Oral Medication and Insulin Therapies

A Practical Guide for Reaching Diabetes Target Goals

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I dedicate this book to my husband, Judd Freeman, who has supported my career as a diabetes educator for more than thirty years. Our breakfast and dinner meals were frequently interrupted with calls from my diabetes patients needing assistance with their insulin dosage. He encouraged me during challenging times while on the national American Diabetes Association executive committee and continues to tolerate my numerous trips around the country.

Table of Contents

Preface .... ***vii***

Chapter 1 **Understanding Diabetes 1**

Normal Metabolism 1
Hormones That Affect Blood Glucose 2
Classifications of Diabetes 5
Type 1 Diabetes Mellitus: Immune Mediated Diabetes 5
Type 2 Diabetes Mellitus: Insulin Resistance Diabetes 8
Impaired Glucose Homeostasis 14
Gestational Diabetes Mellitus 14

Chapter 2 **Risk Factors and Diagnosis of Diabetes 15**

Risk Factors for Diabetes Mellitus 15

Chapter 3 **Clinical Target Goals for Diabetes 19**

Clinical Target Goals for Diabetes: Blood Glucose and HbA_{1c} 19

Chapter 4 **Management of Type 2 with Oral Medications 23**

Management of Type 2 Diabetes Mellitus 23
Oral Medications for Type 2 Diabetes 23
Matching Pharmacology to Pathophysiology 24
Algorithm for Type 2 Diabetes Medical Management 25

Chapter 5 **Insulin Therapy: Types of Insulin 31**

Normal Insulin Action 31
Special Insulin Therapy Tips 32
Action of Insulin 33

Chapter 6 **Insulin Therapy: Regimens** **39**
Insulin Regimens 39
Split-Mixed (Breakfast/Dinner) 39
Split-Mixed (Breakfast/Dinner/Bedtime) 41
Multiple Daily Injections (MDI) 42
Continuous Subcutaneous Insulin Infusion (CSII) Pump 43
Combination Insulin 45

Chapter 7 **Insulin Therapy: Calculating Dosage** **47**
Calculating Insulin Dosage 47
Total Daily Dose 47
Basal Dose of Insulin 49
Multiple Basal Rate Calculations for CSII 50
Meal Bolus 50
Insulin Sensitivity Factor/Correction Formula 52
Unused Insulin Rule 53

Chapter 8 **Troubleshooting Blood Glucose Control** **55**
Balancing Blood Glucose Control 55
Troubleshooting Guide: High Blood Glucose Results 59
SMBG Check During the Night 59
Troubleshooting Guide: Low Blood Glucose Results 64

Bibliography **67**

Resources **69**

Websites **73**

Preface

Oral Medication and Insulin Therapies: A Practical Guide for Reaching Diabetes Target Goals is intended as a support for health care professionals who are assisting people with diabetes to reach target goals. The various oral medications and their mechanisms, as well as the types and regimens of the insulin are discussed. Reaching target goals has been shown to prevent the acute (hypoglycemia and hyperglycemia) and chronic (retinopathy, nephropathy, neuropathy, coronary heart disease and stroke) complications of diabetes.

Oral Medication and Insulin Therapies: A Practical Guide for Reaching Diabetes Target Goals provides guidelines to manage the medical needs of the patient with type 1 or 2 diabetes and is meant only as an estimated initiation guide that should be modified by patient experience and clinical judgment. Time course of action of any insulin can vary in different people, or at different times in the same person; thus, time periods indicated in this text should be considered general guidelines only. Self-Monitoring Blood Glucose (SMBG) before meals, 2 hours after meals, bedtime, and during the night is necessary to ascertain the action of oral medication and insulin for individual patients. The process of reaching target blood glucose (BG) usually requires at least four weeks with significant patient education (utilizing a nurse diabetes educator and dietitian diabetes educator) and coaching of self-management skills.

