

Disorder of the

Anti-Diuretic Hormone

Overview of Antidiuretic Hormone (ADH)

Structure	Function
Hypothalamus	Produces ADH
Posterior Pituitary	Stores and releases ADH

When ADH is released:

- Kidney **reabsorbs water**
- Urine becomes **concentrated**
- Blood volume **increases**
- Serum osmolality **decreases**

Diabetes Insipidus (DI)

- Diabetes Insipidus is a disorder characterized by **deficiency of ADH or inability of the kidneys to respond to ADH, resulting in excessive urination and extreme thirst.**

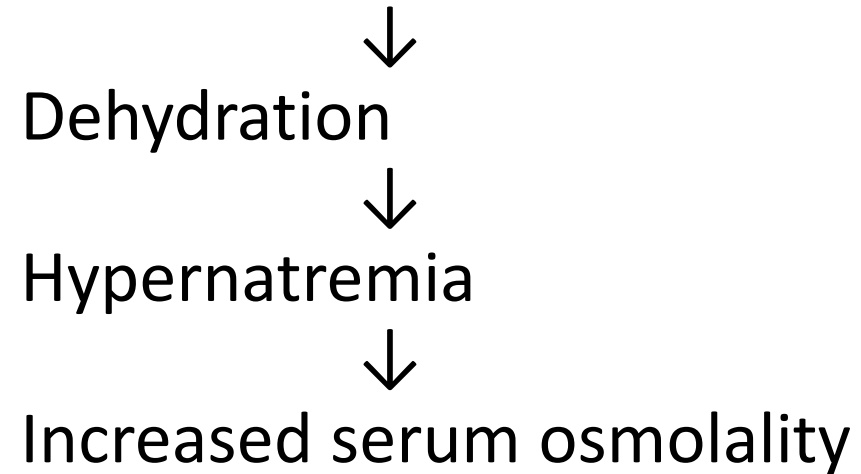
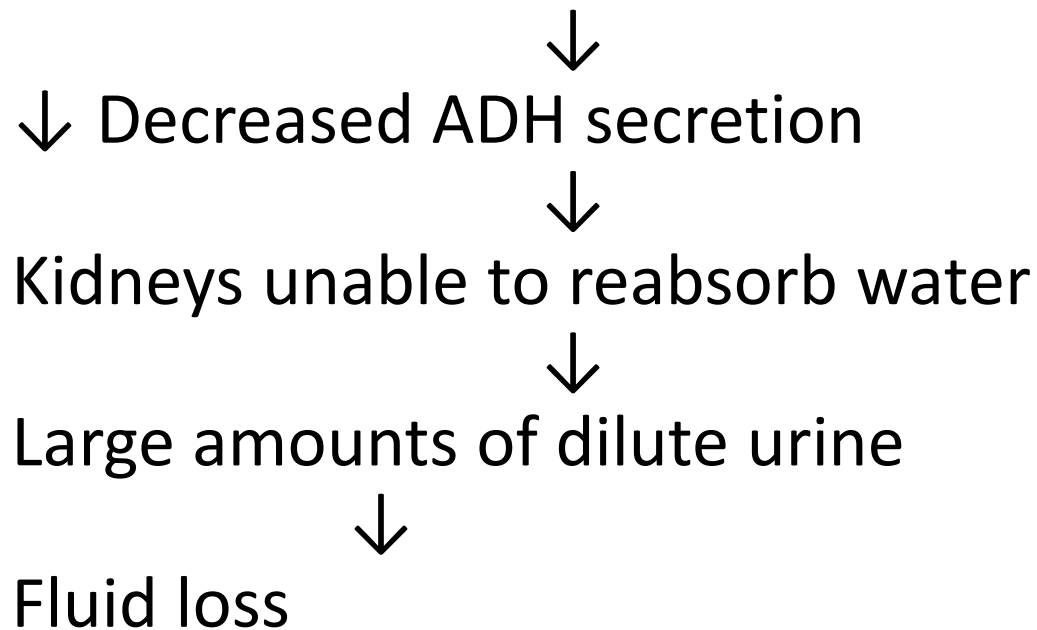
Diabetes Insipidus (DI)

Type	Cause
Central DI	Decreased ADH production from hypothalamus/pituitary
Nephrogenic DI	Kidneys do not respond to ADH
Dipsogenic DI	Excess water intake
Gestational DI	Placental enzymes destroy ADH

Diabetes Insipidus (DI)

- **Pathophysiology**

- Hypothalamus/Pituitary Damage



Diabetes Insipidus (DI)

- **Causes**
- **Central DI**
- Head trauma
- Brain surgery
- Pituitary tumors
- Meningitis
- Encephalitis
- Idiopathic
- **Nephrogenic DI**
- Chronic kidney disease
- Lithium therapy
- Hypercalcemia
- Hypokalemia

Diabetes Insipidus (DI)

Symptom	Explanation
Polyuria	Urine output up to 4–20 L/day
Polydipsia	Excessive thirst
Dilute urine	Low specific gravity
Nocturia	Frequent urination at night
Dehydration	Water loss
Hypotension	Fluid volume deficit
Tachycardia	Compensatory response
Hypernatremia	Sodium concentration increases

Diabetes Insipidus

DIABETES INSIPIDUS

VISUAL PROBLEMS

- if tumor is present

HEADACHE

- if tumor is present
(1 cause of DI)

