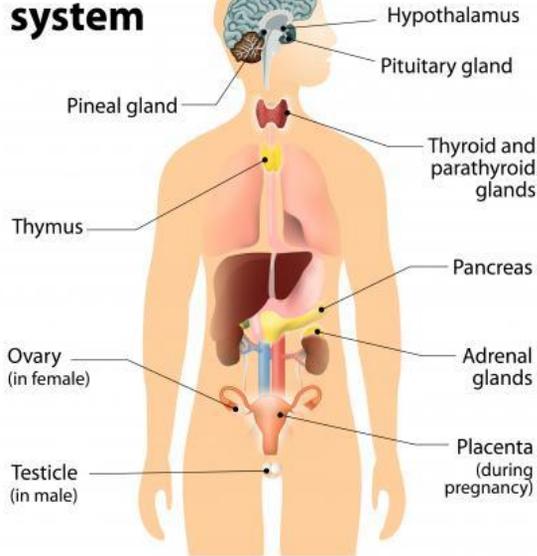


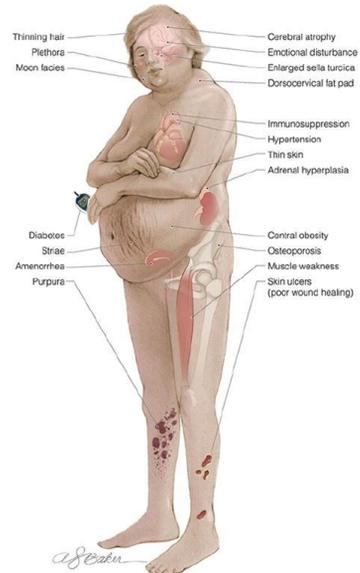
# Endocrine system



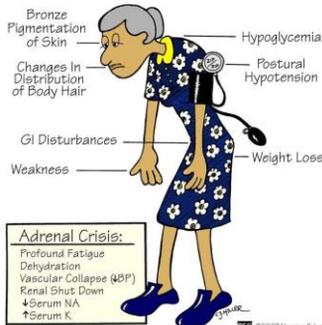
1

## 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*



## ADDISON'S DISEASE



### Primary Hyperaldosteronism

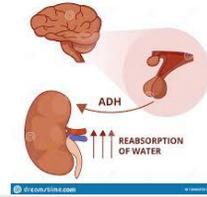
#### Conn disease

- Hypertension
- Hypokalemia
- Metabolic alkalosis

2

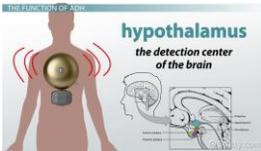
## ADH (Antidiuretic Hormone/Arginine Angiotensin)

- **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?
    - Serum 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 losing too much fluid volume: \_\_\_\_\_
    - **Normally:** pt in shock → urinary output (↑ or ↓ ?)
    - **Diabetes Insipidus:** pt in shock → still polyuria
      - Urine diluted and blood concentrated
        - Serum sodium and Urine Specific Gravity (↑ or ↓ ?)

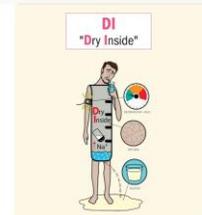


Diabetes Insipidus	SIADH
- High Urinary Output	- Low Urinary Output
- Low Levels of ADH	- High Levels of ADH
- Hypernatremia	- Hyponatremia
- Dehydrated	- Over Hydrated
- Lose too much fluid	- Retain too much fluid

**\* Both will present with excessive thirst**



**PITUITARY:** any problem → think ADH problem  
(some people get SIADH, others get DI)



3

### DI

- Low ADH, Low water in body
- **High UO**, Polyuria
- **High sodium**
- High H&H and serum osmolality from dehydration
- Risk: **Hypovolemic shock**
- TX: DDAVP (ADH)

### SIADH

- High ADH, water intoxication
- **Low UO**, Oliguria
- **Low sodium** (dilutional)
- Low serum osmolality
- Weight gain
- Risk: **Seizures**
- TX: Hypertonic saline

[www.pdbnurseeducationllc.com](http://www.pdbnurseeducationllc.com)

