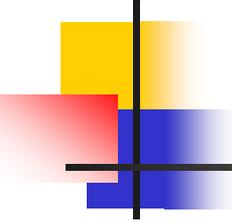


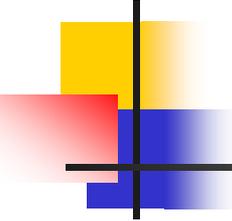
Pancreas Disorders

Williams & Hopper
Chapter 40



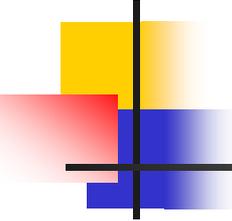
Diabetes Mellitus (DM)

- Group of metabolic diseases in which a defect in either insulin secretion or action results in hyperglycemia.



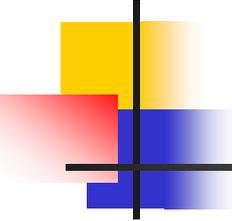
Diabetes Mellitus (DM)

- Chronic disease
 - Carbohydrate metabolism
 - Fat & protein metabolism (lesser degree)
- Inadequate production/utilization of insulin
 - Hyperglycemia
 - Glycosuria
- ~ 20.8 million people w/ diabetes
 - Includes undiagnosed disease



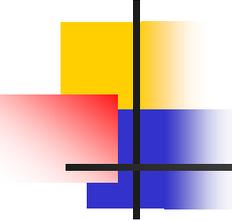
Pathophysiology

- Glucose
 - Used for energy by cells
 - Broken down from dietary carbohydrates
 - Smaller amounts from protein & fats
- Insulin
 - Produced by islets of Langerhans (pancreas)
 - Combines with receptor on cell membrane
 - Allows glucose to enter cell
 - ↓ serum glucose
 - Allows for storage of glucose in liver
 - Glycogen



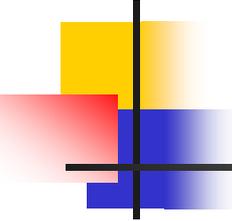
Pathophysiology

- Glucagon
 - Produced in islets of Langerhan
 - Raises blood sugar when needed
 - From storage in the liver
- Insulin & Glucagon
 - Work together
 - Maintain a normal serum glucose



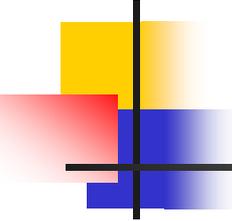
Pathophysiology

- Diabetes Mellitus
 - Faulty production of insulin (hypoinsulinism) and/or
 - Inability of body to utilize insulin
 - Results in **hyperglycemia**



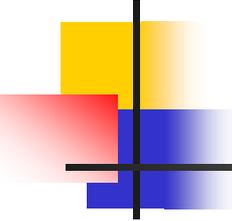
Type 1 DM

- Formerly called IDDM and “juvenile diabetes”
- Beta cells are destroyed
 - *No* insulin secreted
 - Exogenous source is needed
- Cause of destruction
 - Autoimmune component after a viral infection
 - Islet cell antibodies in blood
 - Present in ~ 90% of newly diagnosed pts
- Prone to ketoacidosis



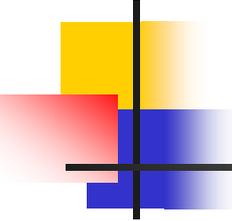
Latent autoimmune diabetes of adulthood (LADA)

- Newly identified type of Type 1 diabetes.
 - Beta cell destruction occurs slower than type 1 diabetes.
 - Sometimes misdiagnosed as type 2 diabetes.



Type 2 DM

- Formerly called non-insulin dependent diabetes mellitus (NIDDM), or adult onset diabetes.
 - Hyperglycemia results from
 - Inadequate amounts of insulin produced (usually combined with)
 - Tissue resistance



Type 2 DM

- Causes

- Heredity

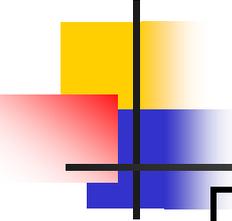
- 90 % of cases

- Obesity

- Contributes to insulin resistance in the tissues

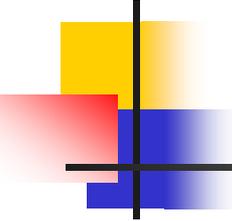
- Stress (trigger more than cause)

- ↑ risk



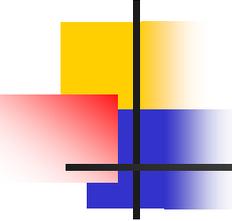
Type 1 & Type 2 comparison

	Type 1	Type 2
<i>Onset</i>	Rapid	Slow
<i>Age of onset</i>	Usually < 40	Usually > 40
<i>Risk factors</i>	Virus, autoimmune response, heredity	Heredity, obesity
<i>Body type</i>	Lean	Obese
<i>Complication due to high blood glucose</i>	Ketoacidosis	Hyperosmolar hyperglycemic nonketotic syndrome
<i>Treatment</i>	Diet, exercise; <u>must</u> have exogenous insulin for survival	Diet, exercise, may need oral hypoglycemics or insulin to control glucose



