications made to the original food pyramid, which can be found at www.mypyramid.gov. Here, one can create an individualized food pyramid, based on individual need.

Timing is Important: *Meal Patterns*

What is our normal pattern of food intake in this society? Typically, we have little or no breakfast, a light lunch, and a large dinner, perhaps quite late by the time the meal is prepared after work and all family members are together. Then what do we do? We sit for the evening, often in front of the TV, and we snack close to bedtime. This picture shows us pushing most of our calories toward late in the day. However, our bodies were not designed to work this way. If one gets out of bed in the morning and does not eat breakfast, the body knows that there is energy going out (for the activities of daily living) and no fuel coming in. This makes the body shut down the metabolism somewhat, in order to be more efficient, and burn fewer calories. This encourages weight gain. Many people find that eating even a small breakfast can help their weight loss efforts. Now as for lunch-time, it would be better for us to have our larger dinner meal at lunch and our smaller lunch meal at dinner time, in other words, shifting calories earlier in the day. Would you drive your car from here to Pittsburgh and then fill the gas tank? No, you would fill the gas tank and then burn it off as you drive. Think of the body as a machine. Give the machine the fuel the machine needs when the machine needs it. Having the larger meal earlier in the day gives the body the time to burn it off. If one cannot switch lunch and dinner, how about a more substantial lunch and a lighter dinner? And now, let's look at the late evening. It can be hard to realize just how much one can consume while snacking and watching television. Food commercials can be triggers for "the munchies," and it can be very difficult to limit one's food intake at that time. However, it's important to realize that we are generally very sedentary at that time of day. Calories that we take in that are above what we burn off during the course of the day will be stored as fat. Many people find that snacking in the evening can be difficult to control, especially if one is tired, stressed, or hungry due to little food intake earlier in the day. One successful dietary effort for many is to limit food intake during the late evening hours. For example, picking a time 3 or more hours before bedtime, after which no food intake will be allowed, can help control this habit of overeating. This one change can make a significant difference in blood glucose levels as well as weight.

Another key concept is that overall patterns of BG control are much more important than short periods of time. In other words, if I have generally good BG control, a small "cheat" will be better tolerated than the same indiscretion during a time of uncontrolled BG. The bigger picture is what's important. As the saying goes, "It's not what you eat between Christmas and New Year's, but what you eat between New Year's and Christmas…"

Get Up and Move! ***Adding Physical Activity***

The term "exercise" is often met with trepidation by our patients. It conjures up visions of finding time one doesn't have, going to a gym one can't afford, and doing painful things one doesn't want to do. However, our approach to this topic can help our patients see that they can, in fact, make changes in daily activity levels that can make a significant difference in both weight and BG levels. The benefits of regular physical activity are many. Yes, it helps my heart (modifying cardiovascular risk factors), it helps me keep my weight down, it helps me sleep better, improves my psychological well-being and it helps keep me "regular." But it also helps my body respond to insulin better, decreasing my body's resistance to insulin. This improves BG control and decreases my need for pharmacological intervention. In fact, adding regular physical activity to one's daily routine over time can literally save a co-payment, in terms of forestalling the need for additional medication for some people. The recommendation is for the "accumulation" of 30 minutes of moderate physical activity on most days of the week (ADA, 2007g). What can we offer to those who have little time or inclination to start an exercise program?

First, it is important for the individual to consult with his or her primary care provider regarding any appropriate screening, evaluation and precautions to be observed. Once this has been addressed, a number of possibilities can be explored with your patient. Probably the safest and least expensive form of physical exercise we can recommend is walking. With the right footwear, this can be a very rewarding and productive effort toward better BG control and weight reduction. Here again, however, making small changes is key. For the person not used to a walking program, starting with even 5-10 minutes per day (or every other day), at a comfortable pace, can begin to make a difference. Walking the neighborhood, the mall, around the house, around the park, or within one's house (the bathroom to the bedroom to the dining room to the bathroom to the bedroom to the dining room…) are some of the creative alternatives that are possible. Keeping a written record of the days or minutes walked, together with planning rewards for goals reached can help motivate most of us to keep with such a program.

One of the newest tools to help motivate our patients is the pedometer, which counts not mileage necessarily, but steps. You clip this little gadget to your belt and start with an assessment of the number of steps taken in a typical day. The goal is then to increase the number of steps, gradually, to achieve your goal. Ten thousand steps are the rough equivalent of five miles, and a goal for some. However, a good first goal is to add 500 steps to one's current number of steps per day. Two thousand steps is roughly a mile, for most people. If I have the goal of "walking more," and have no way to measure what "more" means, I may have less motivation over time to continue my efforts. By being able to measure and follow my progress, I am given a tangible outcome to guide me, and this may help me keep with the program.

Other creative ideas to share with your patients include cycling while watching TV (for example, during the first 10 minutes of the 6:00 news). Walking on a treadmill (while reading a magazine laid across its control board), turning on the TV dance station and dancing, or even turning a bicycle on its end and using one's hands to pedal it are all possibilities to experiment with. For those whose mobility is limited, chair dancing can be an interesting alternative. Sitting in a chair without arms, one simply moves to the music. There are videos available, with varying levels of aerobic intensity. Contrary to what one might think, this can be a significant workout!

The key to any successful exercise program, however, is not just finding an activity that appeals to the individual, but ensuring the commitment to a regular pattern of activity that will continue despite changes in weather, mood, finances or time constraints. One must be willing to make the decision to create the habit, much like other habits we cultivate over time. For example, by the time we become adults, most of us have cultivated the habit of brushing our teeth. At this point in my life, if I get into bed at night and realize I have not brushed my teeth, I must get up and do it. It doesn't matter how tired I am. This habit is so ingrained in me that I must do it. Habit

development takes time and commitment. We need to help our patients move toward making that commitment, helping them to make this important lifestyle change over time.