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1 Understanding Diabetes

Normal Metabolism

Diabetes still remains one of the leading causes of death in the United States because of the complications associated with this metabolic condition. These complications can be prevented by reaching target blood glucose (BG) goals.[1, 2]

Pathophysiology should be understood by the health care professional, as well as the patient with diabetes, in order to comprehend all of the complexities that can alter BG results in diabetes. To begin to understand diabetes use **Figure 1: What is Normal Metabolism.**

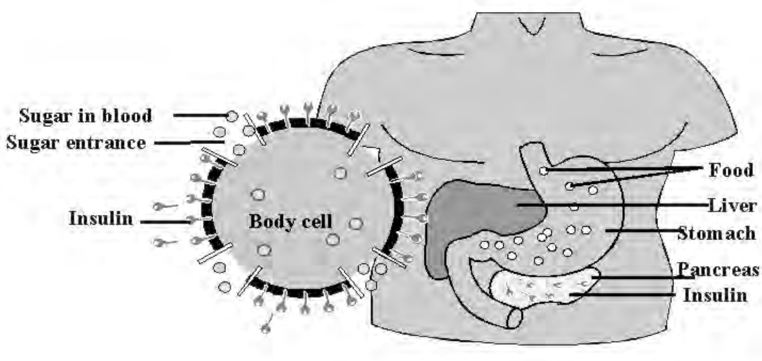

Figure 1: What is Normal Metabolism?

When **food** is eaten, it goes to the **stomach** and changes to **glucose (sugar)**. Everything eaten changes to glucose (even meat is 50% glucose).

Glucose stimulates the beta cells in the islets of Langerhans in the **pancreas** to give off **insulin**. Protein does not require insulin to be utilized.

Insulin is the **key** that opens the **doors (receptor sites)** on the cells to allow the glucose to enter the cell for **energy**.

Energy is needed every second of the day, but food is not eaten every second of the day. Extra glucose not used immediately for energy is stored in the **liver** as **glycogen** (or glucose). If more food is eaten than is burned for energy, that glucose is stored as fat.

Diabetes is a metabolic condition in which either the pancreas produces insufficient insulin or the body cells utilize glucose ineffectively. This results in abnormally elevated blood glucose (BG) levels that, if not controlled, lead to the debilitating complications of diabetes.

Hormones That Affect Blood Glucose

In addition to normal metabolism, **hormones** can alter the BG levels in patients with diabetes. Note **Figure 2: Hormones That Affect Blood Glucose (BG).**

HORMONE	EFFECT ON BG
Insulin	▼
Amylin	▼
Glucagon	▲
Epinephrine	▲
Glucocorticoids	▲
Growth Hormone	▲
Progesterone, HPL, Cortisol	▲

Figure 2: Hormones That Affect Blood Glucose (BG)

Insulin and **Amylin** are normally secreted by the beta cells in the pancreas. Insulin helps to return plasma glucose concentrations to the fasting levels primarily by stimulating the uptake of glucose from the circulation into the muscle and fat cells for storage. Amylin works as a partner hormone to insulin and glucagon, helping to regulate the post-meal increase in glucose concentrations. Trials indicate that amylin injections may reduce the amount of injected insulin required by diabetes patients.

Glucagon is normally secreted by the alpha cells in the pancreas to stimulate the liver to give off glycogen and raise BG levels when a meal is omitted or late.

Epinephrine comes from the adrenal medulla during stress. This also causes the liver to give off glycogen stores. Patients with diabetes demonstrate elevated BG levels during episodes of stress, illness, surgery, etc.

Glucocorticoids stimulate the liver to give off glycogen stores. Cortisol is normally secreted by the adrenal cortex as the long-term stressor causing elevated BG for the diabetes patient. BG can be elevated with interthecal injections, i.e. an injection into the knee of a patient with diabetes caused a BG >500. The patient was not aware that her BG might elevate and she thought something was wrong with her insulin pump. Another patient was given prednisone for asthma which precipitated overt diabetes.