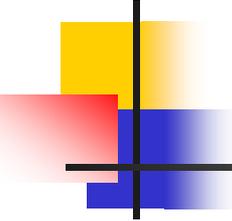
Gestational Diabetes

- Pregnancy (stressor to body)
 - ↑ metabolic demands
 - Triggers onset of DM
 - hyperglycemia
- May occur in women w/type 2 DM risk factors
- Glucose returns to normal after delivery
 - Remains ↑ risk of developing type 2 DM



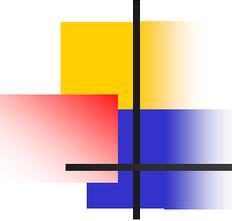
Prediabetes

- Glucose is above normal
- Not high enough to meet diagnostic criteria for DM
- Recently recognized & treated
 - Impaired fasting glucose if oral GTT 140-199 after 2 hours
 - Impaired fasting glucose if FBS is 110-125.



Secondary Diabetes

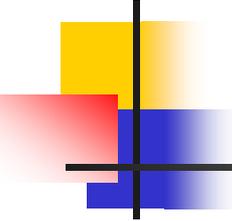
- Development of DM due to another cause
 - Illnesses
 - Pancreatitis
 - Cystic fibrosis
 - Drugs
 - Steroids
 - Thiazide diuretics
 - phenytoin (Dilantin)



Metabolic Syndrome

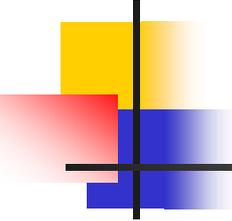
(Syndrome X, Cardiometabolic Syndrome)

- Group of disorders: abdominal obesity, ↑ triglycerides, ↓ HDL, HTN (>130/85)
- Greatly increases the risk of Type 2 diabetes.



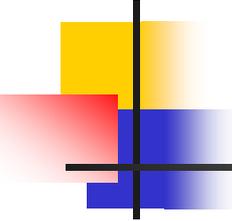
Signs & Symptoms

- Three “P’s” (classic in Type 1)
 - Polydipsia – excessive thirst
 - Polyuria (also nocturia – night time urination) – excessive urination
 - Polyphagia (not as prominent) – excessive hunger (weight loss)
- Glycosuria – glucose in urine
- Fatigue
- Blurred vision
- Abdominal pain
- Headaches
- ↑ ketones in blood & urine (in Type 1 DM)



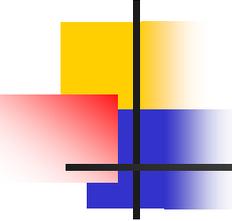
Signs & Symptoms

- (Type 2)
 - May be none at presentation
 - Usually discovered on routine exam, screening or exam for other complaint
 - May be reason many pts undiagnosed
 - Often insidious (slow to develop)
 - Complication from undiagnosed DM may bring the pt for exam & diagnosis
 - Visual trouble
 - Pain in feet or foot ulcer
 - Impotence
 - Cardiovascular disorder



Recommended screening

1. All individuals 45 yrs of age or older
 - If normal – then q 3 years
2. Younger age (arbitrarily > 25) in high risk individuals to be done annually:
 1. Obese
 2. 1st degree relative w/DM of any type
 3. High-risk ethnic group (African American, Hispanic/Latino American, Native American, Asian American, Pacific Islander)
 4. Delivered a baby > 9lbs or dx w/gestational DM
 5. Hypertensive ($\geq 140/90$ mmHg)
 6. HDL ≤ 35 mg/dl and/or triglycerides ≥ 250 mg/dl
 7. Prediabetes
 8. (Have PCOS)
 9. (Have hx of vascular disease)



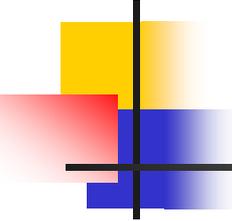
Diagnostic Tests

Diagnosis of diabetes if:

- Fasting Blood glucose
 - ≥ 126 mg/dl (on two separate occasions)

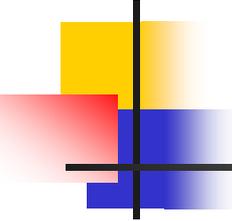
*100 – 125 is considered to be IFG.

- Random (casual) FBS
 - ≥ 200 with symptoms



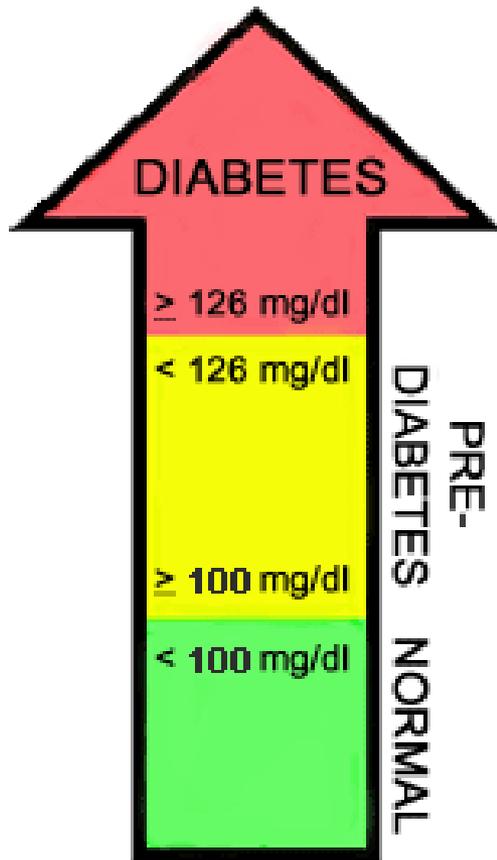
Diagnostic Tests

- Oral glucose tolerance test (OGTT)
 - Client drinks a concentrated carbohydrate drink
 - Measures glucose @ specific intervals
- Diagnosis is made if:
 - ≥ 200 for 2 hr postload
 - *140-199 diagnosed as IGT

