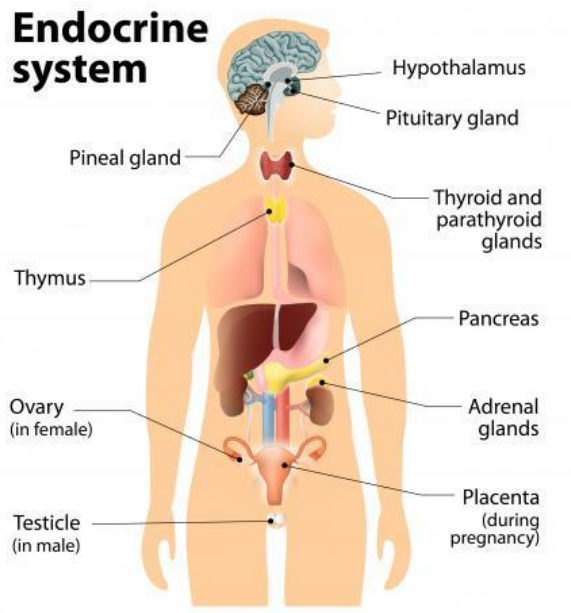


1

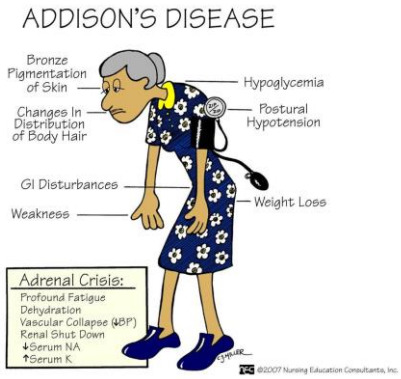
ALDOSTERONE

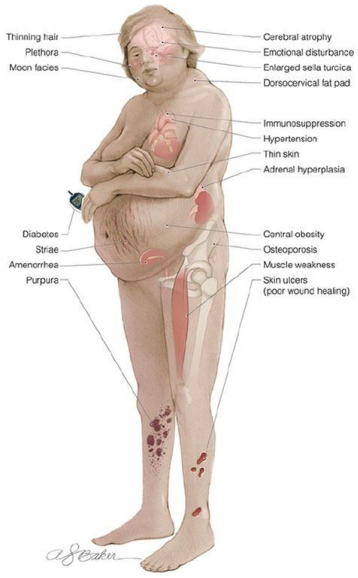
- Normal action: retain _____ and ____
- Too much Aldosterone => retain too much ___ and ___ => fluid volume ___ (excess or deficit?) in the ____ (interstitial or intravascular?) space
  - *Conn's (hyperaldosteronism) and Cushing syndrome => too much Aldosterone*
- Not enough aldosterone => lose **sodium** and **water** => fluid volume ___ (excess or deficit?)
  - *Addison's disease*

Primary Hyperaldosteronism

Conn disease

- **Hyper**tension
- **Hypo**kalemia
- Metabolic **alkalosis**





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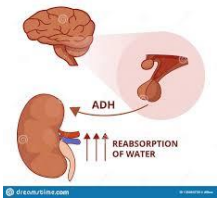
ADH (Antidiuretic Hormone/Arginine Angiotensin)



- **Normal action**: retain _____
- **Too much ADH** => retain too much ___ => fluid volume ___ (excess or deficit?) in the ____ (interstitial or intravascular?) space
  - *Normally: increase in vascular volume => increase in urine output*
  - SIADH: ↑blood volume and ↓urine output
    - Blood and urine will not mirror each other
      - Blood diluted and urine concentrated
    - Hypervolemia or Hypovolemia?
    - Sodium and Urine Specific Gravity (↑ or ↓ ?)
- **Not enough ADH** => cannot properly retain ___ => fluid volume ___ (excess or deficit?) in the ____ space
  - Diabetes Insipidus
    - My #1 concern when loosing too much volume: ___
  - *Normally:* pt in shock => urinary output ↓)
  - Diabetes Insipidus: pt in shock => still polyuria
    - Urine diluted and blood concentrated
    - Sodium and Urine Specific Gravity (↑ or ↓ ?)