Help! Diet and Exercise Aren't Enough! ***The Addition of Medications***

Despite diet and exercise being the cornerstone of therapy for our patients with diabetes, there are times when those therapies are just not enough. This is when pharmacological intervention becomes appropriate. There are a number of different pharmacological approaches available today. The first are the secretagogues, which are so named because they push the beta cells of the pancreas to secrete insulin. Below is a simple review of the most common:

Secretagogues		
Name	**Dose**	**Caution**
Glyburide (Micronase, Diabeta)	2.5-20 mg/d	Elderly, impaired renal or hepatic function
Glipizide (Glucotrol) (Glucotrol XL)	 5-40 mg/d 5-20 mg/d	Elderly, impaired renal or hepatic function
Glimepiride (Amaryl)	1-8 mg/d	Elderly, impaired renal or hepatic function
Repaglinide (Prandin)	0.5-16 mg/d	Elderly, impaired renal or hepatic function
Nateglinide (Starlix)	60-360 mg/d	Elderly, impaired renal or hepatic function

The first two, glyburide and glipizide, are of a class called sulfonylureas. The older generations of these medicines include Diabinese, Orinase, and Tolinase, for example. Some of these older medications had quite a prolonged duration of action (Diabinese in particular), resulting in severe hypoglycemia in some patients. The newer alternatives offer a better safety profile. However, the issue of hypoglycemia remains. Any patient who is given one of the secretagogues **must** be instructed regarding the possibility of hypoglycemia, its signs and symptoms, causes, prevention and treatment. It is also very important for our patients to have access to home blood glucose monitoring as a tool for their safety. Anyone who takes a glucose lowering medication should always carry with them some form of quick acting sugar. For example, glucose tablets are available over the counter, for about $1.00, and provide the right amount of glucose to raise BG levels approximately 50 mg/dl (typically 4 tablets at 4 gm glucose each). If the individual's BG is 50 mg/dl, for example, 100 mg/dl is a safer place to be. Candy, on the other hand, is not generally a good source of glucose to carry for this purpose. Lifesavers, for example, are an alternative, but, one would need to chew 4-6 Lifesavers to ingest the appropriate amount of sugar, which would have a more significant impact on one's dental work than one might like. Sucking on one piece of the candy is just not enough. Other forms of candy, such as a candy bar, are also inappropriate for another important reason. Most candy bars have a significant portion of their calorie composition from fat. The fat in that candy bar will slow the absorption of the sugar, making it a less readily available treatment for hypoglycemia.

As a quick review, the typical symptoms of hypoglycemia are: shakiness, dizziness, lightheadedness, perhaps a feeling that "something is wrong," a headache or even hunger. These symptoms may be different for different people, and may not be consistent in the same person. This is why relying on BG monitoring is much safer than relying on symptoms alone. The ideal treatment is milk (8 oz) or juice (a little under 8 oz). The liquid will work faster than other more complex foods and it is important for the individual to retest the BG in approximately 20 minutes to assess one's progress. If the reason for the low was a missed meal, then the meal should be consumed (after drinking the milk or juice). If it is not mealtime, then the milk should be followed by a small snack (perhaps cheese and crackers, or ½ sandwich). Prevention is key here. For the person who takes a set dose of glucose-lowering medication from day to day, it is important to

maintain some consistency in the timing of the medication, meals, and physical activity, as well as maintaining consistency in the amount of carbohydrates at a given meal from day to day.

A word of caution for our patients: if one has ever had a low BG reading without any symptoms, this asymptomatic hypoglycemia warrants a little investigation. Some people who take insulin for many years do lose some of the early warning signs of hypoglycemia. However, it is sometimes the case that hypoglycemia itself causes a loss of these early warning symptoms. In other words, if an individual is low today at some point, he or she is in danger of being low again within the next 24-48 hours, **with fewer symptoms**. By putting an end to the "bouncing" (repeated lows) one will generally regain at least some of the early warning symptoms of hypoglycemia (Cryer, Davis, and Shamoon, 2003). The way to do this is to test much more often over the next several days (including middle of the night readings) to determine the BG pattern, and make any appropriate medication adjustments. For the person experiencing lows in the middle of the night, without being awakened, this bouncing may result in a high BG by the time one arises in the morning. This is called the Somogyi effect, and needs to be differentiated from the dawn phenomenon. This is because both the Somogyi effect and the dawn phenomenon produce an elevated fasting BG, but they each require different remedies. If I awake with a high fasting BG, it may be due to changing hormone levels and increased glucose release from my liver in the early morning hours (the dawn phenomenon), requiring **more** medication. If, however, my high fasting BG is a bounce from a hypoglycemic event at 4 am, I need **less** medication, not more. My body's glucagon will try to get my BG up, but it's a long, slow process and doesn't always know when to stop, resulting in elevated BG levels (the Somogyi effect) several hours after the low. A vigilant investigation into BG patterns will give one a clearer picture of the problem and the appropriate remedy.

Regardless, any individual with a history of asymptomatic hypoglycemia should be advised to test his or her BG before getting behind the wheel of a car. One's reaction time is certainly altered when in a hypoglycemic state, as is one's judgment and one's ability to do many tasks safely. In addition, once an individual treats a hypoglycemic reaction, it is wise to wait 30 minutes before driving, as it takes that much time for one's reaction time to recover (despite possibly feeling "fine" within a few minutes of treating the low).

Today, evolving technology is allowing us to obtain even more data regarding BG patterns. Continuous Glucose Monitoring Systems (CGMS) measure glucose levels in interstitial fluid, which closely approximate BG levels. They are very helpful in identifying BG trends and alerting one to glucose levels that are above or below set warning levels. They are used in conjunction with fingerstick testing (not in place of), since interstitial fluid glucose levels lag behind BG levels by about 10-15 min. These systems are not perfect, but they can add significant information to one's decision-making process. For example, if my BG level is 80 at bedtime, it would be very useful to know if it was 80 and rising or 80 and dropping, and how quickly. Some of these systems have been approved for up to 7 days use at a time (without changing the insertion site). These systems offer a great deal of hope to those who find it difficult to attain adequate BG control with multiple daily testing as it is done now.

Next, let's take a look at the biguanides. Up until 1995, the sulfonylureas were all we had in terms of oral mediations for diabetes. Metformin (brand name Glucophage) opened the door to another mechanism of action in an oral medication.

