



**DIABETES MELLITUS**  
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**3-01-10**

# DEFINITION

- Diabetes Mellitus is a syndrome of disordered metabolism marked by high levels of blood glucose resulting from defects in insulin production, insulin action, or both.



# PREVALENCE

- Prevalence of diabetes in the US in 2005:
  - 20.8 million; 7.0% of the population.
    - Diagnosed: 14.6 million
    - Undiagnosed: 6.2 million



# FIND THE SWEET PEOPLE

- Test When?
  - Any age if  $\text{BMI} \geq 25$  + any risk factor
  - Anybody  $\geq 45$  years of age



	<b>Sugar</b>	<b>condition</b>
<b>FBS</b>	<100	Normal
<b>FBS</b>	$100 \leq \text{FBS} < 126$	Impaired Fasting Glucose: Increased risk for diabetes
<b>FBS</b>	$\text{FBS} \geq 126$	Diabetes
<b>2-hr (75 gm)</b>	<140	Normal
<b>2-hr (75 gm)</b>	$140 \leq \text{PP} < 200$	impaired Glucose Tolerance: increased risk for diabetes
<b>2-hr (75 gm)</b>	$\text{PP} \geq 200$	Diabetes
<b>Random</b>	$\geq 200 \text{ mg\%}$	Diabetes
<b>HgA1C</b>	< 5.7%	Normal
<b>HgA1C</b>	$5.7 \leq \text{HgA1C} < 6.5\%$	Increased risk for diabetes
<b>HgA1C</b>	$\text{HgA1C} \geq 6.5\%$	Diabetes

In the absence of unequivocal hyperglycemia, criteria should be confirmed by repeat testing.



# DM PRESENTATION

- Polyuria
- Polydipsia
- Recurrent blurred vision
- Paresthesias
- Nocturnal enuresis in very young children.
- Weight loss usually over weeks
- Dizziness and weakness



- In the absence of unequivocal hyperglycemia, these criteria should be confirmed by repeat testing on a different day.
- OGTT is not recommended for routine clinical use.



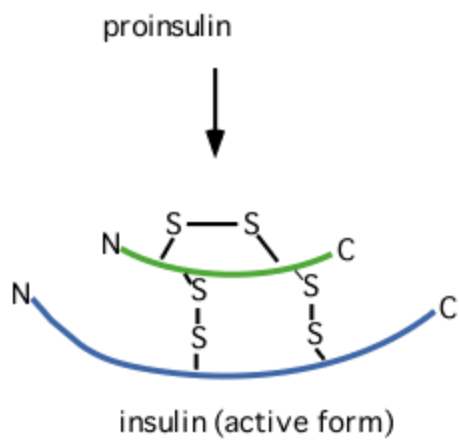
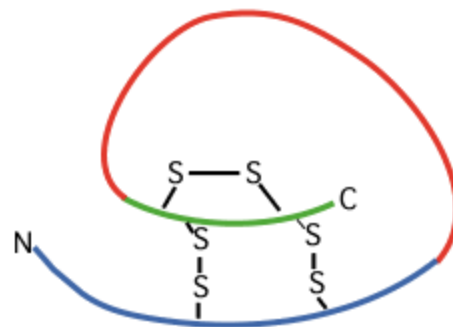
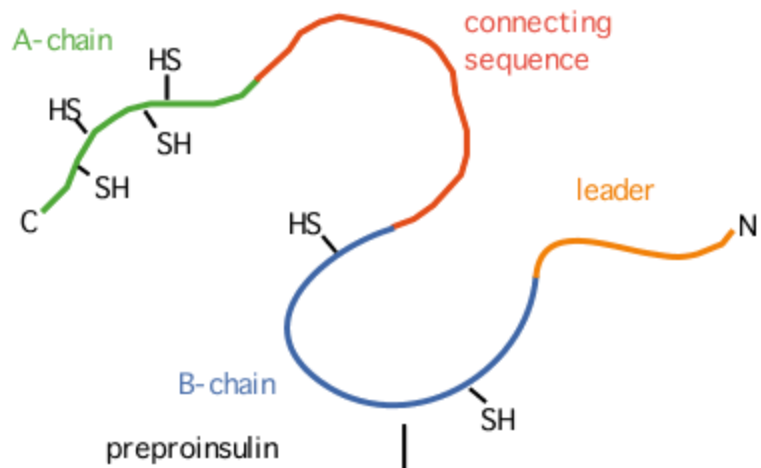
- Type 1 diabetes
- Type 2 diabetes
- Other specific types of diabetes due to other causes, e.g., genetic defects in cell function, genetic defects in insulin action, diseases of the exocrine pancreas, and drug or chemical-induced diabetes.
- Gestational diabetes mellitus (GDM) (diabetes diagnosed during pregnancy)



# DM TYPE 1

- Type 1 diabetes:
- 5-10% OF ALL DIABETICS
- **Immune-mediated** (-cell destruction, usually leading to absolute insulin deficiency)





- islet cell auto-antibodies (ICA), AND auto-antibodies to glutamic acid decarboxylase (GAD-65) are used for definite diagnosis of type 1
- C-peptide
  - ( $t_{1/2}$  ~ 30 min. (insulin ~ 4 min.)
  - Excreted exclusively by the kidney
  - Low variability and high reproducibility of.
  - It measures residual beta-cell function.



- Symptoms develop acutely/subacutely within days to weeks
- More common in younger ages (children and adolescents)
- Absolute Insulin deficiency can lead to Diabetic Keto-acidosis as the presenting symptoms



# DKA

## ○ Symptoms:

- Polyuria, polydipsia, polyphagia, weakness.
- Kussmaul's respirations.
- Nausea and vomiting in 50 to 80 percent of patients,
- Abdominal pain in 30 percent. Coffee-ground emesis in 25 percent of vomiting patients.

## ○ Signs:

- Temperature usually is normal or low.
- Dry mucous membranes, tachycardia, and hypotension,(10% dehydration).
- Breath with fruity odor
- Consciousness ranges from alert to confused to a comatose state <20 percent of patients.



## CASE A

- A 14-year-old male is brought to the Emergency Department via ambulance with a report of the patient being found unresponsive. Copious quantities of black colored vomit were evident.
- His mother told the medics that patient has been feeling tired lately with symptoms of excessive thirst and frequent usage of rest room over last 2 weeks.



