Diabetes

SWEET SIPHON VS SIPHON LACKING FLAVOUR

Diabetes – two completely different problems

- Common symptom of diabetes is peeing a lot

Normal blood sugar level is 3.6-6.2

- Diabetes Mellitus ("sweet siphon") – glucose in urine
  - Type 1 / IDDM / Juvenile onset
    - Autoimmune destruction of beta cells
  - Type 2 / NIDDM / Adult onset
    - Inability to produce enough insulin / respond to insulin to regulate blood glucose

Insulin Dependant Diabetes Mellitus vs Non-IDDM

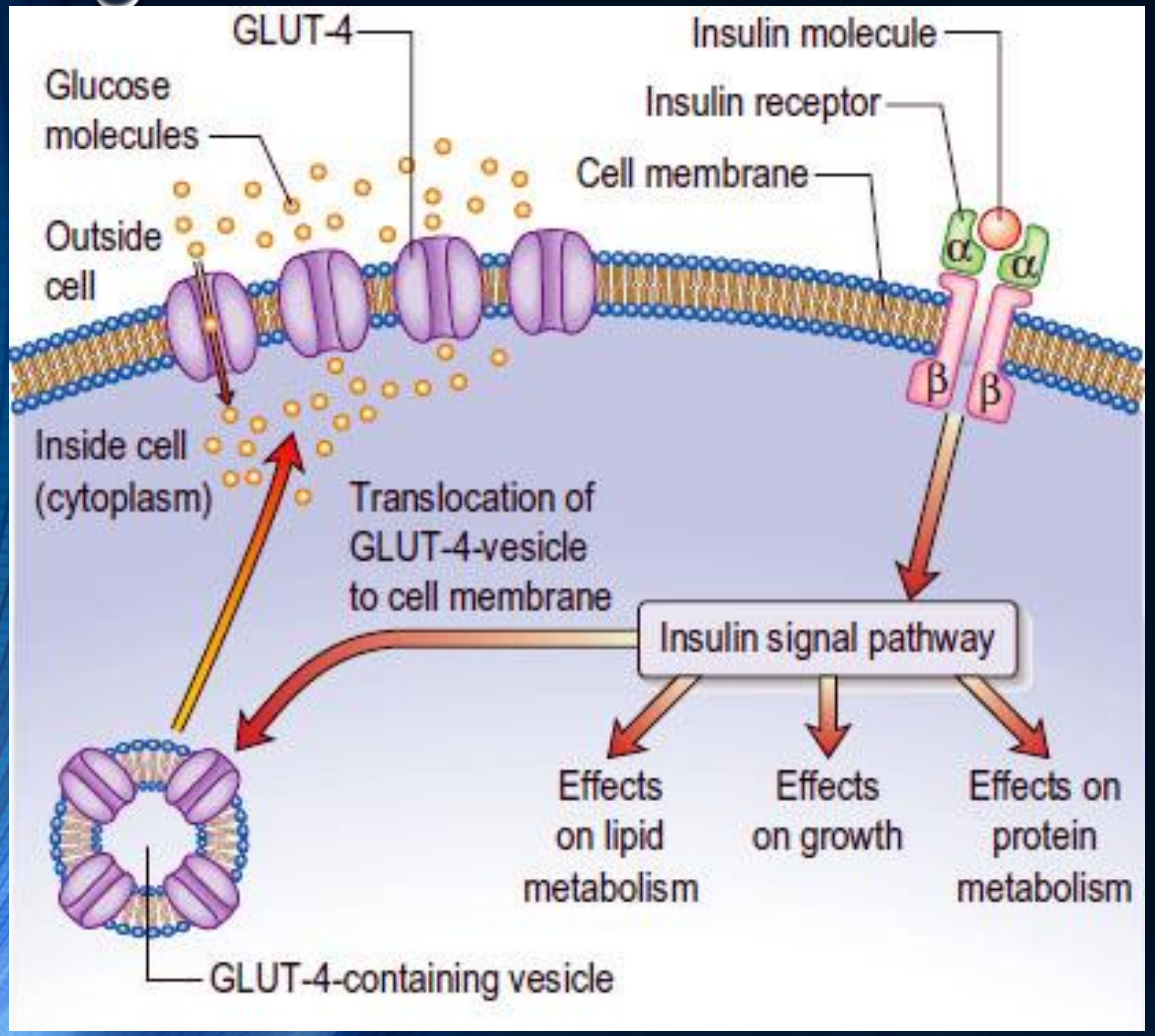
- Diabetes insipidus ("siphon lacking flavor") – dilute, colourless urine
  - Inability to produce or respond to ADH