THIRST

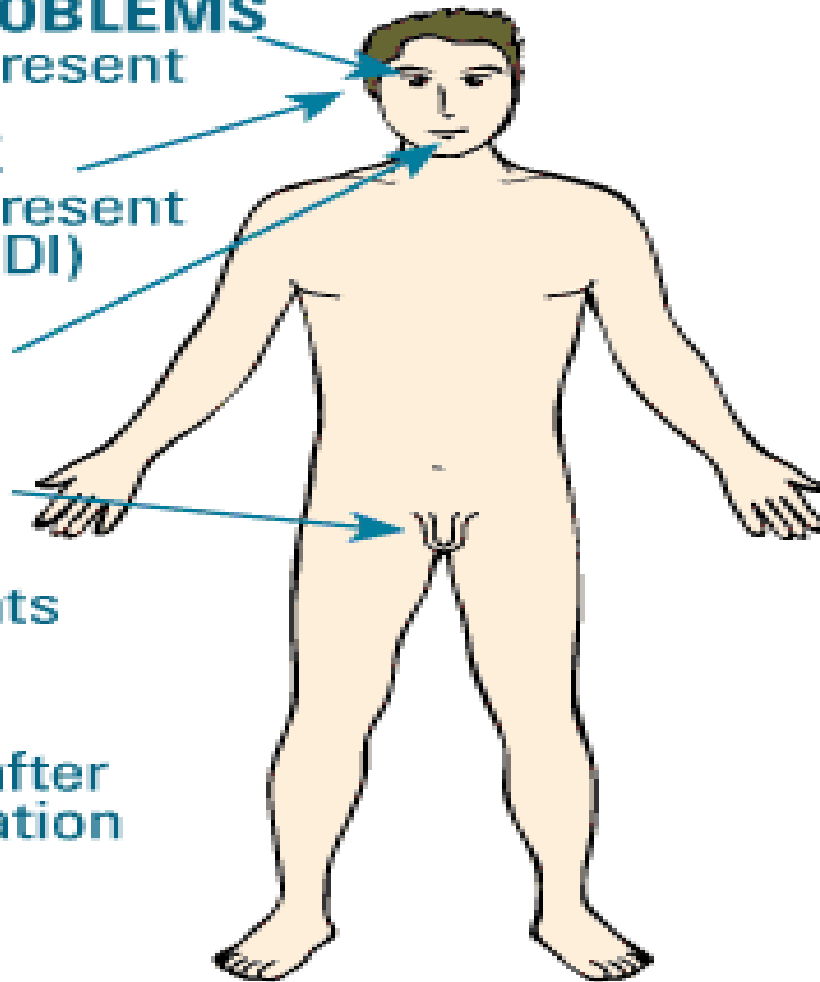
- constant

URINE

- pass very
large amounts

OTHER

- may occur after
recent operation
or car crash



Diabetes Insipidus (DI)

- Diagnostic Tests

Test	Finding
Urine Specific Gravity	<1.005
Urine Osmolality	Low
Serum Osmolality	High
Serum Sodium	High
Water Deprivation Test	Confirms DI
MRI of Brain	Identify pituitary damage

Diabetes Insipidus (DI)

- Water Deprivation Test

- **Procedure:**

1. **Withhold fluids**

2. **Monitor weight and urine concentration**

3. **Administer **Desmopressin****

Diabetes Insipidus (DI)

- **Medical Management of DI**
- **Monitor Fluid Balance**
- Strict **intake and output**
- Monitor urine volume
- **Monitor Vital Signs**
- Hypotension
- Tachycardia
- **Monitor Electrolytes**
- Sodium levels
- Serum osmolality
- **Prevent Dehydration**
- Encourage fluid intake
- IV fluids if severe
- **Medication Administration**
- Desmopressin as ordered
- **Safety Measures**
- Assess mental status
- Monitor neurological changes

Diabetes Insipidus (DI)

- Nursing Diagnosis

Diagnosis	Reason
Fluid volume deficit	Excessive urination
Risk for electrolyte imbalance	Hypernatremia
Impaired comfort	Excessive thirst
Risk for decreased cardiac output	Severe dehydration

Diabetes Insipidus (DI)

- **Patient Education**
- Take medication regularly
- Maintain adequate hydration
- Monitor urine output
- Recognize symptoms of dehydration
- Follow-up laboratory monitoring

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- SIADH is a disorder characterized by **excessive secretion of ADH, causing water retention, dilution of blood sodium, and hyponatremia.**

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- Excess ADH secretion



Kidneys reabsorb excessive water



Water retention



Dilution of sodium in blood



Hyponatremia



Decreased serum osmolality



Cellular swelling



Neurological symptoms

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- Causes of SIADH

- **Neurological Causes**

- Brain tumors
- Head trauma
- Stroke
- Meningitis

- **Pulmonary Causes**

- Pneumonia
- Tuberculosis
- Lung cancer

- **Drug-Induced**

- Antidepressants
- Carbamazepine
- Chemotherapy drugs

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

System	Symptoms
Neurologic	Headache, confusion
Severe hyponatremia	Seizures
Gastrointestinal	Nausea, vomiting
Muscular	Weakness
Fluid status	No edema

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

Test	Finding
Serum Sodium	Low (<135 mEq/L)
Serum Osmolality	Low
Urine Osmolality	High
Urine Specific Gravity	High
Urine Sodium	Elevated

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

Treatment	Purpose
Fluid restriction	First-line treatment
Hypertonic saline (3%)	Severe hyponatremia
Loop diuretics	Promote water excretion
Vasopressin receptor antagonists (Vaptans)	Block ADH effect
Demeclocycline	Reduce ADH response