Biguanides		
Name	**Dose**	**Caution**
Metformin (Glucophage) (Glucophage XR)	 500-2500 mg/d 500-2000mg/d	Impaired renal or hepatic function, elderly, CHF requiring treatment, significant ETOH intake (e.g., binge drinking)

Metformin's primary action is to prevent the liver from pouring out glucose into the circulation first thing in the morning. It does not directly lower BG levels. Instead, it prevents elevations in BG levels. Therefore, hypoglycemia is not generally a concern with this medication when taken alone. This is the one pill we have for diabetes where people tend to lose a little weight, rather than gain. For those individuals who have normal bedtime BG levels and high fasting BG's (without food intake in between), this medication can be a very effective remedy. In other words, for those whose elevated fasting BG's are the result of excess glucose production from the liver in the early morning hours (and not a carry-over from too much food intake the night before), metformin can make a significant difference in those fasting BG levels.

However, metformin is not for everyone. It is important that an assessment of renal and hepatic function be done before the individual is started on metformin, and here's why. Most medications that are taken by mouth eventually leave the body, either through the kidneys and dumped into the urine, or by being detoxified by the liver. If an individual has abnormal renal or hepatic function, these medicines may not be excreted in the normal way and may accumulate in the body. Metformin is a medication, which, if it should accumulate in the blood, may cause a very serious side effect, called lactic acidosis. Lactic acidosis is rare (approximately 0.03 cases per 1000 patient-years), but 50% of cases prove fatal (PDR, 2003). It generally only happens, however, in people who already have abnormal renal or hepatic function and shouldn't be put on metformin to begin with. This is why regular renal and hepatic function testing is so important. The metformin will not hurt one's liver or kidneys (as some of our patients fear), but rather, if one develops kidney or liver problems for some other reason, the metformin may not be safe for the individual.

The person on metformin should be advised that any condition that may cause a change in renal or hepatic function may require a change in metformin therapy. For example, a severe infection, MI or stroke, or severe dehydration may significantly affect renal function and require a temporary discontinuation of the metformin. Drinking alcohol on a regular basis will eventually affect the liver, so some providers will not give metformin to an individual who drinks heavily (e.g., binge drinking).

Our patients should be advised to report any symptoms of lactic acidosis, most of which are not very specific (weakness and fatigue, for example). The one to be aware of, however, is muscle aching, much like the ache that occurs in the calf muscles when one has tried to climb too many stairs. This may feel like flu symptoms, so it is wise to encourage the annual flu shot, and a call to the primary care provider for any unusual muscle aching while on metformin.

Another point to consider, many of our patients with diabetes are on medication for congestive heart failure (CHF). Even patients well controlled on maintenance therapy for CHF, however, may be at risk and caution is necessary.

As for typical side effects, metformin can cause gastrointestinal (GI) upset and diarrhea, which is usually temporary and resolves within the first few weeks of therapy. This is why it is prudent to start the individual with a small dose of metformin and increase the dose gradually. It is also important to advise our patients to take this medication at the end of the meal (not on an empty stomach), in order to help prevent stomach upset.

Thiazolidinediones		
Name	**Dose**	**Caution**
Pioglitazone (Actos)	15-45 mg/d	Impaired hepatic function, CHF NYHA class III or IV cardiac status
Rosiglitazone (Avandia)	4-8 mg/d	Impaired hepatic function, CHF, NYHA class III or IV cardiac status

This class of medication works by decreasing insulin resistance. They help to open those doors (to the cells), so to speak, enabling one's insulin to be more effective. The first generation of these medications included troglitazone (brand name Rezulin), which was taken off the market a few years ago. This was due to a number of cases of hepatic injury due to troglitazone therapy. This newer generation, however, has not been shown to cause hepatic function problems, but it is still appropriate to monitor hepatic function before and periodically during therapy.

Actos (pioglitazone) and Avandia (rosiglitazone) are generally well tolerated, and can be taken at any time of day, with or without food. They can cause weight gain, however, and fluid retention (which is the reason for the caution listed above). It is important to counsel our patients that the action of this class of medication is very slow. One does not generally see a significant change in BG levels within a few days of starting therapy (some do, but most do not). It usually takes up to two or three months to see the full effect of starting therapy with these drugs, and our patients need to know this.

Another consideration is that for those women who are pre-menopausal but not ovulating for some reason, these medications may induce ovulation. Women of childbearing age who normally do not use any form of contraception must be warned that they need to consider contraception if a pregnancy is not desired.

Alpha-Glucosidase Inhibitors		
Name	**Dose**	**Caution**
Acarbose (Precose)	25-300 mg/d	Renal impairment, inflammatory bowel disease, chronic intestinal disease
Miglitol (Glyset)	25-300 mg/d	Renal impairment, inflammatory bowel disease, chronic intestinal disease

This class of medication works by slowing down the absorption of starch from the gut. The carbs are all absorbed, but it just takes longer. Therefore, instead of a significant rise in the postprandial BG, the rise may be more gradual. These medications do have some socially unacceptable side effects (bloating and gas), which limits their usefulness in many individuals.

It is important for our patients to know that the above classes of medications can and often are used in combination. No longer is the use of two or three different medications for diabetes a sign that the diabetes is "really bad." It is appropriate to use several different classes of medication at the same time, as their actions complement each other and can work synergistically with each other. These combinations can ultimately result in better BG control than was possible with oral medication in the past.

<u>Newer Meds</u>

Incretin Mimetics		
Name	**Dose**	**Caution**
Byetta (Exendin-4)	5-10 mcg bid sc	GI diseases

Byetta is a synthetic form of GLP-1, which is a naturally occurring gut hormone. GLP-1 is secreted in response to food intake, and has four main actions:

1. Glucose-mediated insulin release (insulin secreted only in response to food intake)

2. Slowed gastric emptying (which often is accelerated in type 2 diabetes), helping to control postprandial elevations

3. Inhibited postprandial glucagon release (glucagon is often released inappropriately after meals in type 2 diabetes), helping to control postprandial elevations

4. Increased satiety (Byetta works on the satiety areas of the brain, leading to fullness, decreased food intake and weight loss)

Byetta is unique, in that it is the first secreatagogue (medication which stimulates insulin release) without causing hypoglycemia. It is given within one hour before the two largest meals, at least 6 hours apart (usually breakfast and dinner); and is administered subcutaneously, using a disposable pen. The pen itself is functionally the same as the Humalog pen, for those who are familiar with this device. Side effects include nausea, vomiting, diarrhea, which are common, but tend to resolve or improve with time. At the time of this writing, a long acting version of Byetta is undergoing FDA trials.

