#### 

**Nurse Bedside Tests:** VS, Blood glucose checks, urine dip (ketones), 12-lead ECG, blood draws, IV access, catheter insertion.

* **Potassium Levels**
  + K+ moves from intracellular to extracellular space d/t increased entry of hydrogen ions into cells – displacing K+ ions
  + Even though there is depletion of K+ through urine and vomit, plasma K+ levels present normal or high
  + During DKA tx K+ levels fluctuate because insulin promotes uptake into cells

(Jerreat, 2010)

PATHOPHYISIOLOGY OF DKA:

Because there is not enough insulin available to use the glucose in the body, ketones are used to provide energy. Ketones are normal in starvation and can be used as a source of energy by the brain and kidney. In ketoacidosis, ketones are produced faster than they can be used or excreted.

(Jerreat, 2010)

Recreational drugs such as ecstasy, ketamine, methadone and cocaine have been shown to precipitate DKA, as well as alcohol binges. (Jerreat, 2010)

DKA is caused by the absence or markedly inadequate amount of insulin in the body. This results in disorders in the metabolism of carbohydrates, proteins and fats.

**Hyperglycemia**

* **S&S:** polyuria, polydipsia, blurred vision, weakness, headache, orthostatic hypotension, and hypotension with weak, rapid pulse
* Insulin deficiency leads to decreased cell utilization of glucose and the liver increases glucose production
* During times of infection/illness: increase in “stress” hormones (glucagon, epinephrine, norepinephrine, cortisol and growth hormone
  + These hormones promote glucose production by liver and interfere with glucose utilization by muscle, fat & tissue – counteracting insulin
* BG range from >11 - >50 mmol/L
* **Dx:** Blood glucose: >11 mmol/L

**Acidosis**

* **S&S:** hyperventilation, SOB, acetone breath, drowsiness/coma, N&V, abd pain, anorexia
* Severe acidosis has adverse effects on brain and heart
* Presence of ketones induced N&V
* Acetone breath (fruity odour) – d/t elevated ketone levels
* Hyperventilation – Kussmaul respirations – the body’s attempt to decrease acidosis, counteracting effect of ketone buildup
* A low PCO2 level (10-30 mm Hg) reflects respiratory compensation for the metabolic acidosis
* **Dx:** Moderate ketonuria or blood ketone levels >3 mmol/L, and plasma bicarbonate concentration <15 mmol/L or arterial pH <7.3

**Dehydration & Electrolyte Loss**

* **S&S:** tachycardia, hypotension, dry mouth, lips, tongue
* Hyperglycemia leads to polyuria & polydipsia
* Dehydration occurs during the body’s attempt to rid itself of excess glucose - leads to polyuria
  + Kidneys excrete glucose, water & electrolytes (Na & K+)
* Vomiting contributes to dehydration
* Hyperglycemia leads to glycosuria, which increases Na and water loss
* Patients with severe DKA may lose up to 6.5L of water and 400-500 mmol/L of Na, K+ and Cl in 24hr

# Three clinical features of dka:

“DKA”

Diabetic ketoacidosis

Andrew Ferguson – 2013 Western IP

Day, R. A., Paul, P., Williams, B., Smeltzer, S. C., & Bare, B. (2010). *Textbook of Canadian medical-surgical nursing.* Philadelphia: Lippincott Williams & Wilkins.

Donahey, E., Folse, S. (2012). Management of diabetic ketoacidosis. *Advanced Emergency Nursing Journal, 34*(3), 209-215.

Jerreat, L. (2010). Managing diabetic ketoacidosis. *Nursing Standard, 34*(24), 49-55.

**Extra Information:**

* Catheter inserted to monitor urine output and adequate renal function prior to initiating K+ infusion to prevent hyperkalemia
* ECG’s are performed to monitor for arrhythmias r/t abnormal K+ levels

**Reversing Acidosis**

* Ketone bodies accumulate d/t fat breakdown
* Acidosis that occurs in DKA is reversed with insulin
  + Inhibits fat breakdown – stopping acid buildup
* Insulin is usually IV infused slowly at ~5 units/hr
* Q1H BG tests
* **IMPORTANT: When mixing insulin drip, important to flush insulin solution through all tubing and discard first 50 mL of fluid as insulin molecules adhere to IV tubing!**
* Insulin infusion must be infused continuously until SC administration resumes – **Even if BG levels are dropping to normal**
  + Interruption may cause ketones to accumulate, worsening acidosis
  + Rate or concentration of dextrose (D5W) infusion should be changed to accommodate this
  + IV insulin infusion may continue for 12-24 hours until serum bicarbonate levels improve
  + **Bicarbonate infusion in severe acidosis is avoided during DKA tx b/c it could cause further K+ drops**

**Rehydration**

* Fluid replacement important for tissue perfusion and increasing the excretion of excessive glucose by kidneys
* Patients may need up to 6-10 L of IV fluid to replenish losses
* **Initially:** 0.9% NS administered at 0.5-1.0 L/hr for 2-3 hrs
* **Later:** After first few hours, 0.45% NS is used for continued rehydration if BP stable and Na not low (200-500 mL/hr)
* When BG level comes down to 16.6 mmol/L or less fluid can be changed to D5W to prevent major glucose decline
* **Monitor VS frequently!**

**Restoring Electrolytes**

* **K+ is major lyte of concern!**
* Serum K+ level drops during course of treatment of DKA as K+ re-enters the cells – **Frequent monitoring required**
  + Rehydration – increases plasma volume & decreases concentration of K+ and increases excretion in urine
  + Insulin – enhances movement of K+ from extracellular fluid into cells
* **K+ replacement must begin once K+ levels drop to normal**
* **Initially:** Q2-4H ECG’s and K+ lab measurements during first 8 hrs of tx
* K+ tx withheld if presenting with hyperkalemia or not urinating

# Initial treatment of dka

Key Pieces of Information:

1. The majority of patients presenting with DKA have type I diabetes
2. The mortality rate associated with DKA is < 5%
3. Precipitating Factors: Situations increasing stress hormone production - infection, noncompliance, undiagnosed diabetes, pancreatitis, MI, CVA and medications

(Donahey & Folse, 2012)