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- **Nursing Management of SIADH**
- **Fluid Restriction**
- Usually **800–1000 mL/day**
- **Monitor Electrolytes**
- Sodium levels
- Serum osmolality
- **Neurological Assessment**
- Monitor mental status
- Assess for seizures
- **Monitor Intake and Output**
- **Daily Weight**
- Detect fluid retention
- **Safety Measures**
- Seizure precautions

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- Nursing Diagnosis

Diagnosis	Reason
Excess fluid volume	Water retention
Risk for decreased cerebral perfusion	Hyponatremia
Risk for injury	Seizures
Confusion	Cerebral edema

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- **Patient Education**
- Follow fluid restriction
- Monitor weight daily
- Recognize symptoms of hyponatremia
- Avoid medications that trigger SIADH

Comparison: Diabetes Insipidus vs SIADH

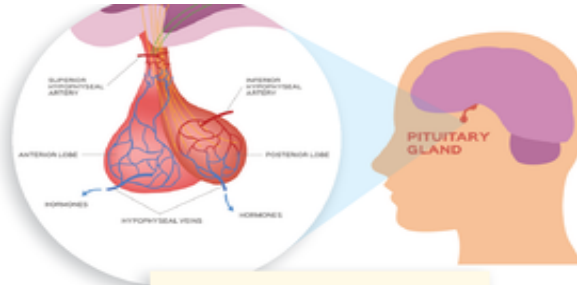
Feature	Diabetes Insipidus	SIADH
ADH Level	Low	High
Urine Output	High	Low
Urine Concentration	Dilute	Concentrated
Serum Sodium	High	Low
Serum Osmolality	High	Low
Urine Osmolality	Low	High
Hydration Status	Dehydration	Fluid overload
Treatment	ADH replacement	Fluid restriction

Memory Tip

- **DI = Dry Inside**
 - Dehydration
 - Dilute urine
 - Decreased ADH
- **SIADH = Soaked Inside**
 - Water retention
 - Hyponatremia
 - Excess ADH

Comparative Chart

SIADH VS. DI



ADH

Anti-Diuretic Hormone
Adds Da H₂O

SIADH
"Soaked Inside"

DI
"Dry Inside"

DI "Dehydrated"
"Die" ADH!
Diabetes Insipidus

SIADH "Soaked"
"Yes" ADDS DA H₂O
Syndrome of Inappropriate Antidiuretic Hormone

7 S's

- S** STOPs urination (LOW urine output)
- S** STICKY & THICK "urine" HIGH Sp. Gravity 1.030+
- S** SOAKED Inside "Low & Liquidy" Labs
HYPO osmolality (LOW)
HYPOnatremia below 135 Na+ (LOW)
- S** SODIUM Low!! (Headache Early Sigr)
- S** SEIZURES key words: Headache, Confusion
- S** SEVERE HIGH blood pressure
- S** STOP ALL FLUIDS + GIVE Salt + Diuretics
(NO IV or drinking) + (IV 3% Saline + Eat Salt)

CAUSES

- S** • Small cell lung cancer
- S** • Severe Brain Trauma (trauma/surgery)
- S** • Sepsis infections of brain (meningitis)

LABS

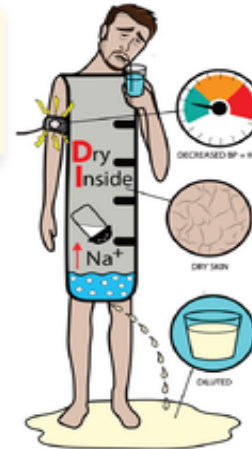
- S** OAKED Inside "Low & Liquidy" Labs
HYPO osmolality (LOW)
HYPOnatremia below 135 Na+ (LOW)
- S** TICKY thick urine" Outside • LOW urine output (STOPS urine)
HIGH specific Gravity 1.030+



- D** • Desmopressin/Vasopressin (synthetic ADH)
- D** • Decreases Urine Output "Pressin" the BP Up!
- CAUTION:** "Headaches" Priority!
Low Na+ (135 or less) > Seizures > DEATH!

NURSING CARE

- Strict I & O
- Daily Weights
(Weight Gain = Water Gain)



7 D's

- D** DIURESE "Drain" fluid (HIGH urine output)
- D** DILUTED urine Low specific Gravity (1.005)
- D** DRY Inside "High & Dry" Labs
HYPER osmolality (HIGH)
HYPERnatremia over 145 Na+ (HIGH)
- D** DRINKING a lot "thirsty"
- D** DEHYDRATED Dry Mucosa & Skin
- D** DECREASED blood pressure
- D** DESMopressin "Vasopressin" (ADH)
Decrease Urine Output
Death by Headache! (Low Na+) 135 or Less

CAUSES

- D** • Damage to brain
(Tumors, Trauma, Surgery)

LABS

- D** RY Inside "High & Dry" Labs
HYPER osmolality (HIGH)
HYPERnatremia over 145 Na+ (HIGH)
- D** I luted Outside "HIGH urine output (Drains urine)
LOW specific Gravity 1.005

- Possible Complications

Diabetes Insipidus	SIADH
Severe dehydration	Severe hyponatremia
Hypovolemic shock	Seizures
Hypernatremia	Cerebral edema
Kidney injury	Coma