Diabetes Insipidus		SIADH
- High Urinary Output		- Low Urinary Output
- Low Levels of ADH		- High Levels of ADH
- Hypernatremia	VS	- Hyponatremia
- Dehydrated		- Over Hydrated
- Lose too much fluid		- Retain too much fluid

* Both will present with excessive thirst

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PITUITARY: any problem => think ADH problem

3

DI	SIADH
• Low ADH, Low water in body	• High ADH, water intoxication
• **High UO**, Polyuria	• **Low UO**, Oliguria
• **High sodium**	• **Low sodium** (dilutional)
• High H&H and serum osmolality from dehydration	• Low serum osmolality
• Risk: **Hypovolemic shock**	• Weight gain
• TX: DDAVP (ADH)	• Risk: **Seizures**
	• TX: Hypertonic saline

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*Severe and rapidly evolving hyponatremia may cause seizures

4

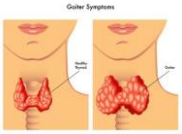
Thyroid

- Produces 3 hormones: T3 - T4 - Calcitonin (↓serum Calcium bringing it back to bones)
- To produce Thyroid hormones we need ____ (vit D or iodine?)
- Thyroid hormone give us energy

HYPERTHYROID: TOO MUCH ENERGY!! (Graves Disease)

S/S:

- Nervous
- Weight (↓ or ↑ ?)
- Sweaty/ Hot
- Exophthalmia
- Attention span (↓ or ↑ ?)
- irritable
- Weight (↓ or ↑ ?)
- Appetite (↓ or ↑ ?)
- GI (some have constipation, some have diarrhea)
- BP (hypertension or hypotension?)*
- Thyroid => hypertrophy (goiter) (why?)



- *everytime you have high BP => work of the heart is increased => worry about heart troubles

5

Thyroid HYPERTHYROIDISM: TOO MUCH ENERGY!! (Graves Disease)

DIAGNOSIS:

- Clinical (Signs and Symptoms)
- Lab test (serum T4 or T3 level would it be increased or decreased?)
- Thyroid scan (radioactive iodine injection)
  (Client must discontinue any iodine containing medication one week prior to the thyroid scan)

TREATMENT:

- **1) Antithyroids**: *
  - Propylthiouracil (Propacil®/PTU®),
  - Methimazole (Tapazole®) =>
  **Stops the thyroid from making TH's (We want this client to become euthyroid)*

*These meds have to be tapered and discontinued (NOT suppose from Hyper to Hypo thyroid)

- **2) Iodine Compounds**: (# from dietary iodine)
  - Decrease vascularity
  - Potassium Iodide (SSKI®),
  - Strong Iodine Solution (Lugol)
    - *(action ≠ than dietary iodine => ↓vascularity of the gland. Can be given pre-op to ↓bleeding)* => _____ (↑ or ↓ ?) **vascularity** of the gland*

*ALL endocrine glands are VERY VASCULAR!

- Give in milk, juice, and use straw

- **3) Beta Blockers**:
  - Ex: Inderal®

=> ↓myocardial contractility => could ↓cardiac output => ↓HR, BP (↓anxiety too)

(Beta blockers block the effects of the hormone epinephrine and norepinephrine)
(can ↓ myocardia contractility => decrease cardiac output => head perfusion – not everybody can handle Beta blockers)

Rule: Do not give beta blockers to asthmatics or diabetics(can't have symptoms of hypoglicemia = can't release epi and norepinephrine

6

HYPERTYRODISM

TREATMENT (CONT.)
4) Radioactive Iodine => destroy thyroid tissue
=> (usually one dose) Given PO (liquid or tablet form)
Rule out pregnancy first
=> Destroys thyroid cells => pt can become _____
✓ Follow radioactive precautions
Stay away from babies for ___hours
Don't kiss anyone for ___ hours
-Watch for thyroid storm (Thyroid storm, thyrotoxicosis, and thyrotoxic crisis are the same)
=> It is hyperthyroidism multiplied by l00
=> Could be a rebound effect post-radioactive iodine

5) Surgery: thyroidectomy (partial/complete)
-Post-op:

- Teach how to support neck
- Positioning => (important)
  - no tension on suture line
  - leave things near the pt (water, phone, etc.)
- Check for bleeding (behind the neck)
- Nutrition (pre & post-op)
- Assess for recurrent laryngeal nerve damage
  - Hoarseness (can have vocal cords paralysis)
  - => if there is paralysis of both cords => airway obstruction will occur requiring immediate trach

✓ Unilateral laryngeal nerve damage = voice changes, including hoarseness
✓ Bilateral laryngeal nerve damage = breathing difficulties and aphonia (inability to speak)

- Keep tracheostomy set at bedside for situations like:
- hypocalcemia
- swelling
- recurrent laryngeal nerve damage
- Teach pt to report any pressure on the site
- Assess for parathyroid removal (PTH => pull Ca from bones => make serum Ca ↑)
  - Worry about electrolytes imbalance => hypocalcemia = muscles rigid, laryngospasm, seizure.