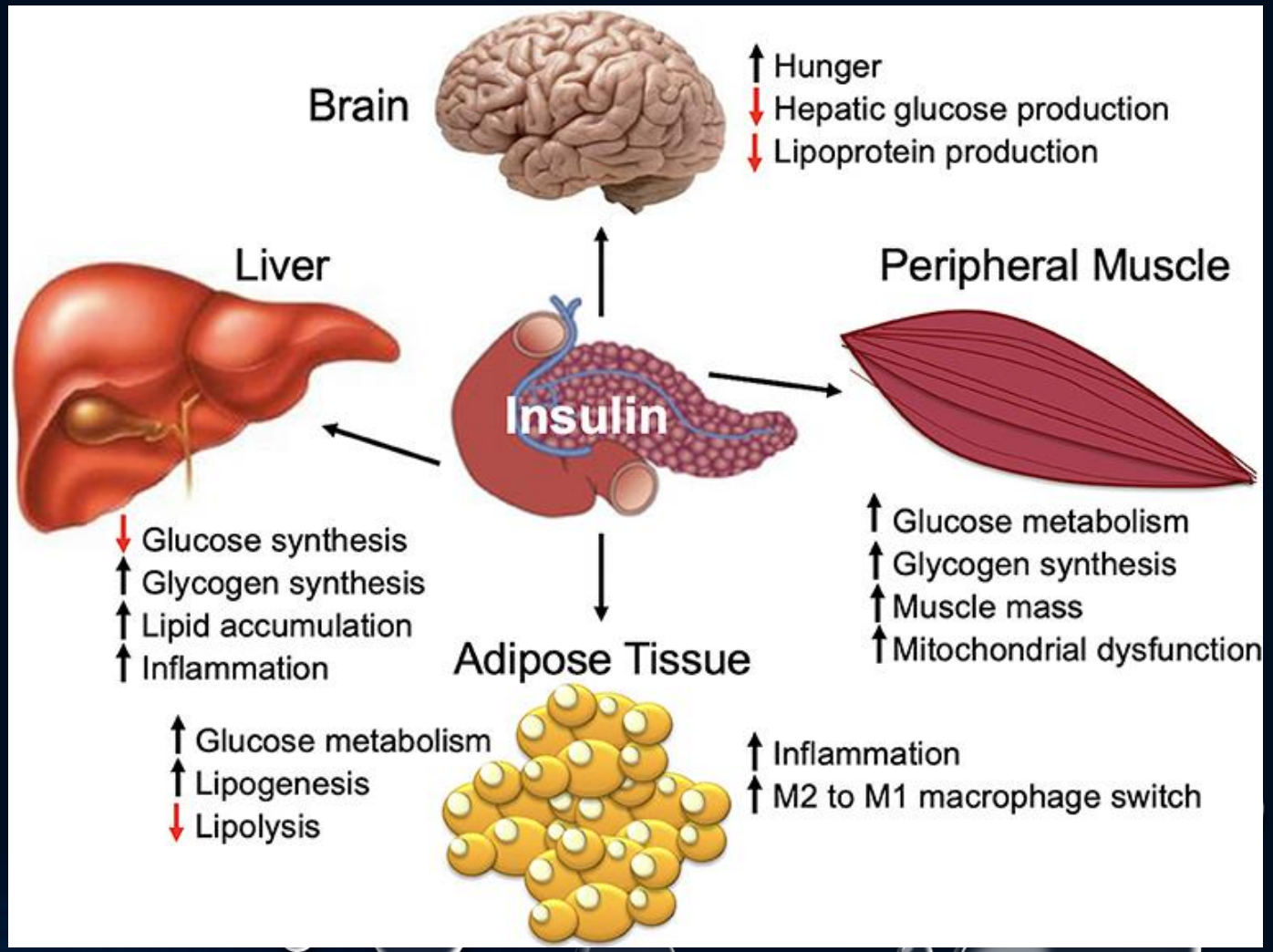
Diabetes

INSULIN DEPENDENT DIABETES MELLITUS (TYPE I)

Lowers sugar by allowing it to enter cells from blood

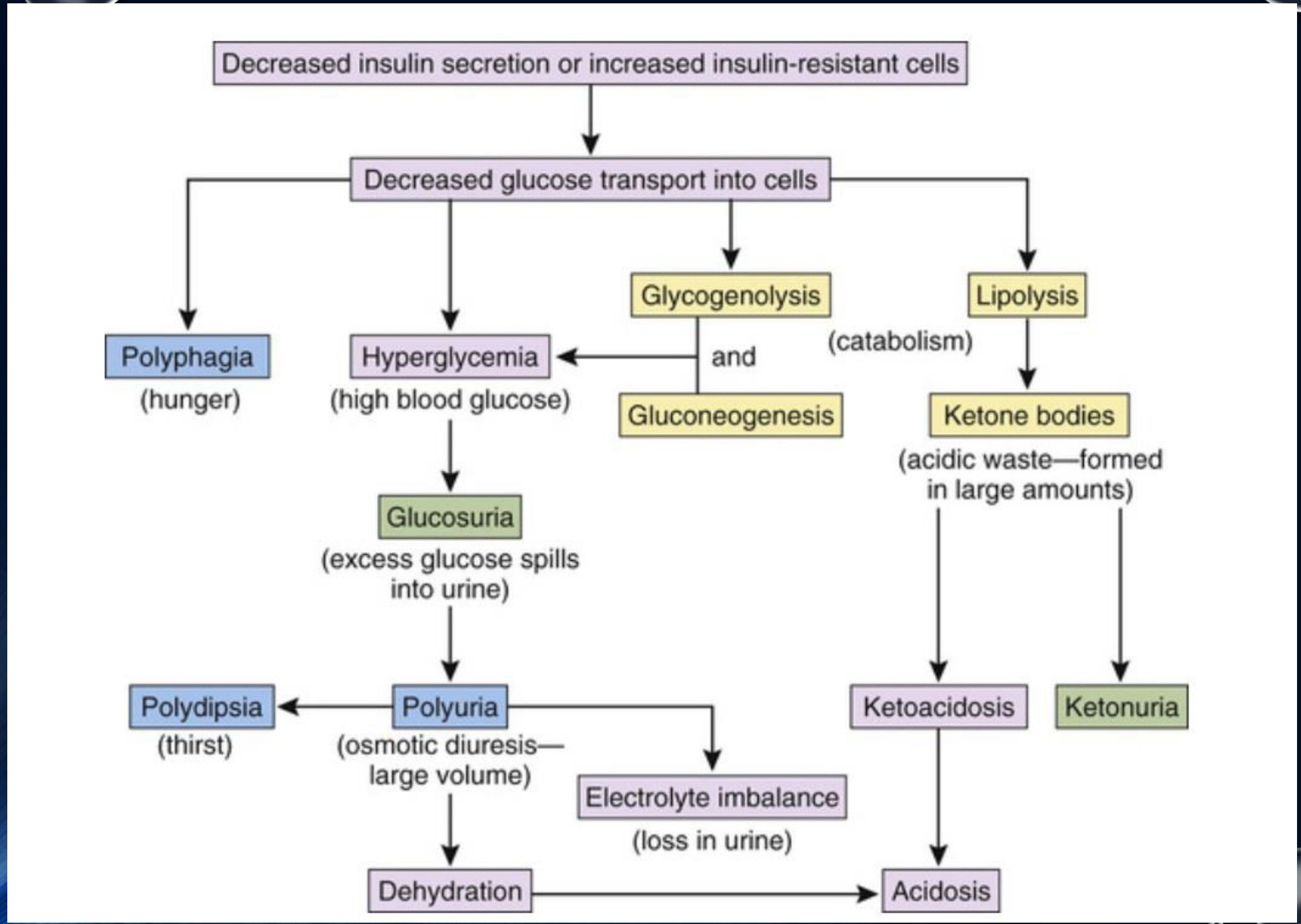
Insulin Effects





Diabetes Mellitus – IDDM

- Etiology
  - Autoimmune destruction of pancreatic beta islet cells, most common in Caucasian pops
- Pathophysiology
  - As blood glucose increases, insulin is not secreted in response to this signal
  - Thus blood glucose remains higher than normal → hyperglycemia
  - Consequences
    - Glucose is freely filtered by kidneys but reabsorption is limited by tubular maximum thus more glucose in urine → glycosuria
    - Glucose has osmotic effect and limits reabsorption of water → polyuria
    - Excessive H_2O loss causes dehydration → increased ADH secretion → polydipsia
    - Nutrients are not stored by cells in same way without insulin → polyphagia