DPP-4 Inhibitors		
Name	**Dose**	**Caution**
Januvia (Sitagliptin)	25-100 mg/d	Renal insufficiency (decreased dose)

As Byetta works by supplying more GLP-1 to the body, Januvia works by inhibiting the breakdown of GLP-1. The body's own GLP-1 is broken down quickly by an enzyme called DPP-4. Januvia inhibits this enzyme from breaking down one's own GLP-1, enabling it to act longer. It has three main actions:

1. Glucose-mediated insulin release
2. Slowed gastric emptying
3. Inhibited postprandial glucagon release

Januvia's actions are much like Byetta, however, it does not have the weight loss effect of Byetta. It is, instead, weight neutral. It is given by mouth, generally 100 mg/d, with or without food. It has a side effect profile of placebo and an interesting list of contraindications: "none." This is a nice alternative for patients needing a secreatagogue but having trouble with hypoglycemia secondary to more traditional medications.

Amylin Analog		
Name	**Dose**	**Caution**
Symlin (Pramlintide Acetate)	15-120 mcg	Hypoglycemia (decrease insulin dose)

Amylin is a naturally occurring hormone, secreted by the beta cells of the pancreas, along with insulin. Its actions include:

1. slowed gastric emptying
2. inhibited postprandial glucagon release
3. increased satiety

Symlin's actions sound very similar to Byetta or Januvia, with an important distinction. Byetta and Januvia are working with gut hormones, where Symlin is a pancreatic hormone. Byetta and Januvia both stimulate insulin secretion, where Symlin does not.

Symlin is indicated **only** for patients already on optimal insulin therapy, that is, multiple daily doses of insulin (either type 1 or type 2 Diabetes), and still not well controlled. It is given via subcutaneous injection at each meal and significant snack (15 gm carbohydrate or more), and cannot be mixed with insulin. This means two injections at each meal, one being insulin, the other Symlin. Nausea and vomiting are significant possible side effects, which is why dosing is started

slowly and increased gradually. Symlin does not offer dramatic results, but may help even out BG levels in the patient already “doing everything right” and still having difficulty achieving good control.

Insulin

There are several new insulin options available today, which make the task of finding the right regimen for the individual much easier than it has ever been. First, let's start with the shorter acting insulins. Regular insulin was all we had until very recently, requiring the individual to take a dose approximately ½ hr before the meal. This is because its onset of action is about 30 minutes. Its peak effect is approximately 2 hours, and its duration of action, theoretically, is 4-6 hours (in actuality the duration can be quite a bit longer). In the real world, most of our patients do not wait that half-hour after injecting to eat their meal, which means the peak of the Regular insulin will come much later than the peak rise in BG due to the meal. Giving enough Regular insulin to cover the immediate postprandial period often results in hypoglycemia several hours later.

There are now three rapid acting insulins available: Insulin Lispro (Humalog), Insulin Aspart (Novolog), and Insulin Glulisine (Apidra). These insulins start to work within a few minutes after administration, have a peak effect about 1 hour later (which more closely mimics the body's peak of BG approximately one hour after the meal), and have a duration of three to four hours. These insulins can be given right at the start of the meal, and in some cases after the meal, when the amount of carbohydrate consumed is known. For example, picture the small child who tells Mom he wants a bologna sandwich, receives an appropriate amount of Regular insulin, and then decides after two bites that he's "full." Here comes the scenario where Mom knows he needs more carbs to feed the insulin, and may then be drawn into a power struggle with the child over what those carbs will look like. With these newer insulins, Mom can offer the sandwich, determine how much is actually eaten, and then give the appropriate amount of insulin to cover the meal (and Mom's own BG level is less affected by stress).

Our attempts to provide the patient with a background, or basal insulin, until recently included the intermediate acting insulins (NPH and Lente) and the longer acting insulin, Ultralente. NPH and Lente have a slow onset (1 hour or more after administration), peak at approximately 6-8 hours (but this can be quite variable), and have a duration of action of up to 24 hours. Because of their variable activity and significant peaks, our patients often found themselves "feeding" their insulin, in other words, making sure they ate a meal at certain times to prevent hypoglycemia. Using NPH or Lente insulin twice a day produces peaks and valleys of blood glucose that make it difficult to attain adequate BG control for many. In addition, the typical pattern involves taking a combination of NPH or Lente together with a shorter acting insulin at breakfast and dinner. The short acting insulins cover those two meals, and the morning NPH or Lente covers most of the middle of the day. However, the evening NPH or Lente, given at dinner time, peaks at 1-2 am, causing middle of the night hypoglycemia in many people. One to two am is the body's normal lowest time of BG. Ideally one's insulin action should not be peaking at this time. A simple solution for some is to move the NPH or Lente component of the evening injection to bedtime. This will then move its peak effect to the morning, when the dawn phenomenon causes a rise in BG (a good time for extra insulin action) and the individual is awake to assess the adequacy of that bedtime NPH or Lente dose.

Ultralente was briefly looked upon as the answer to the basal insulin question. It does provide a more even keel, basal insulin effect. However, it too, has a peak that can be quite variable, making it a poor basal insulin choice for many people.