✓ Eye care is important for a client with hyperthyroidism. If client can't close their eyelids, hypoallergenic tape may be applied to close lid (to help prevent injury or irritation).
✓ Dark glasses may be worn if photosensitivity.
✓ Artificial tears to prevent drying of the eyes.

7

Hypothyroid (Myxedema):

When present at birth it's called cretinism (very dangerous, can lead to slowed mental and physical development if undetected)

S/S:

- No energy, fatigue
- GI => fast or slow?
- weight (gain or loss?)
- hot or cold?
- Speech slow, no expression=> takes energy to articulate words, to move muscles

You may be taking care of a totally immobile client
People with hypothyroidism tend to have ___ HR and ___ BP

TREATMENT:

- Levothyroxine (Synthroid), Thyroglobulin (Proloid), Liothyronine (Cytomel) *
  - Do they take these meds forever?
  - What will happen to their energy level when they start taking these meds? HR and BP?

*If giving IV and pt complains anything related to heart (ex: chest pain) => don't continue – call the dr. ➔ Hypothyroid tend to have Coronary Artery Disease (look for s/s of CAD)

8

Parathyroid Problems

*think Calcium

- The parathyroids secrete ___ which makes you pull calcium from the ___ and place it in the blood.
- If you have too much parathormone in your body the serum calcium level will be___.
- If you do not have any parathormone in your body the serum calcium level will be___.

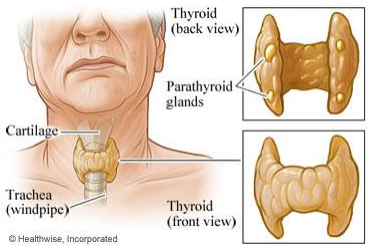
Hyperparathyroidism = Hypercalcemia=Hypophosphatemia

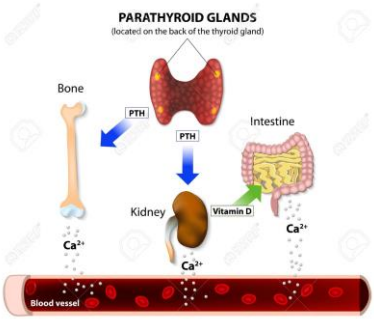
S/S:

- Too much ___
- Serum calcium is ___
- Serum phos is ___
- other S/S

TREATMENT

- Partial parathyroidectomy
  - when you take out 2 of your
    - PTH secretion _____
    - Serum Calcium _____





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Hypoparathyroidism=Hypocalcemia=Hyperphosphatemia:

S/S:

- Not enough _____
- Serum calcium is _____.
  - Worry about low Ca: seizures, laryngospasm, etc.
- Serum phos is _____.
- other S/S

TREATMENT:

- Ca
  => acts like a sedative. When given IV =>monitor heart (can ↓HR and wider QRS)
- When P is too high, can give a Phosphate binder
  - Ex: calcium carbonate, calcium acetate, sevelamer hydrochloride, lanthanum carbonate, and magnesium hydroxide*

*careful => serum Ca ↓

10

Adrenal Problems:

- need your adrenals to handle stress
- You have 2 parts to your adrenal gland: adrenal medulla and the adrenal cortex

Adrenal medulla: (epinephrine, norepinephrine)

- Adrenal Medulla Problems:
  - (major): Pheochromocytoma
    - Benign tumors that secrete epi and norepi in bolus

S/S:

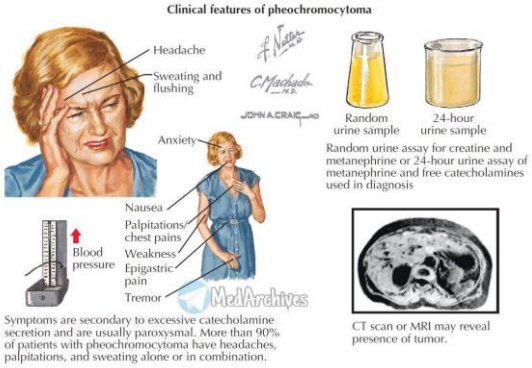
- BP?
- Pulse?
- Flushing/diaphoretic

DIAGNOSTIC

- VMA (vanylmandelic acid) test: a 2 one and you are looking for increased levels of epi/norepi (also called catecholamines)
  - Anything with Vanila can alter the test
- With a 24 hour urine you should ___ the first voiding and ___ the last voiding. Don't miss any ml.
- Other things can alter the test (patients need to stay calm during the test)

