Understanding Diabetes Mellitus

When Being Too Sweet Can Turn Things Sour
How Did You Learn Diabetes?

The Lists:

**Hyperglycemia**
- "Diabetic Coma"
  - Slow onset
  - Frequent urination
  - Thirst
  - Coma (rarely happens)
  - Dry mouth
  - Hunger
  - Abdominal pain
  - Dehydration
  - Kussmaul's respirations
  - Blurred vision
  - Rapid pulse (C-V Shock)
  - B/P low to normal
  - Weight loss
  - Fruity odor(?) keytones(?)

**Hypoglycemia**
- "Insulin Shock"
  - Rapid onset
  - Tachycardia (not shock)
  - Confusion/amnesia
  - Sweating
  - Headache
  - Hunger
  - Irritability/anxiety
  - Tachypnea
  - Loss of consciousness
  - Seizures
  - Coma
How Long Has Diabetes Existed?

... and how do we know?

- **Ancient Greek writings**
  - The 3 main symptoms are listed in ancient texts
  - Polyurina, Polydipsia, Polyphagia
    - Urinating, drinking, eating
  - These are the same symptoms you will look for today
What does “diabetes” mean?

• 'Passing through'
  - Polyuria

• Complete name: diabetes mellitus
  - 'Passing through the sugar'

• The ancients believed that the flesh was 'melting into the urine'

Is there another type of diabetes?

- Diabetes Insipitus
  - “Water passing through the body of unknown cause”
  - Loss of anti-diuretic hormone from pituitary gland
  - Not of pre-hospital interest
Normal Human Physiology

- Body eats food
  - Carbohydrates - Starches
    - Broken into sugars
      - Mono, di and poly-saccharides
  - Body metabolizes carbs for energy
    - Krebs citric acid cycle
    - Left over metabolic wastes
      - Water ($H_2O$) and Carbon Dioxide ($CO_2$)
Normal Human Physiology

- Carbohydrates arrive via blood stream
- Carbohydrates burned INSIDE the cell
- Carbohydrates need help passing through the cell wall
  - Helper molecule - INSULIN

![Diagram showing the process of carbohydrate metabolism](image-url)
Normal Human Physiology

- Insulin is produced in the Pancreas
  - Islets of Langerhans
  - Beta cells – insulin
  - Alpha cells – glucagon
  - Delta cells – somatostatin
    - aka ‘growth hormone-inhibiting hormone’
      - Regulates alpha and beta cells
Blood Sugar Level

- Blood sugar & insulin production oscillates throughout the day
- 'Normal' Blood Sugar Level:
  - 70–120 mg/dL
    - Insulin levels can’t really be checked
Pathophysiology of Diabetes Mellitus

- Body eats food – carbohydrates
- Sugars placed into blood
- Cell wants sugars for energy
- No Insulin – No Sugar into cell
- Sugar stays in the blood (plasma)
Pathophysiology of Diabetes Mellitus

- Sugar stays in blood
  - Blood sugar level rises
    - How high can it go?
      - 300? 600? 800? 1500? Mg/dL

- Body ejects ‘excess’ sugar through kidneys
  - Polyuria – Frequent copious urination
    - How much urine can be produced?
Pathophysiology of Diabetes Mellitus

- Body ejects ‘excess’ sugar through kidneys - **Polyuria**
- Fluid loss will be extreme
  - 1 liter+ per hour
    - Dehydration results
- Body demands WATER - **Polydipsia**
  - Extreme thirst
    - What are they drinking? Sugary drinks?
- Fluid intake can never keep up with urination
Pathophysiology of Diabetes Mellitus

- Body eats food - carbohydrates
- Cell wants sugars for energy
- No Insulin = No Sugar into cell
- Body needs FOOD - produces Hunger
  - Polyphagia
    - Consumed food never enters cells
- Starvation results
  - Loss of weight
  - Loss of energy
Pathophysiology of Diabetes Mellitus

- Body eats food - Polyphagia
- Body needs FOOD
  - No insulin . . .
  - Consumed food (glucose) can never enter the cells
- Starvation results
- **Fluid and weight loss combined**
  - Ancient Greeks believed that the flesh was melting away into the urine . . .
- The CELL (body) MUST SURVIVE
  - Alternative energy pathway needed
Pathophysiology of Diabetes Mellitus

- Alternative energy pathway
  - Cells burn FATS for energy
    - Fatty acids
  - Fats do not need insulin to enter cell
  - Body metabolizes fats for energy
    - Left over metabolic wastes -
      - Keytones and Acids
Pathophysiology of Diabetes Mellitus