More recently, Insulin Glargine (Lantus), and Insulin Detemir (Levemir) have provided a more truly basal insulin effect. They are generally given once a day at bedtime (although can be given at any time of day), and have no peak in most people. Very rarely, Lantus can have a mild peak, about 4 hours after administration. For the person taking Lantus at bedtime and getting a low in the middle of the night, moving the time of administration to the morning can alleviate this problem. Levemir, in many patients, does not adequately cover the 24 hour period and must be given twice a day in many patients. These insulins must be given alone, (not mixed with another insulin in the same syringe). They can, however, be given at the same time as a rapid acting

insulin, but at different sites. A very nice regimen entails once a day Lantus or Levemir, followed by Insulin Lispro, Aspart or Glulisine at each meal, whenever those meals may be. In other words, if lunch one day is at noon, the next day at 3 pm, and the next day lunch just doesn't happen, it's okay. The rapid acting insulin is given to cover the meal, whenever the meal happens to be. In addition, the individual can learn carb counting to determine the necessary dose for that meal and have the flexibility in meal planning to choose a greater variety of meals and meal content. This pattern of taking Lantus or Levemir once a day and covering each meal with a short acting insulin is called basal/bolus therapy (the basal provides background insulin, where the bolus covers the meal) and is the closest thing we have to insulin pump therapy without actually using a pump.

There is now an inhaled form of insulin, called Exubera (Insulin Human Inhalation Powder), which is a short acting insulin, with an action profile much like Regular insulin (its peak effect is at approximately 2 hours, and duration approximately 6 hrs). This insulin comes in a powder form, in a "blister pack," which is placed in a device that will puncture the blister, releasing the powder into an air chamber, which is then inhaled by the patient. The dosing is cumbersome, in that a 1 mg blister equals approximately 3 units of insulin, and a 3 mg blister equals approximately 8 units of insulin. Therefore, 1 mg plus 1 mg plus 1 mg equals more than a 3 mg dose. This will take some getting used to for both providers and patients, but does offer an alternative to insulin injections for those who fear pain. (It should be stated, however, that an insulin injection, given properly, is not painful. Many of our patients, though, will not believe this until they try it!)

Another issue with inhaled insulin is that it is not for everyone. Smokers and those with unstable or poorly controlled lung disease are not candidates for its use. Ex-smokers must wait six months before being a candidate for Exubera therapy, and all patients must have a Pulmonary Function Test (PFT) before and periodically after starting therapy. It remains to be seen whether health insurance companies will cover the cost of these PFT's.

There are some innovations over the last several years that have made insulin administration easier for most people. First, there are insulin mixtures: NPH/Regular based 70/30, Lispro/NPL based 75/25 (Humalog 75/25), and Aspart/Protamine 70/30 (Novolog Mix 70/30), for example. These mixtures combine a short acting and intermediate acting insulin in a fixed combination. They offer an alternative for the individual requiring some short acting coverage at breakfast and dinner in addition to their intermediate acting insulin, but who cannot, for whatever reason, mix insulins in a syringe. The drawback, of course, is that one cannot change the dose of one component without changing the other.

There are some newer tools available as well. For example, insulin pens are becoming more popular. Many are disposable, and consist of a cartridge of insulin in a pen shaped device. One screws on a pen needle, gives an "air shot" (to prime the needle), dials up the dose, and gives the injection. Pens are now available for each of the insulins mentioned above.

The insulin pump is one of the more exciting tools available to people with diabetes. It looks somewhat like a beeper, and is worn on one's belt or clipped to or hidden inside one's clothing. The pump holds a reservoir of rapid acting insulin, and delivers it slowly through a tube connected to one's body via a small catheter. The needle used to introduce the catheter is removed after insertion, leaving only the catheter in place in the skin. This insertion site must be changed every 2-3 days, and eliminates the multiple daily injections that many people need to give otherwise. The small amount of short acting insulin given continuously (every few minute intervals) provides a background, or basal insulin that can be adjusted throughout the 24-hour period. For example, I might need one basal rate from midnight to 3 or 4 am, but may then need a higher basal rate between 4 and 9 am (to cover the dawn phenomenon's increasing BG levels). I may then need a lower rate again throughout much of the rest of the day. I can program several different basal rates throughout the 24-hour period, to meet my varying needs.

When I eat a meal, the process I would go through looks like this: I calculate the amount of carbohydrate I will consume. Based on my **carb ratio** (a formula for the amount of carbohydrates one unit of insulin will cover), I determine how many units of insulin to **bolus** to cover that meal. I deliver the bolus by pressing a few buttons on the pump to give the determined amount. I may also use a **correction factor**, a formula for how much one unit of insulin will drop my BG. For example, if I know my correction factor is 1:50, that means one unit of insulin will drop my BG by 50 mg/dl. If my BG at mealtime is 200, and I would like it to be 100 going into that meal, I would take 2 units of insulin to "correct" my errant BG level.

There are newer pumps available today, making this process even easier. For example, the Medtronic Minimed pump can store one's carb ratio and correction factor information in the pump itself. When preparing for a meal, the individual tests his or her BG, and this reading can be transmitted by radio frequency from the meter to the pump. The individual then enters (on the pump) the amount of carbs that he or she intends to consume, and a calculation is made by the pump's "Bolus Wizard" regarding how much insulin to bolus. This calculation is based on the current BG, the amount of carbs to be consumed, and any contributing insulin action from any previous bolus given within the past few hours. Pumps are certainly becoming easier to use, and there is much to be anticipated in the future.

I'm Comin' Alice! ***The Role of Stress***

This title is actually a melding, in this author's mind, of two past TV sit-coms: Red Foxx's clutching his chest when under stress, exclaiming "I'm comin' Elizabeth!" and Jackie Gleason's response to the stress his wife inevitably caused him "To the moon, Alice!"

