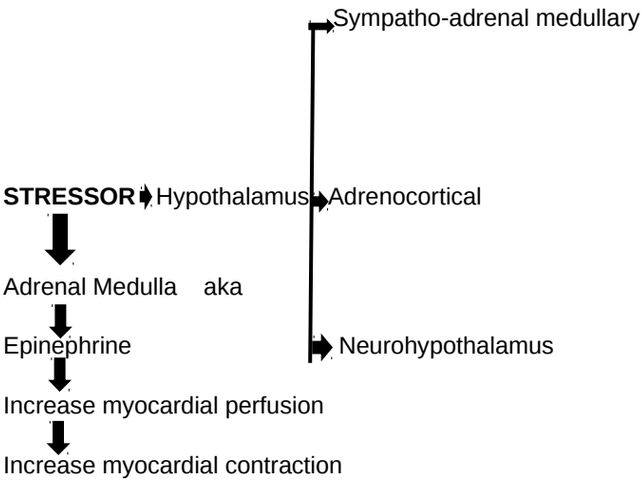
MEDICAL SURGICAL



SYMPATHO-ADRENAL MEDULLARY

⇒ Diaphoresis, ↑ BP, ↑ RR, ↑ Rate & depth of respiration, pallor, cold clammy, weakness, anorexia, oliguria, anuria, ↑ visual acuity, transient hyperglycemia

⇒ **SECRETE:** VC= peripheral arterioles
- **cathecolamines**
- **Epinephrine/Adrenaline** VD= coronary artery
- **Norepinephrine/ Nor-Adrenaline =** VC

EPINEPHRINE

⇒ Dilate pulmonary vessel

⇒ release smooth muscle bronchiole → dilate → hyperpnea

⇒ constrict peripheral arterioles → ↑ peripheral resistance → BP increase

⇒ constrict arterial skin → pallor

⇒ **GLYCOGENOLYSIS** (LIVER) **glucose**

SYMPATHETIC NERVE → sweat gland → sweat

- GIT → gastric secretion → gastric motility
- Urinary bladder M → bladder muscle relax
- Urinary sphincter → close
- Pupils → dilate → visual acuity

Dilate: **mydriatic**
Constrict: **myotic**

GLUCOCORTICOIDS/ steroid
↓
Gluco- NEO- genesis (formation of new glucose) → fats, CHON

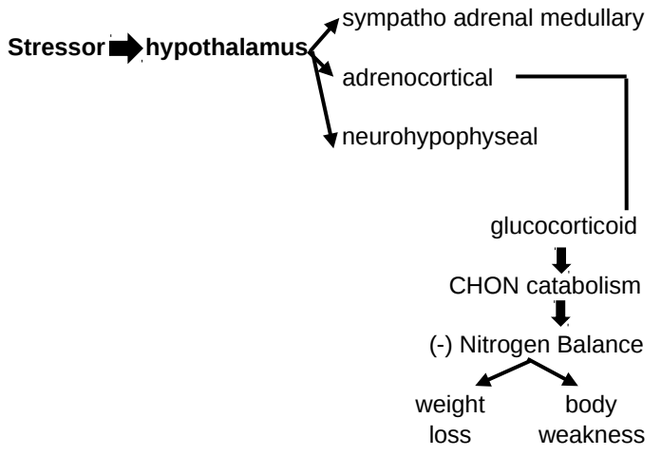
MINERALOCORTICOIDS
↓
Aldosterone
↓
Na & H_2O retention
K excretion

If under STRESS
- **Oliguria-** <400mL for 24h
- **Anuria-** <100 ml for 24h
- **Urinary Suppression-** without UO

PHASES OF CARBOHYDRATE METABOLISM

⇒ **Anabolism**

⇒ **Catabolism**

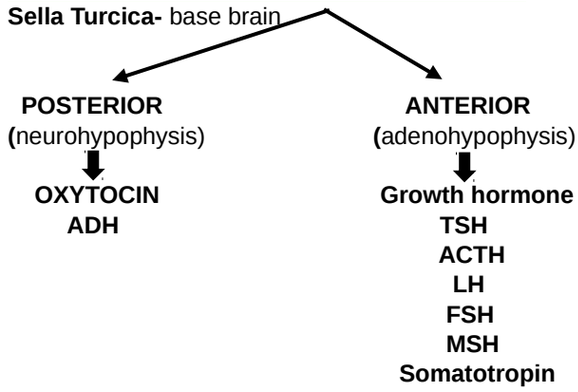


Intake of protein= **Body expenditure**

***Reality:**
ANABOLISM > CATABOLISM
(+) N balance
↓
= more weight gain

HYPOPHYSIS CEREBRI/ PITUITARY GLAND

Sella Turcica- base brain



Stress Response
- **aka** Sympatho Medullary Response or
- Sympatho Adrenal Medullary Response