\*Severe and rapidly evolving hyponatremia may cause seizures

4

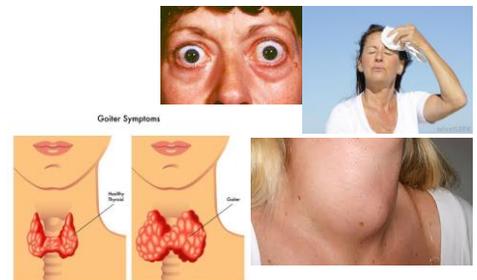
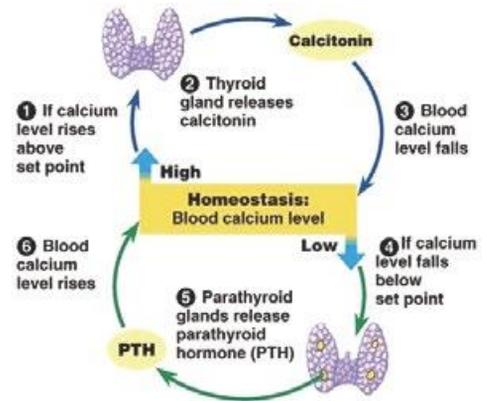
## Thyroid

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

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

**S/S:**

- Nervous
  - Weight (↓ or ↑ ?)
  - Sweaty/ Hot
  - Exophthalmia
  - Attention span (↓ or ↑ ?)
  - Irritable
  - Appetite (↓ or ↑ ?)
  - GI (some have constipation, some have diarrhea)
  - BP (hypertension or hypotension?)\*
  - Thyroid => hypertrophy (goiter) (why?)
- *\*everytime you have high BP → work load of the heart is increased worry → about heart troubles*



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## 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 ↓ ?) **vasculature** of the gland\*

**\*ALL endocrine glands are VERY VASCULAR!**

- Give in milk, juice, and use straw

- **3) Beta Blockers:** ( ↓ Heart Rate and Blood Pressure)

- Ex: Inderal®
- => ↓ myocardial contractility => could ↓ cardiac output → ↓ HR, BP (↓ anxiety too)

(Beta blockers block the effects of the hormones epinephrine and norepinephrine)

(can ↓ myocardia contractility => decrease cardiac output => ↓ brain perfusion – not everybody can handle Beta blockers)

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

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# HYPERTHYROIDISM

## 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 100

=> 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.

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## 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



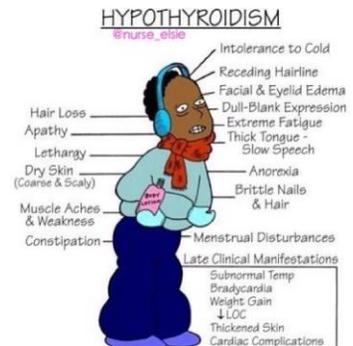
You may be taking care of a totally immobile client

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)



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## 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 \_\_\_.

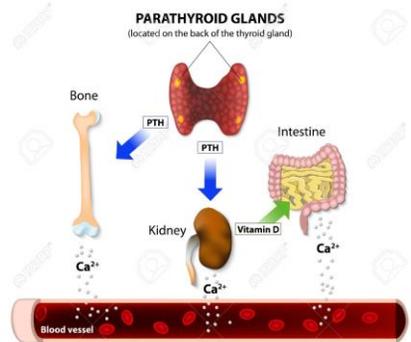
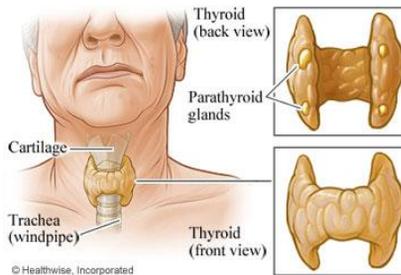
### Hyperparathyroidism = Hypercalcemia=Hypophosphatemia

S/S:

- Too much \_\_\_
- Serum calcium is \_\_\_
- Serum phosphorus 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 phosphorus 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 → when serum Ca ↓

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**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 (vanilylmandelic acid) test: a 24 hour urine specimen is done and you are looking for increased levels of epi/norepi (also called catecholamines)
  - Anything with Vanilla 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 )

**Clinical features of pheochromocytoma**

Random urine sample      24-hour urine sample

Random urine assay for creatine and metanephrine or 24-hour urine assay of metanephrine and free catecholamines used in diagnosis

CT scan or MRI may reveal presence of tumor.