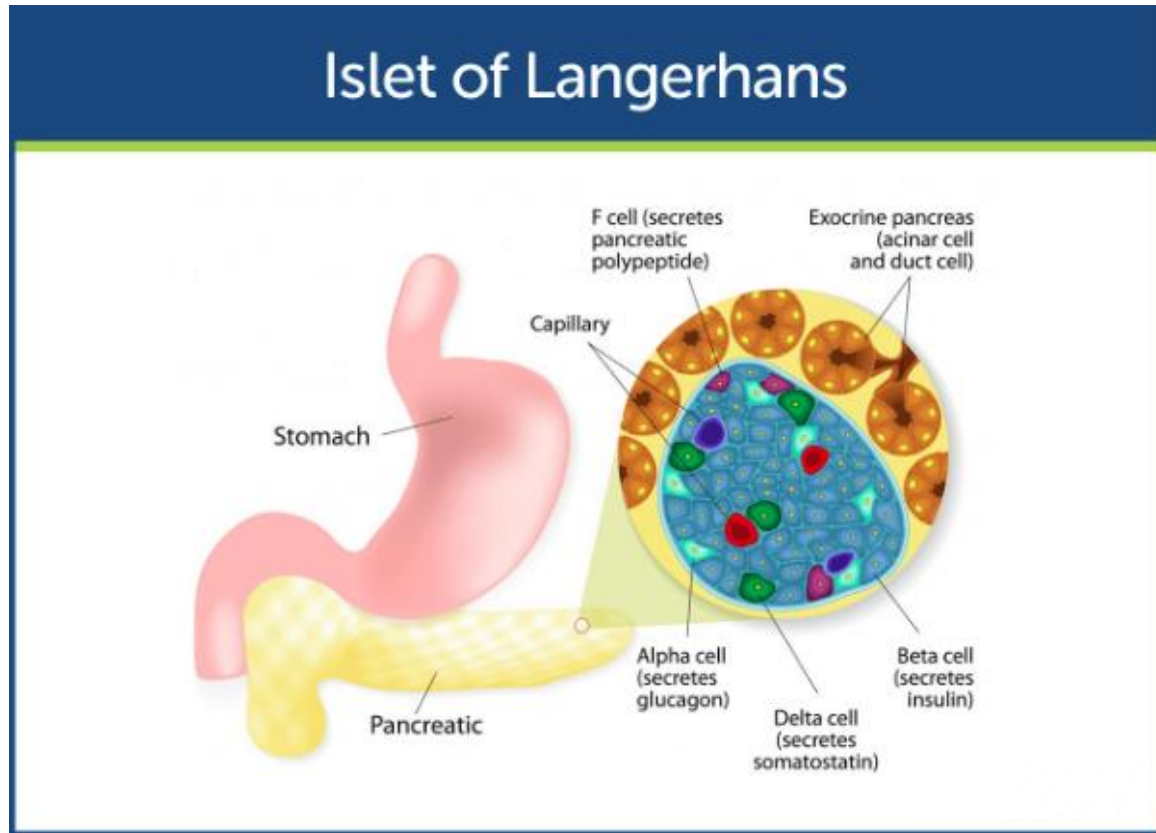
Disorder of the

PANCREAS

DIABETES MELLITUS

- **Diabetes Mellitus (DM)** is a **chronic metabolic disorder characterized by hyperglycemia (high blood glucose)** resulting from:
 - **Defects in insulin secretion**
 - **Insulin resistance**
 - **Or both**
- Insulin is a hormone produced by the **beta cells of the pancreas** that regulates blood glucose levels.

Pathophysiology



Normal Glucose Metabolism

Role of Insulin

Insulin allows glucose to **enter the cells** where it is used for **energy production**.

Process

Glucose from food



Absorbed into bloodstream



Pancreas releases insulin



Glucose enters body cells



Energy production

When insulin is absent or ineffective → **glucose accumulates in the blood** → **hyperglycemia**

Pathophysiology

- Insulin deficiency or resistance



Glucose cannot enter cells



Hyperglycemia develops



Cells lack energy



Body breaks down fat and protein



Ketone production



Metabolic disturbances

Type	Description
Type 1 DM	Autoimmune destruction of pancreatic beta cells
Type 2 DM	Insulin resistance with relative insulin deficiency
Gestational Diabetes	Diabetes occurring during pregnancy
Secondary Diabetes	Due to disease or medications

Type 1 Diabetes Mellitus

- **Type 1 Diabetes Mellitus**
- **Characteristics**
- Usually occurs in **children or young adults**
- **Absolute insulin deficiency**
- Requires **lifelong insulin therapy**
- **Causes**
- Autoimmune destruction of beta cells
- Genetic predisposition
- Environmental triggers

Type 1 Diabetes Mellitus

- **Type 1 Diabetes Mellitus**
- **Characteristics**
- Usually occurs in **children or young adults**
- **Absolute insulin deficiency**
- Requires **lifelong insulin therapy**
- **Causes**
- Autoimmune destruction of beta cells
- Genetic predisposition
- Environmental triggers

Type 2 Diabetes Mellitus

- **Type 2 Diabetes Mellitus**
- **Characteristics**
- Most common type
- Usually occurs in **adults**
- Associated with **obesity and sedentary lifestyle**
- **Pathophysiology**
- Insulin resistance
 - ↓
 - Pancreas produces more insulin
 - ↓
 - Beta cell exhaustion
 - ↓
 - Relative insulin deficiency