# CASE A

- **Vital Signs:** Blood pressure: 101/72; heart rate: 123; respirations: 32; oral temperature: 34.8 °C; pulse oximetry: 100% on room air.
- **General:** An approximately 65 Kg, thin male who responds to simple questions with moans, but is, in general, responsive only to very loud or painful stimuli.
- **Head and neck:** very dry mucous membranes and a moderate amount of dried, black material (Gastrocult positive).
- **Lungs are clear.** His respiratory pattern is that of rapid and deep Breathing.
- **Abdomen exam is negative.**
- **There are no other pathological findings on PE.**



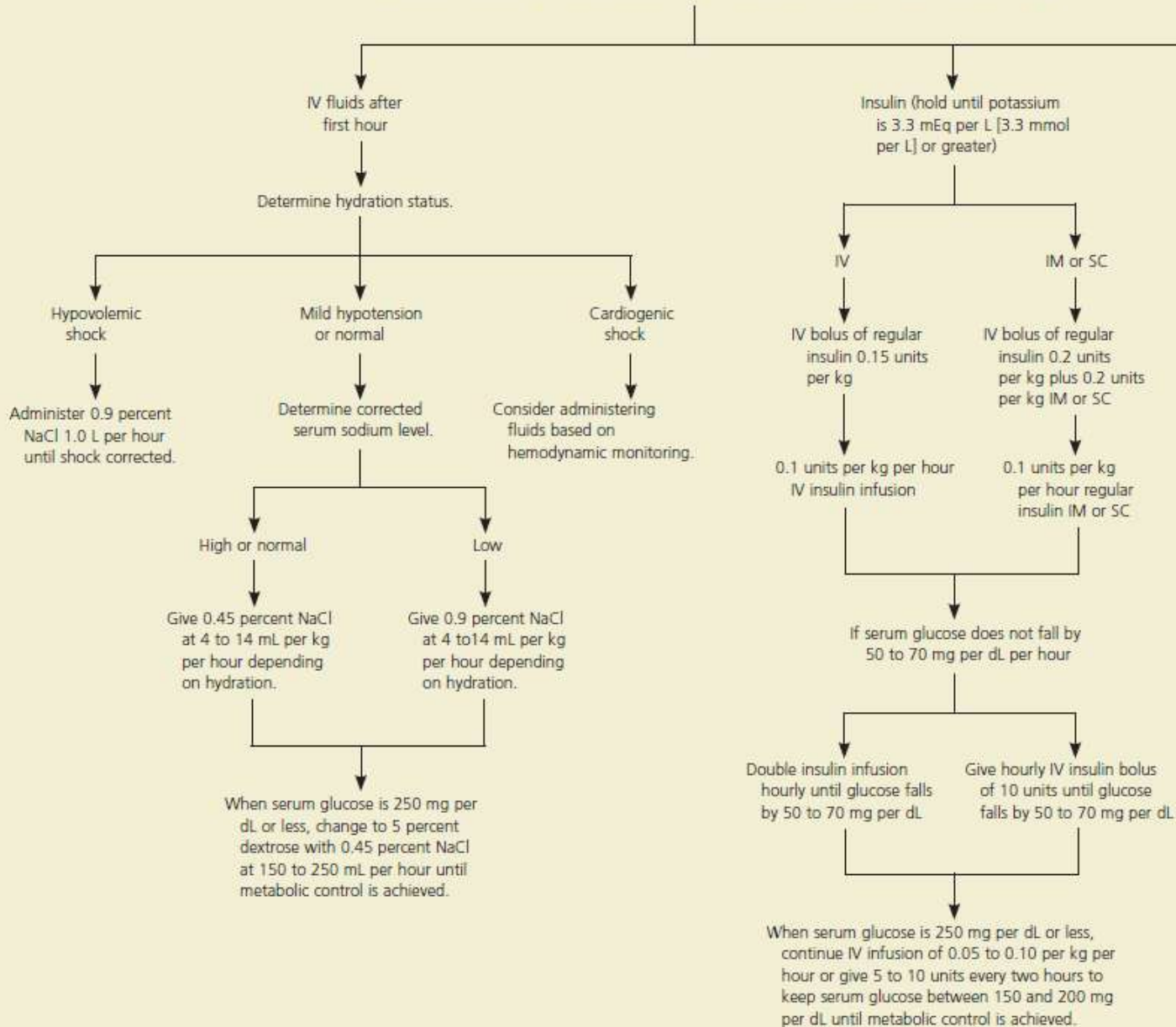
## CASE A

- arterial blood gas pH of 6.92, CO<sub>2</sub> of 9 and a bicarb of 2.
- WBC 62.6 thousand (62,600), hemoglobin of 14.4 mg/dL, hematocrit of 43.5%.
- Na 127, K 5.2, Cl 87, CO<sub>2</sub> <5, BUN 32, cr 1.5, and a blood sugar of 1,582.
- The serum ketones were positive at a dilution of 1:32.



# Management of Adults with Diabetic Ketoacidosis

Perform history and physical examination, order laboratory tests, and evaluate severity of diabetic ketoacidosis. Quickly start 0.9 percent NaCl at 1.0 L per hour (15 to 20 mL per kg) for first hour.



# DM TYPE 1

- Normally the pancreas secretes one unit of insulin/hr in adults.
- Adults need around 20-25 units of insulin per day when NPO.
- Most newly diagnosed patients with type 1 diabetes can be started on a dose of 0.2 to 0.4U/Kg/d, majority will require 0.6 to 0.7 U/Kg/d.



# DM TYPE 2

- Type 2 diabetes
- Insulin resistance with relative insulin deficiency. It accounts for 90–95% of those with diabetes,



## DM TYPE 2

- Insidious onset of hyperglycemia, and may go undiagnosed for many years
- In Women can present as chronic candidal vulvovaginitis, or having delivered large infants (>9 lb, or 4.1 kg).
- In men, Impotence can be the presenting symptom
- Ketoacidosis, seldom occurs, but can occur with stresses.