Symptoms are secondary to excessive catecholamine secretion and are usually paroxysmal. More than 90% of patients with pheochromocytoma have headaches, palpitations, and sweating alone or in combination.

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**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\*\*

**POTASSIUM DEFICIT**

- \* Alkalosis
- \* Shallow Respirations
- \* Irritability
- \* Confusion, Drowsiness
- \* Weakness, Fatigue
- \* Arrhythmias - Tachycardia, Irregular Rhythm and/or Bradycardia
- \* Lethargy
- \* Thready Pulse
- \* ↓ Intestinal Motility, Nausea, Vomiting, Ileus

**HYPERKALEMIA SIGNS AND SYMPTOMS**

**M-U-R-D-E-R**

- M-**u**scle cramps
- U-**r**ine abnormalities
- R-**r**espiratory distress
- D-**e**creased cardiac contractility
- E-**K**g changes
- R-**e**flexes

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

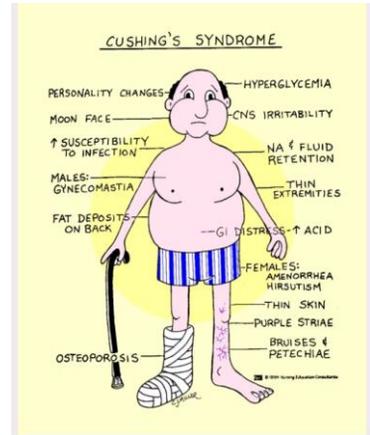
ACTH => think cortisol (steroid)

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)

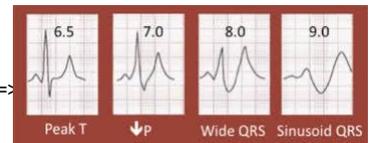


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**Adrenal Cortex Problems:**

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

- Risk of Shock (low aldosterone → loose Na<sup>+</sup> and Water)– 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

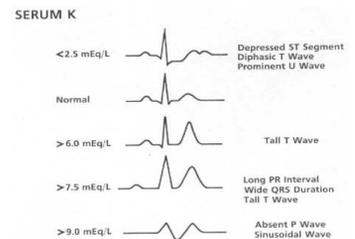


**a) Pathophysiology:**

- 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<sup>+</sup> 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



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## 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  $\uparrow$  blood sugar)
- white patchy area of depigmented skin (vitiligo)
- hypotension (due to  $\uparrow$ capillary permeability and  $\downarrow$ ability for vessels to constrict – losing Na and H<sub>2</sub>O)
- If you checked this client's blood/urine for adrenocorticotropic hormones (cortisol)=> it would be decreased or absent.



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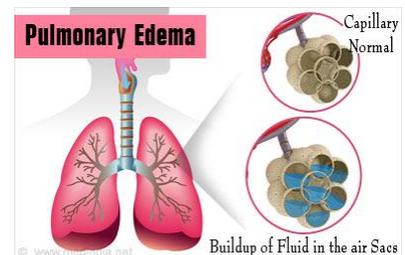
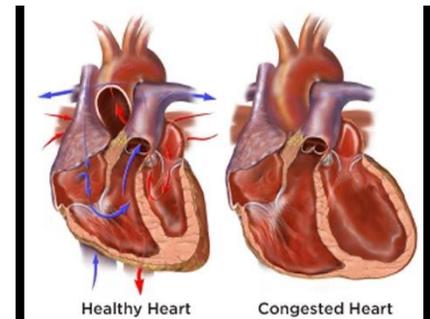
## ADDISON'S DISEASE:

### 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<sub>2</sub>O, 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\*
  - \* (in case of complications, decrease or hold the medication)

#### Rule

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



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➤ 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 1<sup>st</sup>, 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