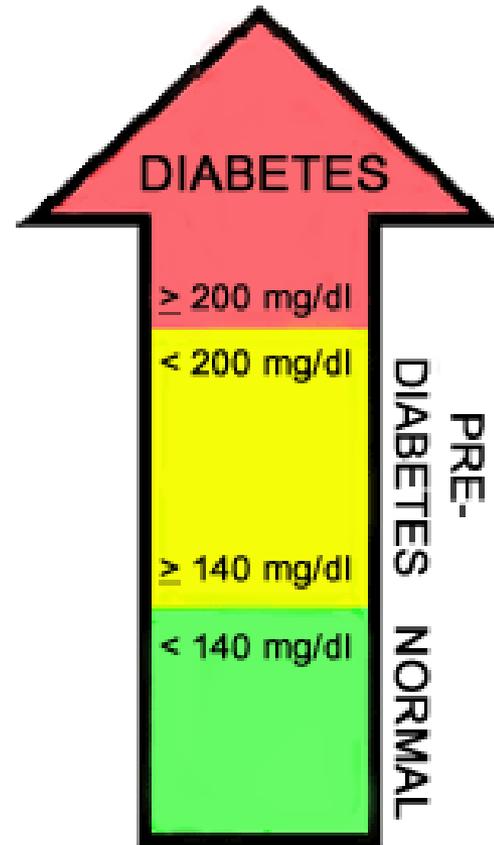


Diagnostic Tests

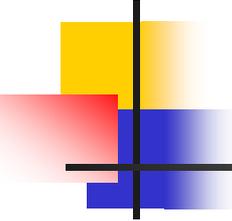
- Glycohemoglobin test (HbA1c)
 - Not primary test for diagnosis
 - Useful for monitoring glycemic control
 - Reflects average blood glucose for past 2-3 months
 - Normal is 4 –7 %



Fasting
Blood Sugar

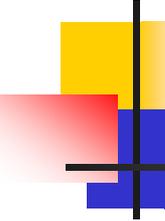


Oral Glucose
Tolerance Test



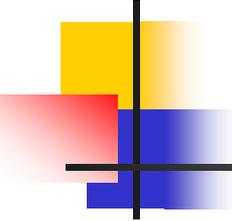
Treatment

- Only cure
 - Pancreas (or islet cell) transplant
- Goal of treatment
 - Glucose control
 - Diet
 - Exercise
 - Oral medications or insulin
 - Blood glucose monitoring
 - Education



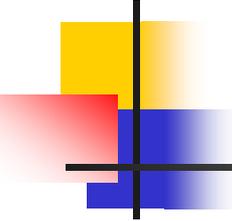
Diet (Medical Nutrition Therapy)

- Consistent consumption of carbs, proteins & fats daily
- Do not exceed the insulin's ability to utilize.
- All diets limit fat intake
- Encourage complex carbohydrates
 - Grains, pastas, vegetables, fruits
 - Limit simple sugars (high GI)
- Should fit pt's lifestyle
- ADA diet – referral to a dietician/diabetes educator
 - Meal plan healthy for anyone
 - Only limits portion size
 - Can continue to eat socially while controlling glucose



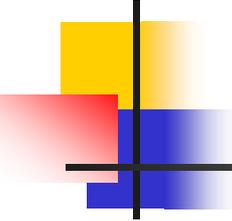
Diet

- MyPyramid
- ADA exchange list
- Carbohydrate counting
- Glycemic Index (GI)



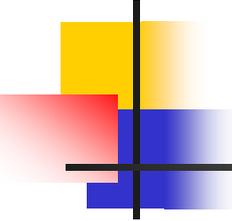
Exercise

- Helps controls blood glucose & lipids
 - ↓ glucose immediately & continuously 24'
 - Improves blood lipid levels & circulation
- Increases insulin sensitivity over time
 - Insulin not needed to allow glucose to enter exercising cells
- Facilitates weight loss



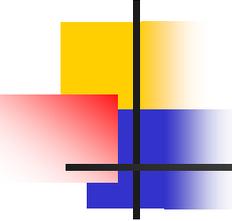
Exercise

- Pts should always carry quick sugar source
- Avoid exercising when glucose usually low
 - Have a protein or carbohydrate snack before exercising (especially if bld glu <100)
- Avoid insulin injection within 1 hr of exercise
- Avoid exercise when glucose > 250 mg/dl + ketones, or if >300 without ketones
 - Insufficient insulin available
 - Glycogen may be released further increasing bld glu.



