

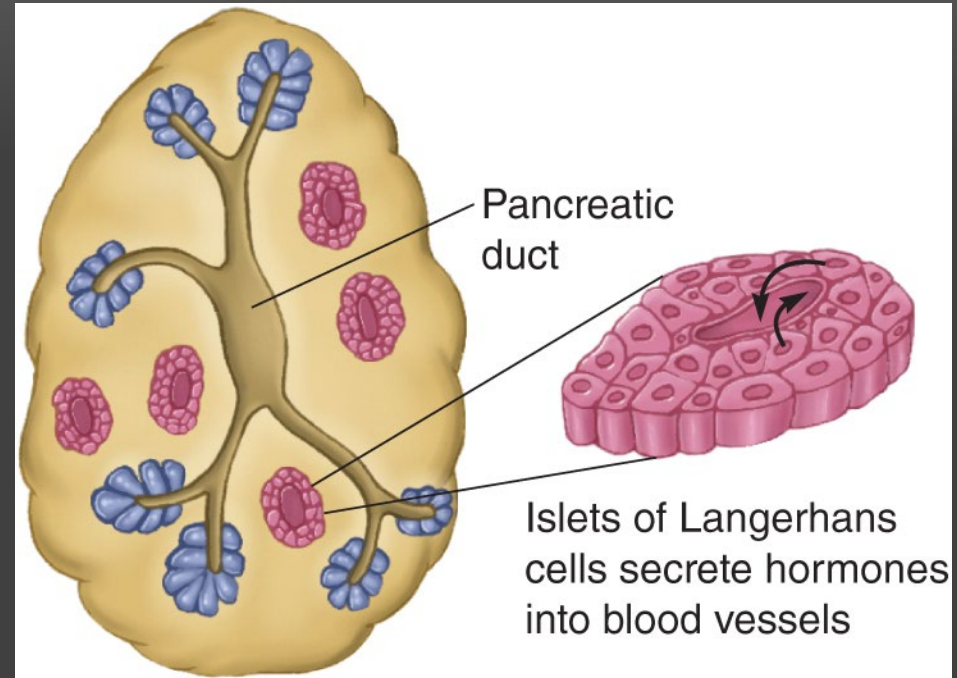
ENDOCRINE EMERGENCIES – DIABETES



Pea Ridge Fire Department
EMS refresher

Components of the Endocrine System

- Pancreas
 - Digestive gland considered both an endocrine and an exocrine gland
 - Exocrine component secretes digestive enzymes into duodenum via pancreatic duct.



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Components of the Endocrine System

- Pancreas (cont'd)
 - Endocrine component comprises the islets of Langerhans.
 - Cell groups within the pancreas that act like “an organ within an organ”
 - Alpha cells secrete glucagon.
 - Beta cells secrete insulin.
 - Delta cells secrete somatostatin.

Components of the Endocrine System

- Pancreas (cont'd)
 - When blood glucose level falls, glucagon is secreted to raise it.
 - When blood glucose level rises, insulin is secreted.

Patient Assessment

- Endocrine emergencies tend to affect many organ systems.
 - Do not take them lightly.

Scene Size-Up

- Address hazards.
- Follow standard precautions.
- Check the home for medications.

Primary Assessment

- Identify and manage life threats.
- Form a general impression.
 - Signs and symptoms depend on affected hormone.
 - Is patient alert?
 - Diaphoresis is a sign of severe distress.

Primary Assessment

- Airway and breathing
 - Ensure patent airway.
 - Investigate abnormal breathing sounds.
 - Assess breathing effort.

Primary Assessment

- Circulation
 - Assess skin color, moisture, and temperature.
 - Obtain blood pressure.
 - If necessary:
 - Administer IV fluid.
 - Replenish blood component.

Primary Assessment

- Transport decision
 - Many patients should be transported to a specialty facility.
 - Provide rapid transport to the closest facility if the patient is unstable.

History Taking

- Especially useful in diabetic emergencies
- If patient is unresponsive
 - Obtain blood glucose level
- Consider signs and symptoms.
 - Undiagnosed or poorly managed diabetes may cause:
 - Polyphagia
 - Polyuria
 - Polydipsia

History Taking

- Ascertain any allergies prior to administering medication.
- Document all medications being taken.