• Blood chemistry changes:
  - Acids (keytones) are dumped into blood
    - pH levels drop
  - DKA Diabetic Keyto-acidosis
    - $H^+$ (acid) combines with bicarbonate $HCO_3^-$
    - Produces water ($H_2O$) and carbon dioxide ($CO_2$)
  - Acid is blown off through the lungs
    - Every $CO_2$ molecule eliminates one acid ion
  - ‘Tachypnea’ - but NOT short of breath!
    - Continuous sighs . . . Kussmaul’s Respiration

$H^+ (aq) + HCO_3^- (aq) \rightleftharpoons H_2CO_3(aq) \rightleftharpoons H_2O (l) + CO_2 (g)$
Pathophysiology of Diabetes Mellitus

- **Blood chemistry changes:**
  - Blood sugar rises - *HYPER-glycemia*
  - Blood becomes 'thicker'
    - Hyperosmolar state
  - Water leaves cells to dilute the high sugar levels
    - As water leaves, Potassium ($K^+$) leaves too
  - **Blood becomes hyperkalemic**
    - Potential for arrhythmias

![Diagram of osmosis with water and sugar molecules moving across a selectively permeable membrane.](image)
Pathophysiology of Diabetes Mellitus

Other Hyperglycemia symptoms?

- Slow onset?
  - Takes days of altered metabolism to build up changes
Other Hyperglycemia symptoms?

- Fruity/keytone smell?
  - Not detectable by everyone
Pathophysiology of Diabetes Mellitus

Other Hyperglycemia symptoms?

• Rapid pulse?
  - Patient is in a HYPOVOLEMIC state
Other Hyperglycemia symptoms?

- Altered LOC?
  - Loss of consciousness/Coma is a LATE sign
  - Occurs in only ~20% of DKAs
Pathophysiology of Diabetes Mellitus

Other Hyperglycemia symptoms?

- Abdominal Pain
  - New onset childhood Diabetes Mellitus commonly mistaken for appendicitis
Why Would Blood Sugar Rise?

1. Not taking meds
   - Insulin
   - Oral hypo-glycemic agents

2. Infection
   - Body needs to metabolize more energy & needs more insulin

3. Physiologic stresses
   - Trauma, MI

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**Figure 3. Pathogenesis of DKA and HHS**

Stress, Infection and/or Insufficient Insulin

- Absolute Insulin Deficiency
- Counterregulatory Hormones
- Relative Insulin Deficiency

- Lipolysis
- FFA to liver
- Ketogenesis
- Alkal reserve
- Ketoadidosis
- Triglyceride
- Hyperlipidemia
- Glucose utilization
- Glycogenesis
- Glycogenolysis

- Hyperglycemia
- Hyperosmolarity
- Glycosuria (osmotic diuresis)
- Loss of water and electrolytes
- Dehydration
- Decreased fluid intake
- Impaired renal function

DKA
HHS

Adapted from ref 1.
Prehospital Treatment - HYPERRglycemia

- Fluids - dehydration is the emergency!
  - NS (wide open?)
- Cardiac Monitor
  - K+ shifts cause arrhythmias
- O₂?
  - Not necessary - tachypnea may imply a need
- Insulin?
  - No, hyperglycemia is NOT the emergency
Type 1 Diabetes

- Body produces NO insulin
- Insulin Dependent Diabetes Mellitus (IDDM)
  - “Shot controlled Diabetes”
  - “Juvenile diabetes”
Sources of Therapeutic Insulin

- Originally recovered from animals
  - Cows & Pigs (bovine and porcine)
- Human insulin now produced by bacteria
  - Lispro (Humalog)
    - Onset 5-15 min Peak 30-90 Lasts 3-4 hours
  - Regular (Novolin)
    - Onset 30 min Peak 2-3 hrs Lasts 6-8 hrs
  - NPH (Novolin-N)
    - Onset 2-4 hrs Peak 4-10 hrs Lasts 16 hrs
  - Glargine (Lantus)
    - Onset 1 hr (no peak) Lasts 24 hours
How Is Insulin Administered?

- Insulin cannot be swallowed!
  - It would be digested in the stomach
- There are no 'insulin pills'
- SQ injections
  - Abdomen & Legs
- Insulin dispensers:
  - Pumps
Insulin Pumps

- **Size of a smart phone** - Filled with quick acting insulin
  - Works like a real pancreas - basal & bolus rate
    - Small catheter under skin
- **Tighter control** - lowers BG and A1C levels
  - 1. Count carbs in meal . . .
  - 2. Take Blood glucose level . . .
  - 3. Add or subtract insulin based on BG levels
- **Pump records information** about amount of insulin administered and can be accessed in field
Continuous Glucose Monitor

- **Continuous Glucose Monitor**
  - Measures glucose in tissue fluid
  - Uses ultra thin needle under the skin – replaced every 3-7 days
    - Not yet as accurate as regular test strips
    - Must be calibrated against test strips
  - Feeds information back to recorder/pump
  - Can set off alarms to check glucose level
Type 2 Diabetes