Growth Hormone from the pituitary gland stimulates the liver to give off glycogen. This causes an elevated BG in diabetes patients. Children experience "roller coaster" BG levels during periods of growth and their BG levels can be challenging to control. Growth hormone is present in both children and adults and may produce the **Dawn Phenomenon (Figure 3).** During the night when all the glycogen stores in the muscle are used, growth hormone stimulates the liver to give off glycogen causing an elevated morning BG in patients with diabetes. Therefore, the HS (hour of sleep) BG is normal, the 3 AM BG is normal, and the morning BG is elevated.

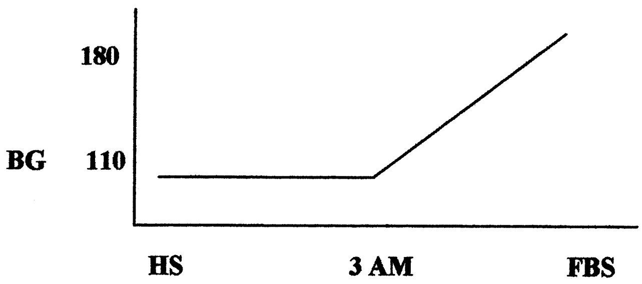

Figure 3: Dawn Phenomenon

Progesterone, HPL, and **Cortisol** are secreted by the placenta during pregnancy. Note **Figure 4: Insulin Requirements During Pregnancy.**[3] Human placenta lactogen (HPL), insulinase enzyme, and cortisol cause insulin resistance. Therefore, the amount of insulin required normally increases at 24–28 weeks gestation. This causes gestational diabetes mellitus (GDM) if the beta cells in the pancreas cannot respond to the increased need for insulin. The woman with diabetes prior to pregnancy has a four-fold increase in insulin requirements during pregnancy. When the baby is born, the insulin requirements return to the non-pregnancy level.

Hormonal levels during menstrual period and menopause can also alter BG levels. Some patients with diabetes experience elevated BG levels five to ten days prior to menses and therefore, need more diabetes medication or insulin.

Figure 4: Insulin Requirements During Pregnancy

Classifications of Diabetes

A panel including members of the National Institutes of Health (NIH), Centers for Disease Control and Prevention (CDC), and American Diabetes Association (ADA) announced the Expert Committee Report in 1997. The report included classifications of diabetes to address the confusion surrounding Non-Insulin Dependent Diabetes Mellitus (NIDDM) patients who require insulin. The new classifications are:

- **Type 1 (Immune-mediated diabetes)** replaces Insulin-Dependent Diabetes Mellitus (IDDM) and Juvenile-onset Diabetes
- **Type 2 (Insulin-resistance)** replaces NIDDM and Mature-onset Diabetes
- **Impaired Glucose Homeostasis** or Pre-Diabetes includes:
    - **IFG (Impaired Fasting Glucose)** is a new category
    - **IGT (Impaired Glucose Tolerance)**
- **GDM (Gestational Diabetes Mellitus)** occurs during pregnancy.

Type 1 Diabetes Mellitus: Immune Mediated Diabetes

Type 1 diabetes is **immune mediated diabetes** and occurs when the beta cells in the pancreas are destroyed. Note **Figure 5: Type 1 Diabetes, Immune Mediated Diabetes.** The destruction of the pancreatic beta cells prevents the normal release of insulin and therefore, causes an abnormal increase in BG.

Many people have reported that type 1 diabetes developed after an episode of severe "flu." Although the onset may seem abrupt, beta cell destruction occurs over a variable interval of months to years and may be triggered by viral infections such as coxsackie B_4 and rubella. Beta cells are damaged by an autoimmune process in which the body produces antibodies against itself. Therefore, the new name "immune-mediated" is more correct and complete.

This classification was formerly called Insulin-Dependent Diabetes Mellitus (IDDM) as the patient "depends" on exogenous insulin injections to survive. The new classification is according to the etiology instead of the treatment of the condition.

This classification was also formerly known as Juvenile-onset Diabetes. Type 1 diabetes usually occurs before the age of 20. However, it can occur at a later age. Therefore, the term Juvenile-onset Diabetes is no longer used. The term **Latent Autoimmune Diabetes of Adulthood (LADA)** is now used to describe the condition in which adults acquire type 1 diabetes.

