# Hyperglycemia and Diabetic ketoacidosis (DKA)


PREPARED BY:

Fares Mlitat
Akram Alawneh
Saja Sawalha
Duaa Jawabreh
Aleen Sawalmeh

COURSE INSTRUCTOR:

Dr.Fatima Hirzallah

# Overview of DM

The term **diabetes** mellitus refers to metabolic disorders characterized by elevated blood glucose concentrations and disordered insulin metabolism .

Normally ,pancreatic insulin secretions rise after food is ingested

In diabetes ,insulin secretion may be inadequate ,cells normally responsive to insulin may be resistant to its effects ,or both.

These impairments result in defective glucose uptake and utilization in muscle and adipose cells and unrestrained gluconeogenesis in the liver .

The result is hyperglycemia ,a marked elevation in blood glucose levels that can ultimately cause damage to blood vessels ,nerves ,and tissues

# Types of DM

Two main types of diabetes ,type 1 and type 2 diabetes

**TABLE 26-3 Features of Type 1 and Type 2 Diabetes Mellitus**

| Feature | Type 1 Diabetes | Type 2 Diabetes |
|---|---|---|
| **Prevalence in diabetic population** | 5–10% of cases | 90–95% of cases |
| **Age at onset** | <30 years | >40 years[a] |
| **Associated conditions** | Autoimmune diseases, viral infection, inherited factors | Obesity, aging, inactivity, inherited factors |
| **Major defect** | Destruction of pancreatic beta cells; insulin deficiency | Insulin resistance; insulin deficiency relative to needs |
| **Insulin secretion** | Little or none | Varies; may be normal, increased, or decreased |
| **Requirement for insulin therapy** | All cases | Some cases |
| **Former names** | Juvenile-onset diabetes<br>Insulin-dependent diabetes | Adult-onset diabetes<br>Non-insulin-dependent diabetes |

[a] Incidence of type 2 diabetes is increasing in children and adolescents; in more than 90% of these cases, it is associated with overweight or obesity and a family history of type 2 diabetes.

**What about Gestational Diabetes?**

# Stress-Induced Hyperglycemia

## BOX 18-1 RISK FACTORS FOR THE DEVELOPMENT OF HYPERGLYCEMIA IN THE CRITICALLY ILL PATIENT

- Preexisting diabetes mellitus, diagnosed or undiagnosed
- Comorbidities such as obesity, pancreatitis, cirrhosis, hypokalemia
- Stress response release of cortisol, growth hormone, catecholamines (epinephrine and norepinephrine), glucagon, glucocorticoids, cytokines (interleukin-1, interleukin-6, and tumor necrosis factor)
- Aging
- Lack of muscular activity
- Relative insulin deficiency/insulin resistance
- Administration of exogenous catecholamines, glucocorticoids
- Administration of dextrose solutions, nutritional support
- Drug therapy such as thiazides, beta-blockers, highly active antiretroviral therapy, phenytoin, tacrolimus, cyclosporine

## Potential adverse consequences

- Immune suppression

- Cerebral ischemia/stroke

- Dehydration/osmotic diuresis

- Impaired wound healing

- Endothelial dysfunction/thrombosis

- Decreased erythropoiesis

- Impaired gastric motility

## Clinical Management

**•Establish euglycemia**

- Target glucoses of 180-140 mg/dL **(According to American Diabetes Association and the American Association of Clinical Endocrinologists study)**

# Acute complications of DM

1- Diabetic Ketoacidosis in Type 1 Diabetes .

2- Hyperosmolar Hyperglycemic Syndrome in Type 2 Diabetes .