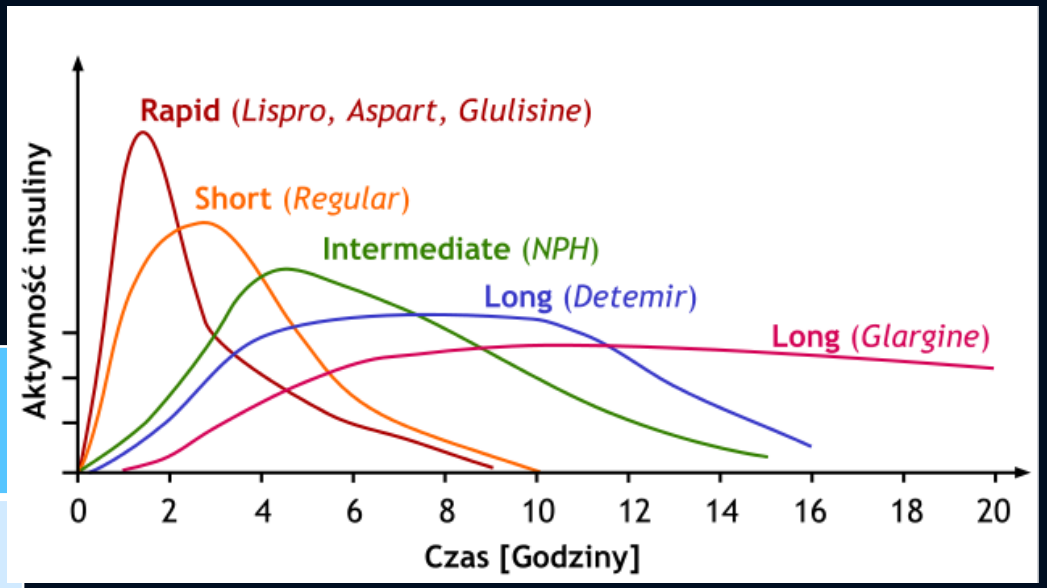

Decreased insulin secretion or increased insulin-resistant cells
Decreased glucose transport into cells
Glycogenolysis
Lipolysis
Polyphagia
(hunger)
Hyperglycemia
(high blood glucose)
and
(catabolism)
Gluconeogenesis
Ketone bodies
(acidic waste—formed in large amounts)
Glucosuria
(excess glucose spills into urine)
Polydipsia
(thirst)
Polyuria
(osmotic diuresis—large volume)
Electrolyte imbalance
(loss in urine)
Ketoacidosis
Ketonuria
Dehydration
Acidosis

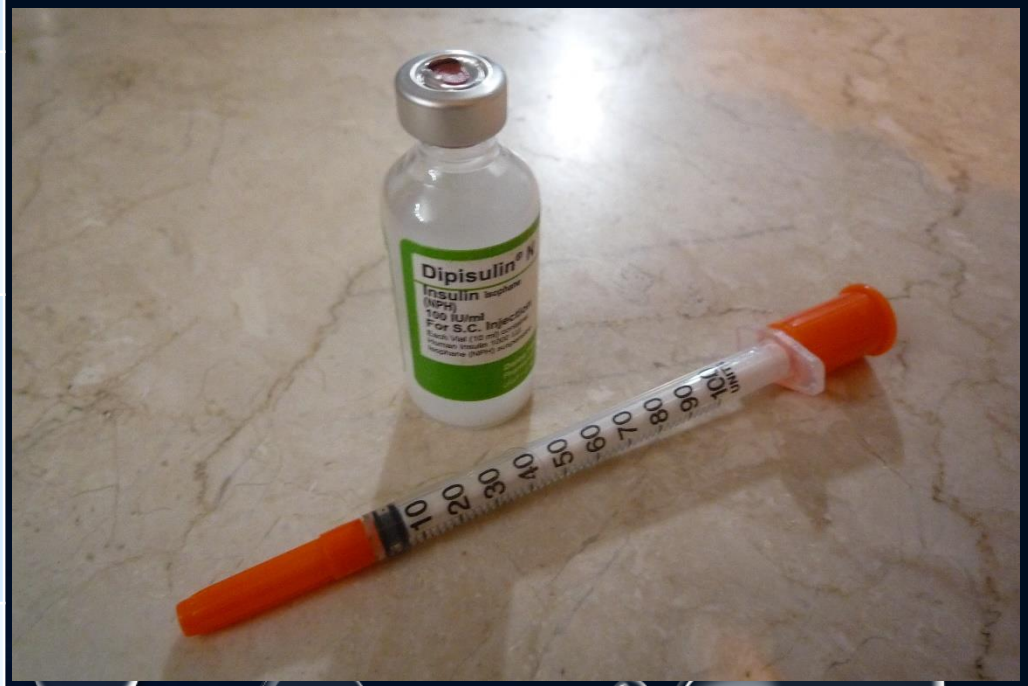
Diabetes Mellitus – IDDM

- Clinical Manifestations
  - Hyperglycemia (>11 mmol/L; normal is <6.2mmo/L)
  - Polydipsia, polyphagia, polyuria (glucosuria)
  - Patients tend to be thin despite eating lots
  - Usually diagnosed around age 14 though signs can be present much earlier
  - Patients may be fatigued, weak, irritable
  - Blurred vision
- Treatment
  - Insulin replacement therapy

Insulin Formulations

Type	onset	Peak	Duration
Rapid – lispro	15 min	30 – 60 mins	< 5 hours
Short – regular (clear)	60 min	2 – 3 hours	5 – 7 hours
Intermediate – isophane / NPH	1 – 4 hours	8 – 12 hours	18 -24 hours
Long – zinc	4 – 8 hours	18 – 24 hours	36 hours





Diabetes

NON INSULIN DEPENDENT DIABETES MELLITUS (TYPE II)