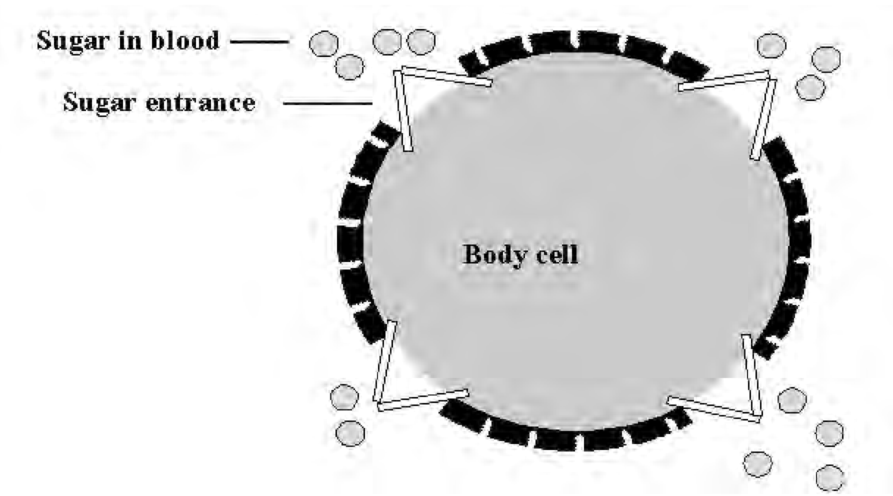

Figure 5: Type 1 Diabetes: Immune Mediated Diabetes

The classic symptoms of type 1 diabetes are:

- Polyuria (frequent urination)
- Polydipsia (increased thirst)
- Polyphagia (increased hunger)
- Unusual weight loss
- Extreme fatigue
- Irritability

When the beta cell in the pancreas secretes the insulin molecule, the C-peptide (connecting peptide) is cleaved off. The laboratory test to measure the amount of C-peptide in the serum ascertains if the amount of insulin is low (as in type 1 diabetes); or normal or elevated (as in type 2 diabetes). Note **Figure 6: Insulin Molecule with the C-Peptide.**

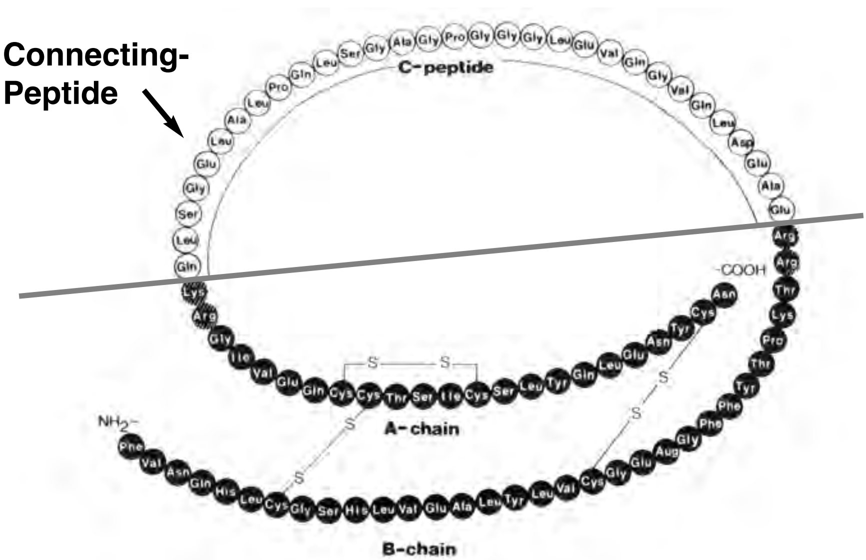

Figure 6: Insulin Molecule with the C-Peptide

The diagnostic key to type 1 diabetes is that the patient is prone to **Diabetic Keto-Acidosis (DKA): Figure 7.** When there is not enough insulin to get the sugar into the cells for energy, fat is burned for energy causing the body cells to become too acid, leading to DKA. This can lead to coma and death if not treated with insulin. DKA also occurs when the BG levels are too high in type 1 diabetes, especially when insulin is omitted.