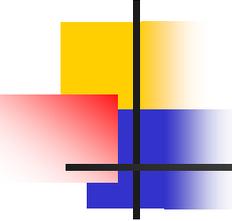
Medications

- Insulin
 - Type 1 DM
 - Produce no endogenous insulin
 - Type 2 DM
 - If diet and/or oral hypoglycemics fail
 - Cannot be given by mouth
 - Insulin is a protein
 - Digested by gastric juices
 - Broken down & inactivated
 - Given subcutaneously
 - IM or IV in emergencies



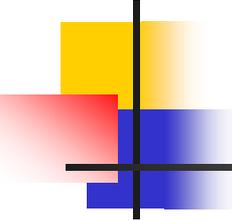
Medications

- Insulin (cont.)
 - Rotate sites
 - Avoids tissue injury
 - Onset of action
 - Time elapsed before glucose starts to decrease
 - Peak
 - Blood glucose at lowest point
 - At risk for hypoglycemia
 - Duration
 - Length of time it takes for insulin effects to cease



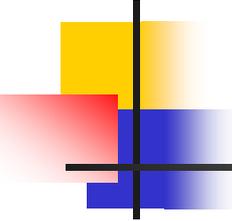
Medications

- Rapid acting insulins (Very short-acting)
 - Humalog (lispro), NovoLog (aspart), Apidra (glulisine)
 - Onset ~15"
 - Peak 30 – 90"
 - Durations ~5 hrs



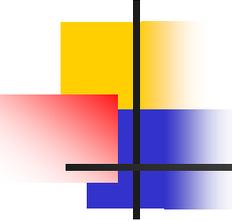
Medications

- Short acting insulins
 - *Regular* - Humulin R; Novolin R
 - Onset
 - 30 – 60"
 - Peak
 - 2-5 hrs
 - Duration
 - ~8 hrs



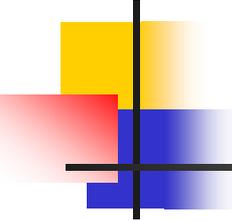
Medications

- Intermediate acting insulins
 - NPH – Novolin N (human); Humulin N (human)
 - Onset
 - 1 to 2 hrs
 - Peak
 - 6 to 12 hrs
 - Duration
 - 18 to 24 hrs



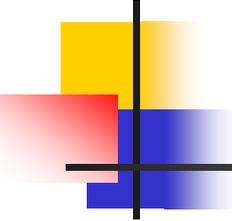
Medications

- Long acting insulins
 - Lantus (insulin glargine)
 - Onset ~ 1 hr
 - Peak – no significant peak
 - Duration – up to 24 hours
 - Levemir (insulin detemir)
 - Onset ~ 2 hrs
 - Peak – no significant peak
 - Duration – up to 24 hours



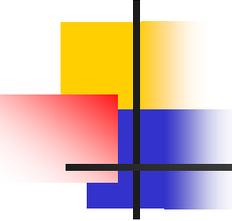
Medications

- Premixed insulins
 - **Humulin 50/50** (50% NPH, 50% Regular), **Humulin 70/30** (70% NPH, 30% Regular), **Novolin 70/30** (70% NPH, 30% Regular), **Novolog Mix 70/30** (70% insulin aspart protamine, 30% insulin aspart), **Humalog Mix 75/25** (75% insulin lispro protamine, 25% insulin lispro)



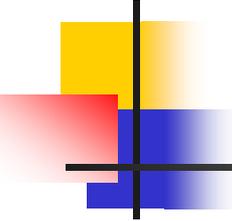
Medications

- Sliding scale
 - Used during stress or hospitalization
 - Dose determined by blood glucose
 - Usually ac & hs
 - Glucose “covered” with regular (short-acting) or rapid-acting insulin



Medications

- Problems with Insulin Use
 - Somogyi effect – Bld glu drops too low (usually at night) causing body to react by increasing glucose levels w/ hormones, then insulin is ↑'ed worsening the problem.
 - Dawn phenomenon – release of cortisol causes ↑ bld glucose in AM.



Medications

- **Insulin Pump**

- Developed ~25 years ago
- Continuous insulin infusion
- Uses Humalog or Novolog
- Basal and boluses are programmed in.
- Can be used for Type 1 or 2

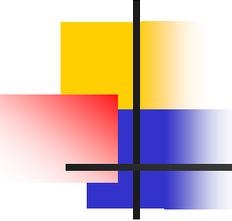
Insulin Pumps: Overview

- Insulin pump therapy, also referred to as continuous subcutaneous insulin infusion (CSII)



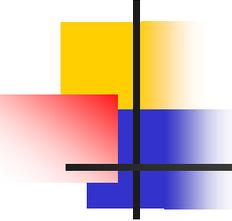
Sample Insulin Pumps





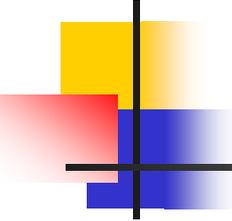
Medications

- **Insulin Pump (cont.)**
- Used for “tight” control – mimic physiologic pattern
 - Basal rate – preset determined by physiologic need
 - Bolus – to cover increases such as mealtime, or unexpected rises



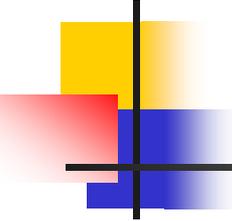
Medications

- **Insulin Pump (cont.)**
- **Candidates**
 - Must have thorough comprehension of pump
 - Ability to self-monitor glucose at least 4X/day
 - Ability to carb count