# DM TYPE 2 RISK FACTORS

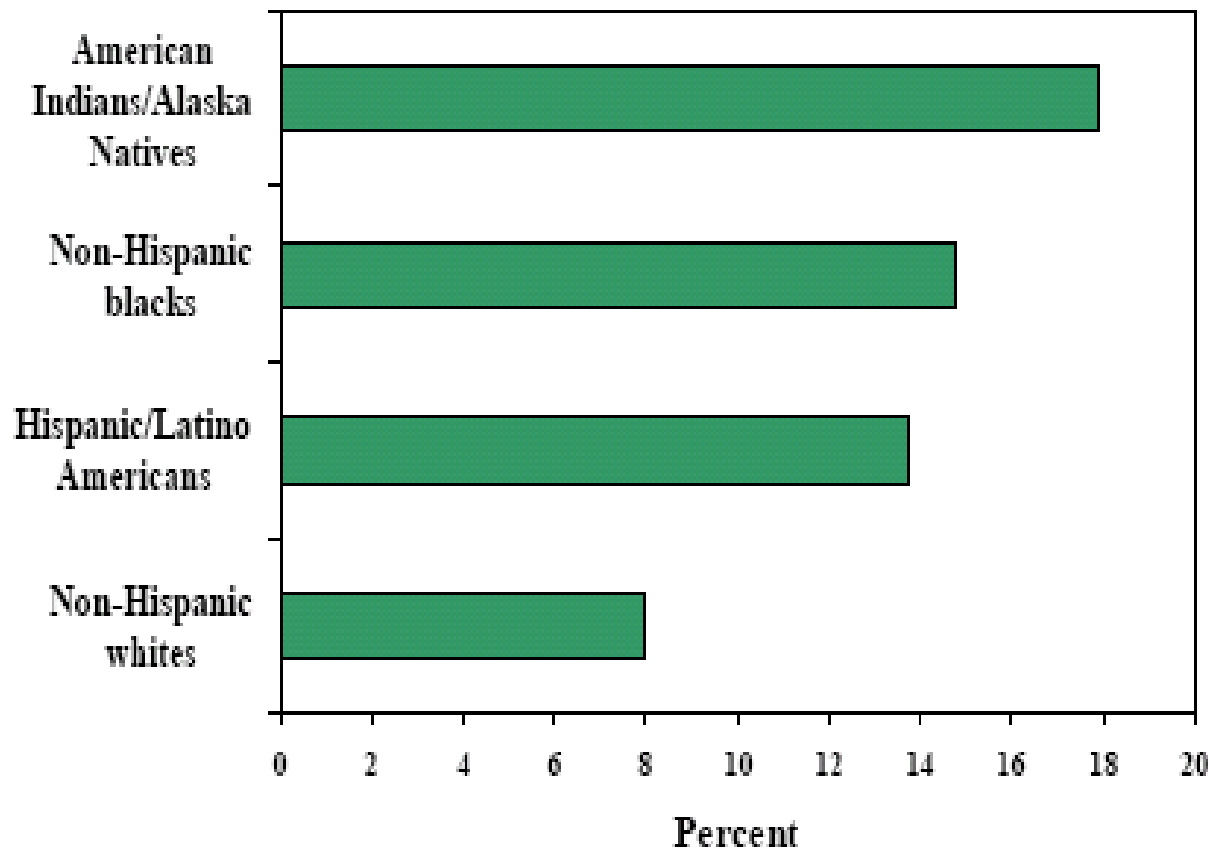
- Age
- Obesity
- Lack of physical activity.
- GDM
- Hypertension
- Dyslipidemia,
- Different racial/ ethnic subgroups.
- Family history



- Centripetal fat distribution has been termed "android"
- High waist to hip ratio
- Hypertension



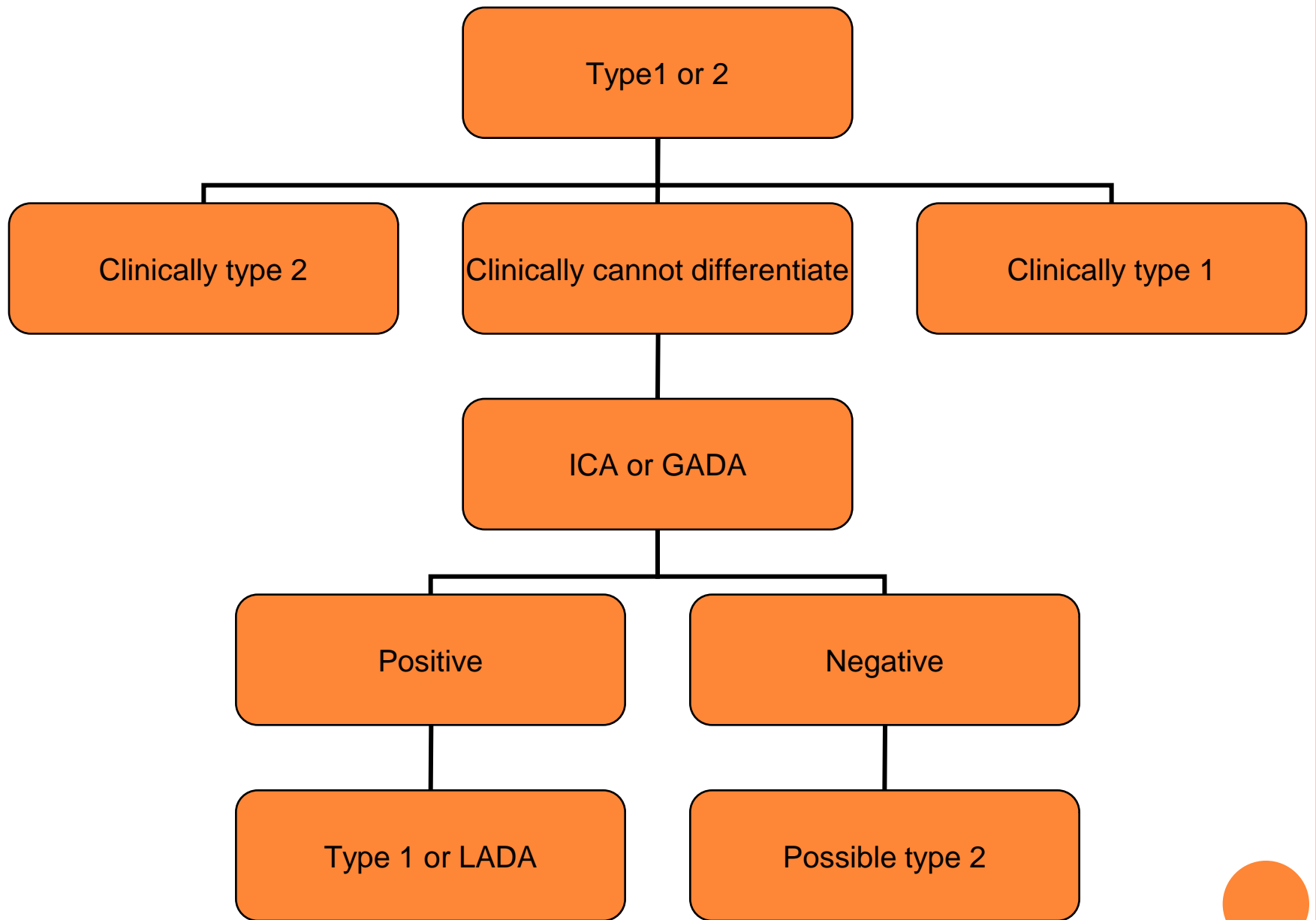
Estimated age-adjusted total prevalence of diabetes  
in people aged 20 years or older, by race/ethnicity—  
United States, 2005



# KETOSIS-PRONE TYPE 2 DIABETES (KPD)

- **Ketosis-prone type 2 diabetes mellitus (KPD)** — "atypical diabetes," "Flatbush diabetes," "diabetes type 1B," and "ketosis-prone type 2 diabetes mellitus"
- Obese
- Diabetic ketoacidosis as first manifestation
- Subsequently found to have type 2 diabetes
- Typically African-Americans, or of African, Hispanic or Caribbean descent.
- Mean age is 40 years, higher prevalence in men.
- Strong family history of type 2 diabetes.
- Ketoacidosis results from transient suppression of beta cell function, the cause for which is unknown ? Glucotoxicity
- Initial treatment as for type 1 diabetes and ketoacidosis.
- Diabetic remission (A1C <6.3 percent, fasting glucose <120 mg/dL) has been reported for 40 to 70 percent after three months
- Long term will need treatment like type 2.





