Diabetic Emergencies…

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Alabama EMS Challenge

THE UNIVERSITY OF ALABAMA AT BIRMINGHAM
Department of Emergency Medicine

Goals and No Disclosures

- Diabetes
  - Definitions
  - Some Pathophysiology and input on current treatments
- Hyperglycemic complications and treatment
  - DKA
  - HHS (HONK)
  - Hyperglycemia
- Hypoglycemic complications , risk factors, and treatment
- Key points in EMS care of the diabetic patient

May not be this…

But , hopefully not this…

Diabetes - Definitions

- A group of metabolic diseases characterized by ***hyperglycemia*** resulting from defects in ***insulin*** secretion, insulin action, or both.
- *In the 1950s, 1/5 people died within 20 years after a diagnosis of type 1 diabetes. 1/3 died within 25 years of diagnosis.*
- Now—can live relatively normal life span

(**before insulin, they all died**)

Type I Diabetes:

- 10% of all Diabetic cases
- Auto immune destruction of *Islet of Langerhans* in Pancreas

  (Isle of Dread)
- So….no or little insulin
  - Usually diagnosed in children and young adults…..*but…*

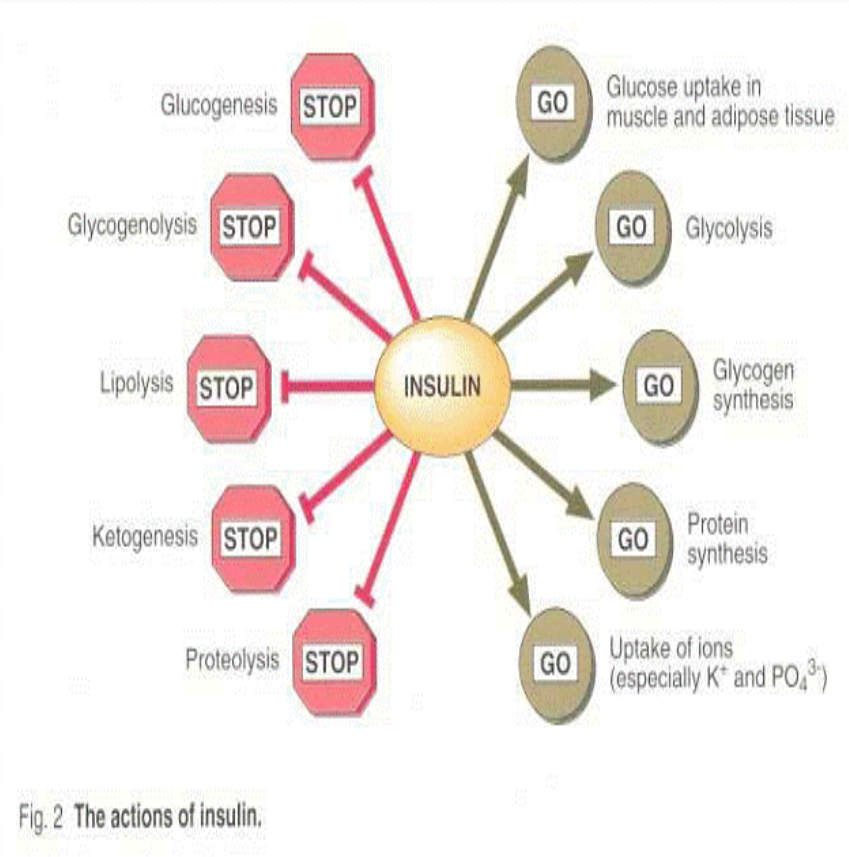
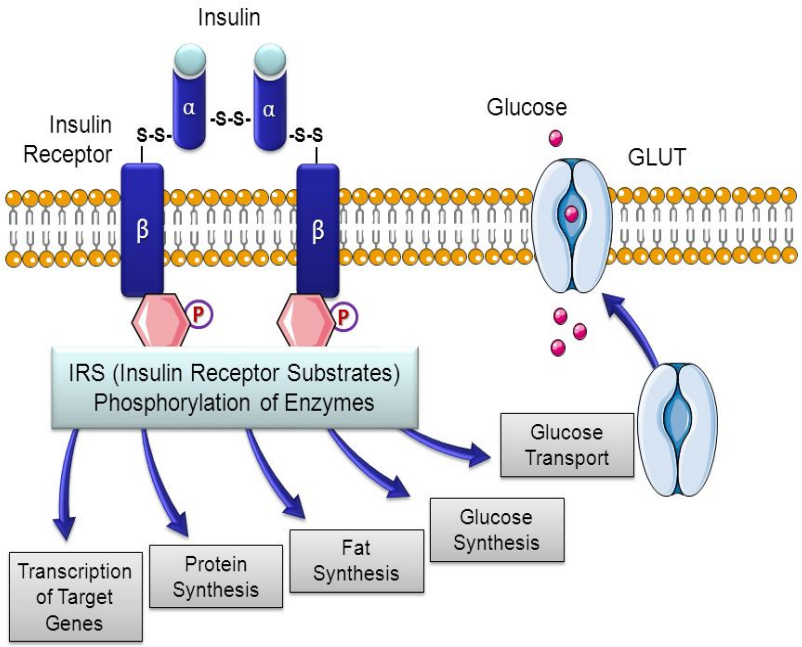


Fig. 2 **The actions of insulin.**



Treatment

On January 11, 1922 a 14-year-old diabetic who lay dying in Toronto , was given the first injection of insulin

Now we have:

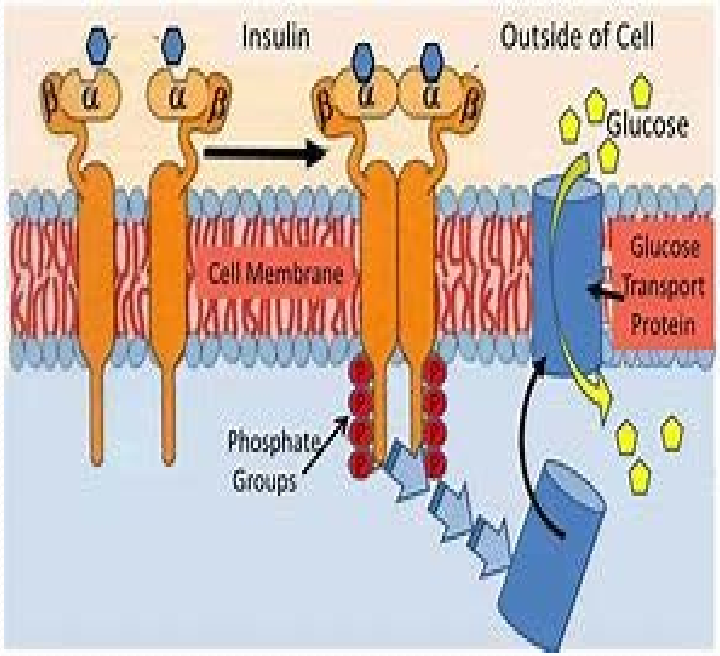
Long acting

Intermediate acting

Short and ultra fast acting

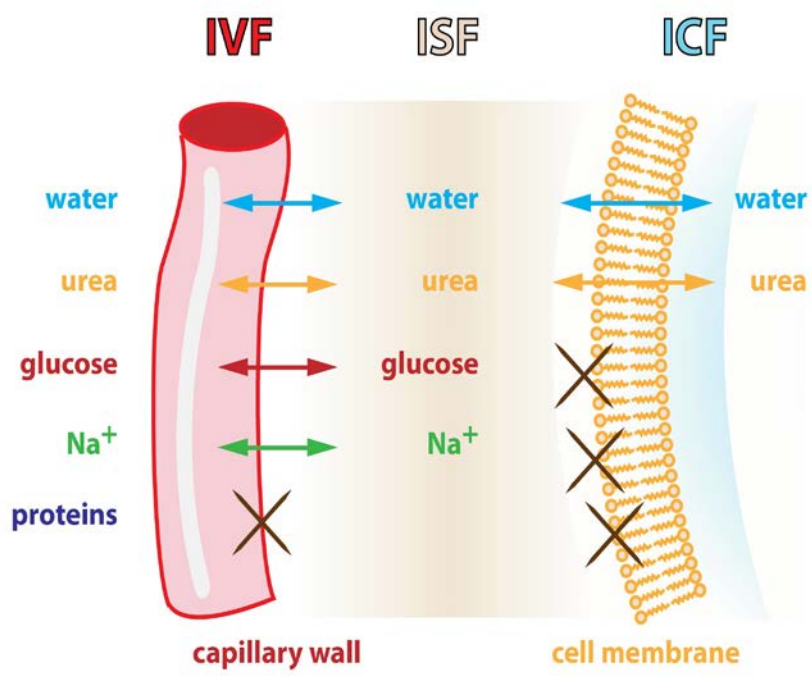
Sliding scales/Infusion pumps

Insulin



Hyperglycemia – Acute or chronic: either way its bad…

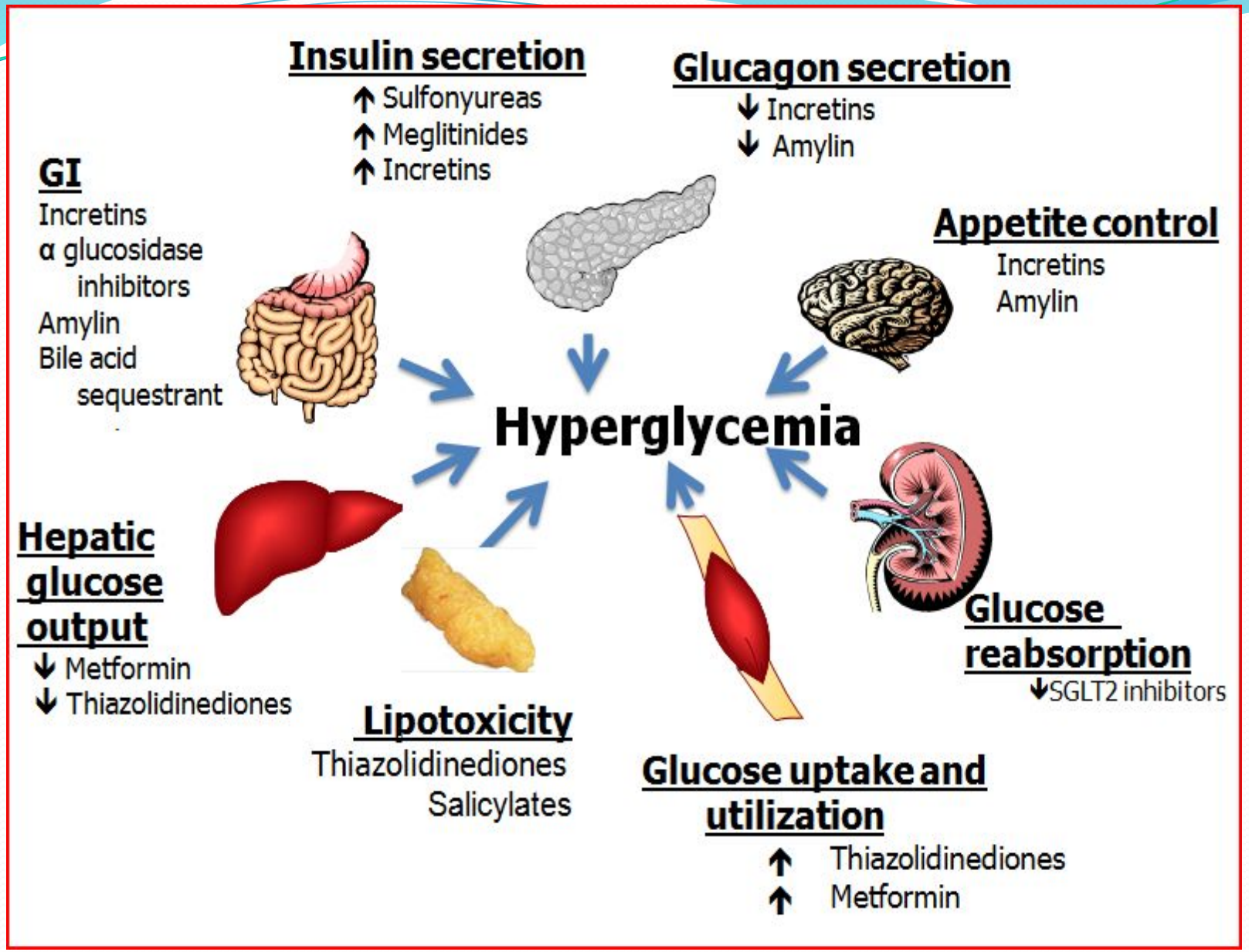
- Present in all types of DM and is a main factor leading to long term complications now.