Stress manifests itself in many ways, and today's busy lifestyle creates some stresses that weren't a normal part of life years ago. We all understand the true emergency, such as a life or death situation, where stress hormones (like epinephrine and cortisol) run rampant. There are stories told about seemingly super-human deeds performed by regular people in emergency situations, like the young woman driving along with her baby in the back seat of her car, who's car then stalls on a train track. When she sees the train coming, she hops out of the car (heart pounding, blood pressure up, pulse racing) and does the seemingly impossible feat of pushing the car across the track and out of harm's way. This sudden outpouring of stress hormones can obviously be lifesaving in such a situation. However, the types of stress we encounter these days are a bit different. The schedule that pits one against the clock, for example, causes lower levels of these stress hormones to be released on a more ongoing basis. If I have a deadline at work that I'm not likely to make, an extra phone call presenting an urgent problem which needs immediate attention, a call from the babysitter that my child is sick, and a monstrous traffic jam outside my window in the direction I'm soon to be heading, my stress level certainly begins to climb. Of course, how I choose to respond to this situation will determine what type of effect this stress may have on my body, but that's a discussion for another time. The point here is that most of us encounter life situations that make demands upon us that are sometimes quite stressful. The resulting change in the body's stress hormones can cause a change in one's BG, usually upwards. There is the occasional individual with diabetes who experiences a consistent drop in BG with stress, but this is rare.

For this reason, our patients must know that stress can and does affect BG levels, and that stress can be defined not only in terms of emotionally charged situations, but also in terms of physical assaults on the body. For example, having a cold, flu or skin infection can result in a significant rise in one's BG. Many of our patients ignore their BG control when they are sick. One may have no appetite, not feel like doing much, and subsequently not check BG levels or even take diabetes medication, assuming no food means no need for medication. However, the individual under the physical stress of illness, despite very little food intake, may need as much if not more medication than usual. Less or no medication, coupled with less frequent BG testing can put the individual at risk of significant loss of BG control. Our patients must be advised to check BG levels more frequently when ill, continue to take his or her medication if possible, and be in contact with his or her primary care provider when questions arise.

Sweet Babies: ***Diabetes in Pregnancy***

Pregnancy is a time during which BG control is critical. “If thou art woman, thou shalt remember that bad blood sugars beget big babies.” (Valentine, Dierman, and Toohey, 1994). There is more to this issue than big babies, however. Diabetes during pregnancy can be classified as either gestational diabetes (diabetes that is discovered during the pregnancy) or pre-gestational (diabetes that was diagnosed before the pregnancy). There are women who develop diabetes (unbeknownst to them), become pregnant, and then discover the diabetes. This is still called gestational diabetes because it is first identified during the pregnancy.

Most women with Gestational Diabetes (GDM) will develop diabetes between the 24th to 28th week of pregnancy, which is why they are screened sometime during that time period. GDM typically resolves with the birth of the baby, but signals an increased risk for type 2 diabetes later in life. The incidence of GDM has been increasing over the past 20 years, by as much as 10-100% in several ethnic groups. This reflects the rise in Type 2 Diabetes in these same populations (especially Native Americans, Asians, Hispanics and African-Americans) (Ferrara, 2007). The other risk factors for GDM are the same as the other risk factors for type 2 diabetes, specifically, aging (having a baby at age 35 rather than 25, for example), a family history of type 2 diabetes and increasing weight. The woman who is at risk for developing GDM is the woman who is already at risk of developing type 2 diabetes, as she is typically already developing some insulin resistance.

If we already know that stress can raise one’s need for insulin, what is the biggest stress that a woman can do to her body? Having a baby, of course! The stress to the body is greater as the pregnancy progresses. This is why GDM tends to show itself toward the end of pregnancy and not the beginning. Pregnancy itself is a “stress test of the pancreas,” in that pregnancy is normally a time of increased insulin resistance due to the hormones of pregnancy (Ryan, 1998). The normal woman will produce increased amounts of insulin to overcome this resistance, but the woman already experiencing some insulin resistance may not be able to produce the extra insulin required to maintain normal BG levels, resulting in elevated BG levels and therefore, GDM. This is also why, in most women, the GDM resolves with the birth of the baby. With delivery, the stress of the pregnancy is gone (as well as all the placental hormones of pregnancy). However, the diagnosis of GDM is a warning that this woman has a significant risk of developing type 2 diabetes within the next several years .

Many women panic, upon first hearing the diagnosis of GDM, because they expect this means their baby is at risk for malformations and genetic defects. This is generally not the case, however. GDM tends to develop later in the pregnancy, well after the period where the baby’s organs are being formed. The concerns here are, first, macrosomia (the big baby). If Mom’s BG is high, there is more glucose going from Mom’s circulation to Baby’s circulation. In “feeding the baby too much,” Baby gets big (maybe 9-10 lb. or more). That’s not an easy delivery for Mom or Baby, and it’s preventable by keeping Mom’s BG controlled. Another risk to the baby is hypoglycemia after delivery. During the pregnancy, Mom’s extra glucose goes to the baby, as we said earlier. However, Baby can make extra insulin to keep Baby’s BG normal. That’s just fine, but Baby gets used to making a lot of insulin. Then comes delivery day: the umbilical cord is cut, taking away Mom's extra glucose. Baby has to shut off Baby’s extra insulin, and that’s hard for a newborn to do, which results in hypoglycemia in the newborn. The next risk to the baby is respiratory distress syndrome. Baby’s lungs are maturing the last several weeks of pregnancy. This maturing process works better in the environment of normal BG control. In addition to these concerns, very high BG levels can (and have) been a cause of fetal demise, especially in the days before GDM was recognized to be a concern, where a woman may be unaware of this condition and not accessing prenatal care.

Treatment of GDM is first and foremost, diet. This diet, however, is not the typical meal plan that would be given to a non-pregnant person. The carbohydrate level is lower (often 40-45% of total

calories), because of a relative carbohydrate intolerance during pregnancy. Protein levels are higher, in comparison, because you're growing a baby. Instead of three big meals, the usual plan is six smaller meals, spreading carbohydrate intake throughout the day. Many women who work intensively with diet can avoid the need for insulin. For those whose BG levels are not controlled by diet alone, currently, insulin is necessary (although there may be an indication for one of the oral hypoglycemics at some point in the future).

After delivery, it is imperative that the woman who has had GDM be re-tested with a 2 hr GTT (with a 75 gm glucose load). This test is ideally done at approximately 6-8 weeks postpartum, in order to ensure that the diabetes is, in fact, resolved. This woman needs to be counseled, however, that she is at an increased risk of developing type 2 diabetes in the future. After GDM, 30-65% of women will develop type 2 diabetes within 10 years (Metzger et al., 2007).. For those who have had a normal postpartum GTT, a repeat GTT is recommended at one year and at least every three years thereafter (Metzger et al., 2007). (In addition, this author suggests a few postprandial spot checks with a BG meter as well, for reasons mentioned earlier).