Secondary Assessment

- Goals with comatose patients:
 - Determine level of consciousness.
 - Look for source of coma.

Secondary Assessment

- Vital signs
 - Look for hypertension and bradycardia.
 - Be alert for abnormal respiratory patterns.
 - Look for respiratory-related motions.

Reassessment

- Continually reassess the patient.
- Be prepared to manage the airway.
- Obtain blood specimens early in patients with diabetes.
- Provide emotional support.
- Monitor cardiac rhythm.
- Recheck vital signs, pupils, and level of consciousness.

Emergency Medical Care

- If patient has altered mental status:
 - Establish IV with 0.9% (NS) or a saline lock.
 - Initiate treatment for reading <60 mg/dL.
 - Give 2mL per kg of D₁₀.
 - If condition does not improve after dextrose and you suspect narcotic overdose, consider naloxone.

Emergency Medical Care

- Transport for comatose patients
 - Intubated: Supine with cervical collar
 - Not intubated: Stable side position
 - Increasing intracranial pressure: Head elevated to 30–45 degrees and midline
 - Keep mouth and pharynx free of secretions.

Glucose Metabolic Derangements

- Endocrine disorders are caused by:
 - Hypersecretion of a gland
 - Insufficient secretion of a gland
- Glucose metabolic derangements
 - Caused by dysfunction of the pancreas

Glucose Metabolic Derangements

- Most endocrine emergencies result in:
 - Compromise of the ABCs
 - Improper fluid balance
 - Deteriorating mental status
 - Abnormal vital signs and blood glucose levels

Diabetes

- A group of complex metabolic disorders
 - Diabetes mellitus
 - Gestational diabetes
 - Hypoglycemia/hyperglycemia
 - Diabetic ketoacidosis
 - Hyperosmolar hyperglycemic syndrome

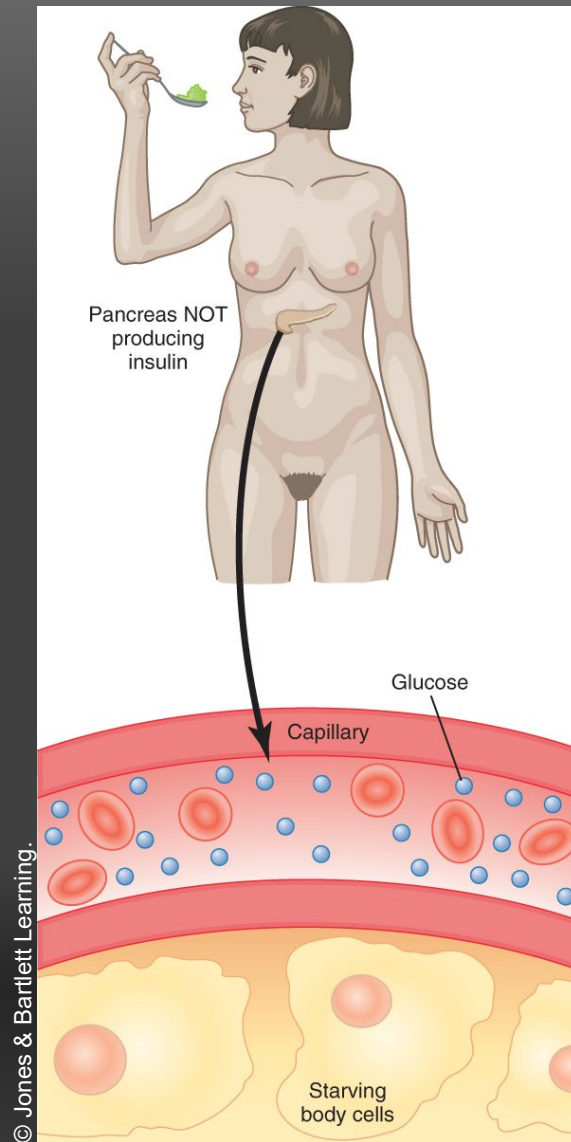
Diabetes Mellitus

- Metabolic disorder in which body's ability to metabolize simple carbohydrates is impaired.
- Characterized by:
 - Polyphagia
 - Polydipsia
 - Polyuria