ENDOCRINE DISORDERS

⇒ **hyperactivity (**hypersecretion)
- tumor within /outside the gland
- failure of kidneys to excrete the hormone
- failure of liver to detoxify/ deactivate the hormone

⇒ **hypoactivity** (hyposecretion)
- congenital absence of 1 gland
- surgical removal of the gland
  - **parathyroidectomy**
  - **adrenalectomy**
  - **idiopathic atrophy of gland**

↓ **APG**

⇒ **PH. dwarfism**

⇒ **FROHLICK's SYNDROME**
- ✓ dwarf, obese, mental retardation, genital atrophy

⇒ **SIMMOND's DISEASE**
- ✓ wizened old man (young but looks old)

⇒ **PITUITARY CACHEXIA**
- ✓ premature senility (absence of spermatogenesis)
- ✓ amenorrhea

↑APG
- ⇒ **ACROMEGALY (**after closure of epiphyseal line)
- ⇒ **GIGANTISM (**before closure of epiphyseal line)

GIGANTISM- prolongation & elongation of long bones
ACROMEGALY
- ⇒ **Hypertrophy**
  - bone thickness
  - deepen voice
  - **prognathism**/ protrusion of jaw
  - lengthening of chin
  - big nose, big ears, thick lips
  - separation of lower teeth- because of overgrowth of mandible
  - **spade-like fingers**
  - enlargement of visceral organs- spleen, liver

Medical Management
- ⇒ **SOMATOSTATIN/** Sandostatin (Ocreotide)
  - ✓ inhibit production of GH

DIABETES INSIPIDUS
- ⇒ disorder in water metabolism
- ⇒ due to ↓**ADH**

prevents renal tubules to reabsorb H_2O

POLYURIA----- 5 to 29 L/ day
Polydipsia **Diluted** ↓**SpGr** ↑**Serum Osmolality**
↓
↑**Na**
(135-145 mEq/L)

Medical Management:

OILY PREPARATIONS
- ❖ **PITRESSIN TANNATE**
- ❖ **Vasopressin** (potent Vasoconstrictor)
  - give by **deep IM**
  - to prevent lipodystrophy, rotate site of administration

LIQUID PREPARATIONS
- ❖ **Desmopressin Acetate**
- ❖ **Lypressin**
  - given by **nasal spray**
  - **Monitor BP** when giving Pitressin, will cause HPN

***First Priority Nursing Responsibility when giving Desmopressin Acetate:**
- ⇒ **Maintain AIRWAY PATENCY** (bec it is given nasal)

CLOFIBRATE (Atomid S/ Clo5)**-** antilipemic drug
- ⇒ not only reduce cholesterol
- ⇒ also has **antidiuretic effect**

SIADH
- ⇒ ↑**ADH**

fluid retention ➡ ↑**IV volume** ➡ **BP**↑
↓
electrolyte dilution
↓
Dilutional Hyponatremia
Dilutional Hyponatremia
↳ into cell ➡ brain tissue ➡ cerebral edema
↳ cellular dehydration

***All are sx of SIADH except: LEG EDEMA**

↑**renal perfusion** ➡ ↑ **GFR** ➡ ↑**UO (**therefore no leg edema)
*also there is dilutional hyponatremia, when you reduce Na, you excrete fluid

DIABETES INSIPIDUS
- ⇒ **nephrogenic-**use of diuretics
- ⇒ **neurogenic**

*hyperactivity- give **Cobalt therapy,** limit hypophysectomy
*Hypoactivity- **supplement**

↑ **ADH** ➡ **~~inhibit~~** **DEMECLOCYLINE/ Declomycin**

or Tetracyline (antimicrobial antibiotic, will inhibit production of ADH)

PTH- essential for reabsorption of Ca & excretion of Phosporus by renal tubules
- essential for blood coagulation
- regulate cardiac rhythmicity