TREATMENT

- Surgery => take out the tumors
- Medications:
  - Methylprednisolone Sodium Succinate (Solu-medrol),
  - Hydrocortisone (Solu-Cortef),
  - Prednisone,
  - Dexamethasone (Decadron)



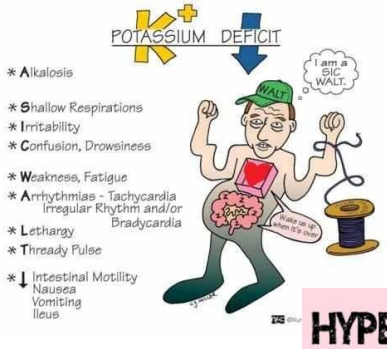
11

Adrenal cortex: (think steroids)

Adrenal Cortex Steroids:

1) Glucocorticoids:

- Change your mood
  - depression, mean, psychotic, insomnia
- Alter defense mechanisms
  - Suppress immune system
- Breakdown protein/fat
  - Arms and legs thin
- Inhibits insulin
  - Blood sugar goes ↑
- Inhibits growth in children



2) Mineralocorticoids *Aldosterone:

- Make you retain _____ & _____
- Make you lose _____

⇒ **Too Much Aldosterone :**

Vascular Space: (excess or deficit?)

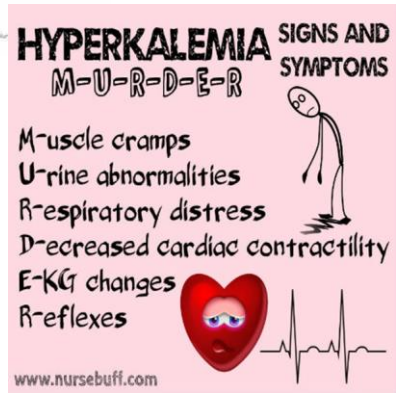
Serum Potassium: (↑ or ↓?) => retain sodium => hypokalemia

⇒ **Not Enough Aldosterone**

Vascular Space: (deficit or excess?)

Serum Potassium: (↑ or ↓?) => hyperkalemia

3) Sex hormones: ** See Cushing's notes**



12

Adrenocorticotropin hormones (ACTH) are made in the pituitary and they stimulate cortisol to be made.

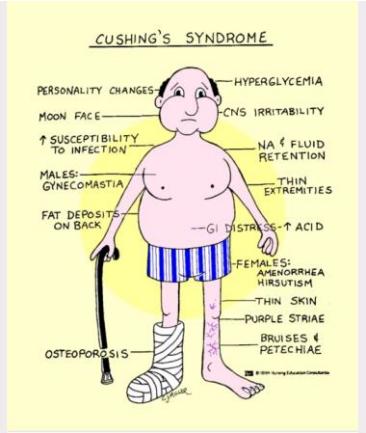
ACTH => think cortisol (steroid)

Cortisol is a hormone of the adrenal cortex.

So, no matter what "fancy" word NCLEX uses => you will get the same result - think steroids

↑ACTH= ↑ Cortisol level

Too many steroids = Hypercortisolism (just another word)

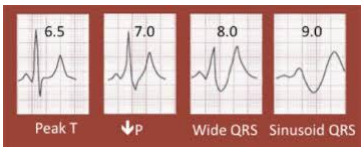


13

Adrenal Cortex Problems:

ADDISON'S DISEASE: (adrenocortical insufficiency-not enough steroids)

- Risk of Shock (low aldosterone)– high Potassium (A major function of aldosterone is to increase urinary potassium secretion. As a result, hypoaldosteronism can be associated with hyperkalemia and mild metabolic acidosis) => *Add steroids*
- *Not enough steroids*
- *Shock*
- *High potassium*



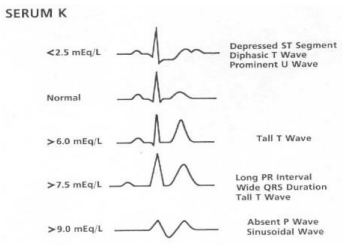
a) Pathophysiology:

- If this client is insufficient do they have enough glucocorticoids, mineralocorticoids, or sex hormones?
- Focus on aldosterone:

-Normally, aldosterone makes us retain ___and ___ and lose ___

Now we don't have enough (insufficient) so we will lose ___ and ___ and retain __

-The serum K+ will be _____



The majority of the **S/S** are a result of the **hyperkalemia** initially.