Diabetes Mellitus

- Glucose: Primary fuel for cellular metabolism
- Body cannot metabolize glucose.
 - Pancreas does not produce enough insulin
 - Cells do not respond to insulin

Diabetes Mellitus



Life-Altering Complications from Diabetes

- Kidney failure
 - Glomeruli become sclerotic.
- Heart disease
 - Lipolysis raises fat level in the blood.
 - Microangiopathy restricts blood flow.
 - Increased risk for silent MI

Life-Altering Complications from Diabetes

- Cerebrovascular disease, stroke, and hypertension
 - Peripheral artery disease
- Eyes
 - Primary cause of blindness.
 - Cataracts
 - Retinal detachment

Life-Altering Complications from Diabetes

- Neuropathy
 - Loss of sensation and function.
- Many conditions can be delayed or prevented with lifestyle changes.

Type 1 Diabetes

- Pathophysiology
 - Generally affects children
 - Environmental factors may be part of the cause.
 - Pancreatic cells do not produce insulin.
 - Daily insulin is required by injection or pump.
 - Strict dietary control must be observed.

Type 1 Diabetes

- Assessment
 - Assess compliance with disease management.
 - With altered mental status, suspect low blood glucose level.
 - Consider additional chronic conditions.
 - Look for sores or infections.

Type 1 Diabetes

- Management
 - Insulin injection is required.
 - Some patients use an insulin pump.
 - Several types of insulin are available.



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Type 2 Diabetes

- Pathophysiology
 - Most common form of diabetes
 - Blood glucose levels are elevated.
 - Often, pancreas produces enough insulin, but body cannot effectively use it.
 - Development is associated with obesity and physical inactivity.

Type 2 Diabetes

- Assessment
 - Symptoms may include:
 - Frequent urination
 - Thirst
 - Blurred vision
 - Frequent infections
 - Cranky, confused, or shaky
 - Unresponsiveness

Type 2 Diabetes

- Management
 - Weight loss helps to control disease.
 - Food intake must be spread throughout the day.
 - Medication/insulin required daily.

Type 2 Diabetes

Table 23-1

Oral Agents Used to Treat Type 2 Diabetes Mellitus

Medication Class	Function	Examples
Sulfonylureas	Stimulate beta cells to produce more insulin	Chlorpropamide (Diabinese) Glipizide (Glucotrol, Glucotrol XL) Glyburide (Micronase, Glynase, DiaBeta) Glimepiride (Amaryl)
Meglitinides	Stimulate beta cells to produce more insulin	Repaglinide (Prandin) Nateglinide (Starlix)
Biguanides	Decrease the amount of glucose produced by the liver	Metformin (Glucophage)
Thiazolidinediones	Increase insulin effectiveness in the muscle and decrease liver glucose production	Rosiglitazone (Avandia) Pioglitazone (ACTOS)
Alpha-glucosidase inhibitors	Prevent the breakdown of starches	Acarbose (Precose) Miglitol (Glyset)
DPP-4 inhibitors	Inhibit the breakdown of GLP-1, a naturally occurring compound in the body that reduces blood glucose levels	Sitagliptin (Januvia) Saxagliptin (Onglyza)

Prediabetes

- Blood glucose levels or hemoglobin A1c levels are above normal levels.
 - Not high enough to be diagnosed as diabetes
- Affects 1 out of 3 US adults, and 90% are unaware of status.
- Without intervention, 15–30% develop type 2 diabetes.