# ASSESSMENT OF GLYCEMIC CONTROL

- **Assessment of glycemic control.**
  - patient self-monitoring of blood glucose (SMBG)
  - HgbA1C measurement.
  - Continuous monitoring of interstitial glucose



# ASSESSMENT OF GLYCEMIC CONTROL

## HgA1C

- Hemoglobin A1C reflects sugar control over the last 2-3 months
- HgbA1C at least two times a year in patients who are meeting treatment goals
- HgbA1C quarterly in patients whose therapy has changed or who are not meeting glycemic goals.



## *SUMMARY OF GLYCEMIC RECOMMENDATIONS FOR NON-PREGNANT ADULTS WITH DIABETES*

A1C

<7.0%\*

Preprandial capillary plasma glucose  
70–130 mg/dl (3.9–7.2 mmol/l)

Peak postprandial capillary plasma glucose  
<180 mg/dl (<10.0 mmol/l)

\*Referenced to a nondiabetic range of 4.0–6.0% using a DCCT-based assay. Postprandial glucose measurements should be made 1–2 h after the beginning of the meal, generally peak levels in patients with diabetes.



# KEY CONCEPTS IN SETTING GLYCEMIC GOALS:

- A1C is the primary target for glycemic control.
  - Goals should be individualized based on:
    - duration of diabetes
    - age/life expectancy
    - comorbid conditions
    - known CVD or advanced microvascular complications
    - hypoglycemia unawareness
    - individual patient considerations
    - More or less stringent glycemic goals may be appropriate for individual patients.
- Postprandial glucose may be targeted if A1C goals are not met despite reaching preprandial glucose goals.



# SELF MONITORING OF BLOOD GLUCOSE: SMBG

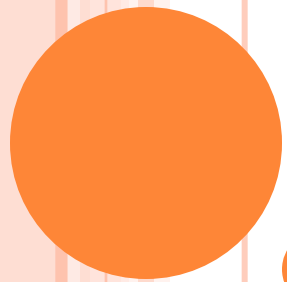
- SMBG:
  - Three or more times daily with multiple insulin injections.
  - Unknown frequency with oral agents or medical nutrition therapy (MNT) alone, (FBS and 2-hr PP)



# GLYCEMIC GOALS

- Less stringent HgbA1C goals may be appropriate for patients with a history of severe hypoglycemia, patients with limited life expectancies, children, individuals with comorbid conditions, and those with longstanding diabetes and minimal or stable microvascular complications.





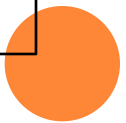
# TREATMENT

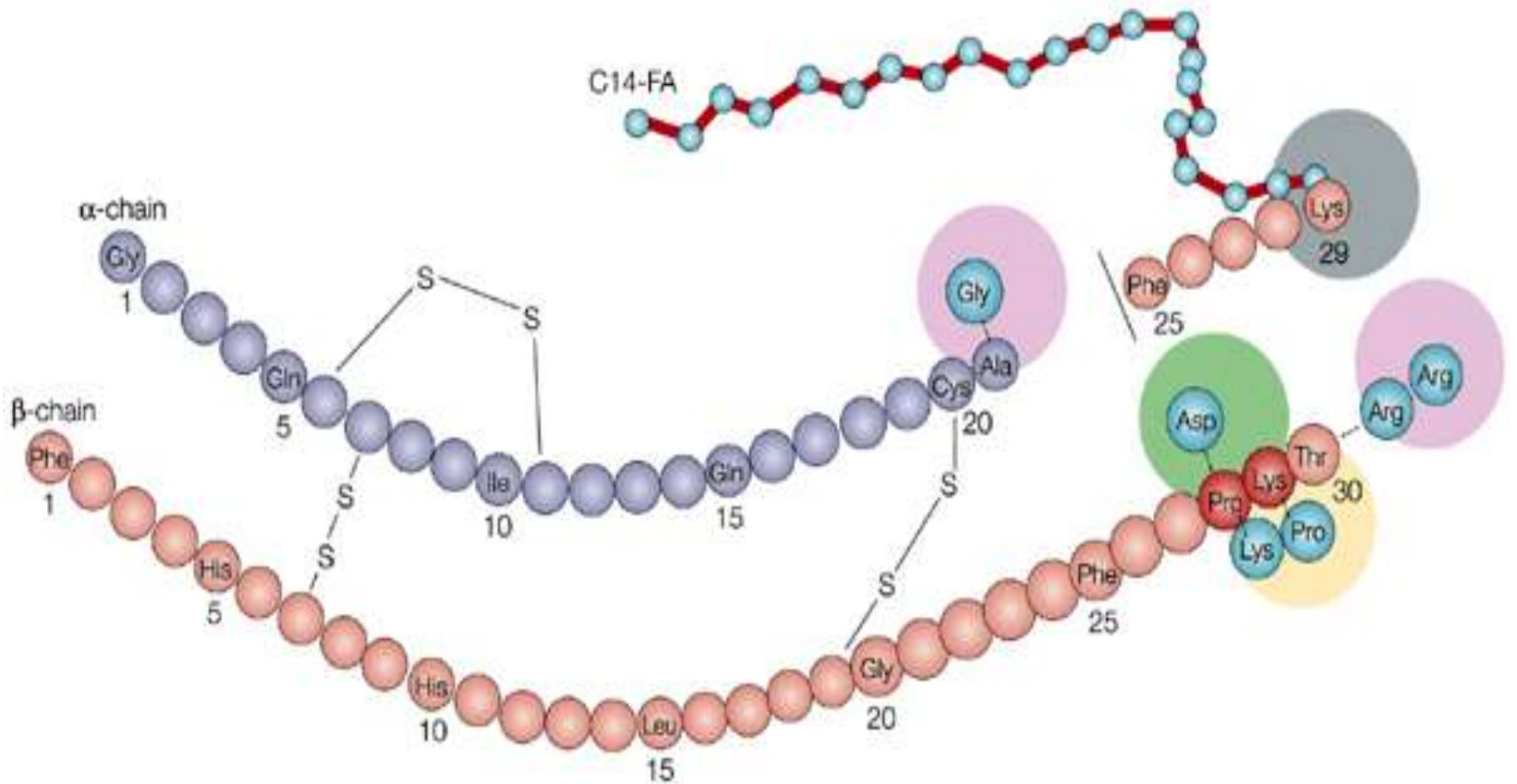
# DM TYPE 1

- Insulin therapy is the only treatment for all patients with type 1 diabetes
- Intermediate or long-acting basal insulin in combination with pre-meal rapid- or short-acting insulin