**Addison's disease**

**ADDISONIAN CRISIS**  
 ACUTE ADRENAL INSUFFICIENCY

- SUDDEN PENETRATING PAIN IN LOWER BACK, ABDOMEN OR LEGS
- SEVERE VOMITING AND DIARRHEA, FOLLOWED BY DEHYDRATION
- LOW BLOOD PRESSURE
- LOSS OF CONSCIOUSNESS

**- HYDROCORTISONE i.v.**  
**- 0.9 % NaCl i.v.**  
**- DEXTROSE**



- Immediate intravenous administration of 100 mg hydrocortisone, followed by 100–200 mg hydrocortisone (in 5% glucose) over 24 h
- Fluid administration, 1,000 ml 0.9% sodium chloride, during the first 60 min
- Further fluid administration guided by central venous pressure
- Monitoring (intensive care unit)

Medscape Source: Neil Rev Endocrinol © 2010 Nature Publishing Group

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**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)

**Cushing's Syndrome: Symptoms**

**3S's**

- S**ugar (hyperglycemia) 
- S**alt (hypernatremia) 
- S**ex (excess androgens) 



The most common cause of Cushing syndrome is taking too much glucocorticosteroid medicine. Prednisone, dexamethasone, and prednisolone are examples of this type of medicine. Glucocorticoids mimic the action of the body's natural hormone cortisol. These drugs are used to treat conditions such as asthma, skin inflammation, cancer, bowel disease, joint pain, rheumatoid arthritis.

Other people develop Cushing syndrome because their bodies produce too much cortisol. This hormone is made in the adrenal glands.

references: <https://www.nlm.nih.gov/medlineplus/ency/article/000410.htm> Upload your own at [nursestips.com](https://nursestips.com)

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## 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 cortisol levels would be \_\_\_\_ (high or low?)



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### 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? (which one is wrong?)**
  - o Glucose (blood sugar ↓ or ↑)
  - o Ketones (fat break down)
  - o Protein?? (proteins are big molecules)



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# 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?)

\*risk of shock

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## Diabetes type 1:

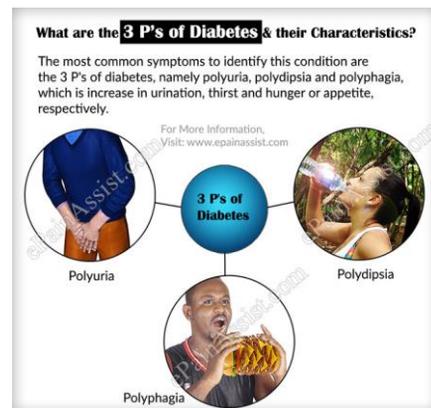
### 2) S/S: Hyperglycemia = 3 p's

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

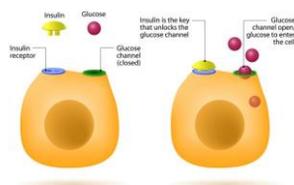
### 3) TREATMENT:

- Insulin (See General Tx)

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



### HOW DOES INSULIN WORK?



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### Somogyi Phenomenon:

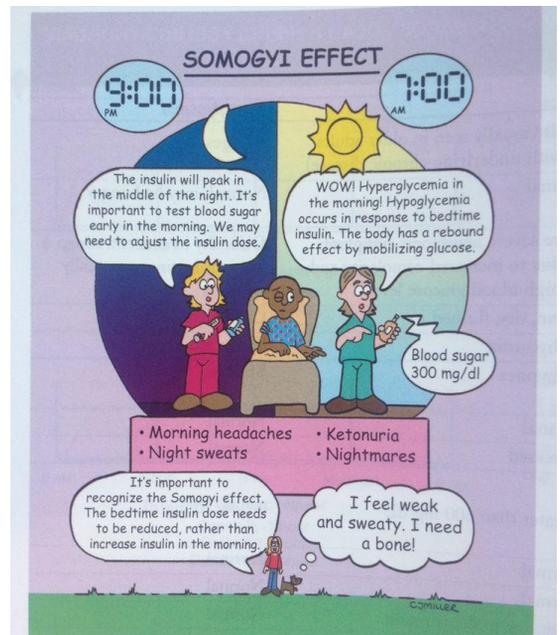
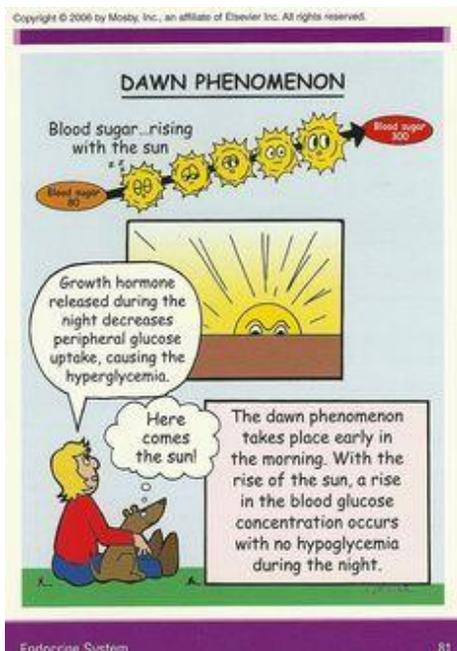
- rebound phenomenon that occurs in Type I diabetic;
- client has normal or  $\uparrow$  BG levels at bedtime and BG drops in early morning hours (~2-3AM);
- Client's body attempts to compensate by producing counter-regulatory hormones to increase BG resulting in hyperglycemia.
- **Treatment:**  $\uparrow$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

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### Different Types of Insulin and How They Work

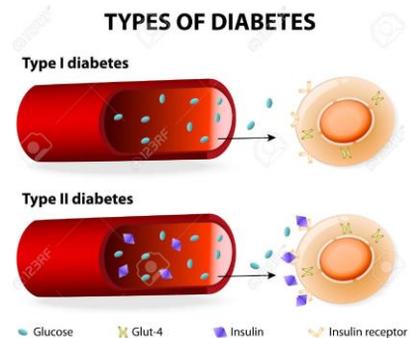
Insulin type	How fast it starts to work (onset)	When it peaks	How long it lasts (duration)
Rapid-acting	About 15 minutes after injection	1 hour	2 to 4 hours
Short-acting, also called regular	Within 30 minutes after injection	2 to 3 hours	3 to 6 hours
Intermediate-acting	2 to 4 hours after injection	4 to 12 hours	12 to 18 hours
Long-acting	Several hours after injection	Does not peak	24 hours; some last longer

Courtesy of the American Diabetes Association

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## Diabetes Type II: (NIDDM)

- **1) Pathophysiology:**
- These clients don't have enough \_\_\_\_\_ or the insulin they have cannot bring glucose into the cell.
- 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 \_\_\_\_.



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- **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. Really 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)

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- **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 uptake by tissues
acarbose (Precose®)	Delay absorption of glucose from GI tract
pioglitazone (Actos®)	Inc glucose uptake in muscles, dec endogenous glucose production

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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)
    - **ILLNESS + DIABETES = DKA**
- Rotation of sites (Rotate **within** an area first)
  - One site at a time (avoid lipodystrophy, scar => less insulin absorption)
- Aspirate?
  - Too much tissue trauma (also heparin and lovenox)

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#### 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

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### 3. Complications of Diabetes:

#### a. Diabetic Ketoacidosis (DKA):

- **Cause:** anything that increases blood sugar (infection, illness, skipping insulin) can throw a client into DKA
- may be the 1<sup>st</sup> 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 CO<sub>2</sub> to compensate for the metabolic acidosis). Also, as the client becomes more acidotic the LOC goes down.

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## 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 arrhythmias)
- 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.

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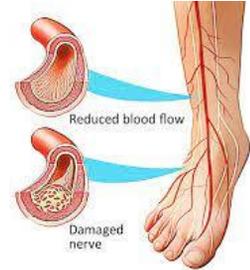
**Hyperosmolar hyperglycemic nonketosis (HHNK) or Hyperosmolar hyperglycemic nonketosis coma (HHNC):**

- 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 Kussmaul's respirations?
    - Happens in Type II => no ketones, no acidosis, no Kussmaul

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 BETWEEN 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