Prediabetes

- Risk factors
 - Age greater than 45 years
 - Being overweight
 - Family history of diabetes
 - Certain racial or ethnic backgrounds
 - Gestational diabetes
 - Physical inactivity

Gestational Diabetes

- Pathophysiology
 - Form of glucose intolerance during pregnancy
 - Increases risk of type 2 diabetes
 - Resolves before delivery for most women
 - Does not produce birth defects

Gestational Diabetes

- Assessment
 - Oral glucose tolerance test is used
 - Hemoglobin A1c test can be used during the first pregnancy visit
- Management
 - Stabilize blood glucose levels.
 - Diet, exercise, blood glucose testing
 - May require insulin injections

Hypoglycemia

- Low blood glucose level
 - 45 mg/dL or less
- Experienced by patients with type 1 and type 2 diabetes
- Requires close monitoring and control of diabetes to prevent long-term complications
- Severe hypoglycemia requires intervention and treatment.

Hypoglycemia

- Pathophysiology
 - Often results from:
 - Too much insulin
 - Too little food
 - Both
 - Counterregulation is body's natural defense to maintain blood glucose at appropriate level.

Hypoglycemia

- Pathophysiology (cont'd)
 - Body's first line of defense
 - Reduce insulin production by the pancreas
 - Increase glucagon production by alpha cells
 - Second line of defense
 - Secretion of catecholamines
 - Last defense
 - Stimulation of autonomic nervous system

Hypoglycemia

- Pathophysiology (cont'd)
 - In type 1 diabetes, islets of Langerhans do not make insulin.
 - Body's first line of defense against hypoglycemia is lost.
 - In type 2 diabetes, the pancreas generates insulin, but:
 - Body may be insulin-resistant
 - Pancreas may not produce enough insulin

Hypoglycemia

- Assessment
 - Common signs and symptoms include:
 - Trembling, rapid pulse, sweat, and hunger
 - Blood glucose <60 mg/dL
 - Unexplainable agitation, irritability, combative behavior
 - Altered mentation or confusion
 - Nausea
 - Weakness
 - Cool, clammy skin

Hypoglycemia

- Assessment (cont'd)
 - Additional signs and symptoms include:
 - Headache
 - Memory loss
 - Incoordination
 - Slurred speech
 - Irritability
 - Dilated pupils
 - Seizures and coma in severe cases

Hypoglycemia

- Assessment (cont'd)
 - Hypoglycemia develops rapidly.
 - Suspect in any diabetic patient with:
 - Bizarre behavior
 - Neurologic signs
 - Coma

Hypoglycemia

- Management
 - Immediately increase blood glucose.
 - Administration of glucose with stroke may exacerbate cerebral damage.



Hypoglycemia

- Management (cont'd)
 - Rule out hypoglycemia with a field glucose test.
 - Administer glucose tablets or sugar if patient is alert and able to swallow.
 - If patient has altered LOC or difficult airway:
 - Aspiration may result with administration of oral substances.
 - If patient has decreased LOC and obtaining a patent IV line is difficult:
 - Administer glucagon 1 mg IM in adults.