	<b>Insulin, Generic Name (Brand)</b>	<b>Onset</b>	<b>Peak</b>	<b>Duration</b>
<b>Rapid Acting</b>	Insulin aspart injection (NovoLog) Insulin lispro injection (Humalog) Insulin glulisine injection (Apidra)	5-15 min	30-90 min	<5 h
<b>Short Acting</b>	Regular (Novolin R)	30-60 min	2-3 h	5-8 h
<b>Intermediate, Basal</b>	NPH (Novolin N)	2-4 h	4-10 h	10-16 h
<b>Long-acting, Basal</b>	Insulin glargine (Lantus)	2-4 h	No peak	20-24 h
	Insulin detemir (Levemir)	3-8 h	No peak	5.7-23.2 h





**Fast-acting analogues**



Insulin lispro



Insulin aspart

**Long-acting analogues**

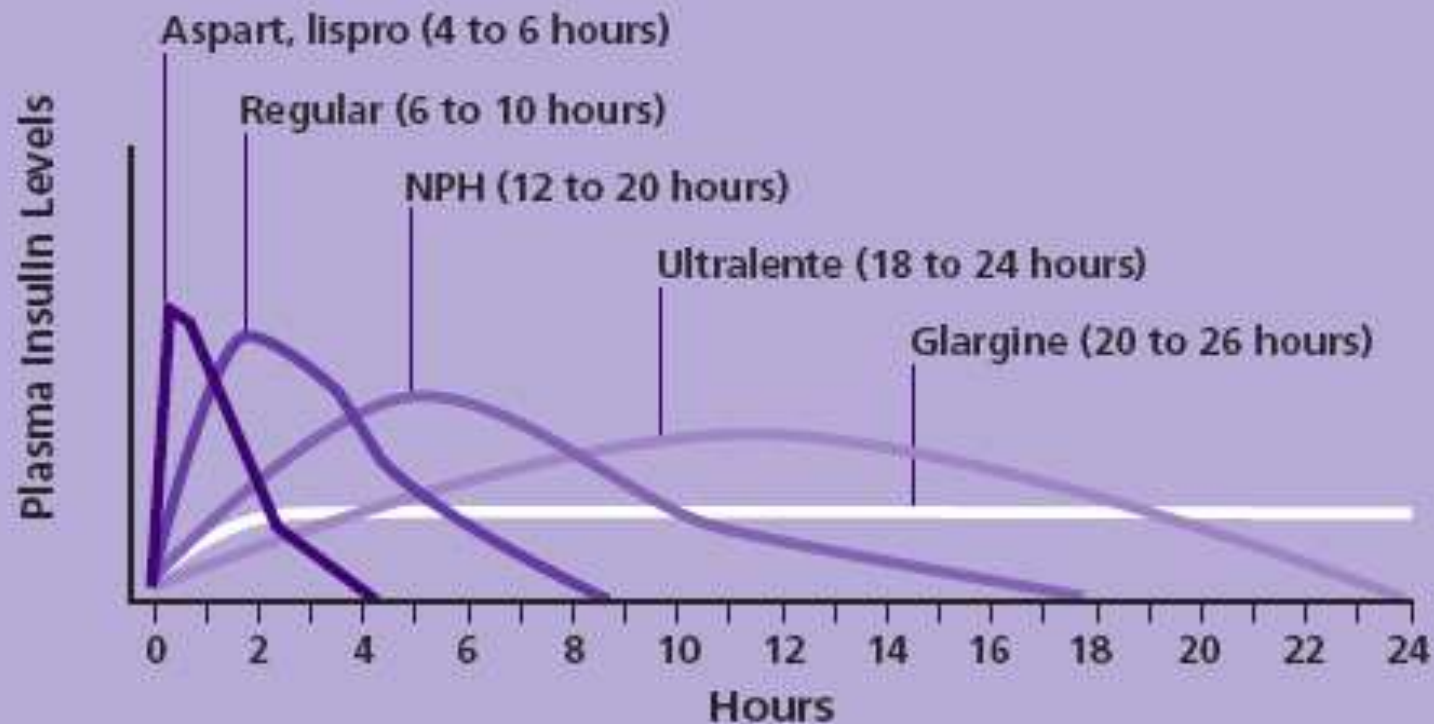


Insulin glargine



Detemir insulin

## Profiles of Human Insulins and Analogues

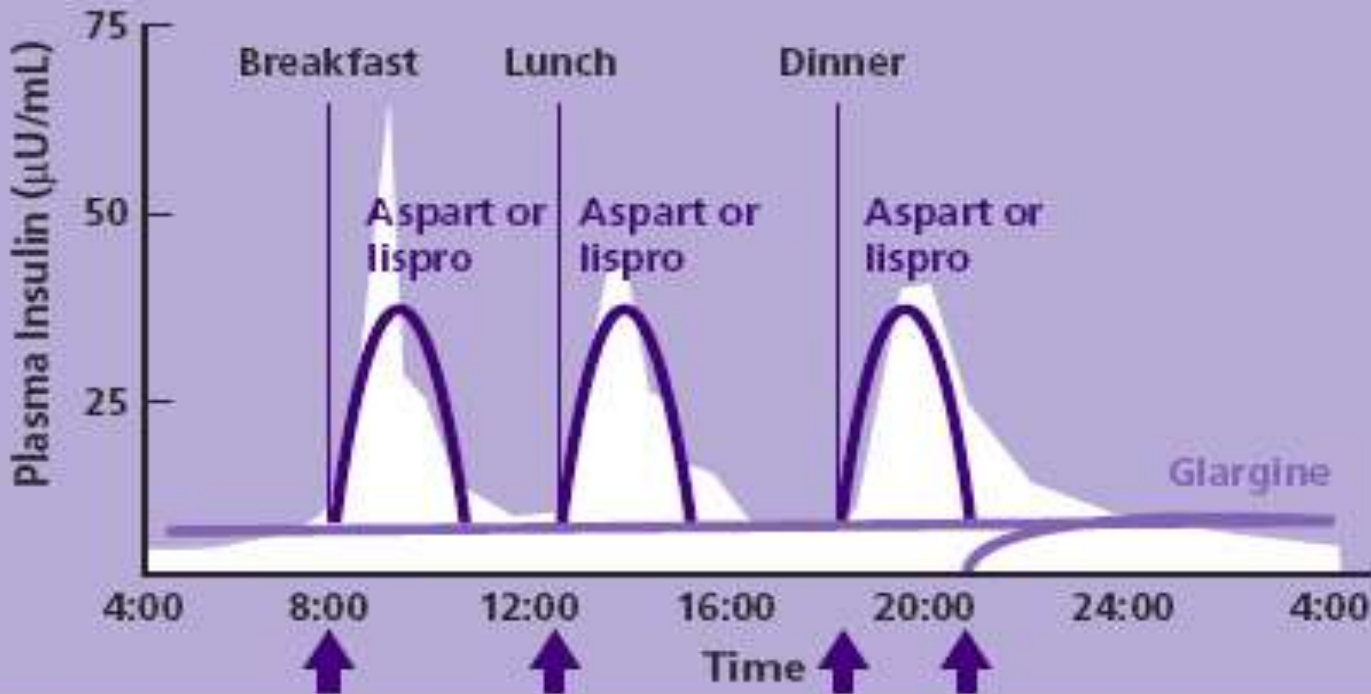


**Figure 2.** Onset of action, peak, and duration of action of exogenous insulin preparations. (Neutral protamine Hagedorn = NPH)