# Diabetic Ketoacidosis (DKA)

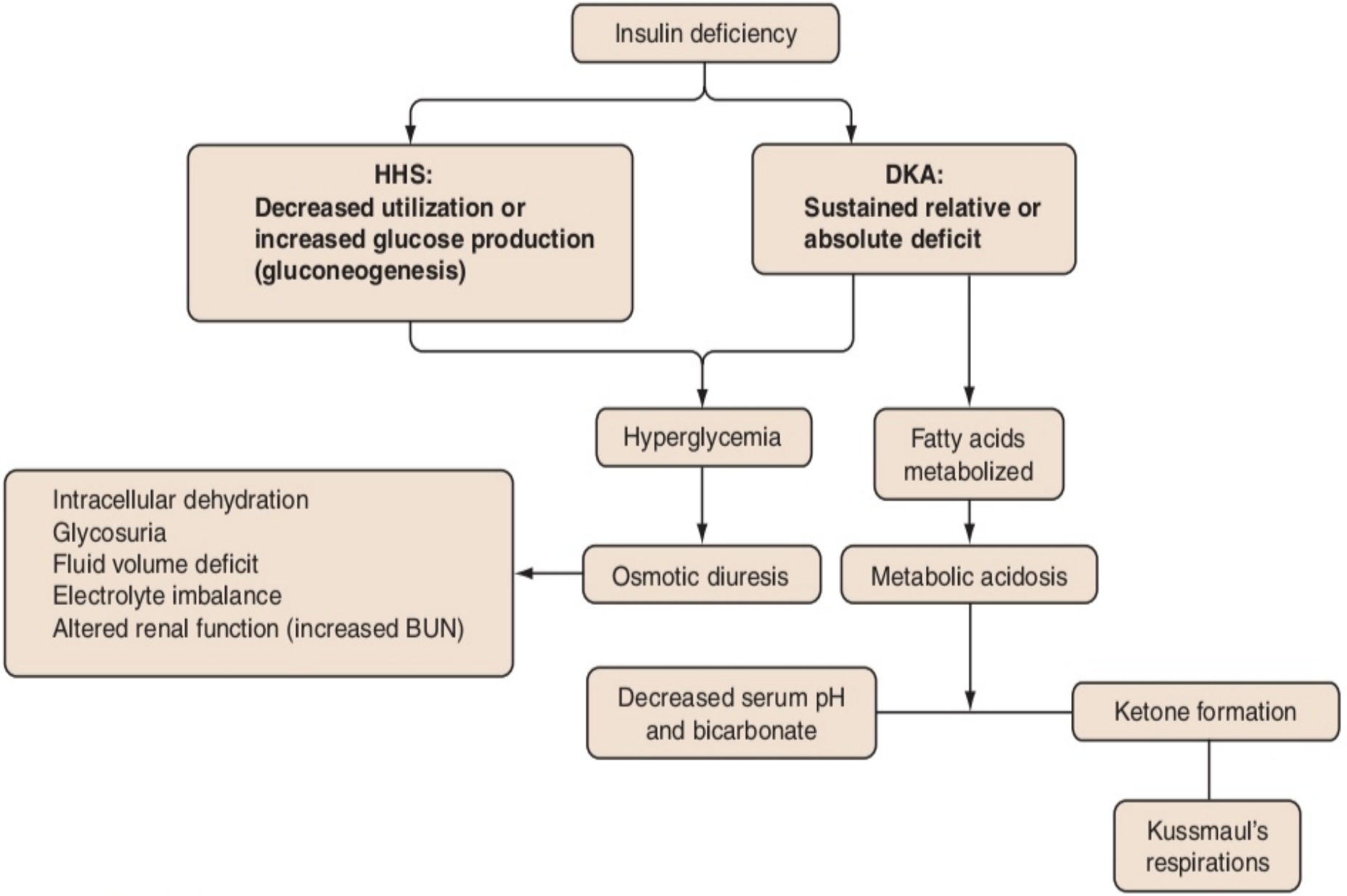



**FIGURE 18-3** Pathophysiology of diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS).

# Diabetic Ketoacidosis (DKA) pathophysiology :

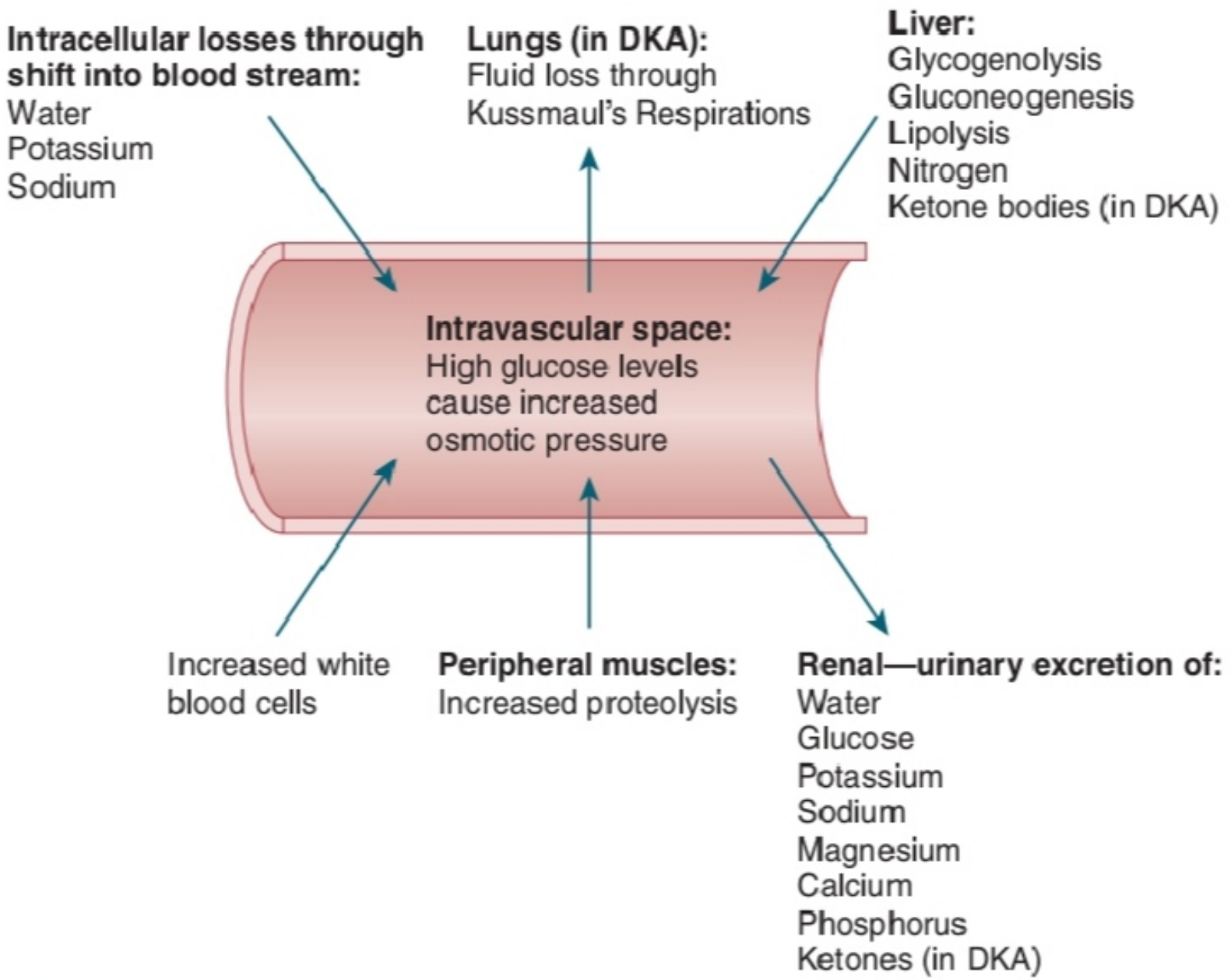



**FIGURE 18-4** Intracellular/extracellular shifts in hyperglycemic crises. *DKA*, Diabetic ketoacidosis.

# Anion Gap

## BOX 18-5 CALCULATION FOR ANION GAP

$$(Na^+ + K^+) - (Cl^- + HCO_3^-)$$

The normal value is 8 to 16 mEq/L. An elevated value indicates the accumulation of acids, such as is present in diabetic ketoacidosis.

*$Cl^-$*, Chloride; *$HCO_3^-$*, bicarbonate; *$K^+$*, potassium; *$Na^+$*, sodium.

# DKA :Etiology

- Initial presentation of type 1 diabetes

- Infections

- Insufficient insulin relative to need

- Severe stress—trauma ,surgery ,acute myocardial infarction( AMI)

- Pregnancy in type 1 diabetes mellitus( DM)

# DKA :Etiology( continued)

**•Missed or reduced insulin**

- Nonadherence to insulin regimen

- Insulin pump failure

- Intentional omission : Eating disorders

**•Medications**

Glucocorticoids

**•Mismanagement of sick days**

# Clinical Presentation of DKA

- Classic signs of dehydration
- Orthostasis
- Polyuria
- Polydipsia
- Polyphagia
- Hyperventilation/Kussmaul's respirations
- Fruity odor to breath
- Flushed/dry skin
- Decreased bicarbonate 15( mEq/L)
- Decreased pH( less than 7.3)

# Clinical Presentation of DKA (cont)

Lethargy/altered consciousness

Abdominal pain/nausea/vomiting

Blood glucose greater than250 mg/dL (May be lower in pregnancy)

Ketonuria/glucosuria

Weight loss( may be profound)

Blood gas changes( metabolic acidosis)

# Diagnosis of DKA :

↑ Plasma glucose (average: 675 mg/dL)
pH <7.30
↓ Bicarbonate
Ketosis
Azotemia
Electrolytes vary with state of hydration; often hyperkalemic
Plasma hyperosmolality (average: 330 mOsm/kg)