-Beginning with muscle twitching, then proceeds to weakness, then flaccid paralysis

14

ADDISON'S DISEASE:

b) S/S:

*Some S/S have an unknown etiology

- anorexia/nausea
- hyperpigmentation- bronzing color of the skin and mucous membranes
- decreased bowel sounds
- GI upset
- hypoglycemia (steroid ↑ blood sugar)
- white patchy area of depigmented skin (vitiligo)
- hypotension (due to ↑capillary permeability and ↓ability for vessels to constrict – losing Na and H_2O)
- If you checked this client's blood/urine for adrenocorticotropin hormones (cortisol)=> it would be decreased or absent.

15

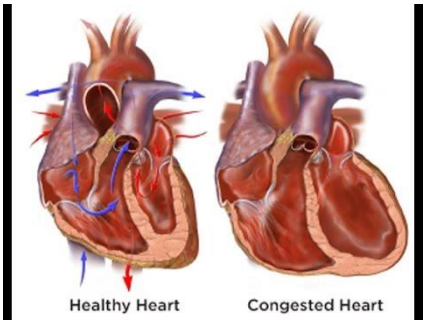
ADDISON'S DISEASE:

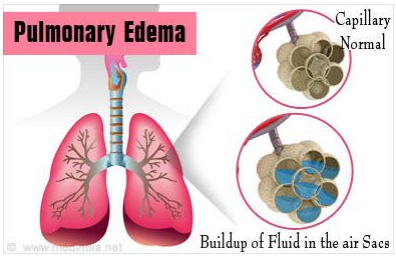
c) TREATMENT:

- -Combat shock (losing __ and___)
- Processed fruit juice/broth (has lots of __)
- I & O and daily weight
- If this client is losing Na & H_2O, their BP will probably be ___
- They will probably be gaining/losing weight?
- Nursing DX: Fluid Volume __ (deficit or excess?) (worry about ___)
- Will be placed on the mineralocorticoid drug Fludrocortisone (Florinef®) (aldosterone) =>
  - WEIGHT is very important in adjusting their medication (will retain __ and __ => watch the weight. I worry about ___ problems and ___ edema

Rule

When on a medicine where weight has to be monitored, keep weight within ± 2 lbs of their normal weight





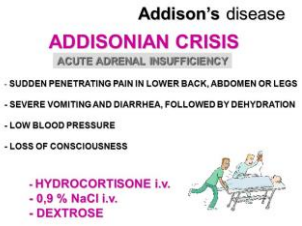
16

➢ If you have a client at home taking Fludrocortisone (Florinef) and the following occurs, what should the CLIENT do?

- (1) Overnight gain of 7 lbs. → what do they do with their AM dose?
  - Too much → decrease dose or hold

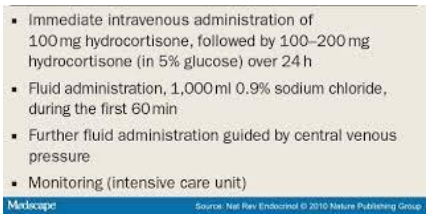
(Test taking strategy: Fluid retention → think heart problems 1st , and pulmonary edema)

- (2) Overnight loss of 7 lbs. what should be done with their dose? (↓or ↥?)
  - Worry about deficit → shock



➢ If the nurse is taking care of a client in the hospital on Fludrocortisone (Florinef, and the following occurs. what should the NURSE do?

- (1) Client has edema or their BP is up *
- (2) Client's BP is steadily going down **



Addisonian Crisis: severe hypotension and vascular collapse
Never stop steroids abruptly

*hold it and call the doctor **increase the dose

17

CUSHING – too many steroids

a) Pathophysiology:

- **Cushing's Syndrome**
- Exogenous administration: someone who is taking steroids for the treatment of asthma or COPD, autoimmune disorders, organ transplantation, chemotherapy, allergic responses.
  - These clients could receive too many steroids as part of the treatment and develop Cushing's Syndrome.

Cushing's disease

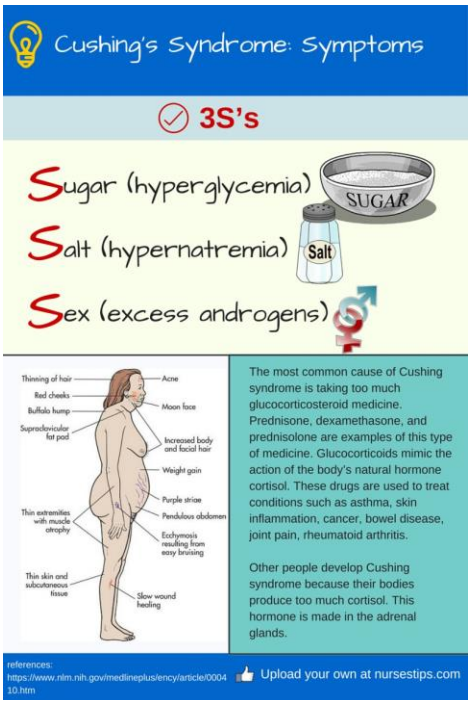
- Endogenous: bilaterally adrenal hyperplasia, pituitary adenoma increases secretion of ACTH, malignancies, adrenal adenoma or carcinoma

b) S/S:

These clients have too many glucocorticoids, mineralocorticoids, and sex hormones.