HYPOACTIVE PARATHYROID- hypocalcemia/ tetany (uncontrolled spasms & hyper irritability)

Tetany
- **Chvostek's-** tap facial nerve (below temple front of ear) see if there's **unilateral spasm of facial muscle**
- **Trosseau's-** occlude blood flow to extremity for 1-2 mins, observe for **carpopedal spasm**

Normal Ca: 4.5-5.5 mEq/L / 8-10 mg/dL

***Which food item is allowed for patients with tetany?**
- ⇒ **Seafood/ seashell**
- ⇒ **NO dairy products** such as milk and cheese, cabbage, broccoli, sardines, tuna, mackerel, yolk- **very rich in Phosphorus**

↓**calcemia** ➡ ↑**phosphatemia**
↑**calcemia** ➡ ↓**phosphatemia**

Medical Management:
- ❖ Ca Lactate
- ❖ Ca Carbonate
- ❖ Ca Chloride 10%
- ❖ Ca Gluconate

***When is the best given time for Ca preparation?**
- ⇒ **AFTER MEAL**
- ⇒ any Ca preparation will stimulate release of hormone gastrin (hormone that stimulates HCl production)

For Ca to be absorbed, Vitamin D is needed.
Commercially Prepared Vitamin D:
- ♥ **DIHYDROTACHYSTEROL**
- ♥ **Calciferol** → **Ergo**, **Chole**
- ♥ **Rocatriol/ Calcifidiol/ Calcidiol**

***E-** tocopherol
***K-** Menadione, Phytonadione

- **CUSHING's SYNDROME**
- **Addison**
- **Conns Disease**

} Adrenal Cortex

- **Pheocromocytoma-** Adrenal Medulla

CUSHING's SYNDROME
- ⇒ hyperactive adrenal cortex
  ↓
  ↑ **SUGAR hormone**
  SALT hormone
  SEX hormone

→ fat

1. Glucocorticoid/ Steroid → gluconeogenesis → CHON

CHON, CHO, fat metabolism

sx: **Hyperglycemia**

Fat ↑ → rate of gluconeogenesis → abnormal fat deposits

↑ lipolysis → ↓ **moon facies, buffalo hump**

CHON → ↑CHON catabolism → tissue starvation

↓

Muscle wasting

↓

Negative Nitrogen Balance

↓

Truncal Obesity
but small and slender
extremities

Buffalo Hump- fatty deposits in your neck and supraclavicular area

2. Adrenal Cortex

- **Aldosterone-** fluid & Na retention

Salt- fluid retention

↓

IV volume

↓

Hypervolemia

↓

↑ BP

* ↑Na, P but ↓K

3. Androgen
Sex Hormones

⇒ Virilization
⇒ Masculine
⇒ Hirsutism
⇒ Oily w/ acne

Medical Management:
cortisol inhibitors:

⇒ amino gluthetimide
⇒ Trilostane
⇒ Metyrapone

ADDISON's DISEASE

↓ **SUGAR hormone** → hypoglycemia
SALT hormone → ↓ → vol → hypovolemia
BP
SEX hormone Na= ↓ , K=↑
-loss of axillary & pubic hair
repolarization → myocardial

↓

contraction irritability

↓

dysrhythmia

↓

cardiac arrest

*level of hormone controlled by **feedback mechanism**

↓ steroid/ sugar → anterior pituitary gland stimulated

↓

↑ACTH

↓

Melanocyte Stimulating effect

↓

Tan complexion- "**BRONZE SKIN"**

Medical Management:

⇒ Prednisone
⇒ Dexamethasone (Decilon forte)
⇒ Hydrocortisone (Solu-cortef)
⇒ Betamethasone (Decadron)

CONN's DISEASE / Primary Aldosteronism

⇒ adenoma of adrenal cortex (benign tumor)

PHEOCHROMOCYTOMA **(**adrenal medulla)

*What will happen hyperactive gland?