Medications

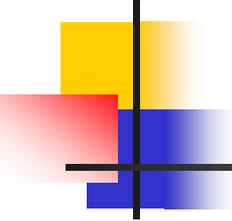
- Oral hypoglycemics
 - Not oral insulin
 - Not useful for type 1 diabetics
 - Depend on some pancreas functioning
 - Can develop tolerance
 - Switch to another type
 - Eventually may no longer work
 - Insulin may become necessary
 - Pt still has type 2 DM
 - Insulin not needed to sustain life like in Type 2 DM



Medications

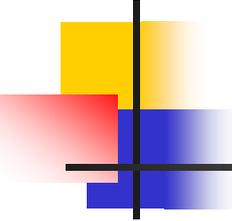
- Sulfonylureas

- Glyburide (Micronase, Diabeta), glipizide (Glucotrol XL), glimipiride (Amaryl)
- Mechanism of action
 - stimulate insulin production from beta cells
- Side effects
 - Contribute to hyperinsulinemia & weight gain
 - Insulin resistance
 - Risk of hypoglycemia
 - May cause beta cell exhaustion quicker
 - Contributing to worsening of diabetes



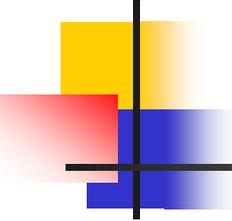
Medications

- Meglitinides - nateglinide (Starlix) & repaglinide (Prandin)
 - Mechanism of action
 - Stimulate insulin secretion from beta cells
 - Similar to sulfonylureas
 - Much more rapid onset of action
 - Starts working within 15 – 30"
 - Shorter duration
 - Starlix – lasts ~ 2hrs
 - Prandin – lasts ~ 4 hrs
 - Side effects
 - hypoglycemia



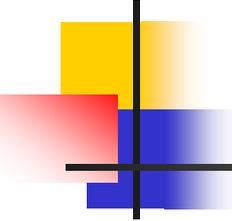
Medications

- Biguanides – Metformin (Glucophage)
 - Mechanism of action
 - Suppresses glucose production in liver
 - Enhances peripheral tissue sensitivity to insulin
 - *Does not* stimulate pancreas to secrete insulin
 - Reduces blood glucose w/o risk of hypoglycemia
 - Often suppresses appetite – weight loss
 - ↓ triglycerides & LDL, slight ↑ HDL
 - Side effects
 - Lactic acidosis – can lead to death
 - Do not use if cr > 1.5 mg/dl
 - Used in combination w/sulfonylureas



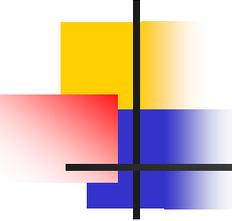
Medications

- Thiazolidinediones – rosiglitazone (Avandia) & pioglitazone (Actos)
 - Mechanism of action
 - Directly reduces insulin resistance in muscles.
 - To lesser degree: Inhibits glucose output by liver.
 - Side effects
 - Weight gain
 - Associated w/liver failure - Monitor LFTs
 - **BOTH HAVE BLACK BOX WARNINGS**



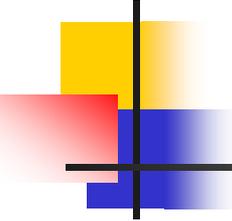
Medications

- Alpha-Glucosidase Inhibitors – Acarbose (Precose) & miglitol (Glyset)
 - Mechanism of action
 - Inhibits absorption of complex carbs from gut
 - No effect on glucose
 - Reduces post prandial hyperglycemia
 - Side effects
 - Flatulence
 - Abd pain
 - diarrhea



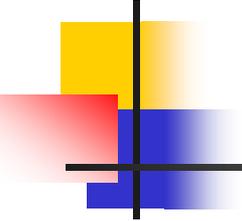
Medications

- Incretin mimetic - exenatide (Byetta)
 - Newest Type 2 diabetes drug
 - Enhances insulin secretion in response to ↑ glucose levels
 - Injectable only - twice daily dose, within 60 minutes before morning and evening meals.



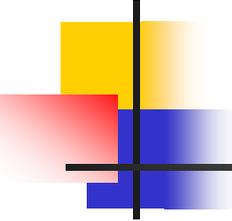
Medications [Newer Agents]

- Symlin (pramlintide acetate)
 - Analog of naturally occurring hormone called amylin.
 - Suppresses glucagon secretion, induces satiety.
 - Given by injection
 - Can be used by Type 1 or 2 patients with IDDM.