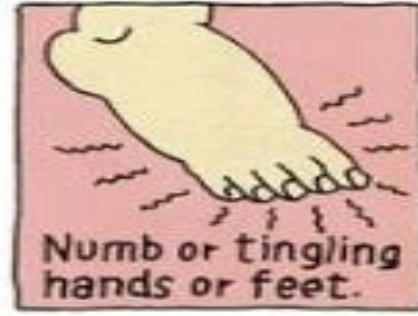
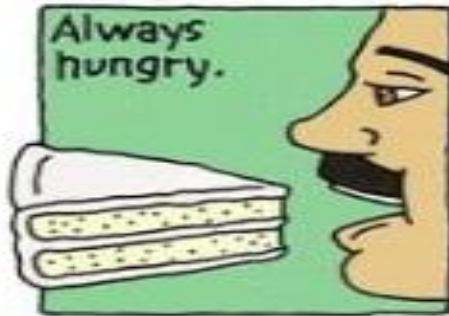
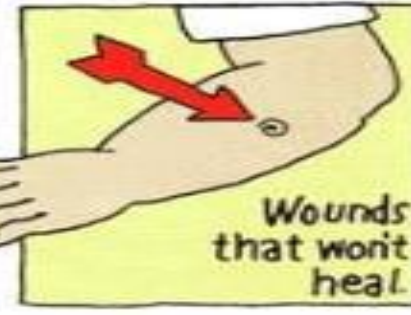
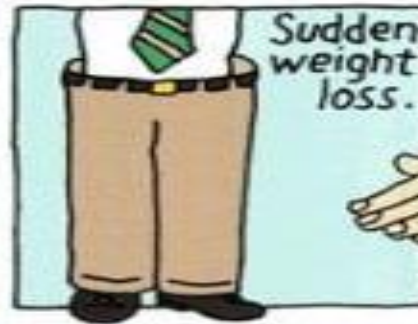
Risk Factors for Type 2 Diabetes

- Obesity
- Family history
- Sedentary lifestyle
- Age over 45
- Hypertension
- Dyslipidemia
- History of gestational diabetes

Clinical Manifestations

DIABETES

KNOW THE SYMPTOMS



Clinical Manifestations

Symptom	Explanation
Polyuria	Excessive urination
Polydipsia	Excessive thirst
Polyphagia	Increased hunger

Diagnostic Criteria

Test	Diagnostic Value
Fasting Blood Glucose	≥ 126 mg/dL
Random Blood Glucose	≥ 200 mg/dL
HbA1c	$\geq 6.5\%$
Oral Glucose Tolerance Test	≥ 200 mg/dL

Glycosylated Hemoglobin (HbA1c)

HbA1c Level	Interpretation
<5.7%	Normal
5.7–6.4%	Prediabetes
≥6.5%	Diabetes

Acute Complications of Diabetes

Diabetic Ketoacidosis (DKA)

- Insulin deficiency



Fat breakdown



Ketone production



Metabolic acidosis

Diabetic Ketoacidosis (DKA)

- Signs and Symptoms of DKA
 - Hyperglycemia
 - Ketonuria
 - Kussmaul Breathing
 - Fruity Breath Odor
 - Abdominal Pain
 - Nausea and Vomiting
 - Dehydration

Diabetic Ketoacidosis (DKA)

- Management of DKA

Treatment	Purpose
IV fluids	Correct dehydration
Insulin infusion	Reduce blood glucose
Electrolyte replacement	Correct potassium imbalance
Frequent monitoring	Prevent complications

Hyperosmolar Hyperglycemic State (HHS)

- Severe hyperglycemia
- Severe dehydration
- No significant ketosis

Hyperosmolar Hyperglycemic State (HHS)

- Symptoms of HHS
 - Extreme Hyperglycemia
 - Severe Dehydration
 - Confusion
 - Coma

DKA vs HHS Comparison Table

Feature	Diabetic Ketoacidosis (DKA)	Hyperosmolar Hyperglycemic State (HHS)
Common in	Type 1 Diabetes	Type 2 Diabetes
Cause	Severe insulin deficiency	Relative insulin deficiency
Onset	Rapid (hours–days)	Gradual (days–weeks)
Blood Glucose	250–600 mg/dL	Often >600 mg/dL
Ketones	Present	Minimal or absent
Acidosis	Present	Absent
Serum Osmolality	Mildly increased	Very high
Dehydration	Moderate	Severe
Respirations	Kussmaul breathing	Normal
Breath odor	Fruity odor	None
Mental status	Alert to drowsy	Confusion to coma
Mortality rate	Lower	Higher

Chronic Complications of Diabetes

Pathophysiology

- **Microvascular Damage**
- **Damage to small blood vessels**

Organ	Complication
Eye	Diabetic Retinopathy
Kidney	Diabetic Nephropathy
Nerves	Diabetic Neuropathy

Microvascular Complications

- **Diabetic Retinopathy**
- Damage to retinal blood vessels.
- Symptoms:
- Blurred vision
- Vision loss
- Blindness

Microvascular Complications

- **Diabetic Nephropathy**
- Damage to kidney filtration system.
- Signs:
- Proteinuria
- Edema
- Renal failure

Microvascular Complications

- **Diabetic Neuropathy**
- Damage to peripheral nerves.
- Symptoms:
- Numbness
- Tingling
- Burning sensation
- Loss of protective sensation

Pathophysiology

- **Macrovascular Damage**

- Damage to **large blood vessels**



- Accelerated **atherosclerosis**



- Complications:

- **Coronary artery disease**

- **Stroke**

- **Peripheral artery disease**

Diabetic Foot

- Due to:

- Neuropathy
- Poor circulation
- Infection

- Signs:

- Foot ulcers
- Gangrene
- Amputation risk

Diabetic Foot Care

Recommendation

Inspect feet daily

Wash and dry feet carefully

Wear comfortable shoes

Avoid walking barefoot

Trim nails carefully

DIABETES MELLITUS

HYPOGLYCEMIA



SLEEPINESS



SWEATING



PALLOR



LACK OF
COORDINATION



IRRITABILITY



HUNGER

HYPERGLYCEMIA



DRY MOUTH



INCREASED
THIRST



BLURRED
VISION



WEAKNESS



HEADACHE



FREQUENT
URINATION

Medical Management of Diabetes

- Management focuses on:
 1. Blood glucose control
 2. Preventing complications
 3. Lifestyle modification