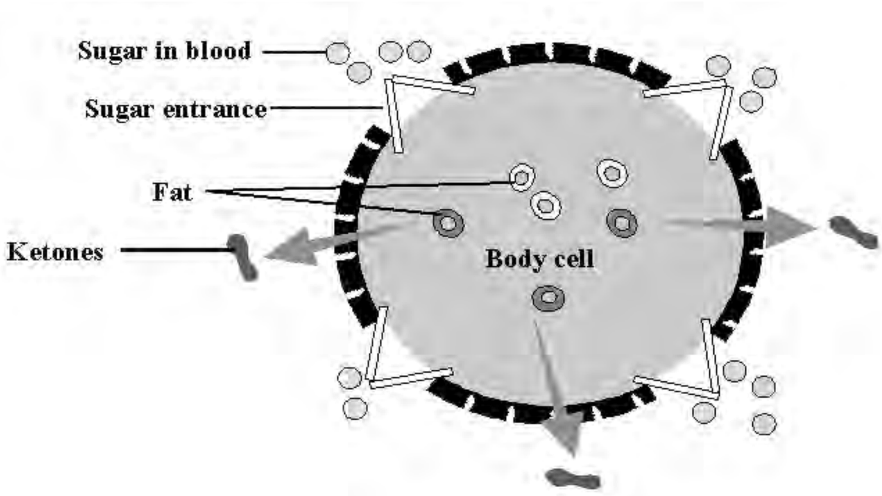

Figure 7: Diabetic Keto-Acidosis (DKA)

In summary, type 1 diabetes is:
• Auto-immune, islet cell antibodies present • Complete insulin deficiency (insulinopenic) • Absolute dependence upon exogenous insulin • Prone to DKA • Lean, recent weight loss • Abrupt onset, usually before age 40 • May occur in elderly

Type 2 Diabetes Mellitus: Insulin Resistance Diabetes

Type 2 diabetes is **insulin resistance diabetes** because the **doors (receptor sites)** on the cells close due to obesity, but the beta cells are not destroyed as in type 1. The pancreas continues to produce insulin, but a blockage occurs which prevents the body cells from utilizing BG. Ninety percent of type 2 diabetes patients are overweight or obese. Note **Figure 8: Type 2 Diabetes Mellitus: Insulin Resistance Diabetes.**

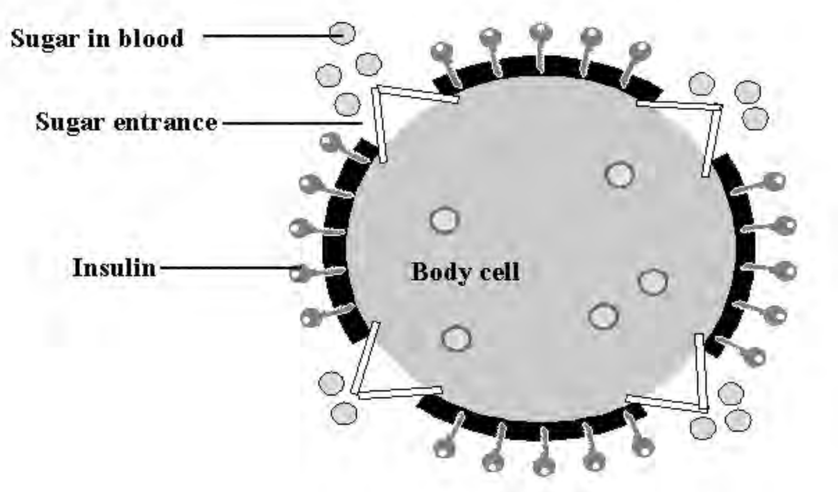

Figure 8: Type 2 Diabetes Mellitus: Insulin Resistance Diabetes

Type 2 was formerly known as Non-Insulin Dependent Diabetes Mellitus (NIDDM) and Mature-onset Diabetes. The name has been changed because many of these patients require insulin to reach target BG levels and type 2 can also occur at a younger age. Obesity and sedentary lifestyle are significant contributory influences to insulin resistance. In the US 65% of our population is overweight or obese. Type 2 diabetes is presently in epidemic proportions because increasing numbers of children are overweight, obese and inactive. Type 2 diabetes increased in youth 125% during 2002.