- End organ damage
  - Neuropathy(osmol)
  - Renal disease
  - Retinal disease
  - Chronic “stressor”
    - (platelet activation)
    - Catecholamines

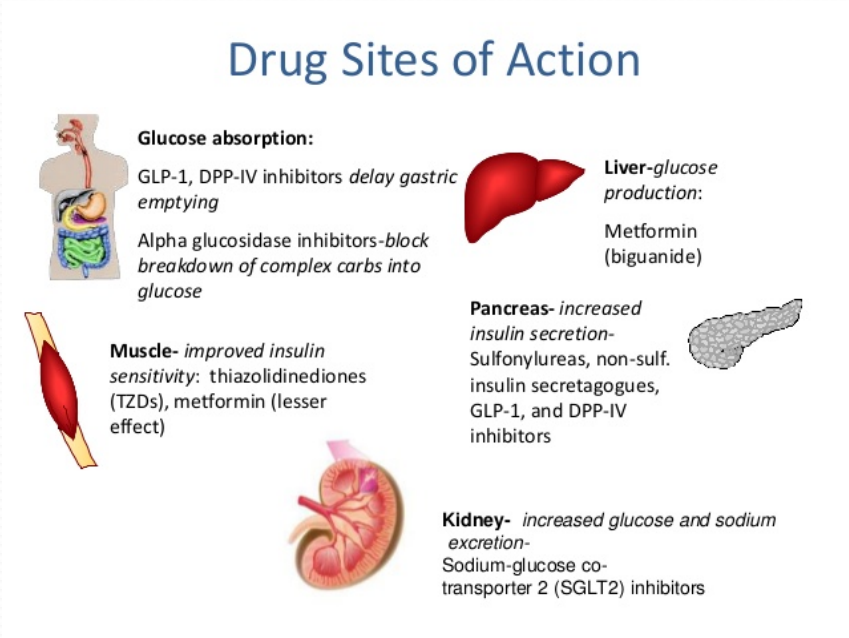
Type II Diabetes:

- ~80-90% of all Diabetic cases
  - failure of circulating insulin to act on various tissues – INSULIN RESISTANCE
  - a "relative" insulin deficiency
    - Insulin may or may not part of treatment at first
  - Usually diagnosed after 40 years old (Ha!)


Insulin secretion
↑ Sulfonyureas
↑ Meglitinides
↑ Incretins
Glucagon secretion
↓ Incretins
↓ Amylin
GI
Incretins
α glucosidase inhibitors
Amylin
Bile acid sequestrant
Appetite control
Incretins
Amylin
Hyperglycemia
Hepatic glucose output
↓ Metformin
↓ Thiazolidinediones
Glucose reabsorption
↓SGLT2 inhibitors
Lipotoxicity
Thiazolidinediones
Salicylates
Glucose uptake and utilization
↑ Thiazolidinediones
↑ Metformin