# Electrolyte Imbalances in DKA

•Hypokalemia( even if serum K +is normal or high ) , Will progress with addition of insulin to treatment regimen

**Insulin“ pushes ”potassium INTO CELLS**

•Phosphate depletion **(Enhanced by insulin administration)**

•Mild hyponatremia

•Elevated BUN/creatinine **(Secondary to profound dehydration)**

# Hyperosmolar Hyperglycemic State

# Pathophysiology of Hyperosmotic Hyperglycemic State

- Decreased use of glucose and/or increased production .

- Hyperglycemia ;increased extracellular osmolality .

- Osmotic diuresis

- Profound dehydration

- No ketoacidosis—hyperglycemia with hyperosmolarity blocks lipolysis

# HHS : Etiology

- Inadequate insulin secretion ;usually with type 2 diabetes .

- Often in geriatric patients with decreased compensatory mechanisms

Stress response .

**- Medications affect blood glucose levels**

Thiazides

Phenytoin

Glucocorticoids

Beta-blockers

Calcium channel blockers

- Enteral and parenteral nutrition

# HHS Clinical Presentation :

- Flushed, dry skin
- Dry mucous membranes
- ↓ Skin turgor (may not be present in elderly)
- Tachycardia
- Hypotension
- Shallow respirations
- Altered level of consciousness (generally more profound and may include absent deep tendon reflexes, paresis, and positive Babinski's sign)

# HHS Diagnosis :

↑ Plasma glucose (usually >1000 mg/dL)
pH >7.30
Bicarbonate >15 mEq/L
Absence of significant ketosis
Azotemia
Electrolytes vary with state of hydration; often hypernatremic
Plasma hyperosmolality (average: 350 mOsm/kg)
Hypotonic urine

# Medical Interventions

1-Respiratory support

2-Fluid replacement

3-Insulin therapy

4-Electrolyte management

5-Treatment of acidosis

6-Patient and family education

# Respiratory support

Assessment of the airway ,breathing ,and circulation is always the first priority in managing lifethreatening disorders.