Blood Glucose Monitoring

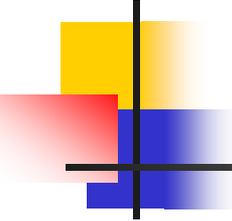
- Blood glucose monitoring (SMBG)
 - Integral part of diabetic care
 - Recommended for ALL insulin dependent (IDDM) pts
 - Use fingers whenever possible
 - *Continuous Glucose Monitoring now available



Blood Glucose Monitoring

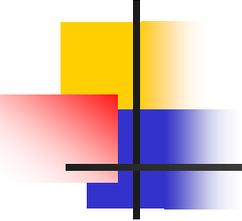
4X/Day - Generally done ac & hs

- Requires education
- Desirable glucose range (ADA recommends preprandial level of 90-130)
 - Decided by physician
 - Different for all pts
- Newer recommendations for testing 2 hours after meals (**post-prandial**) – should be <180



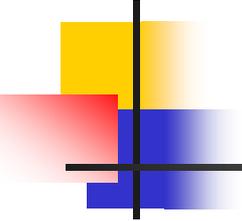
Urine Glucose/Ketone Monitoring

- Urine glucose testing – no longer recommended
 - **To check for ketones**
 - During acute illness & pregnancy
 - Glucose consistently > 300mg/dl
 - When symptoms of ketoacidosis are present
 - Notify MD if present



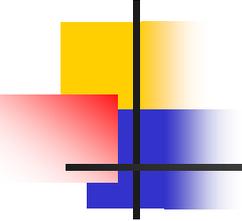
Blood Glucose/Urine Monitoring

- Result interpretation
 - Used to improve glucose control
 - Pt should keep a diary
 - Computer software is available
 - Graphs results
 - Pt education
 - Interpretation of trends



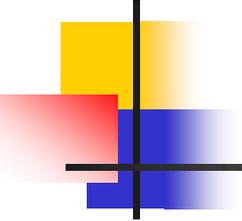
Acute Complications of Diabetes

- HYPERGLYCEMIA
- HYPOGLYCEMIA
- DIABETIC KETOACIDOSIS (DKA)
- HYPEROSMOLAR HYPERGLYCEMIC NONKETOTIC SYNDROME (HHNK)



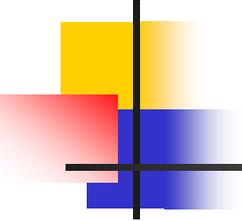
Acute Complications of Diabetes

- Hyperglycemia – high blood glucose
 - Calories eaten > available insulin
 - Stress
 - Release of epinephrine, cortisol, GH & glucagon
 - All ↑ glucose
 - Nurse responsibility
 - Teach pt to recognize s/s of hyperglycemia
 - May be similar to s/s at time of initial diagnosis



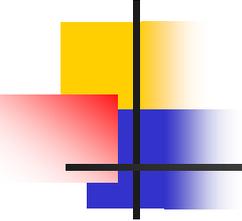
Acute Complications of Diabetes

- Hypoglycemia – low blood glucose
 - Calories < circulating insulin
 - Defined as glucose < 50 mg/dl
 - Some pts can have symptoms at higher levels
 - Sometimes symptoms can occur w/rapid glucose fall – even w/normal or high level
 - Repeated hypoglycemia
 - Can lead to permanent neurological damage
 - **Pt education**
 - Recognition of s/s of low blood glucose



Acute Complications of Diabetes

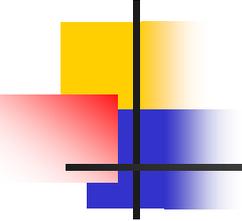
- Hypoglycemia (cont.)
 - Treatment w/ 15g glucose
 - If next meal > 1hr
 - Give protein or complex carb snack
 - Keeps sugar from dropping
 - If symptoms recur
 - Notify MD
 - IV or subq glucagon may be ordered



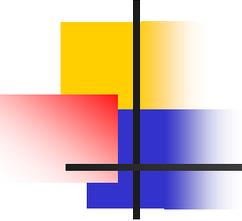
Acute Complications of Diabetes

- Diabetic Ketoacidosis (DKA)
 - Severe insulin insufficiency
 - Signs & Symptoms (in addition to s/s of hyperglycemia)
 - Severe hyperglycemia (250 – 800 mg/dl)
 - Glucose cannot get in cells
 - Cells starve
 - Fat breakdown – attempt to produce energy
 - Releases ketones (very acidic) builds up in blood
 - Kussmaul's respiration
 - Deep, sighing respiration w/fruity odor
 - Body's attempt to ↓ acidosis
 - Blowing off excess carbon dioxide

Acute Complications of Diabetes

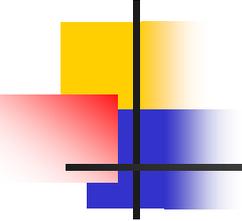


- DKA (cont.)
 - Signs & Symptoms (cont.)
 - Dehydration
 - High glucose = polyuria
 - Hyperkalemia
 - High glucose
 - Causes K⁺ to leave cell & go into blood
 - If not treated
 - Loss of consciousness
 - Death



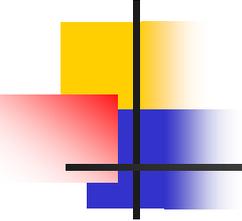
Acute Complications of Diabetes

- DKA (cont.)
 - Treatment
 - IV fluids
 - IV insulin
 - Blood glucose monitoring
 - Monitor K⁺ levels
 - Treatment will allow K⁺ to reenter the cells
 - hypokalemia



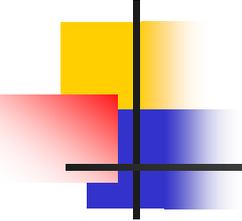
Acute Complications of Diabetes

- DKA (cont.)
 - Prevention
 - Careful blood glucose monitoring at home
 - Urine dipstick w/ glucose > 300 mg/dl
 - Detection of ketones
 - If present – **notify MD**