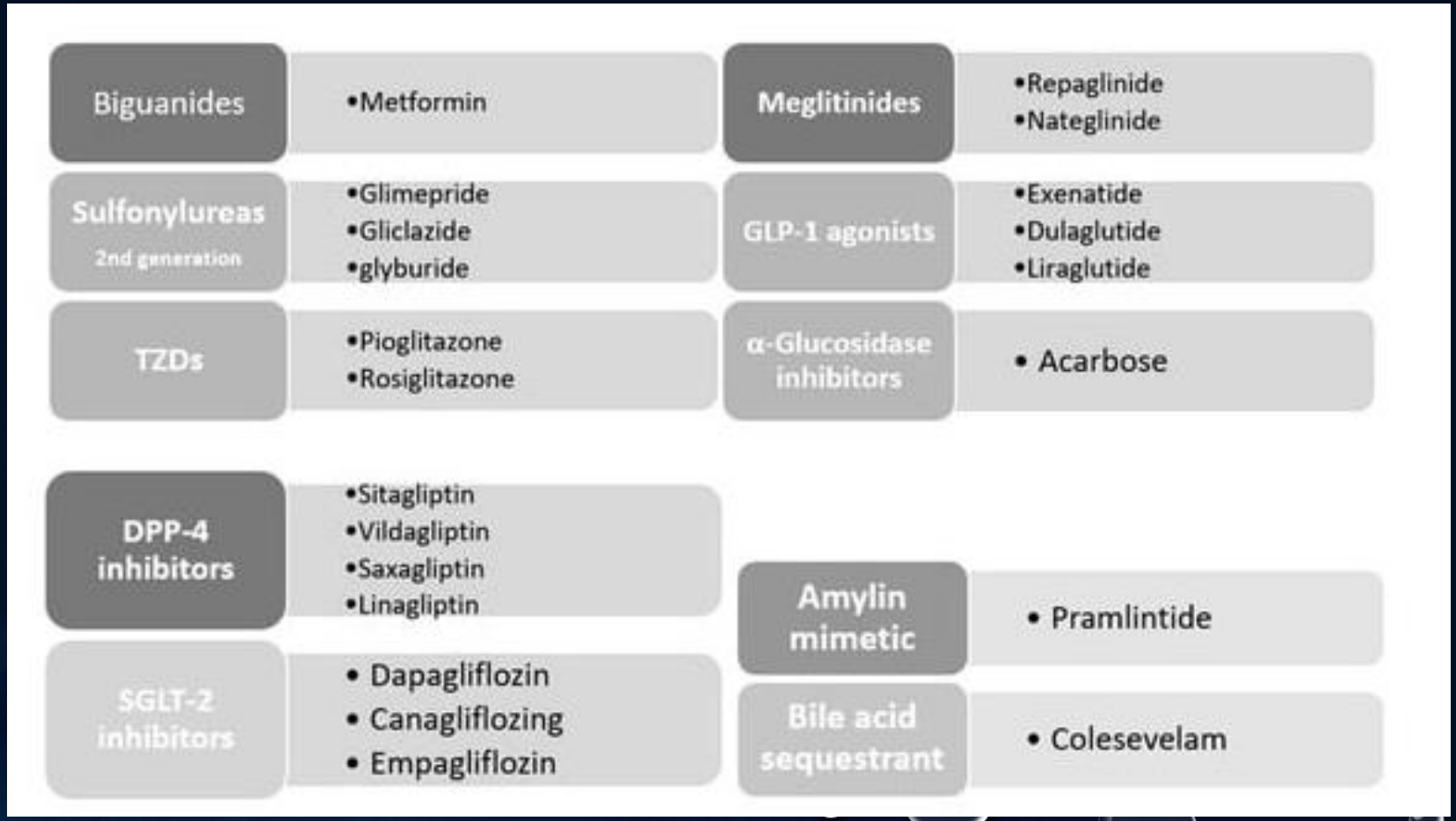
Diabetes Mellitus – NIDDM

- Etiology
  - Skeletal muscle cells, adipocytes and liver cells become less responsive to insulin
  - Pancreas does not secrete enough insulin to adequately reduce blood sugar
  - Cause is unknown but many risk factors
    - Aging (adult onset), sedentary lifestyle, obesity, CHO / lipid rich diets, genetic components
- Pathophysiology
  - As blood glucose increases, insulin is secreted but cannot adequately reduce blood glucose → hyperglycemia (does take longer to reach this level than in IDDM)
  - reduced insulin sometimes causes increased glucagon secretion (lack of inhibition)
  - Consequences
    - Hyperglycemia, glycosuria, polyuria develop for same reasons as IDDM but may be more insidious
    - Polydipsia takes time to manifest

Diabetes Mellitus – NIDDM

- Clinical Manifestations
  - Similar to IDDM but may take longer to develop than in IDDM
  - As obesity is a risk factor, patients tend to be heavier
  - Adult onset used to mean later adulthood but condition is becoming more common in younger individuals
- Treatment – three layers
  - Regular exercise and healthy diet
  - Oral hypoglycemic meds
    - Drugs that increase insulin secretion (incretins, sulfonylureas, meglitinides)
    - Drugs that increase receptor sensitivity (biguanides, glitazones)
    - Drugs that reduce CHO absorption (alpha glucosidase inhibitors)
    - Drugs that promote satiety (amylin, pramlintide, sibutramine)
  - Insulin

Common Oral Hypoglycemic Medication



Diabetes

COMPLICATIONS HYPOGLYCEMIA

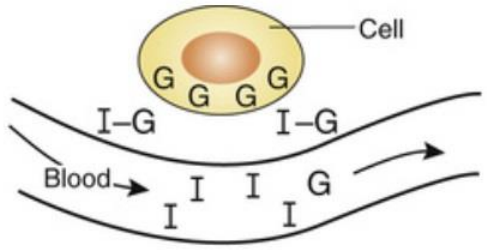
figure out reasons why diabetics can become hypoglycemic

Hypoglycemia

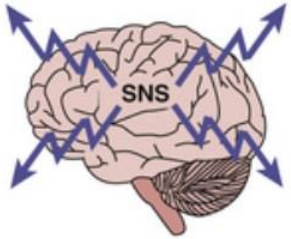
neonates suck at storing sugar, so without constant sugar intake, they become hypoglycemic

- What if the patient takes too much insulin (or oral hypoglycemic medication)?
- Hypoglycemia
  - Tachycardia, diaphoresis, headache, shakiness, irritability, anxiety, drowsiness
- Treatment
  - Source of glucose – OJ, chocolate bar etc
  - Exogenous – Glucagon, Dextrose solution (iv)

Insulin-induced Hypoglycemia



1. Excess insulin in blood
2. Increased transport of glucose into cells
3. Hypoglycemia
   Decreased CNS function
4. Stimulates SNS
5. Increased gluconeogenesis
6. Excess insulin transports glucose into cells