For the woman who has diabetes first, however, the story is a little different. The baby's organs are being formed during the first 7-8 weeks of pregnancy. This is a time during which most women don't even know they are pregnant. The woman in poor metabolic control, by the time she knows she is pregnant, may have sustained significant damage to her developing baby (major birth defects in 5-10% of these women, and spontaneous abortions in 15-20% of these pregnancies) (CDC, 2003). This means that preconception counseling for all women of childbearing age is critical. Potential moms should consult with their primary care and OB providers to make a plan that includes maximizing BG control and effective pregnancy prevention **before** the possibility of becoming pregnant occurs.

Here's the Team, Where's the Captain? ***The Health Care Team***

There is an entire team of professionals available to help the individual with diabetes self-manage his or her condition. These team members may include:

Primary Care Provider	Nurse
Certified Diabetes Educator	Dietician/Nutritionist
Pharmacist	Exercise Physiologist
Social Worker	Podiatrist
Ophthalmologist	Family
Counselor	

The team captain position, however, is a slot reserved for the individual with diabetes. There is probably no other medical condition where the person's own involvement in his or her self-care is so critical. If I have diabetes, what I eat, how much and when, how and if I take my medication, how much physical activity I get and how I cope with the stresses of life all affect my BG levels. I'm the one who needs to make it all work. I have the help of the members of the health care team, but I'm the one who holds the ultimate responsibility for my self-care.

How do we help our patients become educated health care consumers? A reasonable plan of care will include the following (ADA, 2007h):

Annual Physical Examination: This is the PCP's opportunity to care for the whole patient, not just the particular parts that are focused on during brief follow-up visits. Our patients should expect (and ask for) testing of renal function, lipid levels, a urine for microalbumin, a referral for a dilated eye exam and a thorough foot exam. In addition, immunizations are updated as appropriate (flu vaccine, pneumovax, Tetanus/Diphtheria, for example), and the possible use of aspirin is discussed.

Interim visits (often every three months): HgbA1c; lipids, renal, and/or hepatic function tests as appropriate; blood pressure; foot exam; and evaluation of BG control.

We ask a lot of our patients with diabetes: we ask them to learn about this chronic condition, learn about medications and how to take them properly (sometimes by injection), make changes in diet and physical activity, use the health care system effectively, and cope with having this chronic disease. This is no small task, and there is one member of the healthcare team who holds a very important role. This is the Certified Diabetes Educator. The CDE is not only the educator, but is also the interpreter, the motivator, the coach and the encourager. The CDE is the partner who helps the individual to learn problem solving techniques and how to incorporate healthy self-care behaviors into everyday life. Ideally, every person with diabetes will have access to a diabetes self-management education program as well as individual consultation with a CDE as needed. After this initial learning, once a year follow-up with the CDE can provide the necessary review, update, jump-start if necessary, and fine-tuning that will keep the individual in control of his or her diabetes. The American Diabetes Association highlights this need: "Today, self-management education is understood to be such a critical part of diabetes care that medical treatment of diabetes without systematic self-management education is regarded as inadequate." (ADA, 2007i).

There are a number of community resources available to our patients. Listed here are just a few:

American Association of Diabetes Educators

1-800-338-3633 www.diabeteseducator.org

The American Association of Diabetes Educators is a very good resource for patient information, as well as for locating a Certified Diabetes Educator in your patient's area.

American Diabetes Association

1-800-DIABETES www.diabetes.org

The Americans Diabetes Association is a wonderful resource for all types of information regarding diabetes. For example, there is a section for those who have been newly diagnosed with diabetes. There is also a tool available called PHD, or Personal Health Decisions. This is a health risk calculator, where one can enter height, weight, cholesterol level, blood pressure, etc. and view a calculated assessment of current risk for complications such as heart disease, stroke, etc. One can also see how these risks may change with various interventions (for example, how much does my risk for a heart attack decrease if I quit smoking?).

In addition, this website offers an extensive collection of cookbooks, along with many other resources.

National Diabetes Education Program

1-800-438-5383 www.ndep.nih.gov

The National Diabetes Education Program also offers an assortment of diabetes information, as well as free, downloadable patient education materials, including the "ABC's of Diabetes." This approach reminds our patients to think about pursuing regular Hemoglobin A1c's (A), Blood Pressure checks (B) and Cholesterol checks (C).

One particular program to be aware of is the "Small Steps Big Rewards: Your Game Plan to Prevent Type 2 Diabetes." Individual kits are free, and include an informational booklet, a fat and calorie counter and activity record.

What's Wrong With This Picture? ***Access to Diabetes Services***

With the potentially devastating consequences of uncontrolled diabetes, one would think that effective prevention and self-management strategies would be foremost in health care planning and resource allocation. However, this has not been the case. Many health insurance companies will cover an individual's laser surgery, cardiac bypass surgery, renal dialysis and amputation, but will not cover the much less expensive self-management education that can empower the individual to prevent these complications. Yes, there is legislation that approaches this goal; however, there are significant restrictions that stand in the way of many people receiving these necessary services. Medicare covers diabetes self-management education only when provided by an American Diabetes Association "recognized" program. The process by which a program becomes ADA recognized is complex and not easily manageable for smaller or lone providers, particularly in more rural settings. Medicaid does not pay for diabetes education as a stand-alone, billable service. It is assumed that this education happens within the context of the 10 or 15-minute primary care office visit. This creates quite a challenge for the practitioner who would like to offer diabetes self-management education but is limited by reimbursement issues.

There may be hope, however. One example is Independent Health, a major health insurer in Western New York. This company is currently credentialing Certified Diabetes Educators (CDE's) directly to provide diabetes self-management education services. Independent Health is also implementing a provider incentive program, which will work with PCP's to become more aggressive with targets such as A1c's, BP and lipid levels. Perhaps more health insurers will follow suit and provide their members with better access to diabetes self-management education programs in the future.