- Airway and breathing may be supported through the use of oral airways and oxygen therapy.

In more severe cases ,the patient may be intubated and placed on ventilatory support.

- Prevention of aspiration is accomplished by elevating the head of the bed.

-Nasogastric tube suction may be considered in a patient with impaired mentation who is actively vomiting.

# Fluid replacement

- Dehydration may have progressed to hypovolemic shock by the time of admission .

- In DKA ,the typical water deficit approximates 100 mL/kg ,and it may be as high as 200 mL/kg in HHS

- Right atrial pressure or pulmonary artery pressure monitoring may also be instituted to evaluate fluid requirements and to monitor the patient’s response to treatment.

**Fluid replacement (Cont)**

- Normal saline %0.9( NS )is the fluid of choice for initial fluid replacement because it best replaces extracellular fluid volume deficits .

-Fluid replacement usually starts with an initial bolus of 1 L of 0.9% NS. This is followed by an infusion of 15 to 20 mL/kg during the first hour .

**The goal is to** replace half of the estimated fluid deficit over the first 8 hours. The second half of the fluid deficit should be replaced during the next 16 hours of therapy so that the volume is restored in most patients within the first 24 hours of treatment

**The effectiveness of fluid replacement is evaluated by:**

hemodynamic status ,intake and output ,laboratory measures ,and assessment of the patient's general physical condition ,particularly mental status .

**Fluid replacement (Cont)**

- IV fluids are rapidly infused until the patient's blood pressure and serum sodium level normalize.

- If the serum sodium is elevated or normal ,IV fluid is changed to hypotonic saline %0.45(NS )and infused at slower rates to replace intracellular fluid deficits.

- When the plasma glucose level approaches 200 mg/dL %5 ,dextrose is added to fluids to prevent hypoglycemia and assist in the resolution of ketosis

**Point to be in consideration during fluid replacement :**

- Significant improvements in hyperglycemia may be seen with fluid resuscitation before initiation of insulin therapy.

- Hyperglycemia resolves more quickly than ketosis .

- Hypervolemia must be prevented ,especially in patients with ischemic heart disease ,heart failure ,or acute kidney injury.

- Fluid overload from overaggressive fluid replacement can be prevented by monitoring breath sounds and performing cardiovascular assessments .

- Rapid fluid administration may also contribute to cerebral edema, a complication associated with DKA ,which could result in seizures and coma **(Treatment of acute cerebral edema usually involves administration of an osmotic diuretic( e.g. %20 ,mannitol solution)**

**Insulin therapy :**

- **Before** starting insulin therapy ,fluid replacement therapy must be underway and the serum potassium level must be greater than 3.3 mEq/L .

-An **initial** IV bolus of 0.1 units/kg of regular insulin is administered ,followed by a **continuous** infusion of 0.1 units/kg per hour to achieve a steady decrease in serum glucose levels of 50 to 75 mg/dL per hour , not more than ,to prevent cerebral edema, which could result in seizures and coma

- Serum glucose levels are monitored every 1 to 2 hours

-While receiving an intravenous insulin infusion, patients should be NPO .

- When glucose values are less than 200 mg/dL ,insulin infusion rates may be decreased to 0.02 to 0.05 units/kg per hour and maintained to keep the glucose value in the range of 150 to 200 mg/dL

# Insulin therapy (Cont)

- **Patients may be transitioned to subcutaneous insulin** when the blood glucose is 200 mg/dL or less and **when two of the following criteria are met**