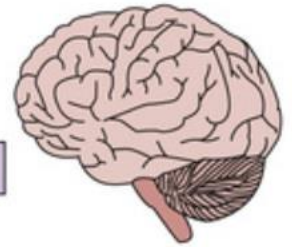
Clinical signs
- weakness, confusion
- pallor
- diaphoresis
- tremors
- tachycardia

6. Excess insulin transports glucose into cells

I = Insulin
G = Glucose

Glucose intake
Return to normal state

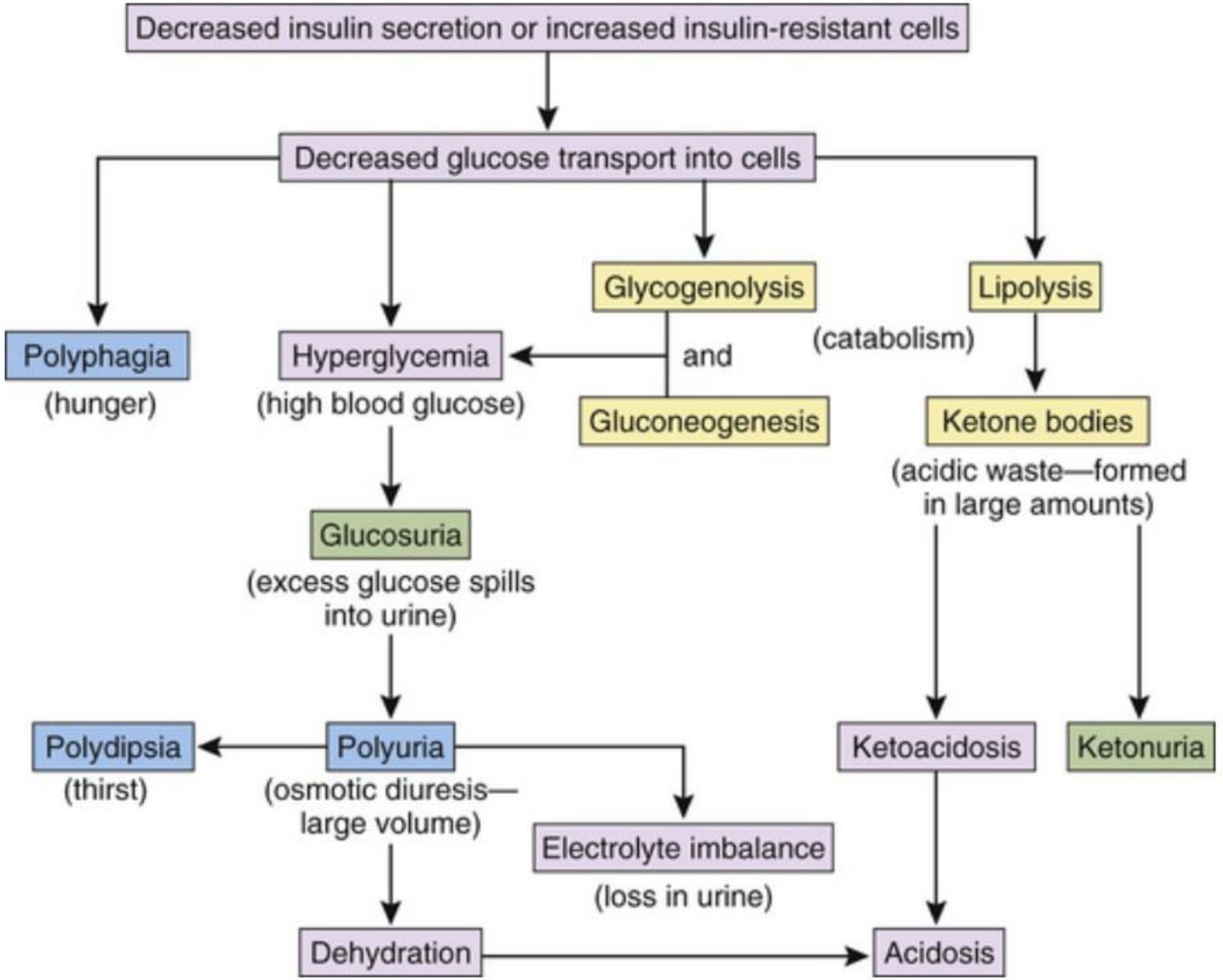
7. No glucose intake
8. Blood glucose levels decrease further
9. Neurons cannot function
10. Coma and death