Oral Treatment's

- Sulfonylurea
- Metformin
- Invokana

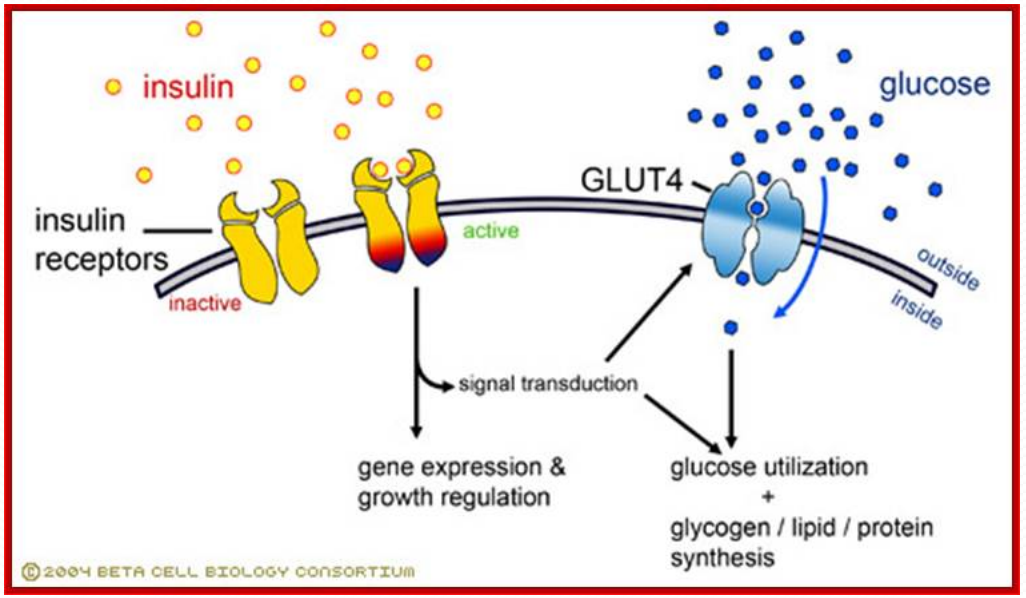


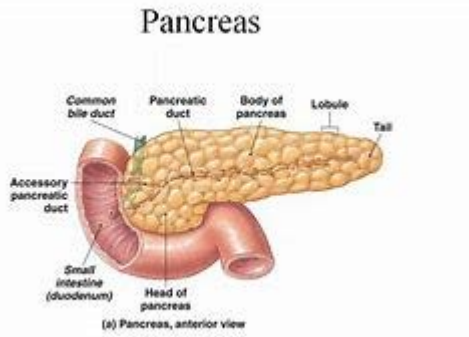
Treatment

- Sulfonylurea

  Wear it out…

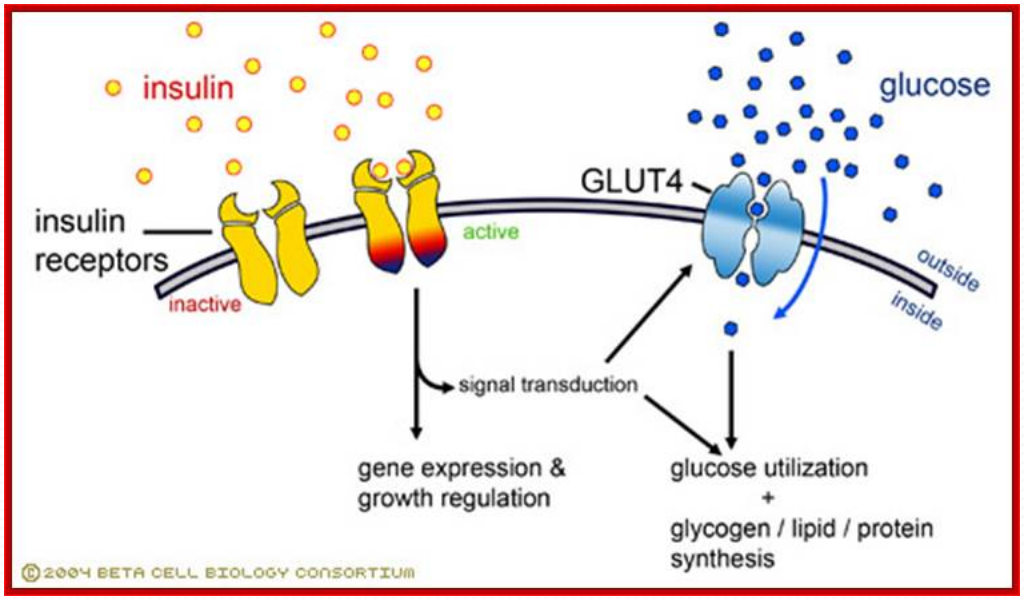
  Hypoglycemia

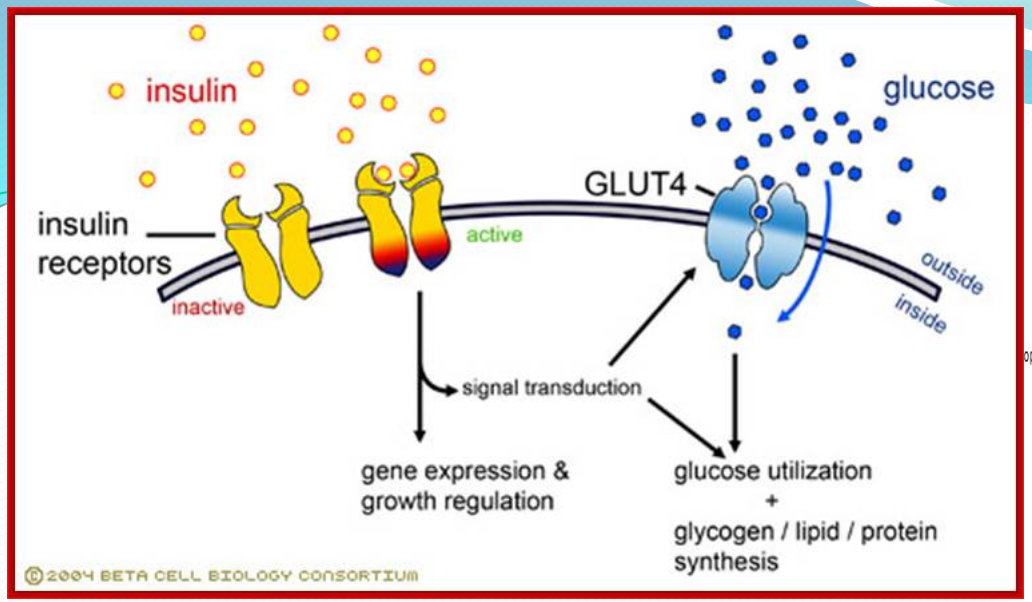




Metformin

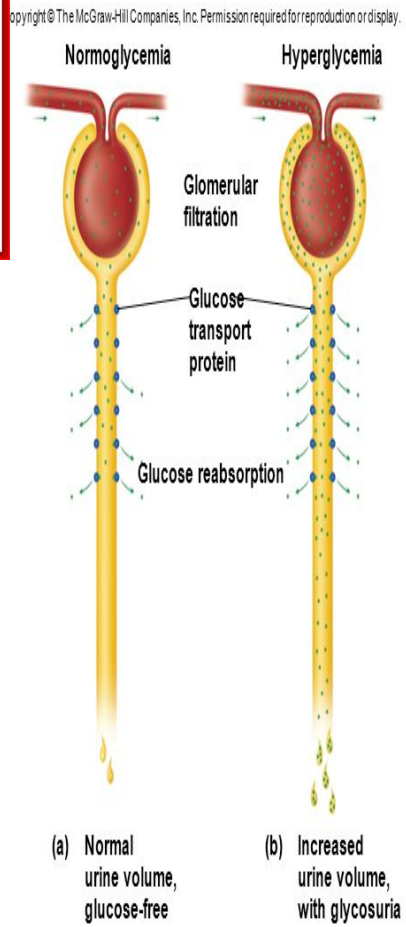
- Lactic Acid





- Invokana
  - Sweet pee…

Transport Maximum

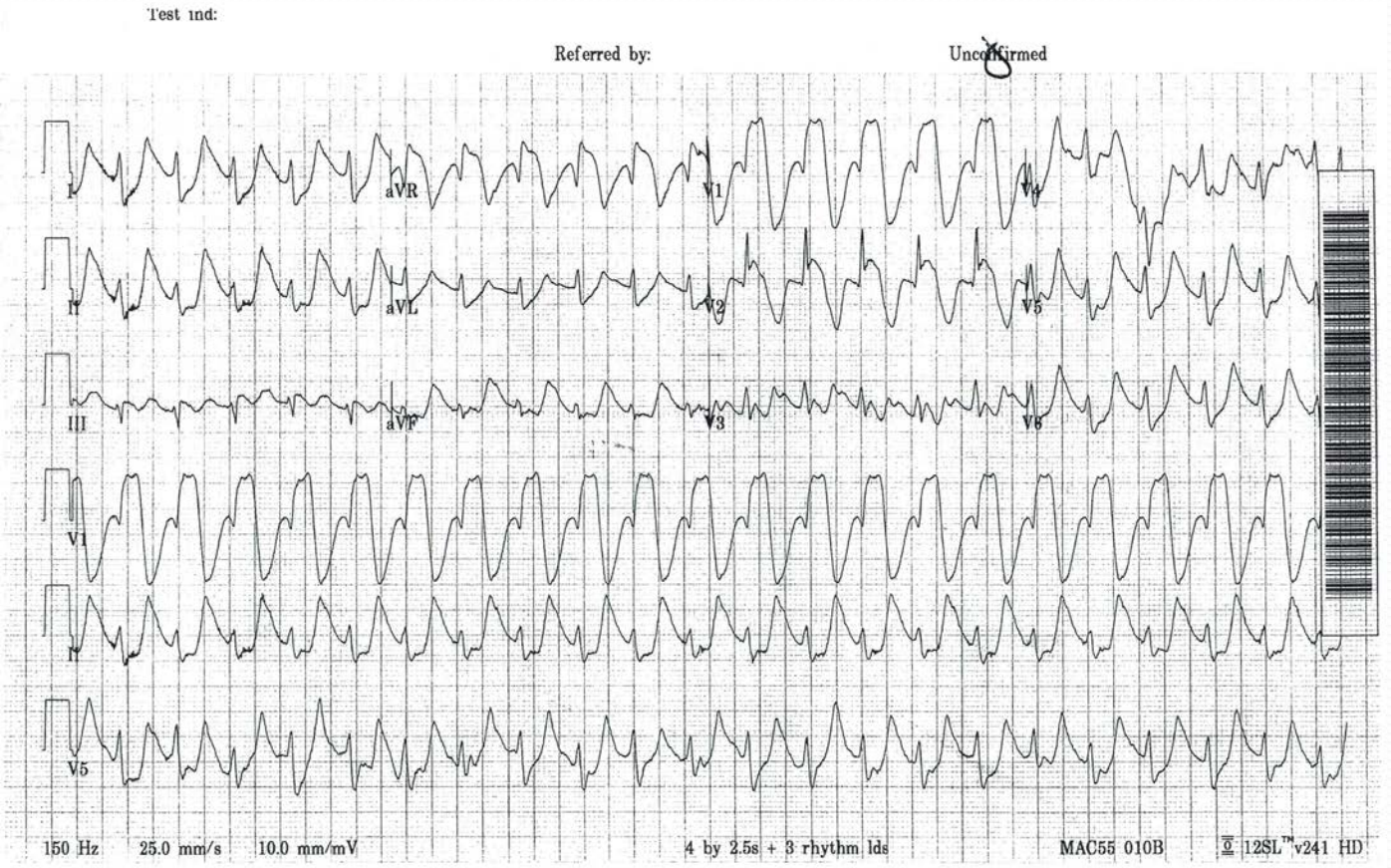


- there is a limit to the amount of solute that the renal tubules can reabsorb
- limited by the **number of transport proteins** in the plasma membrane
- if all transporters are occupied as solute molecules pass, excess solutes appear in urine
- **transport maximum** is reached when transporters are saturated

- Sulfonylurea
  - Hypoglycemia
  - Wear it out
- Metformin
  - Lactic
- Invokana
  - Sweet pee…
  - Hypoglycemia
  - Dehydration with good glucose control

- 50 y/o male call for AMS
- Vital Signs
  - Pulse 124/ resp 34/Bp 95/60, Spo2 96% , end tidal 9, D-stick “Hi”
- Exam
  - Alert, “Ethyl” , dry,

- Altered….
- ? PMH… “sugars”
- Vital Signs
  - HR 124
  - Bp 95/60
  - RR 30 +
  - Sp02 96
  - D-stick “Hi”
  - EtC02- 9



What's up?

DKA

Diabetic Ketoacidosis

- Metabolic derangements in DKA are indirect or direct consequences of the *lack of insulin!* (initial DM diagnosis or med non compliance)
- Insulin insufficiency = inability of glucose to enter cells = hyperglycemia and cellular starvation
- Cellular starvation = release of stress hormones (glucagon) = and increased free fatty acids
- Free fatty acids = ketone bodies = acidosis