Hyperglycemia and Diabetic Ketoacidosis

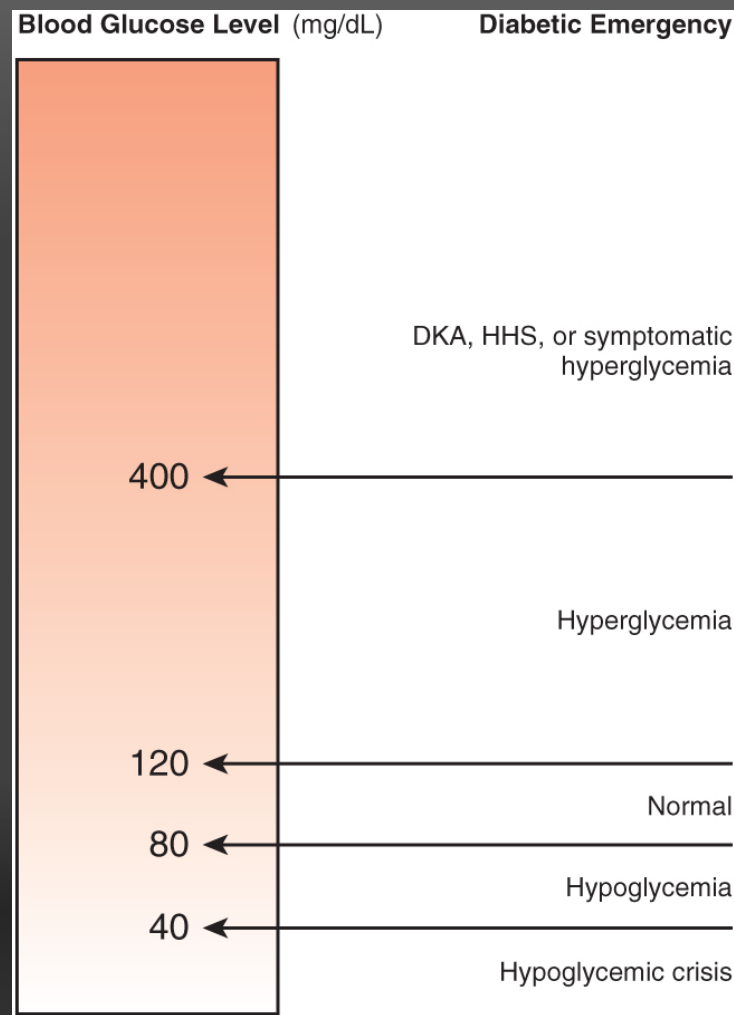
- Pathophysiology
 - Hyperglycemia
 - Classic symptom of diabetes
 - Early signs: Excessive thirst and urination
 - Occurs when blood glucose exceeds 120 mg/dL
 - Onset may be rapid or gradual.
 - Can be caused by:
 - Excessive food, insufficient insulin, infection or illness, injury, surgery, emotional stress

Hyperglycemia and Diabetic Ketoacidosis

- Other causes of hyperglycemia
 - Dawn phenomenon
 - Somogyi effect
- Patients with type 2 diabetes may go undiagnosed for several years.

Hyperglycemia and Diabetic Ketoacidosis

- Untreated hyperglycemia will progress to DKA.
 - Occurs when certain acids accumulate because insulin is not available



Hyperglycemia and Diabetic Ketoacidosis

- Pathophysiology (cont'd)
 - Rising blood glucose leads to massive osmotic diuresis.
 - Deficiency of insulin prevents cells from taking up the extra glucose.
 - Ketones in bloodstream cause a decrease in the blood's pH and result in acidosis.
 - In type 2 diabetes, DKA is rare because insulin is still present.

Hyperglycemia and Diabetic Ketoacidosis

- Assessment
 - Signs and symptoms of hypoglycemia and hyperglycemia can be similar.
 - Hypoglycemia signs and symptoms can include:
 - Blurred vision
 - Polyuria, polydipsia, polyphagia
 - Orthostatic syncope
 - Frequent infections
 - Skin ulcerations

Hyperglycemia and Diabetic Ketoacidosis

- Hyperglycemia usually progresses slowly, with LOC deteriorating gradually.
 - Signs and symptoms include:
 - Polyuria, polydipsia, polyphagia
 - Nausea and vomiting
 - Tachycardia
 - Deep, rapid respirations (Kussmaul respirations)
 - Warm, dry skin and dry mucous membranes
 - Fruity odor of ketones on the breath
 - Abdominal pain, hypotension, and fever

Hyperglycemia and Diabetic Ketoacidosis

- Patients may exhibit:
 - Thin or dehydrated appearance
 - Warm, dry skin
 - Orthostatic hypotension
 - Supine hypotension
 - Fatigue
 - Altered mental status
 - If patient is in coma state, look for head injury, stroke, or drug overdose.