⇒ ↑**aldosterone** in Conns
- s/sx: fluid retention

⇒ ↑**cathecolamines, Epi, NE** in Pheochromocytoma
- s/sx: tachycardia, pallor etc

5 H's of PHEOCHROMOCYTOMA

⇒ **HYPERTENSION**
⇒ **HEADACHE**
⇒ **HYPERGLYCEMIA**
⇒ **HYPERMETABOLISM**
⇒ **HYPERHIDROSIS (**Epinephrine causes profuse sweating)

Surgical Management:

⇒ Adrenalectomy
⇒ Cobalt Therapy

Laboratory Test to primarily diagnose pheochromocytoma

❖ **VMA- Vanillylmandellic Acid** (silent y)
- ✓ evaluate level of catecholamine in blood & urine
- ✓ **Blood= 0.2 – 0.9 mg/dL**
- ✓ **24h Urine= 0.2 – 7 mg**

THYROID DISORDERS

***T**hyroid- anterior aspect just below larynx
*T3- triiodothyronine
*T4- thyroxine

Plasma:

⇒ iodide + tyrosine (aa) = **thyroglobulin**
(storage form of thyroid hormone)

↓

T3 & T4 (circulation)

PG (mastergland) → APG- release several trophic hormones
→ TSH → thyroid gland → T_3T_4
→ ACTH → adrenal cortex → SSS hormone

1. **PBI- Protein Bound Iodine Test**
- ✓ blood test to evaluate for amount of iodine attached to the protein molecule of the blood
- ✓ **Normal Value:** 4.8 mcg
  - ➢ ↑ hyperthyroidism
  - ➢ ↓ hypothyroidism
- ✓ How do you prepare patient?
  - ➢ **2-3 days,** see to it that client is not taking any food/ drug containing iodine (seafood, iodized salt etc.) before the blood test
  - ➢ **Cough syrup** is also rich in Iodine

2. T3 & T4 determination
T3= **70-170 mcg %**

❖ more potent than T4, don't bind in protein
❖ can readily influence cell

T4= **4.7-11 mcg %**
***result** is directly proportional to thyroid function

3. TSH test

⇒ inversely proportional to thyroid function because of negative feedback mechanism
⇒ **TSH= 0.4-6.11 mcg/ml**

↑**TSH** ↓T_3 T_4 **=** send messages to APG for APG to be stimulated to release TSH

⬇TSH ⬆T_3 T_4 = send messages to APG to inhibit prod'n of TSH

***TSH level is high-** hypothyroidism
low- hyperthyroidism

***TSH stimulates production of thyroid hormones.**
Low TSH:
*thyroid gland is making too much thyroid hormone, which is suppressing the TSH

High TSH:
***thyroid gland is not making enough thyroid hormone

4. **BMR Determination**
- ⇒ evaluate O_2 consumption when client is at rest (basal)
- ⇒ placed on NPO for 12 hours
- ⇒ then clamp nostril

5. TBMR –Theoretical Basal Metabolic Rate
- ⇒ **Pulse Pressure + PR/min – 111**
- ⇒ **ex: BP: 170/110 = 60 + 92 -111 = ⬆41**
- ⇒ **Normal: 20-30**
- ⇒ not reliable, hypothetical

6. Radioactive Iodine Uptake
- ⇒ evaluate amount of RAI^{131} "cocktail" stored by the thyroid gland & excreted by your kidney
- ⇒ **Uptake: 15-40%**
- ⇒ **Urine: 40-80%**
- ⇒ Prep: no foods or drugs containing Iodine

PO RAI^{131} "cocktail" ➡ 24h urine
(8 millicuries)
⬇
Scanner
⬇
Evaluate RAI^{131} in urine

liquid, unpleasant **brassy** taste that is why it is called cocktail

⬇0.78 → **Hypo thyroidism**
⬆4.6 → **Hyper thyroidism**

- ⇒ If normal thyroid gland, **urine** should contain only **3.2-6.4 millicuries** (40% of 8 & 80% of 8)
- ⇒ **Uptake** should be **1.2-3.2 millicuries (**15% of 8 and 40% of 8)
- ⇒ **Uptake** is directly proportional
- ⇒ **Urinary Excretion** is inversely proportional

7. Thyroid Scan
- ⇒ evaluate the amount of RAI^{131} stored by thyroid gland to determine size, shape & function of thyroid gland

A. ONSET sx
- ❖ **Cretinism**-childhood
- ❖ **Myxedema-** adulthood

B. CAUSE
- ❖ **Primary-** Thyroid Gland ➡ T_3T_4
- ❖ **Secondary-** Hypoactive APG ➡ ⬇TSH

***Is it possible for a client who has dwarfism to have hypothyroidism? **YES, Secondary cause (APG)**

Sx of Hypothyroidism:
- ⇒ stunted growth & development
- ⇒ delayed onset of puberty
- ⇒ mentally sluggish
- ⇒ poor memory
- ⇒ unable to tolerate extreme cold weather
- ⇒ Weight gain