- Dehydration
- Acidotic—Anerobic for several reasons
- Hyperkalemia
- Lack of insulin –Infection
- Acute MI/ CVA—Stress response
- Trauma/Surgery—Stress response/dehydration
-
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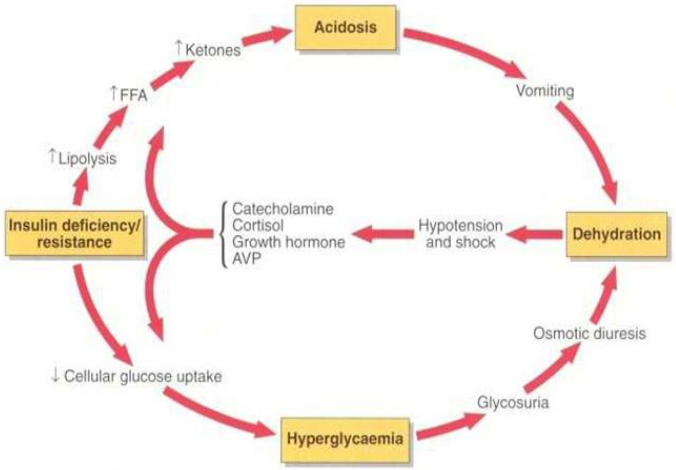
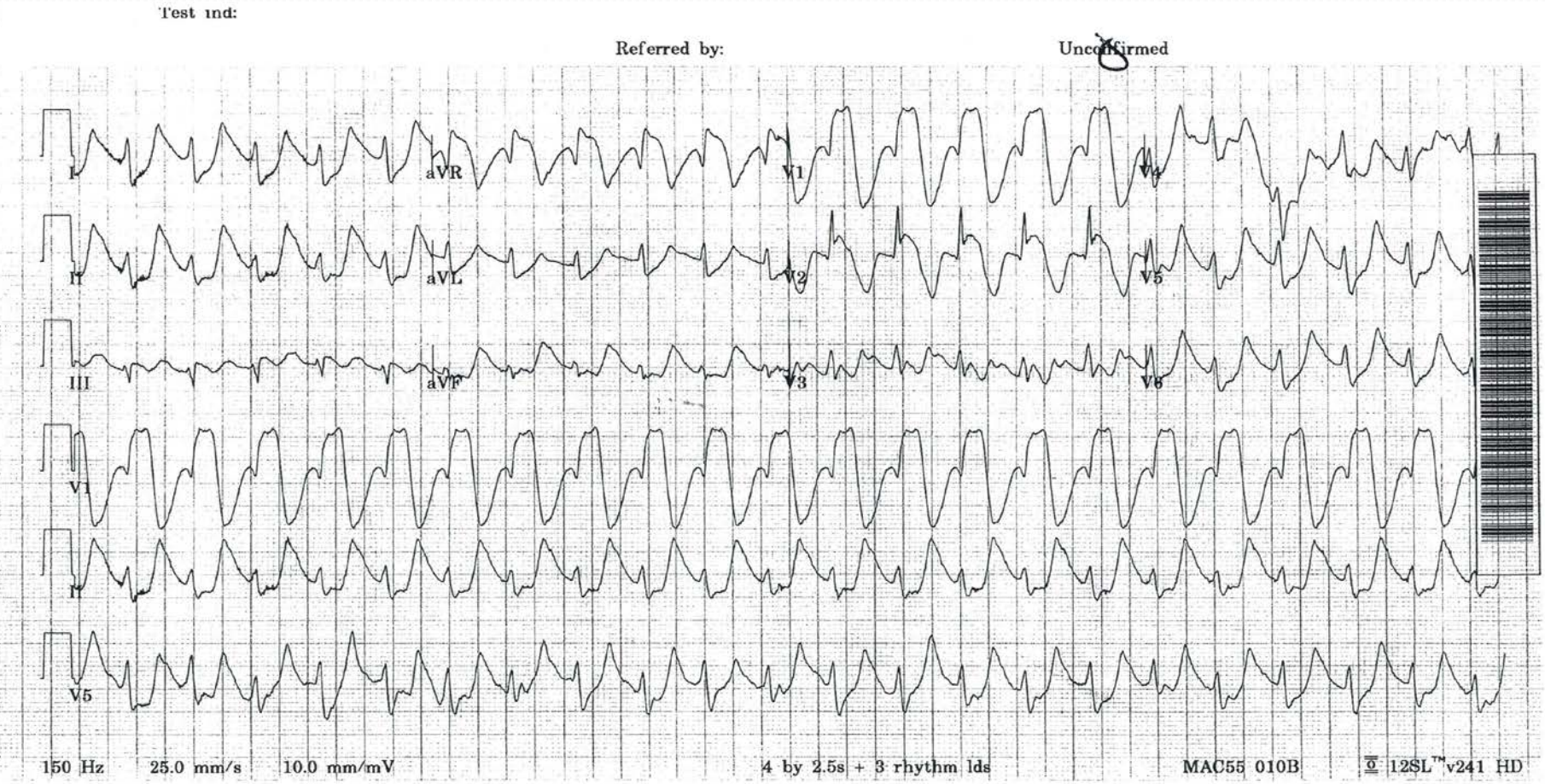
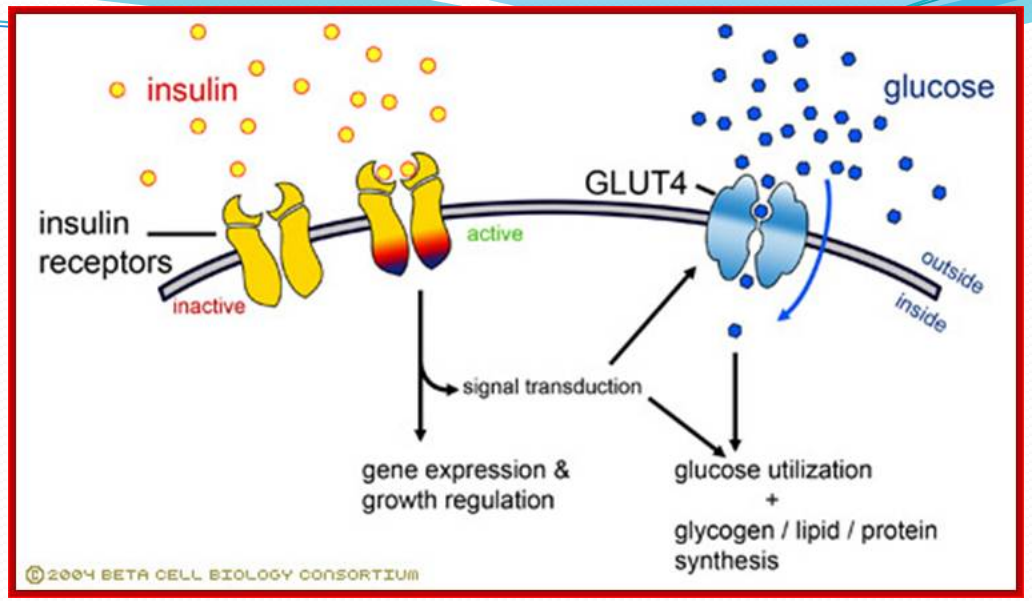


Fig. 1 The development of diabetic ketoacidosis.