*Reprinted with permission from the American Diabetes Association's Clinical Education Program "Insulin Therapy for the 21st Century."*

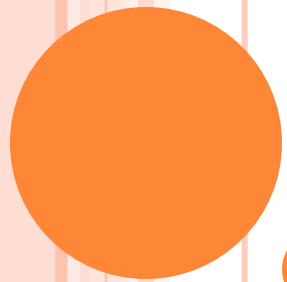


## Basal-Bolus Therapy Using Glargine and Aspart or Lispro



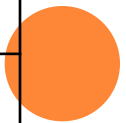
**Figure 7.** Basal plus meal-related regimen using glargine plus aspart or lispro.

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## TREATMENT TYPE 2

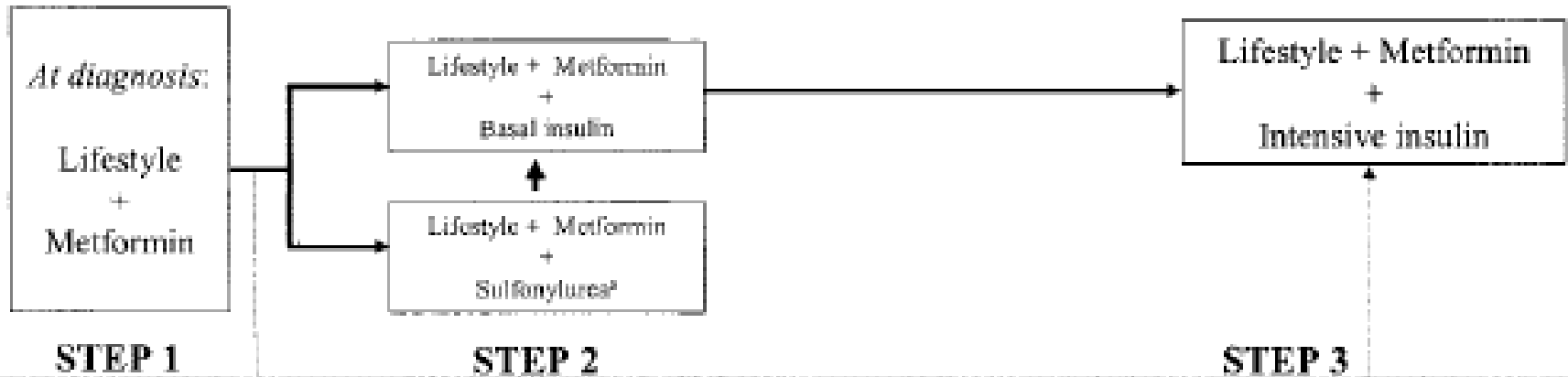
<b>Drug Class</b>	<b>Agent</b>		<b>Drug Class</b>	<b>Agent</b>
<b>Sulfonylurea</b>	<b>first generation</b>		<b>Biguanides</b>	<b>Metformin (Glucophage)</b>
	<b>Acetohexamide (Dymelor)</b>			
	<b>Chlorpropramide (Diabinese)</b>		<b>Thiazolidinediones</b>	<b>Rosiglitazone (Avandia)</b>
	<b>Tolazamide (Tolinase)</b>			<b>Pioglitazone (Actos)</b>
	<b>tolbutamide (Orinase)</b>			
			<b><math>\alpha</math>-glucosidase inhibitors</b>	<b>Acarbose (Precose)</b>
	<b>second generation</b>			<b>Miglitol (Glycet)</b>
	<b>Glyburide (micronase)</b>			
	<b>Glipizide (Glucotrol)</b>		<b>Dipeptidyl-peptidase 4 inhibitors</b>	<b>Sitagliptin (Januvia)</b>
	<b>Glimepiride (Amaryl)</b>			
<b>meglitinides</b>	<b>Repaglinide (Prandin)</b>		<b>Incretin-like</b>	<b>Pramlintide</b>
	<b>Nateglimide (Starlix)</b>			<b>Exenatide (Byetta)</b>



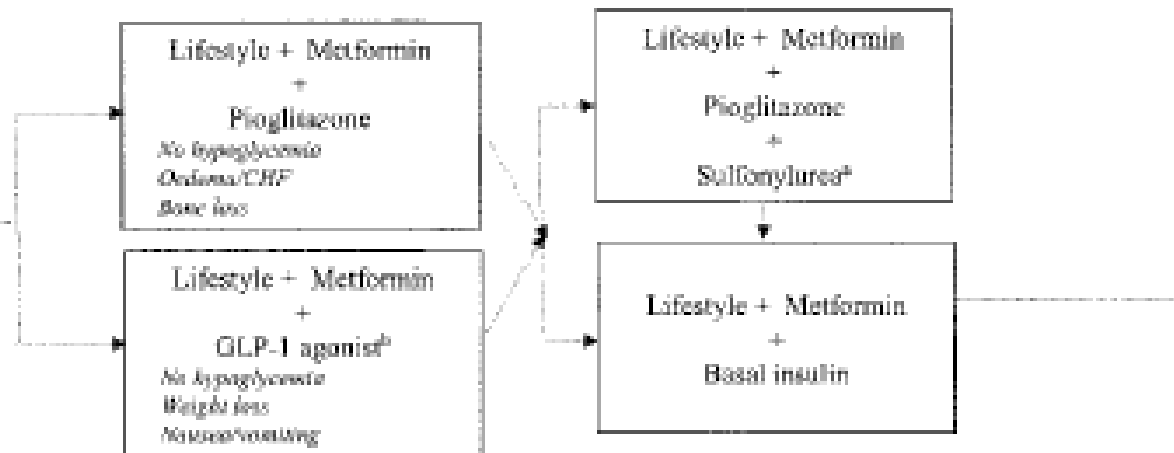
<b>Drug Therapy</b>	<b>Hemoglobin A1c</b>
	<b>Reduction, %</b>
<b>Sulfonylureas</b>	<b>0.9 to 2.5 (1.5)</b>
<b>Biguanide (metformin)</b>	<b>1.1 to 3.0 (1.5)</b>
<b>Thiazolidinediones</b>	<b>1.5 to 1.6</b>
<b><math>\alpha</math>-Glucosidase inhibitors</b>	<b>0.6 to 1.3</b>
<b>Dipeptidyl-peptidase 4 inhibitors</b>	<b>0.8</b>
<b>Noninsulin Injectables</b>	
<b>Pramlintide</b>	<b>0.43 to 0.56</b>
<b>Exenatide</b>	<b>0.8 to 0.9</b>