Back to the Future: *The Challenge*

About 20 years ago, there was a saying in medicine: “If you know syphilis, you know medicine.” That was because syphilis was common, and affected many of the body’s systems. Well, that saying can now be said of diabetes: “If you know diabetes, you know medicine.” Because of the epidemic diabetes has become, and the effect it has on so many systems of the body, if one has a good working knowledge of diabetes and its care, one has a bit of a handle on medicine in general. As the complications of this epidemic continue to claim the good health of so many in our population, it is time that we, as a medical community, become more aggressive, both with medical management of diabetes, as well as with patient education and prevention efforts. We need to be relentless in our efforts at helping our patients to live well with diabetes and not suffer from this chronic disease.

Conclusion

Diabetes has become an epidemic in this country (and the world), with significant human and financial costs. Current trends suggest a continuing increase in the numbers of people who will be affected by this disease in the future, due to an aging population, increasing obesity and sedentary lifestyle. Diabetes is a progressive disease, due to beta cell failure that progresses over time (coupled with continued insulin resistance). The good news is that with aggressive blood glucose control and control of other risk factors, the incidence of diabetes complications can be significantly reduced.

Managing diabetes is a complex process, with multiple possible treatment strategies. However, no matter what pharmacological or other therapy may be introduced, the cornerstone of therapy for the individual with diabetes remains diet, physical activity and self-management education. The inescapable reality is that the responsibility for diabetes self-care rests on the individual with diabetes.

A closing thought:

> "*Never doubt that a small group of thoughtful committed citizens can change the world; indeed, it's the only thing that ever has*."--Margaret Mead

We, as nurses, have the opportunity and responsibility to be those thoughtful committed citizens, as well as essential members of the healthcare team, for our patients with diabetes.

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Taming the Diabetes Monster: Diabetes Care, Treatment and Management
Course Exam

After studying the downloaded course and completing the course exam, you need to enter your answers online. **Answers cannot be graded from this downloadable version of the course.** To enter your answers online, go to e-leaRN's Web site, www.elearnonline.net and click on the Login/My Account button. As a returning student, login using the username and password you created, click on the "Go to Course" link, and proceed to the course exam.

1. The person who has diabetes and no history of heart trouble is at the same risk for a myocardial infarction as the person with heart disease and no diabetes.

    A. True
    B. False

2. Principal risk factors for type 2 Diabetes, which accounts for 90-95% of the cases of diabetes, are:

    A. Hyperlipidemia, hypertension and Type A personality.
    B. Hypertension, family history of diabetes, stressful lifestyle.
    C. Aging, family history of diabetes, obesity and sedentary lifestyle.
    D. Sedentary lifestyle, family history, hypertension and hyperlipidemia.

3. The person who has Type 2 diabetes lacks insulin; the body makes antibodies against its own beta cells thereby destroying their ability to make insulin. A daily insulin injection is generally needed to survive.

    A. True.
    B. False.

4. Once a diagnosis of Diabetes is made, 50% of people already have at least one complication of the illness.

    A. True.
    B. False.

5. The Metabolic Syndrome includes all the following EXCEPT:

    A. Fasting blood glucose equal to or greater than 110; waist circumference greater than 40" in men or greater than 35" in women.
    B. Blood pressure equal to or greater than 130/85; triglyceride levels equal to or greater than 150.
    C. Weight greater than 30 lbs beyond the average for one's height.
    D. HDL levels below 40 in males or below 50 in females.

6. The Hemoglobin A1c test, or glycohemoglobin test represents a 24 hour average blood glucose level over a period of about 6-8 weeks prior to the test. This provides a more representative picture of actual blood glucose levels over time than does the fingerstick blood glucose measurement, which indicates the blood glucose value at that moment.

    A. True.
    B. False.

7. Persons with diabetes who have hypertension have increased risk for heart disease. Current guidelines for control of blood pressure indicate that readings above what level constitute pre-hypertension or hypertension?

    A. 140/95
    B. 120/80
    C. 120/70
    D. 130/90

8. Diabetes is a risk factor for kidney disease. The standard urinalysis identifies protein in the urine beginning at 300 mg/gm creatinine, when nephropathy is already present, so it is not an effective test identify early changes in kidney function as a result of diabetes.

    A. True.
    B. False.

9. Carbohydrates impact blood glucose levels; proteins and fats have little impact. Carbohydrate equivalents to 1 slice of bread (15 gm) include all the following EXCEPT:

    A. 1 small baked potato.
    B. 1/3 cup rice.
    C. 1 cup milk.
    D. 1 cup of corn.

10. Diet is an essential part of treatment and management of diabetes. One representative way of approaching the healthier meal planning recommendations of the American Diabetes Association is illustrated by the “pie” or “plate” chart, with:

    A. 50% carbohydrate, 25 % protein and 25% fat.
    B. 25% vegetables, 25% carbohydrate, 25% fat and 25% protein.
    C. 50% protein, 25% carbohydrate and 25% vegetables.
    D. 50% vegetables, 25% carbohydrate and 25% protein.

11. Exercise helps the body respond to insulin better, thereby decreasing insulin resistance, which improves blood glucose control.

    A. True.
    B. False.

12. The Alpha-Glucosidase Inhibitors are medications that:

    A. Reduce insulin resistance.
    B. Slow down the absorption of carbohydrates in the gut.
    C. Push the beta cells in the pancreas to produce more insulin.
    D. Are synthetic insulin.

13. Incretin mimetics are medications that

    A. Stimulate insulin secretion.
    B. Frequently cause hypoglycemia.
    C. Result in weight gain.
    D. Are used in place of insulin in those with type 1 diabetes.

14. Stress (both emotional and physical) has little or no impact on blood glucose levels.

    A. True.
    B. False.

15. Gestational Diabetes places both mother and baby at risk for complications. All the following are complications of gestational diabetes EXCEPT:

    A. Macrosomia, making delivery difficult for both mother and baby.
    B. Hypoglycemia in the infant, immediately following birth.
    C. Respiratory distress in the infant.
    D. High risk for birth defects.