Insulin Therapy

Type	Onset	Peak	Duration
Rapid-acting	15 min	1 hr	3–5 hrs
Short-acting	30 min	2–4 hrs	6–8 hrs
Intermediate	1–2 hrs	6–12 hrs	18 hrs
Long-acting	1–2 hrs	None	24 hrs

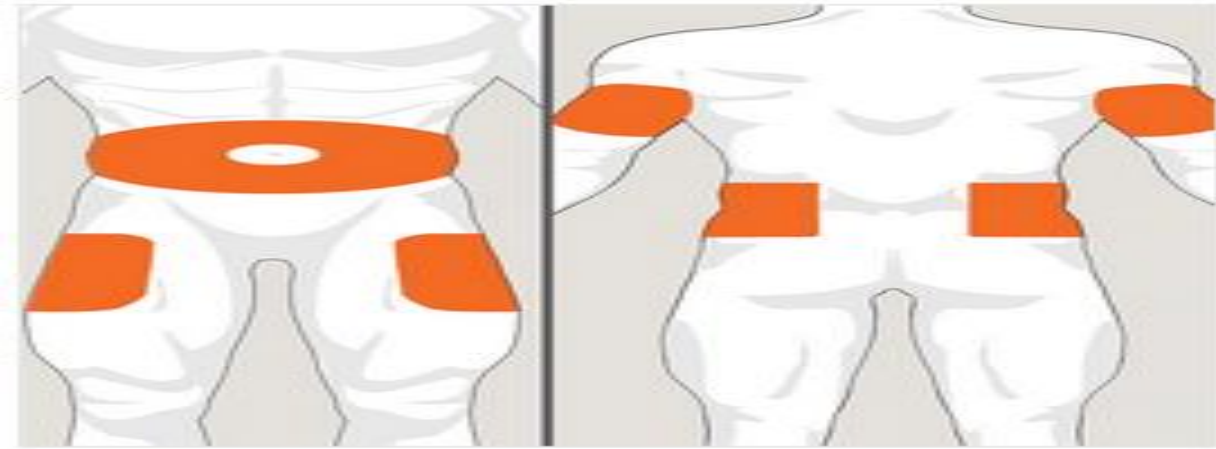
DIABETES MELLITUS - INSULIN THERAPY

Insulin Drug Chart

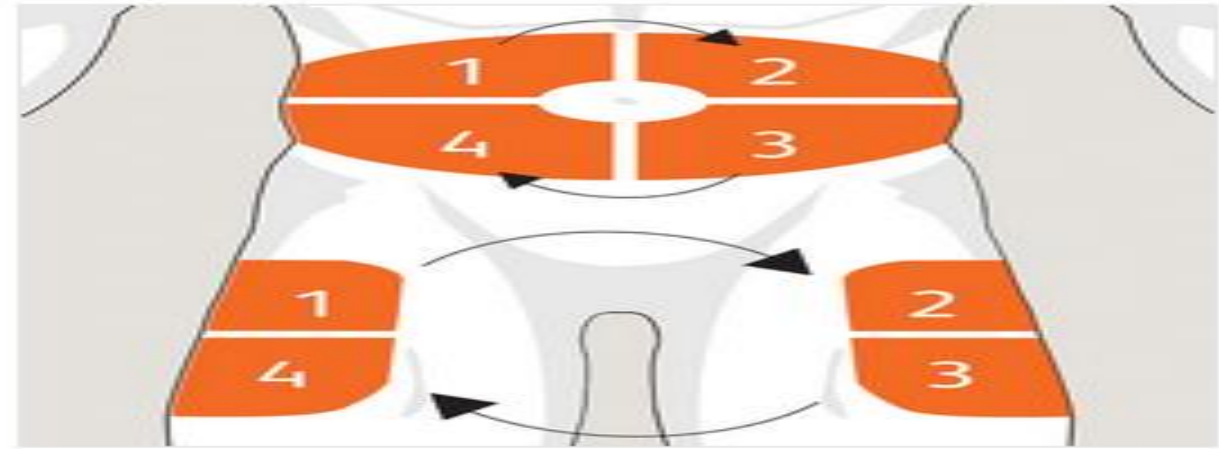
Insulin	STARTS TO WORK IN (hours)	Peak Action (hours)	Duration of Action (hours)	MAXIMUM Duration (hours)
Rapid-Acting				
Lispro (Humalog)	15 TO 30 MINUTES	1 to 2 HOURS	3 to 6 HOURS	4 to 6 HOURS
Aspart (Novolog)	15 TO 30 MINUTES	1 to 2 HOURS	3 to 6 HOURS	4 to 6 HOURS
Glulisine (Apidra)	15 TO 30 MINUTES	1 to 2 HOURS	3 to 6 HOURS	4 to 6 HOURS
Short-Acting				
Regular	30 MINUTES TO 1 HOUR	2 to 4 HOURS	3 to 6 HOURS	6 to 8 HOURS
Intermediate-Acting				
NPH	2 to 4 HOURS	8 to 10 HOURS	10 to 18 HOURS	14 to 20 HOURS
Long-Acting				
Glargine (Lantus)	1 to 2 HOURS	None	19 to 24 HOURS	24 HOURS
Detemir (Levemir)	1 to 2 HOURS	None	19 to 20 HOURS	20 HOURS