## Tier 1: Well-validated core therapies



## Tier 2: Less well-validated therapies



# INSULIN RX

- FBG >250 mg% or persistent random BG > 300 mg or HgbA1c > 10%
- Start Basal insulin RX:
  - Long
  - Intermediate
  - 0.2 mg/Kg/day
  - Increase by 2 units every 3 days



# STARTING BASAL/BOLUS THERAPY

- Total daily insulin Dose (TDD)=  $0.5 \text{ U} \times \text{wt in Kg}$
- Preprandial (20%)  $0.1 \text{ U/kg}$  before each meal
- Basal dose (40%)  $0.2\text{U/Kg}$  at bedtime



# CORRECTION BOLUS (SUPPLEMENT)

- Must determine how much glucose is lowered by 1 unit of short- or rapid-acting insulin
- This number is the Correction factor (CF)
- Use the 1700 rule to estimate the CF
- $CF + 1700/TDD$
- Atlanta Diabetes Associates @[www.adaendo.com](http://www.adaendo.com)



## EXAMPLE

- For a 70 Kg diabetic.  $TDD = 0.5U/Kg = 0.5 \times 70 = 35$  units.
- Then  $CF = 1700/35 = 48.6$  around 50.
- Meaning 1 unit will lower the BG by around 50 mg/dL



## CASE 1

- A 35 y.o. Native American man came to your office for a physical. He had the complaints of polyuria, polydipsia. Not taking medication.
- BP 125/80; P 85, Temp 97.8
- On Physical exam you found:





Dr. Dhanraj  
Srinivas

# CASE 1

- Na 133; K 3.9; CL 101; CO2 22; BUN 35; Cr 1.2; FBG 240; Ph 7.42
- What is your diagnosis?
- Why Hyponatremic?
- Is it Type 1 or 2
- Do you need to do antibody testing?



# CASE 1

- **How would you initially treat this patient?**
- A. Diet and exercise alone
- B. Diet and exercise plus Metformin
- C. Diet and exercise plus an incretin mimetic
- D. Diet and exercise plus insulin



# MECHANISMS

- Drugs that stimulate endogenous insulin secretion
- Drugs that reduce insulin resistance
- Drugs that delay the absorption of carbohydrate from the gastrointestinal tract.



# METFORMIN

- Metformin works by reducing hepatic glucose output
- Monotherapy with Metformin is associated with weight loss (or no weight gain)
- No hypoglycemia



# METFORMIN SIDE EFFECTS

- Side effects include
- gastrointestinal distress such as abdominal pain, nausea, and diarrhea. up to 50% transient
- lactic acidosis unknown with Metformin in the absence of other underlying diseases, particularly renal insufficiency. (three cases per 100,000 patient-years.)



# CONTRAINDICATIONS

- Patients with elevated serum creatinine levels,  $\geq 1.4$  mg per dL in women and  $\geq 1.5$  mg per dL in men.
- Hold Metformin on the day of IV contrast, hold for 48 hours or until the renal function has returned to baseline.
- Cardiogenic shock



# CONTRAINDICATIONS

- Septic shock
- Congestive heart failure that requires pharmacologic therapy
- Severe liver disease
- pulmonary insufficiency with hypoxemia
- severe tissue hypoperfusion
- Alcohol abuse



# METFORMIN FOLLOW UP

## ○ Monitoring

- Serum creatinine at initiation
- Fasting plasma glucose at 2 weeks
- HbA1c at 3 months



# SULFONYLUREAS

- Stimulate insulin release from beta cells
- Start with the lowest effective dose and titrate up at 1-2 week intervals
- Most of the effect at one half of the maximum dose
- Failure
  - Primary in 20-25% of patients
  - Secondary in 5-10 % per year.



# SULFONYLUREAS: SIDE EFFECTS

- Hypoglycemia is the most worrisome:
  - Glipizide (Glucotrol) and Glimepiride (Amaryl) are associated with a lower incidence.
- All sulfonylureas have been associated with weight gain.



# SULFONYLUREAS

- Fasting plasma glucose at 2 weeks
- HgbA1c at 3 months



# THIAZOLIDINEDIONES

- Rosiglitazone: Avandia®
- Pioglitazone: Actos®



# THIAZOLIDINEDIONES

- Enhance insulin sensitivity in muscle and adipose tissue (some inhibition of hepatic glucose production).
- No effect on insulin secretion
- Activate peroxisome proliferator-activated receptor gamma (PPAR $\gamma$ ), a nuclear receptor that regulates the production of proteins involved in glucose and lipid homeostasis



# THIAZOLIDINEDIONES

- Thiazolidinediones as monotherapy are not associated with Hypoglycemia.
- Significant weight gain has been reported.
- Safe in patients with impaired renal function.
- Not to be used when transaminase  $> 2.5 \times$  NI.
- Mild to moderate edema in 5 to 7% of patients.
- Avoid in heart failure, NYHA class III or IV.



# THIAZOLIDINEDIONES

- Decrease in glucose may not be apparent for 4 weeks
- Maximum efficacy of dose may not be observed for 4-6 months
  -



# THIAZOLIDINEDIONES

- The FDA recommends to monitor transaminases every 2 month for 1st year
- Following one year the incidence of transaminase elevations is similar to placebo.



# ALPHA-GLUCOSIDASE INHIBITORS

- Alpha-glucosidase inhibit the enzyme alpha-glucosidase found in the brush border, which cleaves more complex carbohydrates into sugars.
- The largest impact of these drugs is on postprandial hyperglycemia.



## ALPHA-GLUCOSIDASE INHIBITORS

- Abdominal discomfort, bloating, flatulence and diarrhea
- Three to four weeks should be allowed before increasing the dosage of these agents.



# INCRETIN HORMONES

- Incretin hormones, including glucagon-like peptide-1 (GLP-1) and glucose-dependent insulintropic polypeptide (GIP), are released by the intestine throughout the day.
- Levels are increased in response to a meal.
- These hormones are rapidly inactivated by the enzyme, Dipeptidyl-peptidase 4 (DPP-4).



# INCRETIN HORMONES

- GLP-1 and GIP increase insulin synthesis and release from pancreatic beta cells by intracellular signaling pathways involving cyclic AMP.
- GLP-1 also lowers glucagon secretion from pancreatic alpha cells, leading to reduced hepatic glucose production.
- GLP-1 has multiple effects on the stomach, liver, pancreas, and brain that work in concert to regulate blood glucose



## EXENATIDE (BYETTA®)

- The amino acid sequence of exenatide partially overlaps that of human GLP-1.
- Exenatide has been shown to bind and activate the known human GLP-1 receptor in vitro.