1- venous pH is greater than7.30

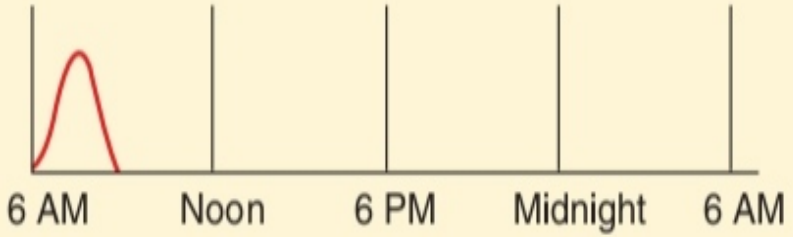
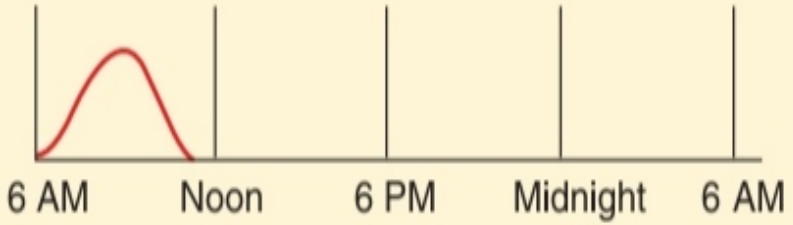
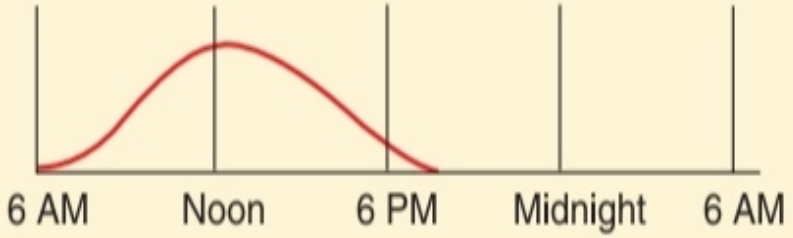
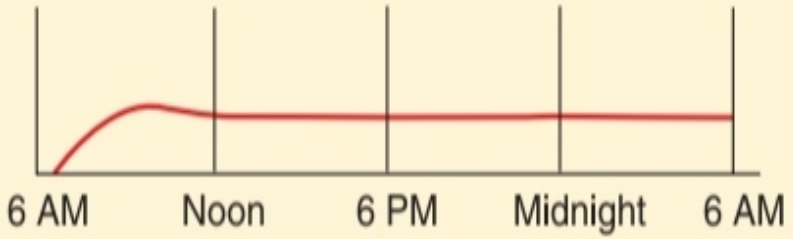
2- serum bicarbonate level is greater than 15 mEq/L,

3- calculated anion gap is 12 mEq/L or less

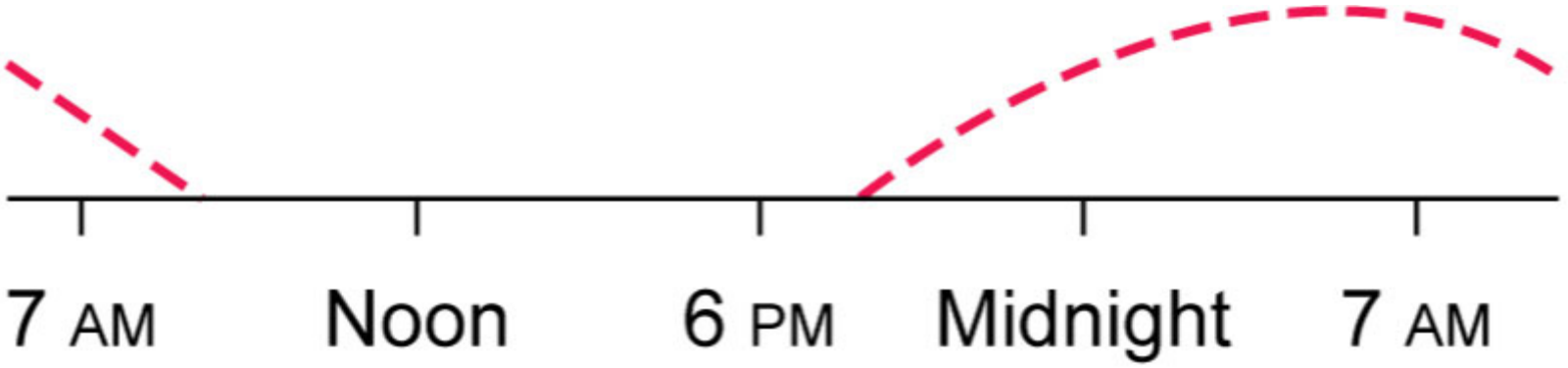
Glucose levels are monitored at least every 6 to 8 hours while a patient is receiving subcutaneous insulin.

- In patients with HHS ,insulin infusion rates may be decreased to 0.2 to 0.5 units/kg per hour when the glucose values reach 300 mg/dL.

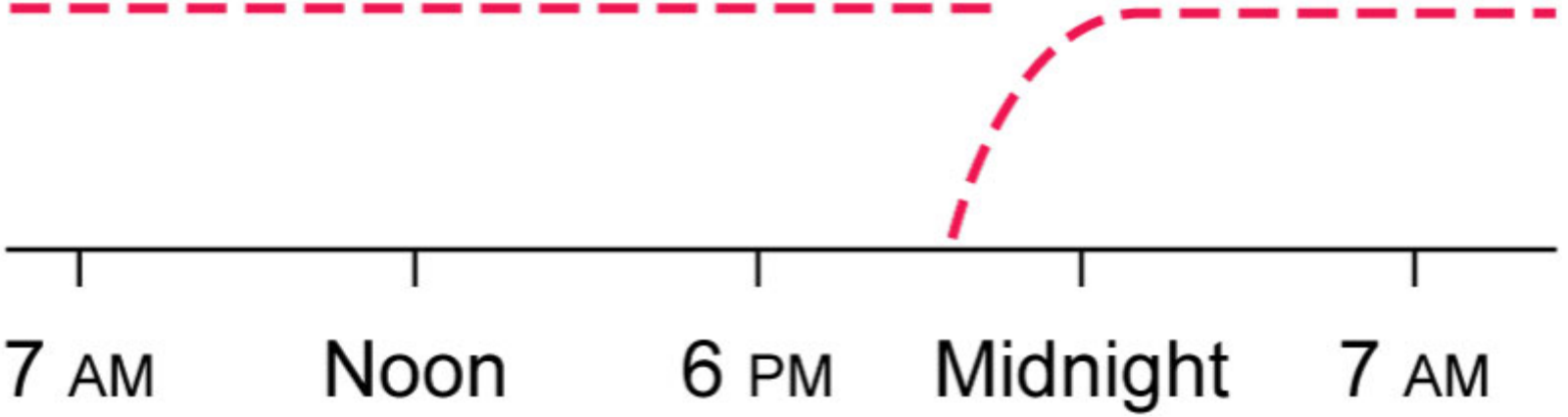
- Target glucose values of 200 to 300 mg/dL should be maintained until the patient's mental status improves ,at which time the patient may be transitioned to subcutaneous insulin therapy.