TOO MANY GLUCOCORTICOID

- growth arrest
- thin extremities/skin (cortisol can promote lipolysis)
- increased risk for infection
- hyperglycemia
- psychoses to depression
- moon faced (can be due to fat redistribution or fluid retention
- truncal obesity (fat redistribution; lipogenesis)
- buffalo hump (fat redistribution)



18

CUSHING's S/S:

TOO MANY SEX HORMONES

- oily skin/acne
- women with male traits
- poor sex drive (libido)

* High levels of adrenal steroids interfere with the ability of the pituitary gland to secrete LH and FSH and for the testes to make testosterone.

TOO MANY MINEROLOCORTICOID

- high BP ↔ Fluid Volume ____ (excess or deficit?)
- CHF
- weight gain

- Since this client has too much mineralocorticoid (aldosterone), the serum K+ will ____ (holding Na and water -> lose K+)

- If you did a 24 hour urine on this client the <u>cortisol</u> levels would be ___ (high or low?)

19

c) Cushing's TREATMENT:

- adrenalectomy (unilateral or bilateral)
  - if both are removed => lifetime replacement
- quiet environment (anytime your steroids are disturbed, you can't handle stress)
- What does this client need in their DIET pre-treatment?
- K+ (more? less?)
- Na (more? less?)
- Protein (more? less?) (are breaking down)
- Ca (more? less?) (steroids ↓serum Ca by making you excrete it through GI tract take Ca from the bones => brittle bones)
- Avoid infection (immune suppress)
- What might appear in their urine?
  - Glucose (blood sugar ↓or ↑) 

  - Ketones (fat break down)
  - Protein?? (proteins are <u>big molecules</u>) 

20

Diabetes:

1. Classification: **Normal Lab Value Blood Glucose: 70-110mg/dL**

- Type 1: (IDDM)

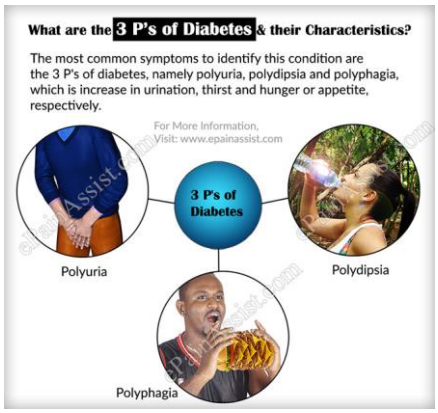
- **Pathophysiology:**
- They have little or no insulin
- Usually starts in childhood
- First sign may be _____.
- Patho:
  - You have to have insulin to carry glucose out of the vascular space over to the cell.
  - Since there is no insulin, the glucose just builds up in the vascular space (blood/serum), the blood becomes hypertonic and pulls fluid into the vascular space
  - the kidneys filter excess glucose and fluids (polyuria and polydypsia) the cells are starving so they start breaking down protein and fat for energy (polyphagia)
  - when you break down fat you get ___ (acids)
  - Now this client is acidotic (respiratory or metabolic?)

21

Diabetes type 1:

2) S/S: Hyperglycemia = 3 p's

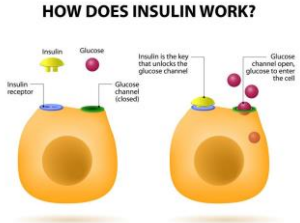
- **P**olyuria (with accompanying weight loss)
  - Kidneys trying to get rid of sugar particles, but also lose water => polyuria leads to shock
- **P**olydypsia (thirst)
- **P**olyphagia (hunger – brain isn't having enough glucose But even eating more, don't have enough insulin)



3) TREATMENT:

- Insulin (See General Tx)

Will oral hypoglycemia agents such as Glipizide (Glucotrol®), or Glyburide (Micronase®/Diabeta®) work in this client?