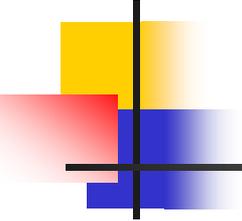
Acute Complications of Diabetes

- Hyperosmolar, Hyperglycemic, Nonketotic Syndrome (HHNKS)
 - Occurs primarily in Type 2 DM
 - Stress/illness (most common cause)
 - Causes ↑ glucose level
 - **600 – 2000** mg/dl
 - ↑ glucose causes polyuria
 - Profound dehydration
 - Blood become *Hyperosmolar* or concentrated



Acute Complications of Diabetes

- Hyperosmolar, Hyperglycemic, Nonketotic Syndrome (HHNKS)
 - Treatment
 - IV fluids
 - Insulin
 - Glucose monitoring
 - Prevention
 - Home glucose monitoring
 - Notify MD w/high sugars
 - ↑ fluids if glucose rises – esp w/stress and illness



Long Term Complications of Diabetes

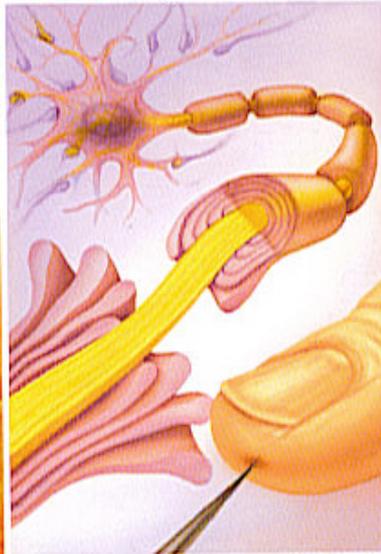
- Systems/Organs usually affected
 - Circulatory system
 - Nervous system
 - Eyes
 - Kidneys
- The Diabetes Control & Complications Trial (DCCT)
 - Study comparing intensive glucose control to conventional treatment
 - Tight control = glucose between 70 – 120
 - Revealed pts had 39 – 76 % less long term complications

Long term complications of DM:

Loss of Vision



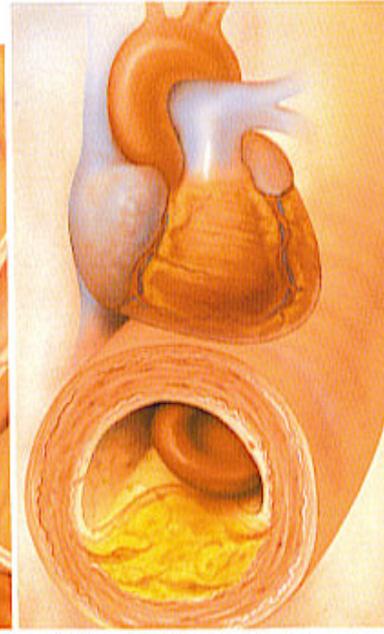
Nerve Damage



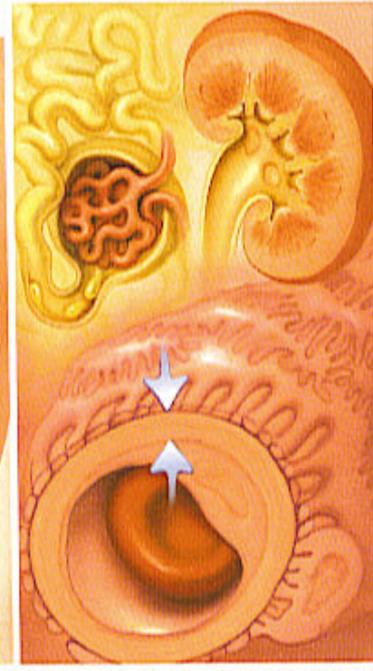
Poor Circulation

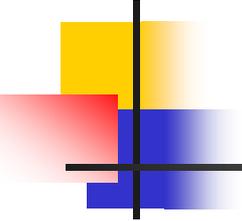


Heart Disease



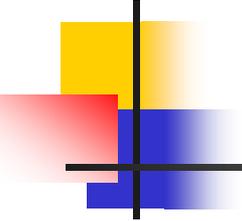
Kidney Failure





Long Term Complications of Diabetes

- Circulatory System
 - Developmental of premature atherosclerosis & arteriosclerosis
 - Higher incidence of stroke & MI
 - 2 -4 X's than general population
 - Leads to poor circulation in lower extremities
 - Slow & impaired healing of cuts or sores
 - Leading cause of leg amputation
 - Pt education re: feet care

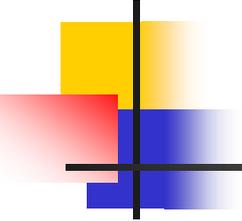


Long Term Complications of Diabetes

- Eyes
 - Retinopathy
 - Directly related to severity & duration of hyperglycemic episodes
 - Disease of small blood vessels in eyes
 - Small hemorrhages
 - Can lead to blindness
 - Causes ~ 12,000 to 24,000 new cases of blindness each year
 - Higher incidence of cataracts
 - **Annual eye exam very important!**