The symptoms of type 2 diabetes are:

- Any symptoms of type 1 diabetes
- Frequent infections
- Blurred vision
- Cuts/bruises slow to heal
- Tingling or numbness in hands/feet
- Recurring skin, gum, or vaginal/bladder infection
- No symptoms

Figure 9: Pathophysiology of Type 2 Diabetes shows the various areas that cause the hyperglycemia (elevated BG):

1. During the night the liver gives off an excess of glycogen or **increased hepatic glucose output** leading to elevated Fasting Blood Sugar (FBS) in the morning.
2. Insulin resistance due to receptor and post-receptor cell defects thought to be due to obesity causes **decreased peripheral glucose uptake** at the muscle leading to elevated BG before meals (AC).
3. Insulin deficiency due to **decreased insulin secretion** from the beta cell leading to elevated BG two hours postprandial or after eating (2 H PC).
4. Too much food causes **increased glucose influx**. This leads to elevated BG 2 H PC.

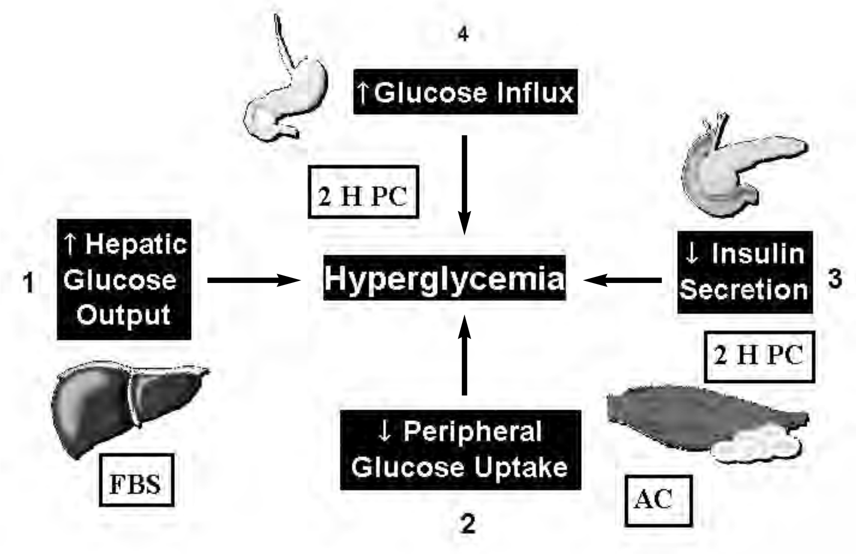

Figure 9: Pathophysiology of Type 2 Diabetes

Note **Figure 10: Natural History of Type 2 Diabetes.**[4] The natural history of type 2 diabetes is illustrated more graphically in this figure. In the period preceding the onset of frank diabetes (characterized by impaired glucose tolerance) macrovascular risk (cardiovascular disease (CVD), coronary artery disease (CAD), peripheral vascular disease (PVD) and stroke (CVA)) is already very pronounced **(note large lower arrow)**. Microvascular risk (retinopathy, nephropathy, and neuropathy) begins after the onset of hyperglycemia.

The two lower graph lines illustrate fasting and postprandial (after eating) glycemia; the earliest abnormality in patients with type 2 diabetes is primarily observed in the postprandial state **(note large upper arrow)**. This abnormality remains more significant than the fasting plasma glucose abnormality for the duration of the disease.