Image via pintarest.com

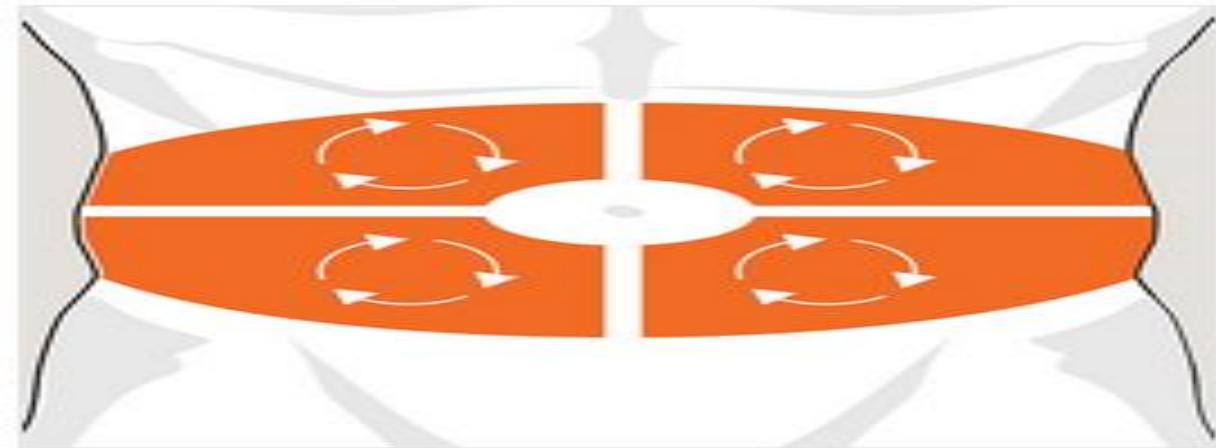
DIABETES MELLITUS - INSULIN THERAPY



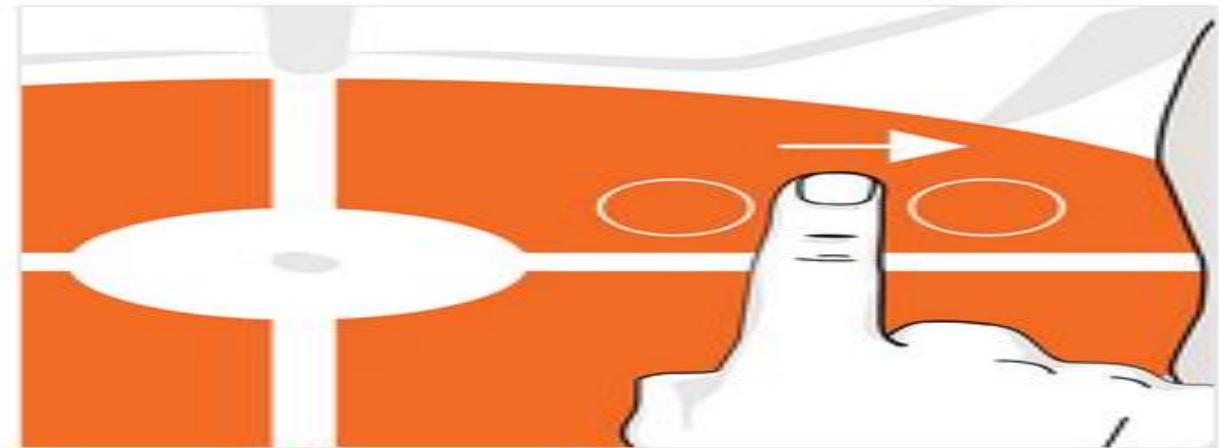
1. Choose an area.



2. Divide that area into four sections.



3. Select an injection site in a section to start injecting. Use one section per week.



4. Inject one finger width away from your last injection.

DIABETES MELLITUS - INSULIN THERAPY

• Horizontal Pattern



• Curve Pattern



• Zig Zag Pattern



• Crisscross Pattern



Insulin in the stomach

DIABETES MELLITUS - INSULIN THERAPY COMPLICATIONS

Feature	Dawn Phenomenon	Somogyi Phenomenon
Basic Cause	Early morning hormone surge	Rebound from nighttime hypoglycemia
What happens at night?	Blood sugar stays normal	Blood sugar drops (hypoglycemia)
Early morning glucose	High	High
Mechanism	Cortisol, growth hormone \uparrow \rightarrow insulin resistance	Low glucose \rightarrow body releases glucagon & epinephrine \rightarrow rebound hyperglycemia
Time	3 AM – 8 AM	Usually after midnight hypoglycemia
3 AM blood glucose	Normal or slightly high	LOW
Key Problem	Not enough insulin in early morning	Too much insulin at night

Key Diagnostic Clue

-  Check **3 AM blood glucose**

Result	Interpretation
Normal or High	Dawn Phenomenon
Low	Somogyi Phenomenon

Clinical Signs

- **Dawn Phenomenon**
- High fasting blood sugar
- No night symptoms
- **Somogyi Phenomenon**
- Night sweats
- Nightmares
- Morning headache
- Fatigue upon waking

Nursing Management

Condition	Management
Dawn Phenomenon	Increase evening insulin or adjust timing
Somogyi Phenomenon	Reduce nighttime insulin or give bedtime snack

DIABETES MELLITUS - INSULIN THERAPY COMPLICATIONS

- **LOCAL ALLERGIC REACTIONS**
- **INSULIN LIPODYSTROPHY**

DIABETES MELLITUS - ORAL HYPOGLYCEMIC MEDICATIONS (OHA)