Hyperglycemia and Diabetic Ketoacidosis

Table 23-2 Comparison of Hypoglycemia and Hyperglycemia			
	Hypoglycemia	Hyperglycemia: Diabetic Ketoacidosis ^a	Hyperglycemia: HHS ^b
Food intake	Insufficient	Excessive	Excessive
Insulin dosage	Excessive	Insufficient	Insufficient
Onset	Rapid, within minutes	Gradual—hours to days	Gradual—days to weeks
Skin	Pale and moist	Warm and dry	Warm and dry
Infection	Uncommon	Common	Usual
Thirst	Absent	Intense	Very intense
Hunger	Intense	Excessive	Excessive
Vomiting	Uncommon	Common	Uncommon
Breathing	Normal or rapid	Rapid, deep (Kussmaul respirations)	Tachycardia
Odor of breath	Normal	Sweet, fruity (nail polish remover/acetone smell)	No ketone bodies are produced; therefore, there is no fruity odor
Blood pressure	Low	Normal to low	Hypotension
Pulse	Rapid, weak	Normal or rapid and full	Rapid, weak
Level of consciousness	Irritability, confusion, seizure, or coma	Restless merging to coma	Restless merging to coma
Urine: Sugar	Absent	Present	Present
Urine: Acetone	Absent	Present	Absent
Blood glucose level (mg/dL)	<60	>250	>600
Response to treatment	Immediately after administration of glucose	Gradual, within 6 to 12 hours following medical treatment	
Abbreviation: HHS, hyperosmolar hyperglycemic syndrome			
^a Usually associated with type 1 diabetes			
^b Usually associated with type 2 diabetes			
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Hyperglycemia and Diabetic Ketoacidosis

- Management
 - Insulin therapy may be delivered at the hospital.
 - If hypotensive, administer isotonic fluids.
 - Monitor cardiac rhythm.
 - Sharply peaked T waves may require administration of sodium bicarbonate.
 - Sine wave may require administration of calcium chloride or gluconate.

Hyperosmolar Hyperglycemic Syndrome (HHS)

- Pathophysiology
 - Also called hyperosmolar nonketotic coma (HONK)
 - Occurs primarily with type 2 diabetes
 - Characterized by
 - Hyperglycemia
 - Altered mental status, drowsiness, and lethargy
 - Visual or sensory deficit
 - Partial paralysis or muscle weakness

Hyperosmolar Hyperglycemic Syndrome (HHS)

- Pathophysiology (cont'd)
 - Few patients present in comatose state.
 - Most have severe dehydration and focal or global neurologic deficits.
 - Clinical features of HHS and DKA overlap.
 - Often develops in patients with diabetes who have a secondary illness that leads to reduced fluid intake

Hyperosmolar Hyperglycemic Syndrome (HHS)

- Assessment
 - Patients do not experience ketoacidosis.
 - Onset can take up to weeks.
 - Most have a history of diabetes.
 - Neurologic changes are possible
 - Drowsiness and lethargy
 - Delirium and coma
 - Focal or generalized seizures

Hyperosmolar Hyperglycemic Syndrome (HHS)

Table 23-3

Comparison of Hyperglycemic Conditions

	HHS/HONK	Diabetic Ketoacidosis
Glucose	>600 mg/dL	>250 mg/dL
Arterial pH	>7.3	<7.3
Ketone bodies	Absent	Present
Type of diabetes mellitus	Type 2	Type 1

Abbreviations: HHS, hyperosmolar hyperglycemic syndrome; HONK, hyperosmolar nonketotic coma

Data from: Hyperosmolar Hyperglycemic Nonketotic Syndrome (HHNS). American Diabetes Association website. <http://www.diabetes.org/living-with-diabetes/complications/hyperosmolar-hyperglycemic.html>. Updated December 6, 2013. Accessed March 23, 2017; Hemphill RR. Hyperosmolar Hyperglycemic State. Medscape website. <http://emedicine.medscape.com/article/1914705-overview>. Updated August 3, 2016. Accessed March 23, 2017; Westerberg DP. Diabetic ketoacidosis: Evaluation and treatment. *Am Fam Physician*. 2013 Mar 1;87(5):337-346; Gosmanov AR, Gosmanova EO, Cannon ED. Management of adult diabetic ketoacidosis. *Diabetes Metab Syndr Obes*. 2014;7:255-264; and Chiasson JL, Aris-Jilwan N, Bélanger R, et al. Diagnosis and treatment of diabetic ketoacidosis and the hyperglycemic hyperosmolar state. *CMAJ*. 2003 Apr 1; 168(7):859-866.

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Hyperosmolar Hyperglycemic Syndrome (HHS)

- Management
 - Address dehydration and altered mental status.
 - Manage airway.
 - A bolus of NS is appropriate for nearly all who are clinically dehydrated.

Thank you



Please continue to the next section