The fasting plasma glucose concentration begins to rise after the postprandial rise in glucose and is coincident with the increase in hepatic glucose production, which, in turn, is coincident with the decline of endogenous insulin secretion. Insulin resistance, which is

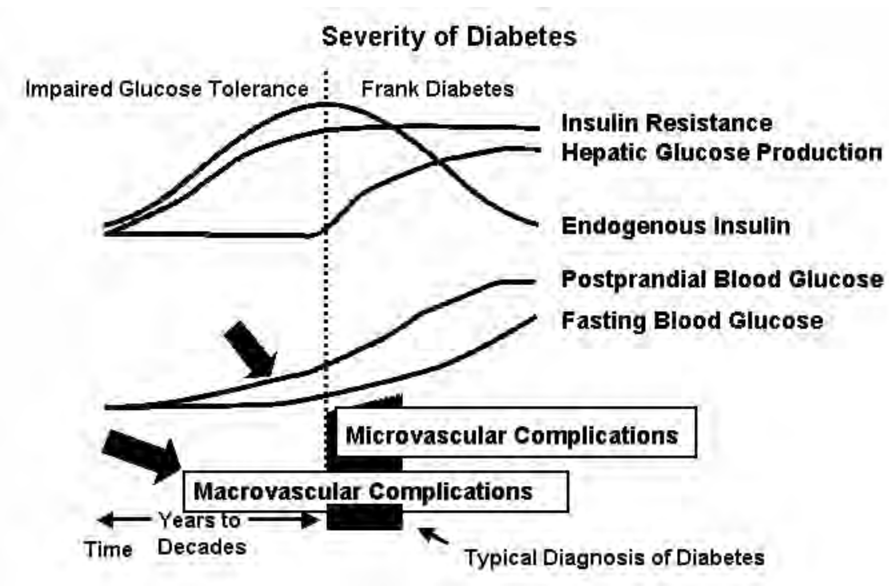

Figure 10: Natural History of Type 2 Diabetes

fully expressed in the stage of impaired glucose tolerance, tends not to change very much over the course of disease. The insulin resistance causes **hyperinsulinemia** (increased endogenous insulin levels and C-peptide in the blood).

From this pathophysiologic scheme of progressive beta-cell failure, it is clear that over time all type 2 patients will require either increased endogenous insulin production or exogenous insulin therapy.

Note **Figure 11: Loss of 1st-Phase Insulin Secretion in Type 2 Diabetes.**[5] The left figure shows phases of insulin release based on the IV Glucose Tolerance Test (GTT) in normal subjects.

- The 1st-phase (acute release) depends on immediate-releasable insulin stores, reflected as a sharp increase in insulin response in the first ten minutes.
- The 2nd-phase partially depends on insulin stores and protein synthesis within the beta cell, shown as persistent increase in insulin concentration from ten to one hundred twenty minutes.

The right figure shows the loss of 1st- and 2nd-release insulin secretion based on IV GTT in patients with type 2 diabetes. The loss of release of insulin secretion represents one of the earliest detectable manifestations; no clinically evident disease exists without this defect, despite presence of insulin resistance. Therefore, the BG 2 H PC is elevated.

Normal vs. type 2 diabetes

Insulin Levels	**Normal**	**Type 2 Diabetes**
After glucose	↑ >7-fold	↓ 2-fold
Peak	within few min	~20 min
Decline	within min after peaking	60 min
Reach baseline levels	within 45 min	>120 min