- Non-insulin dependent diabetes mellitus NIDDM
  - “Pill controlled diabetes”
  - “Adult onset diabetes”
  - “Stable diabetes”
- Body produces SOME insulin
  . . . . But not enough
- Body has an increased resistance to insulin
- “Diet controlled Diabetes”
  - Weight controlled
Oral Hypoglycemic Agents

- Some diabetic Pills:
  - chlorpropamide (Diabinese)
  - glimepiride (Amaryl)
  - glipizide (Glucotrol, Glucotrol XL)
  - glyburide (DiaBeta, Glynase, Micronase)
  - nateglinide (Starlix)
  - repaglinide (Prandin)
  - sitagliptin (Januvia)
  - tolazamide
  - Tolbutamide
  - acarbose (Precose)
  - metformin (Glucophage)
  - miglitol (Glyset)
  - pioglitazone (Actos)
  - rosiglitazone (Avandia)
How Diabetic Pills Work

- (1) Agents which increase the amount of insulin secreted by the pancreas
  - Diabinese, Orinase (overdose?)
- (2) Agents which increase the sensitivity of target organs to insulin & increase uptake of glucose
  - Metformin, Glucophage, Avandia, Actos
- (3) Agents which decrease the rate at which glucose is absorbed from the gastrointestinal tract - Slow absorption better matches natural insulin
  - Glyset, Precose, Glucobay
  - Rare usage in USA
Type 2 Diabetic Crisis

- **Hyperglycemic hyperosmolar syndrome (HHS)**
  - Hyperglycemic hyperosmolar coma
  - Nonketotic hyperglycemic hyperosmolar coma (NKHHC)
  - Hyperosmolar nonketotic coma (HONK)

  *Body produces SOME insulin.*

- **Patient did not take their pills.**
  - Blood sugar rises - **HYPERglycemia**
    - Polyuria
    - Polydipsia
  - Body **CAN** still metabolize some sugars - **Body does not need to burn fats**
  - Body **DOES NOT** produce acid - **No acidosis - No Tachypnea**

  *Patient may not feel 'sick'***
Gestational Diabetes

- Diabetes only when pregnant
  - Pancreas not strong enough to support mom and baby
- “Type 2 diabetes”
  - Treatment = Insulin
    - Oral hypoglycemic agents - birth defects
  - Baby ‘porks up’ on mom’s high blood sugar
    - Large birth weight babies
- Mom may become diabetic later in life
- No increased risk of diabetic child
HYPOglycemia

- First case of Diabetic hypoglycemia – 1923
- A complication of the treatment of Diabetes:
  1. ‘Insulin’ overdose
     - Drew up wrong amount or type
  2. Taking normal insulin without eating
     - Missed meals / delayed meals
     - Travel, holidays, special events
  3. Increase exercise, physical activity, sports
Pathophysiology of Hypoglycemia

- Blood sugar drops
  - “Insulin shock”
  - Below ~65 mg/dL
- Diabetics may react at higher levels
  - Due to chronic hyperglycemia
  - (Efforts to use glycogen are not effective)
- Blood Sugar may drop to ‘zero’
Hypoglycemia – Glucagon

- **Glucagon**
  - Normal body hormone
  - 1 mg injected
- Converts liver Glycogen into Glucose
  - Glycogenolysis
Pathophysiology of Hypoglycemia

- Blood sugar drops
- CNS (brain) does not store sugar
  - Neurons are first cells affected
- Confusion, disorientation, irritability
  - Dysphoria - amnesia
  - Loss of consciousness
  - possible seizures
Hypoglycemia - Sympathetic Nervous System

- Blood sugar drops
- Body responds with only rescue system available
- **Sympathetic nervous response**
  - Epinephrine release
    - Epi attempts to release liver glycogen
    - Tachycardia - nervousness
    - Vaso-constriction
    - Sweating
- Diabetic nerve damage may blunt the SNS response
Prehospital Treatments

- Assessment shows low blood sugar . . .
- Oral sugar??
  - Glucose paste
    - 1” between cheek & gum . . . .
  - Soda pop? - Not diet soda!
    - Simple sugars quickly absorbed
  - Orange juice?
    - Complex sugars may be slower to digest
- Candy?
  - Hard candy is easy to store
    - slower to digest
Prehospital Treatment - Hypoglycemia

- Assessment shows low blood sugar:
- **50% Glucose in solution** - 50cc
  - D-50 (25 Gms of sugar)

- Administration
  - Slow IVP into large vein

- Precautions
  - Do NOT allow infiltration into tissues

- Complications
  - Tissue necrosis

- **D-50 expressed as blood sugar?**
Ways you become HYPOglycemic:

- Have you ever been hypoglycemic?
Hypoglycemia in the NON-diabetic

- Liver disease
  - Cant store glucose
    - Hepatitis?
    - Alcoholic?
- Lack of Adrenal gland function
  - Addison's disease
    - Corticosteroids
  - Low thyroid disease
  - Hypo-pituitary disease
- Pancreatic tumor
  - insulinaoma
Sugar Testing (Urine)

- Originally all blood needed to be sent to lab
  - Results could take days to weeks
- Urine was the only available out of hospital (home) test
- Clinitest® – copper sulfate reacts with sugar
  - Urine in test tube
  - Add Clinitest® tablet, wait for boiling to stop, compare color
Sugar Testing (Urine)

- **Acetest**
  - Tablets to detect Keytones
    - Darker purple means more keytones
- **Dipsticks made things easier**
  - But you were still testing urine
- Not an accurate reflection of current blood sugar level
Field *Estimation* of Blood Sugar

- Your assessment should predict the sugar level **BEFORE** any testing!
  - High, Low or Normal
- Ask pertinent questions . . . .
  - Poly, Poly, Poly?
  - LOC?
  - Abdominal pain?
  - Angry irritable behavior?
  - What were you doing before you got 'sick'?
  - Vital signs?
    - C-V shock? Tachycardia?
Field Testing of Blood Sugar

- **Blood glucose meters**
  - Take small blood sample
    - Arterial? Venus? Capillary?
    - Capillary beyond a finger?

- **Accurate testing:**
  - Follow manufacturers' instructions
  - Quality checks on machine
    - Use the ‘check/control’ solution
  - Ensure the strips are correct for the machine
  - Do the results match your assessment?
Normal Diabetic Blood Sugar

• Normal Blood Sugar = 70-120 mg/dL
• Normal Blood Sugar for a diabetic?
  - 70-120 mg/dL
    • Hypo could kill before hyper so older DM Tx has taught to keep blood sugar ‘a little high’
    • Modern DM Tx tries to keep BS no higher than 120 to avoid complications

Hemoglobin A1c test
Some glucose is bound to Hemoglobin
Reflects long term blood sugar levels
Normal 4% - 6%
Diabetic 7% or less
Long Term HYPERglycemia

- Long Term HYPER-glycemia alters cellular mitochondrial function
  - Endothelial proliferation - thickens walls of blood vessels
  - Alterations in lipid oxidation - increased blood cholesterols
  - Hypercoagulability - increase in micro clots

- All result in poor blood flow
  - Microangiopathy - capillaries
  - Macroangiopathy - arteries
How Organs Are Affected

- Organs with smallest blood vessels most at risk for damage
  - Kidneys
  - Eyes
  - Heart
  - Skin
  - Brain
  - Peripheral nerves
How Organs Are Affected

- **Kidneys**
  - Destruction of vasculature
  - Malfunction of filtration process
- #1 cause of kidney failure leading to dialysis
How Organs Are Affected

- **Eyes**
  - Thin vessels
    - Overgrow
    - Bleed
- **#1 cause of preventable blindness**

![Diabetic Retinopathy](image)

- Normal retina
- Retinopathy
- Macula
- Optic disk
- Hemorrhage
- Aneurysms
- Microaneurysms
- Hemorrhages
- Exudate
- Vitreous
- Retina
- Abnormal blood vessels
How Organs Are Affected

• Heart
• Coronary artery disease - Atherosclerosis
  - Angina
  - Ischemia
  - Infarction
• Diabetes major risk factor
How Organs Are Affected

- **Brain**
  - Inadequate perfusion leading to ischemic strokes
How Organs Are Affected

- **Peripheral nerves**
  - Poorly perfused
- **Nerves die** . . .
  - Numbness
  - Tingling
  - Lack of pain
    - No warning after injury
      - Skin breaks
      - Silent MI
    - No follow up treatment by patient
How Organs Are Affected

- Skin - Poor perfusion
  - Thinner
  - Easily torn & injured
Diabetic Amputations

- Nerve damage makes feet numb
- Injury occurs - no pain felt
- High blood sugar feeds bacteria
  - Infection worsens
- Wound poorly perfused
  - Less repair proteins
  - Few white blood cells
- Necrosis - Gangrene
- DM #1 cause of non-traumatic amputations
Diabetes and Prehospital Care

• Diabetes was once 100% fatal within weeks
  - Not understood - No treatments
• 1940s to 1970s diabetics survived but died from MI, renal failure, cancer, etc.
  - Some of these problems were caused by poor control
• Number of diabetics in society is increasing
• Greater chance of finding DM in patient's history
  - It may or may not be related to the chief complaint
  - No longer can you just “give sugar” to all diabetics

• 21st Century Paramedics must truly UNDERSTAND diabetes pathophysiology in order to provide competent pre-hospital care when.

... being too sweet can make things go sour