Test ind:
Referred by:
Unconfirmed
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
150 Hz
25.0 mm/s
10.0 mm/mV
4 by 2.5s + 3 rhythm lds
MAC55 010B
12SL™v241 HD

Treatment

D stick Usually > 250 (higher)

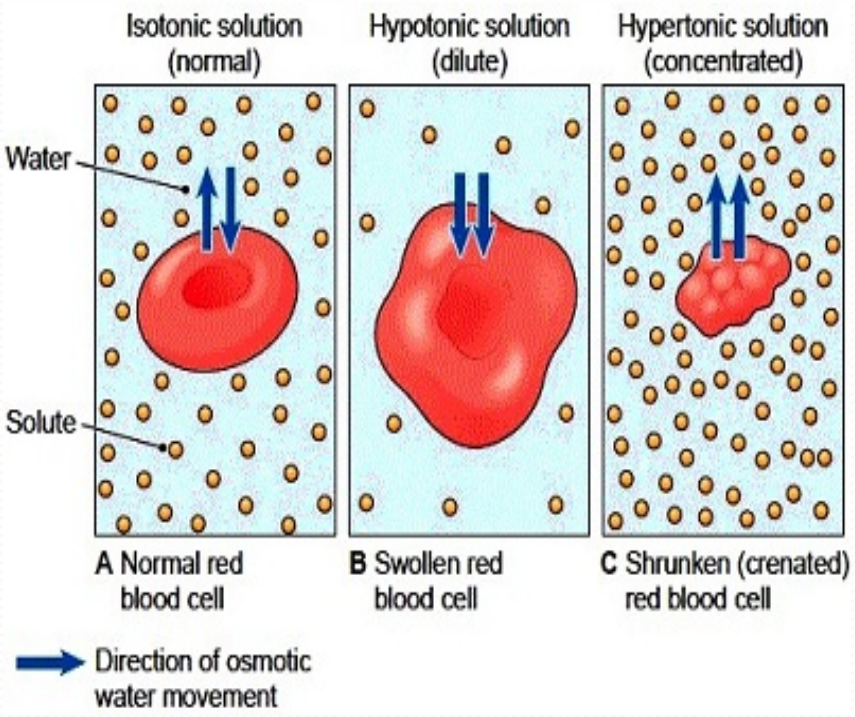


- **IVF**
- Calcium
- Insulin !!!!
- Sodium Bicarb
- Did I say fluids…
- Respiratory control ?
  - These patient's usually have large volume deficits

- What can you do in the field?
- Can u diagnosis this ? Yep…

HHS/ HONK

- Hyperosmolar Hyperglycemic State
- Characterized by progressive hyperglycemia and hyperosmolarity typically found in a debilitated patient with poorly controlled or undiagnosed type II diabetes
- Development of HHS related to:
  - Insulin resistance or deficiency or BOTH
  - Increased hepatic gluconeogenesis and glycogenolysis
  - Osmotic diuresis and dehydration

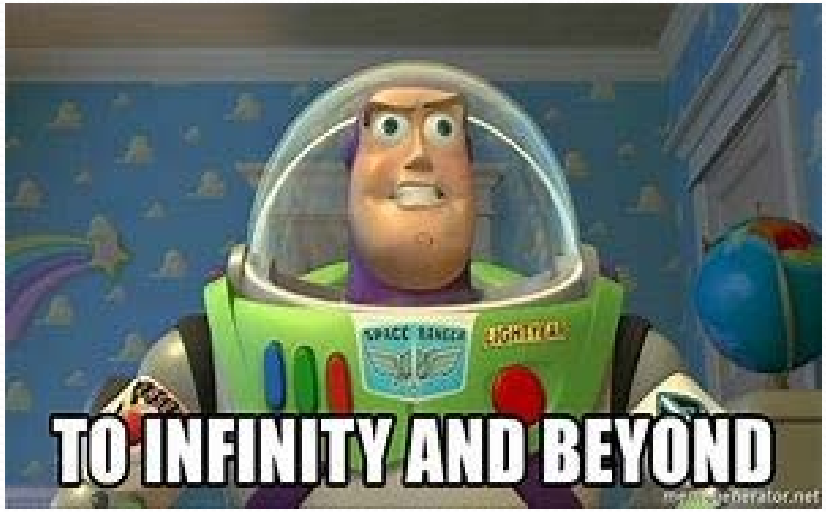


HHS Pathogenesis

- Mild hyperglycemia and stress -> increased insulin resistance -> increased insulin levels ->increased gluconeogenesis and glycogenolysis -> increased glucose without ketones -> increased osmolality -. Increased dehydration and diruesis with sodium depletion
- Key points: Hyperglycemic, Hyperosmolar, NO ACIDOSIS. (***But…***)
- HHS usually develops more slowly – sometimes over weeks

Etiology of HHS

- Infection
- MI
- CVA
- GI Bleed
- Uremia
- Medications
- Trauma
- Pulmonary Embolism
- Heat related illness
- Rhabdomyolysis
- Non compliance with insulin therapy
- New onset diabetes