22

Somogyi Phenomenon:

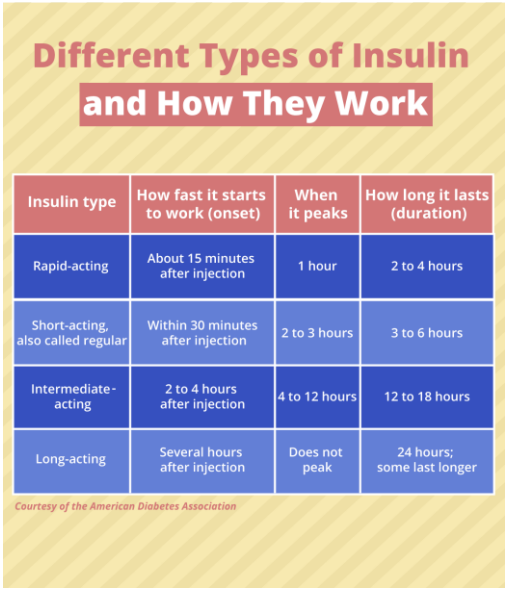
- rebound phenomenon that occurs in Type I diabetic;
- client has normal or ↑ BG levels at bedtime and BG drops in early morning hours (~2-3AM);
- Client's body attempts to compensate by producing counter-regulatory hormones to increase BG resulting in hyperglycemia.
- **Treatment:** ↑ bedtime snack and ↓intermediate acting insulin (NP insulin, Lente insulin)

Dawn Phenomenon:

- Resulting from a decrease in the tissue sensitivity to insulin that occurs between 5-8 AM (pre-breakfast hyperglycemia);
- caused by a release of nocturnal growth hormones
- **Treatment:** give intermediate-acting insulin (NPH insulin, Lente insulin) at 10PM

Insulin is the only hormone that lowers the blood glucose level in your body

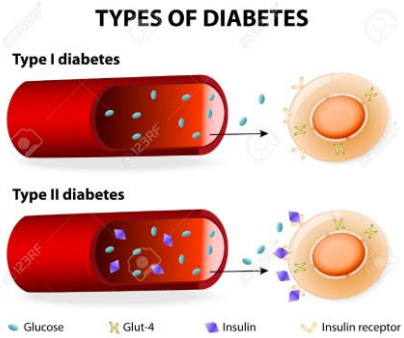
23



24

Diabetes Type II: (NIDDM)

- **1) Pathophysiology:**
- These clients don't have enough _____ or the insulin they have is not good.
- These clients are usually ___weight. They can't make enough insulin to keep up with the glucose load the client is taking in.
- This type of diabetes is not as abrupt as Type I.
- It's usually found by accident; or the client keeps coming back to the doctor for things like a wound that won't heal, repeated vaginal infections, etc. (bacteria, fungus, like sugar)
- **2) Treatment:**
- Start with diet and exercise, then add oral agents, then add ___.



25

- **2. General Treatment of Diabetes: (Type I and Type II)**
- **a. Diet:**

Majority of calories should come from:

- *complex carbohydrates 55-60%
- *fats 20-30%
- *protein 12-20% (limit => diabetics tend to have renal disease)
  - Diabetics tend to have _____ disease.
  - Why are diabetics prone to CAD? Sugar destroys vessels just like fat.

- High fiber diet (keeps blood sugar steady; client may have to decrease insulin)
- High fiber slows down glucose absorption in the intestines, therefore, eliminating the sharp rise/fall of the blood sugar

- **b. Exercise:**
- Some people wait until blood sugar normalize to begin exercise (if glucose goes ↓ => eat sugar => gluc. Realy high => extremes cause vascular damage.
- What should the client do pre-exercise to prevent hypoglycemia? Eat something
- Exercise when blood sugar is at its highest or lowest?
- Exercise same time and amount daily (establish routine)

26

c. Medications:

- How do oral hypoglycemic agents work? Some stimulate pancreas to make insulin

*note: not all oral hypoglycemic agents stimulate the pancreas to make insulin

Medication	Action
chlorpropamide (Diabinese®)	Stimulates release of insulin from pancreas
glipizide (Glucotrol®, Glucotrol XL®)	Stimulates release of insulin from pancreas
glyburide (Diabeta®, Glynase®)	Stimulates release of insulin from pancreas
metformin (Glucophage®, Glucophage XR®)	Dec rate of hepatic glucose production and changes the glucose uptake by tissues
acarbose (Precose®)	Delay absorption of glucose from GI tract
pioglitazone (Actos®)	Inc glucose uptake in muscles, dec endogenous glucose production

27

How is the insulin dose determined?