***S/sx specific for Hypothyroidism: MYXEDEMA**
Patho Myxedema
⬇**T3 & T4**
⬇
⬇ Activity of sebaceous gland
⬇
accumulation of mucopolysacharide
⬇
non pitting face
dry/waxy edema
enlarged thickened skin



Medical Management for Hypothyroidism:
- ❖ **LEVOTHYROXINE (**thyrax, eltoxin, Synthroid)
- ❖ **Thyrolar, Euthroid (**Liotrix)
- ❖ **LIOTHYRONINE** (Cytomel)

GRAVES DISEASE synonymous with
- ❖ **Basedow Disease**
- ❖ **Parry's Disease**
- ❖ **Thyrotoxicosis** (toxic Goiter)

LATS- Long Acting Thyroid Stimulator

LATS ➡ gammaglobulin → iodine accumulation
→ thyroid hyperplasia

EPS- Exopthalmos Producing Substance

EPS ➡ APG
EYE SIGNS:
- ❖ **Exophthalmos-** protrusion of eye balls
- ❖ **Lid lag** (sleepy eye)
- ❖ **Proptosis**- downward displacement of eyeball
- ❖ **infrequent blinking**
- ❖ **fixed stare**
- ❖ **Periorbital edema**
- ❖ **VON GRAEFE's sign-** failure of eyelids to follow he movement of eyes when patient looks down





Von Graefe's sign
• The upper eye lid lags the eyeball as the patient is asked to look downwards.



HYPERTHYROIDISM

- ⇒ ⬆T3 & T4 - hyperactive, hypermetabolic
- ⇒ Overexcitability of SNS- **VS above normal,** tremors, palpitations, diaphoresis, nervousness
- ⇒ ⬆appetite ➡voracious but losing weight
- ⇒ daily pimple sign
- ⇒ infrequent blinking
- ⇒ a fixed stare

*in mountaneous area, **endemic/ non toxic goiter**
- ❖ goiter due to iodine deficiency

Treatment Modalities

1. Anti-thyroid
- ⇒ block synthesis of T3 & T4
- ⇒ **Tapazole/ Methimazole**
- ⇒ **PTU- Propylthiouracil**
- ⇒ **Neomercazole/ Carbimazole**

Prolonged use: AGRANULOCYTOSIS

- ✓ patient can complain of sore throat (indicate URTI), dyspnea (LRTI) or fever

Best parameter to evaluate toxicity to PTU: DIFFERENTIAL COUNT, if not on the choices, CBC

2. Iodine Preparations:
- ⇒ **Lugol solution (SSKI/ KISS)-** reduce vascularity, ⬆firmness of gland, promote storage of T3 & T4

3. Administer Adrenergic Blocking Agent
- ⇒ **Propanolol**

4. RA^{131}

5. Surgery
- ⇒ **THYROIDECTOMY**
- ⇒ **Radical/ Total Thyroidectomy-** entire lobes (left & right)
- ⇒ **Partial/ Subtotal Thyroidectomy-** 5/6 of 2 lobes
- ⇒ **Sistrunk**- for thyroglossal cyst
- ⇒ **Thyroid Lobectomy**
- ⇒ **Isthmusectomy**

Nursing Interventions:
- ❖ **DIET: HIGH CALORIC DIET- ⬆ CHO, CHON, PUFA**
- ✓ to restore the loss glycogen reserve
- ❖ no colas, caffeinated beverages, no tea (stimulant) except for green tea (non stimulating)
- ❖ Quiet & calm, restful environment to provide physical and mental rest.
- ❖ tell client that even with treatment/ surgery, eye signs will not completely regress, it's for life.