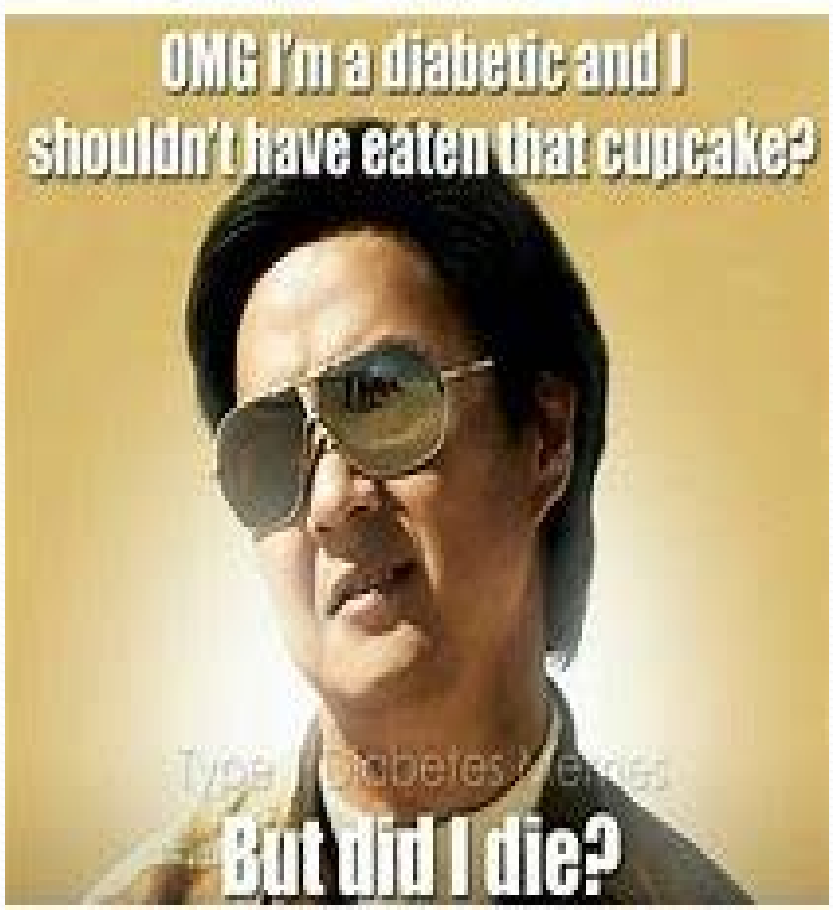
HHS: Clinical Presentation

- Most pronounced and consistent findings in these patients are Neurologic
  - 10% actually present in coma
  - Most have some degree of AMS
  - **Focal Neuro deficits** are ***NOT uncommon***
- Typical presentation is a myriad of vague complaints
  - Weakness, fatigue, dehydration, anorexia, blurry vision, exacerbation of co morbid disease
- Sings of dehydration are usually clinically apparent
  - Tachycardia, postural hypotension, dry membranes,

HHS Initial Evaluation and Management

- D stick …diagnose it… HR/RR/Capnography
- As with DKA, let your history guide you as to potential cause….
- ABC's!
  - These patients most often have some degree of neurological manifestation – even coma!
- Fluid bolus
  - The average fluid deficit in these patients is 8-12 L

Hyperglycemia



- Acute hyperglycemia defined as BG greater than 300
- Absence of ketosis
- Without labs this can be difficult to identify but let your clinical picture guide you
  - Sick/ Not sick, end tidal
- Can present in several ways!
- Lab abnormality in an otherwise asymptomatic patient
- Can be secondary to poor DM management or initial presentation of DM or ATP(acute Twinkie poisoning)

Hyperglycemic Spectrum: Take home points

- A good history can go a long way
- Prompt diagnosis of some eliciting causes is vital to save lives – EKG!
- IV fluids/ IV Fluids / IV Fluids
- In the intubated DKA patient – hyperventilate

  (That's the only patient population to hyperventilate)

  D-stick <"high" and not sick-hyperglycemia

  D-stick > "high" and not sick- HONK

  D-stick > 250 ish and sick, Increased HR/RR ->DKA

Hypoglycemia

- Clinical Definition:
  - Symptoms consistent with diagnosis
  - Low glucose level
  - Symptoms resolve with glucose administration
- Some estimate that over 99% of hypoglycemic episodes occur in diabetic patients on insulin therapy / Sulfonylureas
- Although there is individual variation, signs and symptoms of severe hypoglycemia do not usually occur until the BG is less than 50: (ha)
  - ***Diaphoresis***
  - ***Tremulousness***
  - ***Tachycardia***
  - ***Altered sensorium***
  - ***Focal neurological deficits***
  - ***Seizures***

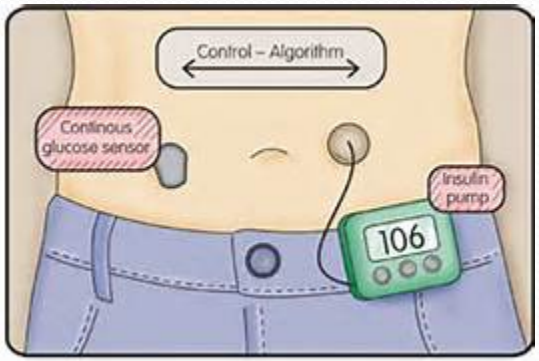
Case

34 y/o female, HX DM , has Insulin pump, 55 Kg

HR 94, BP 112/68, spo2 96%, glucose “low”

diaphoretic , non verbal, can’t get IV access

What you do?



Hypoglycemia: Evaluation and management

- D stick
- Obtain a history if able
- If patient is awake and alert – they can eat
- IV glucose is the most effective treatment(is there too much ?)
- IM glucagon can be used when IV access is not readily available and / or if the patient is **not** elderly or alcoholic (decreased glycogen stores)
- rectal meds ?

Hypoglycemia: Some take home points

- Obtain a history!
- Hypoglycemia is usually a complication of treatment!
  - Most frequently insulin and sulfonylurea's
- ***Patients who are hypoglycemic due to sulfonylurea medications (glipizide, glyburide) they may rebound after treatment! (Like Narcan)***
  - Push these patients for transfer
  - They can PRT but document your discussion
- Remember there is both long and short acting insulin
  - Ask the patient what they took! ***Look it up….***
- If the patient is awake, oral glucose is fine
- Remember – *hypoglycemia is one of the H's and T's*

Bonus points….

- 84 y/o abd pain, hx of HTN, but not DM :)
- Glucose 400, Resp Rate 32,spo2 98, end tidal 9
- BP 90/60 and HR 116
- EKG with prolonged PR and peaked t waves, wide QRS…

Case: Confusion

What's going on?

What you do ?

- 84 y/o abd pain, hx of HTN, but not DM :)
- Glucose 400, Resp Rate 32,sp02 98, end tidal 9
- BP 90/60 and HR 116
- EKG with prolonged PR and peaked t waves, wide QRS…

Diabetic Mimics

- Sick people get naked…and a complete exam….more history
  - Abd exam tight, tender…recent "colon infection " now with bowel perforation -->
    - Stress response
    - Septic
    - Needs the OR…
  - But our initial treatment is still OK !!!!
    - Unless we miss the belly exam…

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