| INSULIN PREPARATION | ONSET, PEAK, DURATION | EXAMPLE |
|---|---|---|
| **Rapid-acting**<br>lispro (Humalog)<br>aspart (NovoLog)<br>glulisine (Apidra) | *Onset:* 15 min<br>*Peak:* 60-90 min<br>*Duration:* 3-4 hr |  |
| **Short-acting**<br>Regular (Humulin R, Novolin R, ReliOn R) | *Onset:* ½-1 hr<br>*Peak:* 2-3 hr<br>*Duration:* 3-6 hr |  |
| **Intermediate-acting**<br>NPH (Humulin N, Novolin N, ReliOn N) | *Onset:* 2-4 hr<br>*Peak:* 4-10 hr<br>*Duration:* 10-16 hr |  |
| **Long-acting**<br>glargine (Lantus)<br>detemir (Levemir) | *Onset:* 1-2 hr<br>*Peak:* no pronounced peak<br>*Duration:* 24+ hr |  |

# Common insulin regimens




From Michel B. Nursing management of diabetes. In Lewis SL, Dirksen SR, Heitkemper MM, eds. *Medical-Surgical Nursing: Assessment and Management of Clinical Problems*. 8th ed. St. Louis: Mosby. 2011.

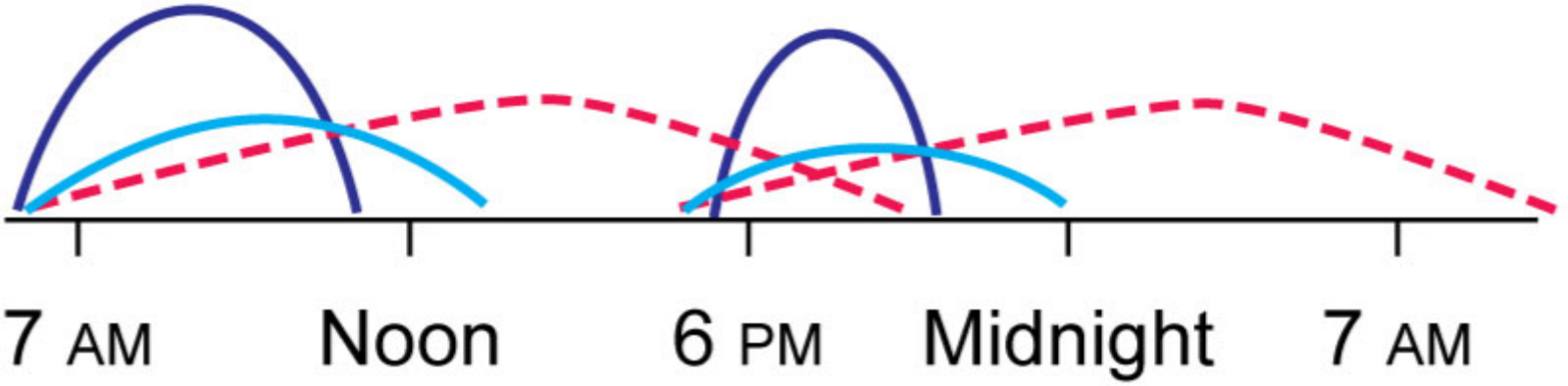



From Michel B. Nursing management of diabetes. In Lewis SL, Dirksen SR, Heitkemper MM, eds. *Medical-Surgical Nursing: Assessment and Management of Clinical Problems*. 8th ed. St. Louis: Mosby. 2011.