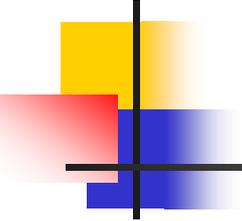
Diabetic Retinopathy





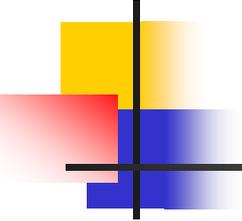
Long Term Complications of Diabetes

- Kidneys
 - Nephropathy
 - Damage to small blood vessels in kidneys
 - Kidneys unable to remove waste products
 - Occurs 20 – 30% of diabetics to some degree
 - Correlates with episodes/duration of hyperglycemic episodes & length of DM
 - Major cause of end-stage renal disease (ESRD)
 - ~ 25% of all pts with end-stage renal disease
 - Dialysis or kidney transplant may become necessary



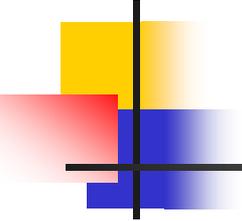
Long Term Complications of Diabetes

- Nephropathy (cont.)
 - Treatment
 - Teach importance of glucose control
 - Routine urine tests
 - Microalbuminuria – small amts protein in urine
 - Indicates kidney damage
 - Low protein diet
 - ACE inhibitor ex: ramipril (Altace), ARB valsartan (Diovan)



Long Term Complications of Diabetes

- Neuropathy
 - Damage to nerves
 - Due to chronic hyperglycemia
 - Signs & symptoms
 - Numbness & pain in extremities
 - Impotence, erectile dysfunction in males
 - **Gastroparesis** (delayed stomach emptying)
 - Nerves to GI system also affected
 - Tx: antidepressants, anticonvulsants, pregabalin (Lyrica), etc.



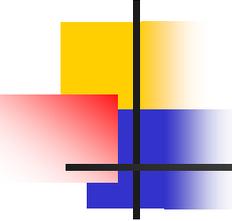
Long Term Complications of Diabetes

- More prone to infection
 - Slow healing
 - Poor circulation
 - For adequate blood supply
 - Poor systemic absorption of IV antibiotics
 - Topical antibiotics are used if localized infection
 - Sluggish immune response
 - WBC may be ineffective
 - Presence of hyperglycemia
 - Higher incidence of periodontal (gum) disease
 - Caused by bacteria in plaque
 - Teach good oral hygiene



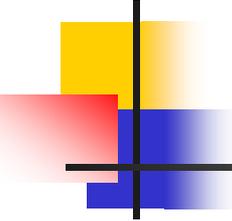
A number of foot problems can occur in patients with neuropathy resulting from long-standing diabetes. Shown here are a friction blister with accompanying cellulitis (A), a trophic ulcer of the great toe (B), and gangrene (C).





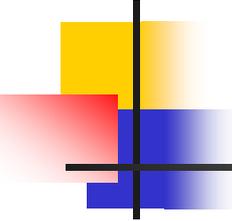
Special considerations: Diabetic Undergoing Surgery

- Surgery = Stress
 - ↑ blood glucose
 - More frequent glucose monitoring
 - Sliding scale of coverage
 - MD may order IVs w/glucose or insulin
 - Closely monitor pt for hyper/hypoglycemia



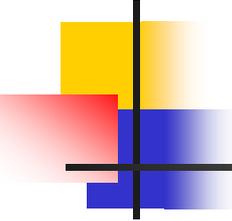
Nursing process

- Assessment
- Nursing diagnosis
- Planning & implementation
- Evaluation



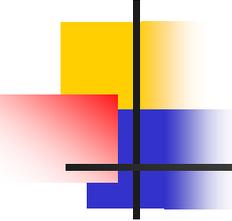
Patient Education

- Begin as soon as possible
 - Videos often helpful
- Teach self care
 - Involvement of family/significant others
- Many institutions utilize a certified diabetes educator
- Referral to outpt diabetes classes
- Elderly may need special assistance
- Medic Alert bracelet



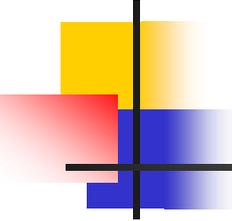
Reactive Hypoglycemia

- Abnormally low blood glucose
 - Usually < 50 mg/dl
 - Not in presence of diabetes mellitus
 - May be warning sign of impending DM
- Pathophysiology
 - Increased secretion of insulin by pancreas
 - Response to eating
 - More insulin produced than needed



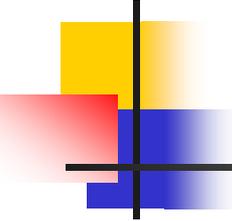
Reactive Hypoglycemia

- Signs & Symptoms
 - Shaking
 - Sweating
 - Palpitations
 - Headache
 - Chills
 - Confusion



Reactive Hypoglycemia

- Diagnosis
 - 5 hour glucose tolerance test
 - Low glucose levels at 2 & 5 hrs – makes diagnosis
 - Pts may also be taught to monitor glucose at home
 - Done am, 2hrs pc & hs
 - Taken to MD for interpretation and diagnosis



Reactive Hypoglycemia

- Treatment
 - Frequent small meals
 - Avoid fasting
 - Avoid simple sugars
 - Can cause rapid rise and fall in glucose
 - High-protein, low carbohydrate diet