Diabetes

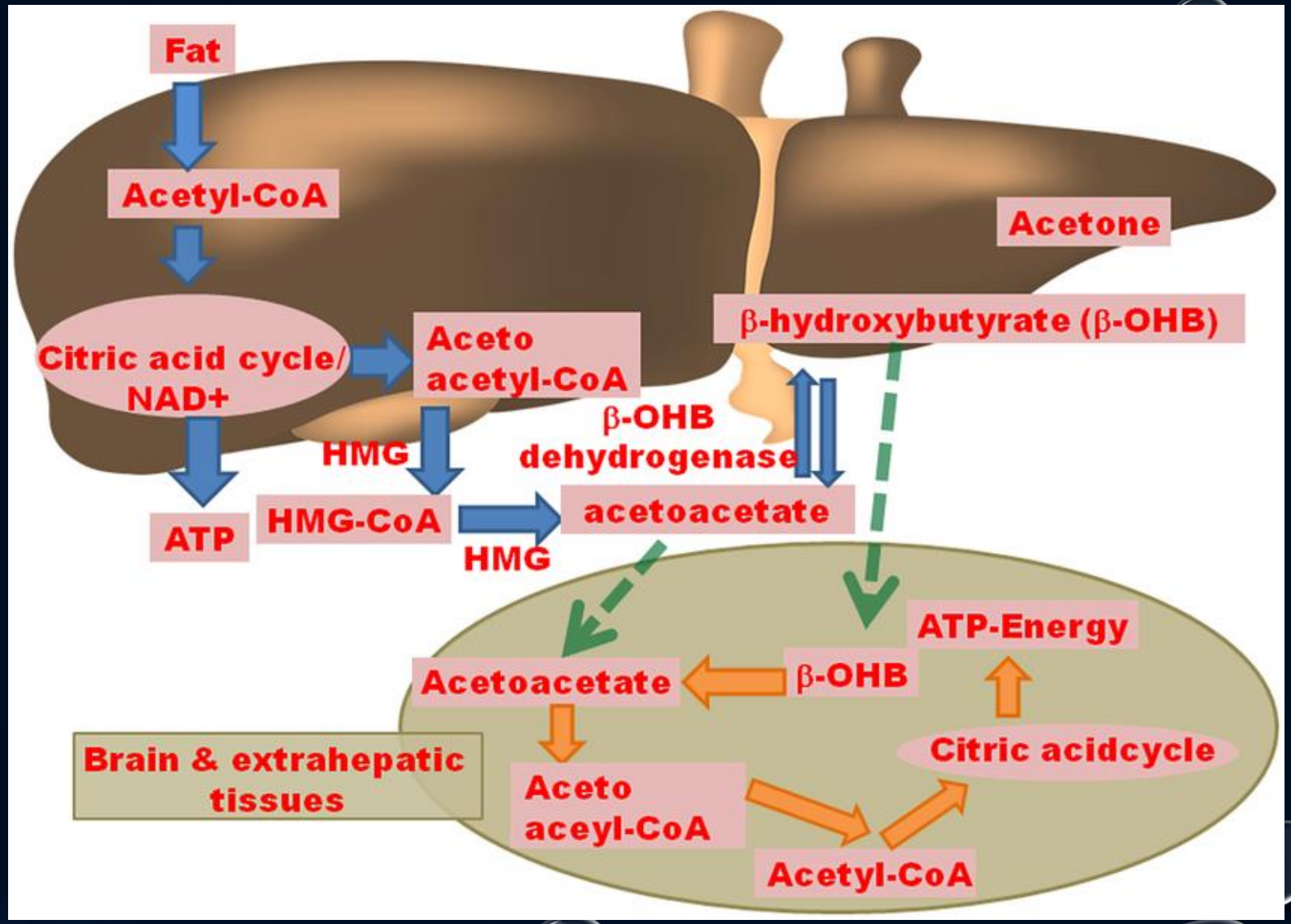
COMPLICATIONS: HYPERGLYCEMIA

RELEVANT
know all these terms



Ketoacid Production

1. Lipolysis (triglycerides broken into free fatty acids
2. Beta-oxidation: fatty acids to Acetyl-CoA in liver mitochondria
3. HMG-CoA Pathway: Acetyl-CoA cannot enter Citric Acid Cycle (limited oxaloacetate) goes into ketogenic pathway. 2 x Acetyl-CoA combine to HMG-CoA
4. Ketone Formation: HMG-CoA cleaved (HMG-CoA lyase) ito acetoacetate (ketoacid)
5. Acetoacetate converted to other two: b-hydroxybutyrate, acetone (excreted breath and urine)



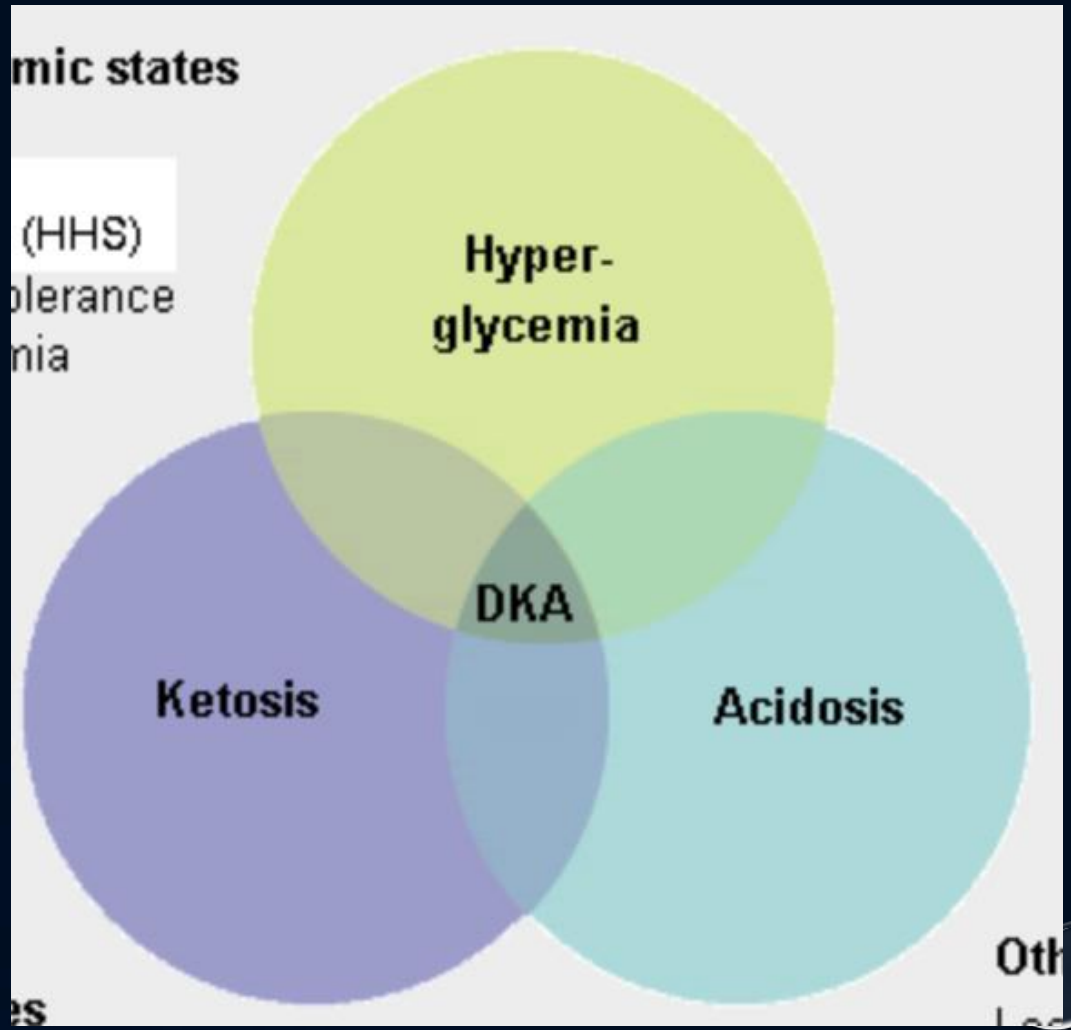
Diabetic KetaoAcidosis (DKA)

Acidosis causes potassium to leak out of cells. So blood potassum levels stay normal, but cells become hypocholemic