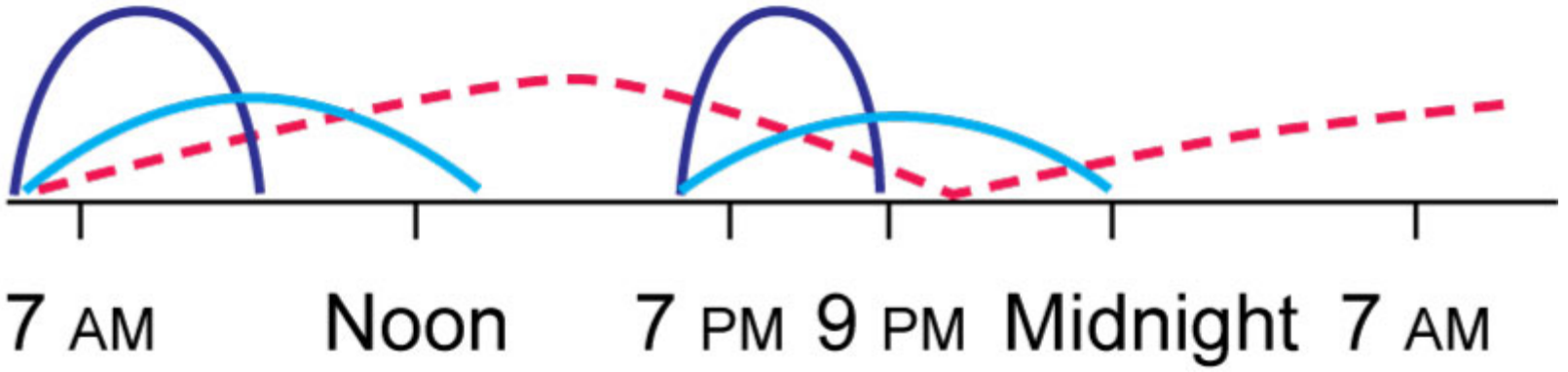
# Common Insulin Regimens. Once a day.

**Key:**

- Rapid-acting (lispro, aspart, glulisine) insulin.
- Short-acting (regular) insulin.
- Intermediate-acting (NPH) or long-acting (glargine, detemir) insulin.




From Michel B. Nursing management of diabetes. In Lewis SL, Dirksen SR, Heitkemper MM, eds. *Medical-Surgical Nursing: Assessment and Management of Clinical Problems*. 8th ed. St. Louis: Mosby. 2011.

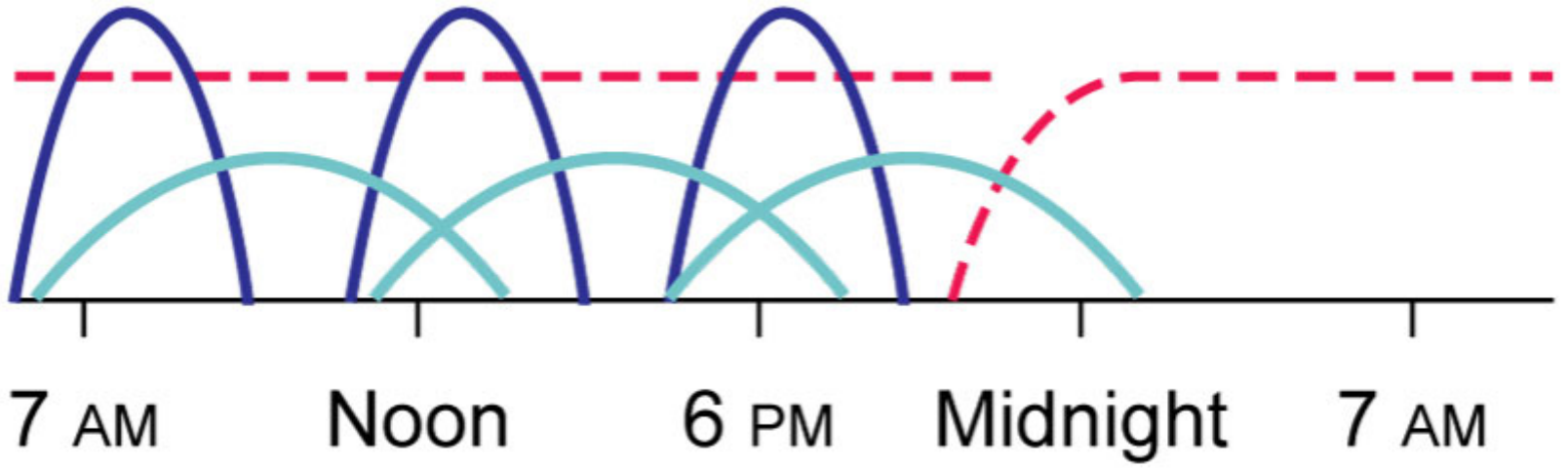



From Michel B. Nursing management of diabetes. In Lewis SL, Dirksen SR, Heitkemper MM, eds. *Medical-Surgical Nursing: Assessment and Management of Clinical Problems*. 8th ed. St. Louis: Mosby. 2011.