Type 2 Diabetes Medications

*ROH = Risk of Hypoglycemia

Drug Class	Examples	Mechanism of Action	Side Effects	ROH*	Contraindications
Biguanides	Metformin	Increase insulin sensitivity; Decrease hepatic gluconeogenesis	Nausea, vomiting, diarrhea, vitamin B12 deficiency, lactic acidosis (rare)	No	CKD, heart disease, liver disease, metabolic acidosis
Thiazolidinediones	Pioglitazone	Increase insulin sensitivity; Decrease hepatic gluconeogenesis	Weight gain, fluid retention, heart failure, bladder cancer risk, fractures, increase HDL	No	Heart failure, osteoporosis, history of bladder cancer
Sulfonylureas	Glipizide	Stimulate insulin secretion by inhibiting/closing beta cell ATP-sensitive K ⁺ channels	Hypoglycemia, weight gain	Yes	CKD, hepatic impairment
SGLT-2 Inhibitors	Dapagliflozin	Decrease glucose reabsorption in the kidney; Increase glucose excretion	Weight loss, thirst, increased urination, UTI risk, AKI	No	Renal impairment
DPP-4 Inhibitors	Sitagliptin	Increase GLP-1, which increases insulin secretion	GI upset, headaches, URIs, joint pain, risk of pancreatitis	No	Pancreatitis, heart failure, angioedema, DKA
GLP-1 Mimetics	Exenatide	Increase insulin secretion; Inhibit glucagon secretion	Nausea, vomiting, diarrhea, pancreatitis, weight loss, AKI	No	Pancreatitis, CKD, medullary thyroid cancer, gastroparesis
Insulin	"Basal"	Exogenous insulin provided	Weight gain	Yes	Hypokalemic drugs, dose adjusted for renal/liver failure

Oral Antidiabetic Drugs

Drug Class	Mechanism
Biguanides (Metformin)	Decreases glucose production
Sulfonylureas	Stimulate insulin release
DPP-4 inhibitors	Increase incretin hormones
SGLT2 inhibitors	Increase glucose excretion
Thiazolidinediones	Improve insulin sensitivity

Nutritional Management

- Goals:
- Maintain normal blood glucose
- Achieve healthy body weight

Recommendation

Balanced diet

Controlled carbohydrate intake

High fiber

Limit sugar and refined carbohydrates

Reduce saturated fats

Exercise Management

- Benefits:
- Improves insulin sensitivity
- Helps weight control
- Reduces cardiovascular risk
- Recommended:
- **150 minutes of moderate exercise weekly**
- Examples:
- Walking
- Cycling
- Swimming

DIABETES MELLITUS - HYPOGLYCEMIA ASSESSMENT

Mild Hypoglycemia	Moderate Hypoglycemia	Sever Hypoglycemia
Hunger	Confusion	Difficulty Arousing
Nervousness	Double Vision	Disoriented Behavior
Palpitations	Drowsiness	Loss of Consciousness
Sweating	Emotional Changes	Seizures
Tachycardia	Headache	
Tremors	Impaired Coordination	
	Inability to Concentrate	
	Irrational or Combative Behavior	
	Lightheadedness	
	Numbness of the Lips and tongue	
	Slurred Speech	
70mg/dL (4.0mmol/L)	40mg/dL (2.2mmol/L)	20mg/dL (1.1mmol/L)

DIABETES MELLITUS - ACUTE COMPLICATION

Simple Carbohydrates to treat Hypoglycemia

- Commercially prepared glucose tablets
- Hard Candies
- 4 tsp of sugar
- 4 sugar cubes
- 1tbsp of honey or syrup
- ½ cup of fruit juice or regular soft drinks
- 8oz of low fat milk
- 6 saltine crackers
- 3 Graham Cracker

- **DO NOT ATTEMPT TO ADMINISTER ORAL FOOD OR FLUIDS TO THE CLIENT EXPERIENCING A SEVERE HYPOGLYCEMIC REACTION WHO IS SEMICONSCIOUS OR UNCONSCIOUS AND IS UNABLE TO SWALLOW**

DIABETES MELLITUS - HYPOGLYCEMIA – INTERVENTIONS (15/15 Rule)

Nursing Management of Diabetes

- **Assessment**
- Monitor:
- Blood glucose levels
- Vital signs
- Signs of complications
- Neurological status

Nursing Interventions

Intervention	Rationale
Monitor blood glucose	Detect hyper/hypoglycemia
Administer insulin	Control blood glucose
Provide patient education	Promote self-care
Monitor feet daily	Prevent ulcers
Encourage healthy lifestyle	Prevent complications

Nursing Diagnosis

Diagnosis

Imbalanced nutrition

Risk for unstable blood glucose

Risk for infection

Impaired skin integrity

Deficient knowledge

- Patient Education
- Patients should learn:
 - Blood glucose monitoring
 - Proper insulin administration
 - Healthy diet
 - Exercise
 - Foot care
 - Medication adherence

Easy Memory Tips

- **Type 1 DM = “NO insulin”**
- Needs insulin **forever**
- Risk for **DKA**
- **Type 2 DM = “NOT working insulin”**
- Insulin is present but **ineffective**
- Risk for **HHS**

Management Overview

Area	Key Points
Diet	Control carbohydrates
Exercise	At least 30 mins/day
Medication	Insulin or oral drugs
Monitoring	Check blood glucose regularly
Education	Lifelong self-care

Diabetic Foot Care

Do	Avoid
Inspect feet daily	Walking barefoot
Keep feet clean/dry	Tight shoes
Wear proper footwear	Ignoring wounds