Acidosis causes hyperventilation, resulting in Kussmaul Respration

keotacids also cause GI upsets

- Life-threatening condition
  - Without insulin, cells mobilize glycogen stores and then lipid stores
  - FFA circulate in blood then converted to KETO-ACIDs in liver then into blood
  - Limit to cellular absorption of keto-acids so they begin to accumulate in blood
  - Keto-acids cause excess of H+ in blood → metabolic acidosis
    - Transient hyperkalemia (why?)
    - Increased rate and depth of breathing (why?)
    - Hypovolemia
    - Keto-acids (ketone bodies) have fruity smell → fruity smelling breath



Hyperosmolar Hyperglycemic Syndrome (HHS)

- Old Name - Non-ketotic hyperosmolar hyperglycemic syndrome (NKHH)
  - Poor control of glucose and poor hydration leads to increased concentration of glucose in blood
    - Meds like diuretics and glucocorticoids can contribute
    - Infections also increase risk
  - Because some insulin is present, lipolysis and metabolism of lipids for use as an energy source is suppressed → no ketosis
  - Lack of glucose uptake into skeletal muscle cells and adipocytes promotes gluconeogenesis and glycogenolysis → further increased blood glucose
  - Blood glucose can reach as high as 600 mg/dL (normal is < 140 mg/dL)
  - Excessive fluid loss can increase ECF osmolarity to > 320 mosm/L
  - Much higher mortality rate (up to 20%) than DKA (<1%)
    - Without acidosis, it is more difficult to identify compared to DKA
    - Excessive fluid loss decreases BP, causes EL imbalances (why might hypokalemia occur?)

CAD 33.3
normal = 7.8

Diabetes Mellitus – General Chronic Complications

- Vascular complications
  - Macrovascular / macroangiopathy – increased risk of atherosclerosis, stroke
  - Microvascular / microangiopathy
    - Retinopathy – increased risk with aging; progresses to neovascularization which eventually causes fibrosis, retinal detachment and blindness
    - Nephropathy – leads to chronic renal failure (irreversible loss of nephrons) due to glomerulosclerosis (initial increased filtering leading to damage to glomeruli through I / N / F process); hypertension
- Increased risk of infection
  - Poor circulation, increased glucose supports bacteria, poor healing due to reduced insulin
- Neuropathies
  - Autonomic – GI, bladder, tachycardia, postural hypotension
  - Sensory – loss of sensation in feet and lower legs → ulcerations, infections, tissue necrosis

HHS vs DKA

both cause extreme dehydration. Whole brain dehydration leads to seizures and altered LOA

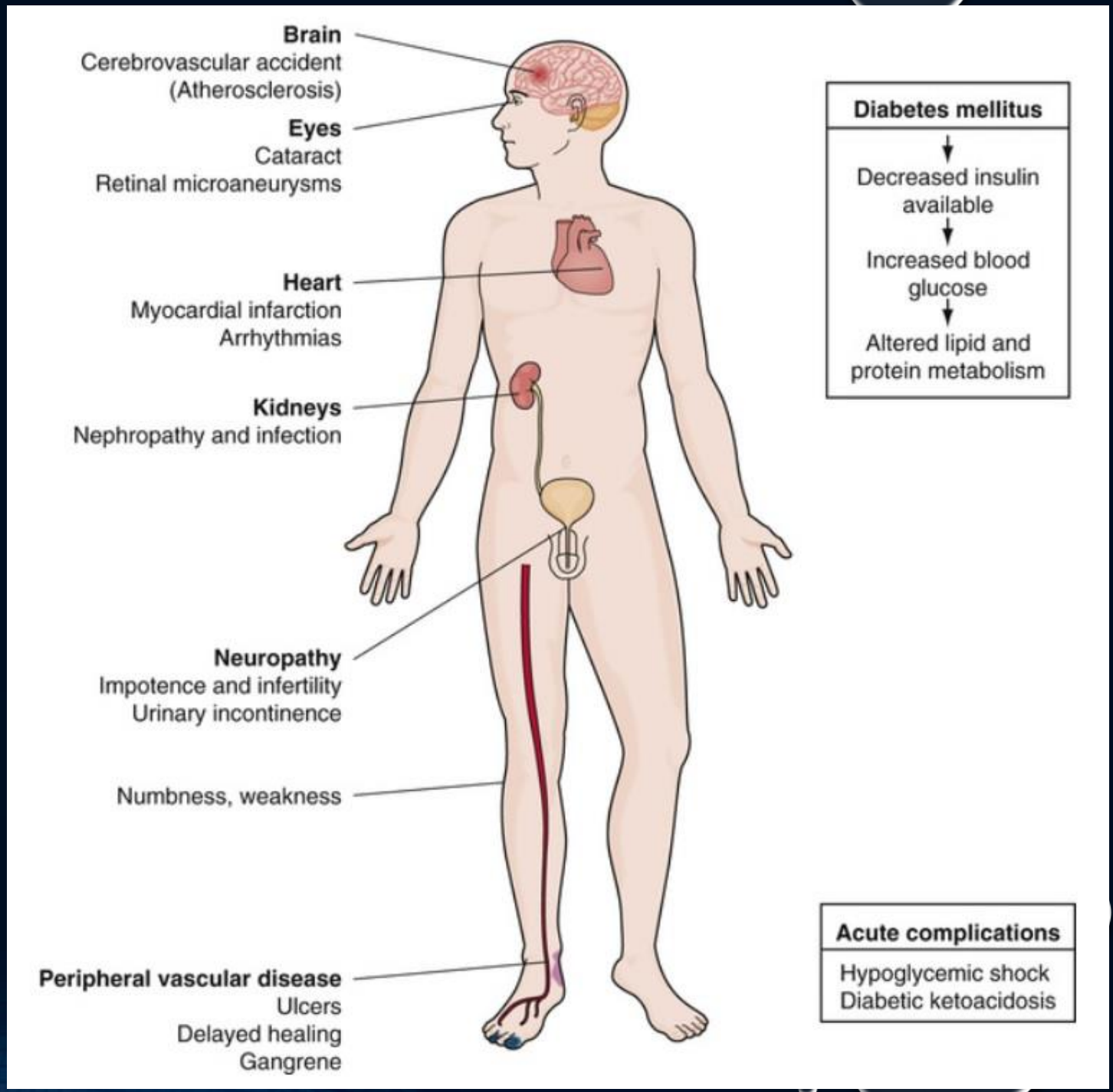
Type 2 more likely for this. why?