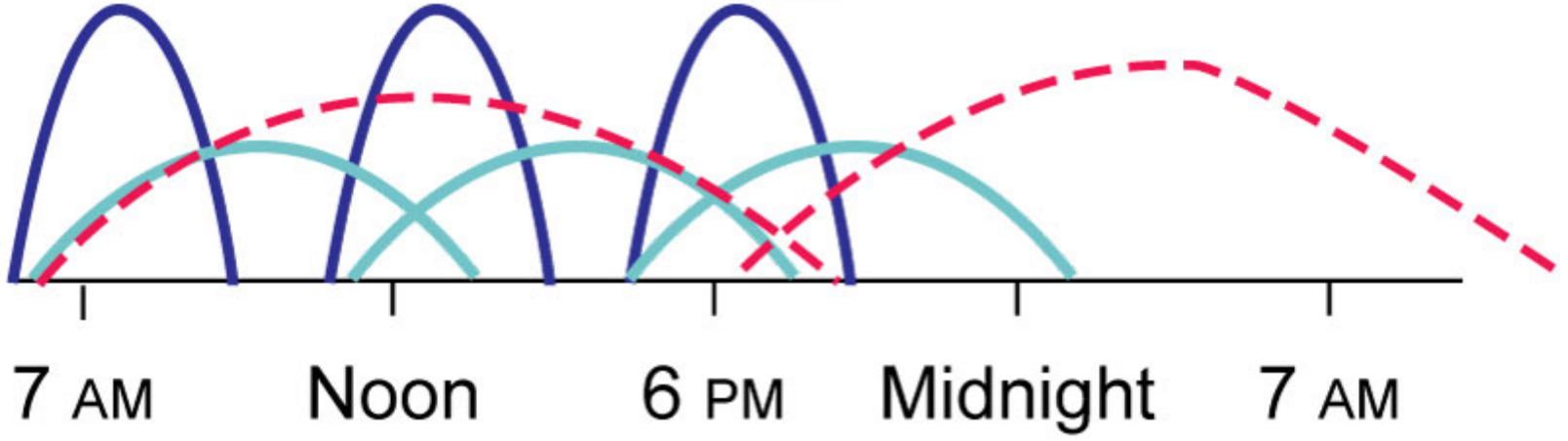
# Common Insulin Regimens. Twice a day

**Key:**

- Rapid-acting (lispro, aspart, glulisine) insulin.
- Short-acting (regular) insulin.
- Intermediate-acting (NPH) or long-acting (glargine, detemir) insulin.




From Michel B. Nursing management of diabetes. In Lewis SL, Dirksen SR, Heitkemper MM, eds. *Medical-Surgical Nursing: Assessment and Management of Clinical Problems*. 8th ed. St. Louis: Mosby. 2011.




From Michel B. Nursing management of diabetes. In Lewis SL, Dirksen SR, Heitkemper MM, eds. *Medical-Surgical Nursing: Assessment and Management of Clinical Problems*. 8th ed. St. Louis: Mosby. 2011.

# Common Insulin Regimens. Basal-bolus.

**Key:**

- Rapid-acting (lispro, aspart, glulisine) insulin.
- Short-acting (regular) insulin.
- Intermediate-acting (NPH) or long-acting (glargine, detemir) insulin.

# Electrolyte management.

- Potassium ,phosphate ,chloride ,and magnesium replacement may be required ,especially during insulin administration.

- Osmotic diuresis in DKA and HHS results in total body potassium depletion , the potassium deficit may be greater in HHS.

- Insulin therapy will promote translocation of potassium into the intracellular space resulting in a further decrease in serum potassium levels.

-Serum potassium levels should be maintained between 4 and 5 mEq/L during the course of therapy.

-In the event that the patient is admitted with hypokalemia ,insulin herapy should be withheld until potassium values exceed 3.3 mEq/L.

**Electrolyte management (cont)**

- The need for potassium therapy is based on serum laboratory results. In the absence of renal disease ,insulin replacement and monitoring begins afterthe first liter of IVfluid has been administered,the serum potassium level is greaterthan 3.3 mEq/L ,and the patient is producing urine.

- At that point 20 ,to 30 mEq of potassium may be added to each liter of fluid administered .

-Electrocardiographic (ECG) monitoring for cardiac dysrhythmias and assessment of respiratory status is also important during potassium administration

**Electrolyte management (cont)**

- Total body phosphorus levels are also depleted by osmotic diuresis ,but serum phosphate levels may remain in the normal range.

Insulin therapy may cause further reductions in phosphate levels. Phosphate replacement occurs when there is associated respiratory or cardiac dysfunction

-Phosphate replacement is used with extreme caution in patients with renal failure because these patients are unable to excrete phosphate and typically have underlying hyperphosphatemia

# Treatment of acidosis

Acidosis is a hallmark feature of DKA. However ,multiple studies have shown that treatment with sodium bicarbonate is often not beneficial and may increased risk of hypoglycemia ,cerebral edema ,cellular hypoxemia secondary to decreased uptake of oxygen by body tissues ,worsening hypokalemia ,and development of central nervous system.

Therefore sodium bicarbonate is not routinely used to treat acidosis unless the serum pH is less than 6,9 , Bicarbonate replacement is used only to bring the pH up to7.0 ,but not to normal levels

# Patient and family education.

- A primary intervention to prevent DKA is patient education.

- Managing blood glucose levels with diet ,exercise ,and medication is a priority.

- Monitoring of hemoglobin A1c levels three to four times per year provides an indication of the patient's long-term control of blood glucose levels ,changing insulin needs ,and indications of psychosocial or behavioral factors that may impact control ,including coping issues such as diabetes-related distress and depression

- The importance of a regular eating schedule, exercise, rest, sleep, and relaxation must be emphasized.

THANK YOU®