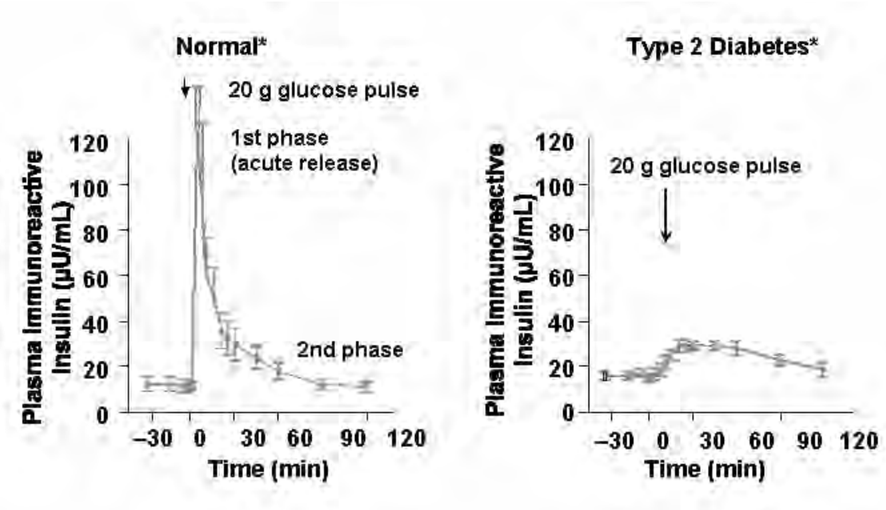

Figure 11: Loss of 1st-Phase Insulin Secretion in Type 2 Diabetes

"Syndrome X" or Metabolic Syndrome is associated with insulin resistance, compensatory hyperinsulinemia, obesity (abdominal or visceral), dyslipidemia (elevated triglycerides, decreased HDL, elevated LDL) and hypertension. Syndrome X has an increased rate of coronary heart disease (CHD). According to Dr. DeFronzo of San Antonio, the treatment for Syndrome X is 3 fold: exercise, exercise, and exercise! Weight loss, exercise and oral medication can decrease the resistance of the cells to insulin action.

In summary type 2 diabetes is:

- Not absolutely dependent upon exogenous insulin
- Progressive condition, gradual onset
- May be relatively free of classical symptoms
- Not prone to DKA
- Strong family history of diabetes mellitus
- Usually obese or history of obesity, sedentary lifestyle
- Usually diagnosed after age 40, but epidemic in youth
- Higher incidence in African-American, Hispanic-American, Native-American, Asian-American, Pacific Islander

Impaired Glucose Homeostasis

Impaired Glucose Homeostasis includes **Impaired Fasting Glucose (IFG)** and **Impaired Glucose Tolerance (IGT)**. These terms refer to a metabolic stage intermediate between normal glucose homeostasis and diabetes, now referred to as **"Pre-diabetes."** Impaired Glucose Tolerance (IGT) was formerly called "borderline diabetes." These patients are at potential risk for developing the chronic complications of diabetes and Coronary Heart Disease (CHD).[6]

Gestational Diabetes Mellitus

Gestational diabetes mellitus (GDM) is defined as any degree of glucose intolerance with onset or first recognition during pregnancy, (Note **Figure 4: Insulin Requirements During Pregnancy**). The definition applies regardless of whether insulin or only diet modification is used for treatment or whether the condition persists after pregnancy. Forty to sixty percent of gestational diabetes patients will develop overt type 2 diabetes in 7 to 10 years post partum. If they are lean and fit, the risk factor is 25%. The chance of developing type 1 within one year is 3–7%.

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Diabetes Interview	www.diabetesinterview.com
Diabetes Self Management	www.diabetes-self-mgmt.com
Insulin Pumpers	www.insulin-pumpers.org
Joslin Diabetes Center	www.joslin.harvard.edu
Juvenile Diabetes Foundation	www.jdrf.org
National Diabetes Education Program (of NIH & CDC & Prevention in children for health care professionals, parents, school personnel, media)	www.ndep.nih.gov
National Institute of Diabetes & Digestive and Kidney Disease	www.niddk.nih.gov

National Diabetes Information Clearinghouse (Part of NIH for diabetes patient education booklets)	www.niddk.nih.gov/diabetes
Online Diabetes Resources by Rick Mendosa	www.mendosa.com/diabetes.htm
The International Diabetes Foundation	www.idf.org
The American Association of Diabetes Educations	www.diabeteseducator.org
The American Diabetes Association	www.diabetes.org
The American Diabetes Association Education Recognition	www.diabetes.org/recognition