# ADMINISTRATION

## EXENATIDE (BYETTA®)

- start at 5 mcg Bid, increase to 10 mcg twice daily after 1 month
- SQ within the 60-minute before the morning and evening meals (6 hours or more apart), nevr with a meal
- The exenatide should be stored in the refrigerator when not in use
- Side effects: Nausea
- No hypoglycemis
- Weight loss



# SITAGLIPTIN (JANUVIA<sup>®</sup>)

- Sitagliptin is a DPP-4 inhibitor
- Side effects: Nausea and cold-like symptoms
- No hypoglycemia
- Weight neutral



# SITALGLIPTIN (JANUVIA<sup>®</sup>)

- Fasting plasma glucose at 2 weeks
- HbA1c at 3 months
  -



# CASE 1

## ○ Patient Assessment, Week 10

- A1c: 7.2%
- Weight: lost 2 more pounds
- Patient has stabilized at a blood glucose level of 160 mg/dL.



# CASE 1

- **At this point, would you:**
- A. Continue the therapy, it has made a difference
- B. Add a second oral agent
- C. Add exenatide
- D. Add insulin



## CASE 1

- The patient is started on 5 micrograms (mcg) bid of exenatide using a pen and a 5mm pen needle.
- Dose was increased to 10 mcg Bid after 1 month
- Possible nausea side effects of exenatide are discussed.



# CASE 1

- **Patient Assessment, Week 18**
  - **A1c is down to 6.6%**
  - **Weight: lost 9 more pounds**
  - **Experienced some nausea on exenatide but was able to continue therapy.**
  - **Latest mean blood glucose is down to 125 mg/dL**
  - **Rate of weight loss and drop in A1c are gratifying, and her A1c is expected to continue declining.**
- **Patient is praised for her success. Her goals for the next several months are discussed, and follow-up visits are scheduled at three-month intervals.**



- Statin therapy should be added to lifestyle therapy, regardless of baseline lipid levels, for diabetic patients:
  - with overt CVD (A)
  - without CVD who are over the age of 40 and have one or more other CVD risk factors. (A)
- For lower-risk patients than the above(e.g., without overt CVD and under the age of 40), statin therapy should be considered in addition to lifestyle therapy if LDL cholesterol remains above 100 mg/dl or in those with multiple CVD risk factors. (E)




	<b>Goal LDL-C</b>	<b>TG</b>	<b>HDL men</b>	<b>HDL women</b>	<b>Recommendations</b>
<b>Diabetes without overt CVD</b>	<100 mg/dL	< 150 mg/dL	>40 mg/dL	>50 mg/dL	
<b>Diabetes + CVD</b>	< 70 mg/dL	< 150 mg/dL	>40 mg/dL	>50 mg/dL	initiation of statin therapy regardless of baseline LDL- C levels, attempt to reach LDL-C < 70 mg/dL).

# HYPERTENSION

- Goals systolic blood pressure <130 mmHg. (C) (ADA) Diastolic blood pressure <80 mmHg. (B) (ADA)
- Should be treated with a regimen that includes either an ACE inhibitor or an ARB.



Test	How	Start	Frequency	Comments
urinary albumin	spot urine albumin/creatinine ratio (preferred)	At diagnosis	yearly	If Micro- or macroalbuminuria, use either ACEIs or ARBs
Serum Creatinine	Blood test		yearly	
Eye exam	ophthalmologist or optometrist	At diagnosis	yearly	
screen for neuropathy		At diagnosis	yearly	
Comprehensive foot examination	Inspection, foot pulses, and monofilament testing plus (testing vibration, pin-prick, or ankle reflexes)		Yearly	
Pneumovax		At diagnosis	Once	<p>≥ 2 years of age.  Revaccination ≥ 64 years of age (&lt;5 years ago). Repeat vaccination nephrotic syndrome, chronic renal disease, and other immunocompromised states, such as after transplantation. (C)</p> 
Influenza Vaccine		At diagnosis	Yearly	≥ 6 months of age

**Definitions of abnormalities in albumin excretion Category Spot collection (mcg/mg creatinine)**

<b>Normal</b>	<b>&lt;30</b>
<b>Microalbuminuria</b>	<b>30–299</b>
<b>Macro (clinical)-albuminuria</b>	<b>≥300</b>



# ANTIPLATELETS

- Aspirin therapy (75–162 mg/ day) as a primary prevention strategy in
  - Men 50 years of age
  - Women 60 years of age
  - Plus who have at least one additional major risk factor (family history of CVD hypertension, smoking, dyslipidemia, or albuminuria). (C)



## ANTIPLATELETS

- Use aspirin therapy (75–162 mg/day) as a secondary prevention strategy in those with diabetes with a history of CVD. (A)
- Aspirin allergy, clopidogrel (75 mg/day) should be used. (B)
- ASA (75–162 mg/day) and clopidogrel (75 mg/day) is reasonable for up to a year after an acute coronary syndrome (B)



# CASE 1

- BP 138/87; P 85, Temp 97.8
- **Lipid Profile** Total: 215 mg/dL LDL: 135 mg/dL  
HDL: 53 mg/dL Triglycerides: 130
- **Kidney Profile** Creatinine: 0.9 mg/dL  
Microalbuminuria: none



# CASE 1

- **Liver Function** ALT: normal AST: normal
- **Cardiovascular** within normal limits
- **Eye Exam** Normal
- **Foot Exam** Normal pulses and sensation



# CASE 1

- How about his Blood Pressure?
- Repeat a level in another setting.
- Repeat 138/87
- How would you treat?
- May try life style.
- Blood pressure remained 138/78
- What is the preferred medicine?



## CASE 1

- Was started on Lisinopril 10 mg Po Qd.
- In 2 weeks his blood pressure was 120/70 mm Hg, but he developed cough.
- What is the next step
- You started him on ARB.



# CASE 1

- What do you need to do now?
- Statin therapy.
- What is your goal?
- LDL<100



# CASE 1

- Does he need ASA
- How much?



- You did funduscopy on that patient which was negative.
- Do you still need Oph consult?
- If yes, when?



# CASE 1

- What vaccinations would you recommend?
- Influenza
- Pneumococcal
- Tdap



# INPATIENT GOALS FOR BLOOD GLUCOSE LEVELS

- Critically ill patients: Insulin therapy should be initiated for treatment of persistent hyperglycemia starting at a threshold of 180 mg/dl (10 mmol/l). Once insulin therapy is started, a glucose range of 140 –180 mg/dl is recommended



# INPATIENT GOALS FOR BLOOD GLUCOSE LEVELS

- Non–critically ill patients: There is no clear evidence for specific blood glucose goals. If treated with insulin, the premeal blood glucose target should generally be 140 mg/dl (7.8 mmol/l) with random blood glucose 180 mg/dl (10.0 mmol/l), provided these targets can be safely achieved.



# INPATIENT GOALS FOR BLOOD GLUCOSE LEVELS

- More stringent targets may be appropriate in stable patients with previous tight glycemic control. Less stringent targets may be appropriate in those with severe comorbidities.  
(E)



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