Cusmos respirationscC

type 1 more likely for this than HHS

DKA	HHS
Hyperglycemia	Severe hyperglycemia (elevated osmolality)
Metabolic acidosis	pH > 7.3
Ketonemia (elevated beta-hydroxybutyrate)	Minimal or negative ketonemia
Volume depletion	Profound volume depletion
Young > Elderly, T1DM > T2DM Acute presentation	Elderly > Young; T2DM, 20% without hx of T2DM illness
Acute presentation	Longer, protracted course of illness

Diabetes Mellitus – Chronic Complications



Diabetes

DIABETES INSIPIDUS

Not diabetes symptom. Could be added infection

NRB at 10

Scenario - 78F on couch, altered LOA

Apartment
lying on couch
A - patent
B - fast, even, normal WOB
C- fast, weak radial
AVPU - V
Dry skn

T - 38
HR - **130**, sinus tachy
RR - **24**
BP - **82/60**
SPO2 - **91**
GCS - 13
pupils - 3+
BG - **high**
ETCO2 - 30
Clear auscultation

Pmx:
Hypotension
Osteoporosis
Diabetes
Cholesterol
No known allergies

Mx:
Tylenol
Metroprolol
Furosemide
metformin
glyburide
lipitor

Neuro exam:
Normal

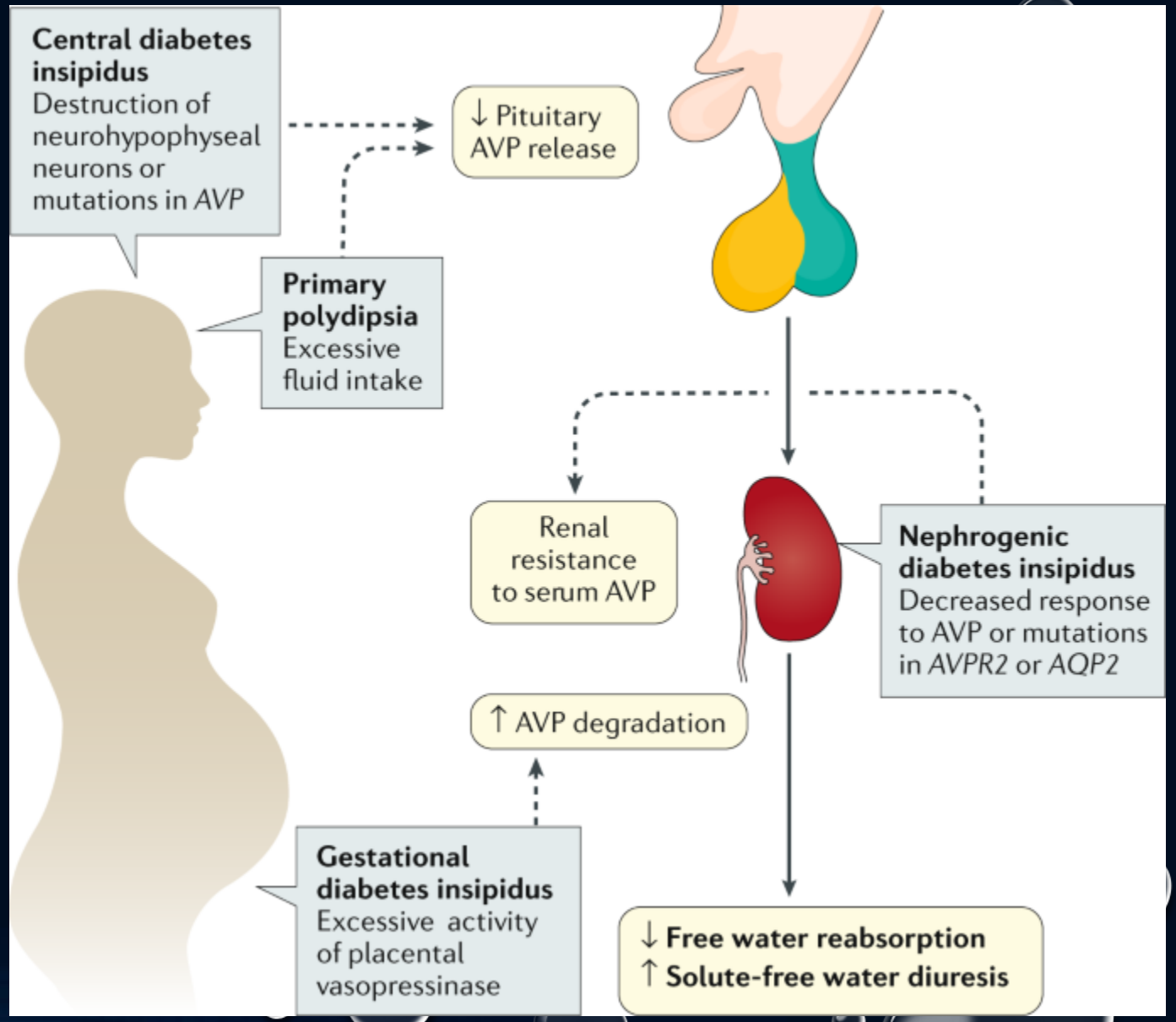
Check feet for open wounds (possible elderly neglect):
Cellulitis (skin infection)

Smell for necrotic tissue, UTI, acetone breath.

Infection developed into Sepsis. Sepsis confused her, caused her to neglect medications. Resulting in unstable BP And BG. Stress caused increase in Blood sugar.

Diabetes Insipidus

- Etiology
  - Central – inability to produce and secrete ADH – idiopathic, tumour, surgery, trauma
  - Nephrogenic – inability to respond to ADH; lack of reabsorption of water
  - Dipsogenic – constant thirst → big water load = lots of pee
  - Gestational – with pregnancy, placenta produces enzyme that metabolizes ADH
- Pathophysiology
  - ADH is responsible for creation of aquaporin channels to facilitate reabsorption of water; without proper ADH function, much fewer aquaporins = decreased water reabsorption



Diabetes Insipidus

- Clinical Manifestations
  - Excessive thirst
  - Excessive peeing (dilute urine) > 3L / day
    - risk of dehydration and EL imbalances, especially hypokalemia (why?)
- Treatment – it depends
  - Central – exogenous ADH / desmopressin
  - Nephrogenic – deal with primary problem, thiazide diuretics can alter Na+ intake which can support more water reabsorption
  - Dipsogenic – not sure
  - Gestational – desmopressin