- the dose is increased until the blood sugar is normal and until there is no more ____ and ____ in the urine

- Reg (clear) + NPH® (cloudy) …. which one do you draw up first?
- What is the only type of insulin you can give IV?
- BLOOD TEST: Hemoglobin A1C:
  - gives an average of what your blood sugar has been over the past 3 ___.
  - For people with diabetes, the ideal goal for their HgbA1C is 7% or less
- Client should eat when insulin is at its (peak or low?) <=>PEAK: think HYPOGLYCEMIA
- What happens to your blood sugar when you are sick/stressed? (up or down?)
  - normal pancreas can handle these fluctuations; an increase in the blood sugar when sick/stressed is a normal reaction to help us fight the illness/stressor
  - But if diabetic, what happens? (INSULIN DOSE: higher insulin dose with higher blood sugar)
    - ILNESS + DIABETES = DKA
- Rotation of sites (Rotate _____(within or without?) an area first)
- One site at a time (avoid lipodystrophy, scar => less insulin absorption)
- Aspirate?
  - Too much tissue trauma (also heparin and lovenox)

28

d. Hypoglycemic/Hyperglycemic Episodes:

- What are the S/S of hypoglycemia?
  - Shaky; nervous; ↑ pulse; cold/clammy; nausea; confusion; headache
    - Hypoglycemia is very dangerous in Diab. => no compensatory mechanism
- What should the client do?
  - Eat simple sugar (ex: milk)
- After the blood sugar is up, what should they do?
  - Eat complex carb and protein (ex: cheese)

You enter a diabetic client's room and they are unconscious...do you treat this client like he is hypo or hyperglycemic?
  - Why? (risk for brain damage)
- If client is awake, give food.
- D5W (hard to push; and if you have a choice you need a large bore IV/angiocath)
- Injectable glucagon (GlucaGen®) (used when there is no IV access; given IM)
- **Prevention:** (1) eat
- (2) take insulin regularly
- (3) snacks

29

3. Complications of Diabetes:

a. Diabetic Ketoacidosis (DKA):

1) Pathophysiology:

- anything that increases blood sugar (infection, illness, skipping insulin) can throw a client into DKA
- may be the first sign of diabetes.
- have all the usual S/S of Type I diabetes

- **Patho:**
- Not enough insulin => blood sugar goes sky high => Polyuria, Polydypsia, Polyphagia => fat breakdown (acidosis) => Kussmaul's respirations (trying to blow off C02 to compensate for the metabolic acidosis). Also, as the client becomes more acidotic the LOC goes down

30

2) Treatment:

Find the cause

- Hourly blood sugar and K+
- IV insulin - Insulin decreases ___ & ___ by driving them out of the vascular space into the cell.
- ECG (hypoglycemia and hypokalemia => worry about arrythmias)
- Hourly outputs (remember: Polyuria, then oliguria, then anuria => Shock)
- ABG's (prevent metabolic acidosis)
- IVF's => Start with NS....then when the blood sugar gets down to about 300 switch to D5W to prevent throwing the client into hypoglycemia
  - I have to bring back the pt's BP too
  - Add K+ => Anticipate that the doctor will want us to add ___ to the IV solution at some point.

31

Hyperosmolar hyperglycemic nonketosis (HHNK) or Hyperosmolar hyperglycemic nonketosis coma (HHNC):

- Non-ketotic => look like DKA but no ___ (acidosis? alkalosis?)
  - Making just enough insulin so they are not breaking down body fat => No fat breakdown no ___ no ketones => no ___ Will this client have Kussmau's respirations?
    - Happens in Type II => no ketones, no acidosis, no Kusmaul

In the NCLEX® world: Type 1=> DKA Type II => HHNK

COMPLICATIONS

Vascular Problems:

- Will develop poor circulation everywhere due to vessel damage (sugar irritates the vessel lining; accumulation of sugar will decrease the size of vessel lumen therefore decreasing blood flow)

1) Diabetic retinopathy (retina very vascular)

2) Nephropathy (kidneys very vascular)

Neuropathy:

1) Sexual problems: impotence/decreased sensation

2) Foot/leg problems: pain/paresthesia/numbness

-Review of Diabetic Foot Care (TOE NAILS CUT: _____; SHOES ____; AVOID HARSH CHEMICALS; DRY BTWEEN TOES; INSPECT EVERYDAY)

3) Neurogenic bladder: (bladder does not empty properly).

The bladder may empty spontaneously (incontinence) or may not empty at all (retention)

4) Gastroparesis: (stomach emptying is delayed so there is an increased risk for aspiration)

Increased Risk for Infection:

↑ sugar = ↑ risk for infection

32