Presence of Eye sign:

⬆ **corneal dryness** ➡ ulceration ➡ blindness
- ⇒ artificial tears
- ⇒ saline
- ⇒ dark eye glasses
- ⇒ remind to **elevate head** to reduce periorbital edema

*__After total thyroidectomy, what will be the basis of all nursing problems and diagnosis?__ r/t hypothyroidism

PREOPERATIVE NI:
- ⇒ Days before surgery:
- ⇒ Prepare psychologically & physically, explain what, how, why it is to be done. (finding exact words given by the doctor)
- ⇒ **Common cause of fears pre-op:** anesthesia, death, losing vital part of the body, financial
- ⇒ Tell to stop smoking and alcohol drinking
- ⇒ Reduce body weight
- ⇒ Correct electrolyte imbalance (Na, K, Ca)
  - ⬇ **Na➡⬇IV vol ➡⬇BP**
  - **K-** repolarization
  - **Ca-** depolarization
- ⇒ **Blood loss is 15-25%**

***Normal circulating blood volume:** 4-6L
-**NCLEX- 4-8L**
- ⇒ Also control blood sugar, if not,
  - increase **risk for post op infection**
  - **will delay wound healing**
- ⇒ A day before surgery, secure informed consent
- ⇒ Place on NPO, to prevent **intraoperative aspiration**
- ⇒ Teach and demonstrate right technique (DBE & CE exercises)
- ⇒ Prepare arrival of cardiologist, anesthesiologist for CP clearance

On the day of surgery:
- ⇒ hygienic needs of preop client
- ⇒ accomplish preop checklist

Narcotic Analgesic
Phenergan (Promethazine)- anti-emetic, anticholinergic
Diazepam- evaluate RR

*__Most used Narcotic Analgesic preop?__
- ⇒ DEMEROL (Meperidine HCl)
- ⇒ Nubain

Antidote: Naloxone (Narcan)

Anticholinergic: Atropine Sulfate- reduce amount of bronchial secretions
- ⇒ evaluate PR <90bpm
- ⇒ If PR is 110/min- continue giving the drug

BURN ➡ stress ➡ activate response ➡ GIT ➡⬇g. motility ⬇ no peristalsis ⬅ paralytic ileus

Nursing Responsibilities
POST THYROIDECTOMY
- ⇒ **ESTABLISH A PATENT AIRWAY**
- ⇒ **Position: SEMI FOWLERS**

*****If there's high fowlers in the choices, don't it will cause strain in the neck muscles ➡ tension on the suture line

*Where's incision? **collar line/ curvilinear**
- ⇒ Turn side to side
- ⇒ Monitor VS until stable
- ⇒ Promote adequate nutrition

*****When do you allow to eat? upon return of gag reflex and patient is fully awake
- ⇒ Promote adequate elimination from the bowel to bladder

*when do you expect to urinate? **6-8hours after the procedure**
*****If it's 6 hours already and patient still has no UO, **check if bladder is distended**
- ⇒ To shorten recovery period- **encourage early ambulation** (boosts morale)
- ⇒ prevent post op complication

*****When will you allow patient to get out of bed? **As soon as possible as long as VS are stable**
*****What if 2 days post op, still unstable? **remind to still be in bed.**

CABG- anterothoracotomy
Incision: sternum down to xiphoid w/ stripping of sternum

Biliary- right Kocher/ right subcostal incision (ilalim ng kanan ng tadyang)
MRM- Y elliptical/ Halsted
Lumbotomy (urological procedure)- flank

COMPLICATIONS: specific
- ❖ **Tetany**
- ❖ **Hoarseness-** accidentally removed laryngeal nerve, **If only 1 nerve** is removed
- ❖ **Aphonia-** removal of bilateral (**2 nerves)** laryngeal nerves
- ❖ **Bleeding**
- ❖ **Respiratory Obstruction**
- ❖ **Thyroid Crisis/ Storm**

1 removed- no tetany
2 glands removed- mild sx of tetany
3-4 glands- (+) Chvostek's & Trosseau's

Hematoma- failure to administer KISS and forget to tie/ ligate bleeding post op

Bleeding Post Op

⇒ Check dressing, **slip hand under nape** to check for dampness
⇒ **If patient complains of TIGHTNESS & CHOKING sensation** it means 100% there is bleeding
⇒ **Evaluate VS-** when patient is bleeding, ⬇ BP and ⬆ PR (rapid, weak, feeble and thready)
⇒ Rapid RR but shallow, pale, cold, clammy, diaphoretic

Airway Obstruction Post Op

⇒ secondary to bleeding and accumulation of tracheobronchial secretions
⇒ Laryngospasm
⇒ Laryngeal edema
} only Management: **Prepare to emergency tracheostomy**

▪ to **establish an artificial airway** because of laryngospasm and edema secondary to anesthesia, hypocalcemia and surgical trauma

Thyroid Crisis/ Thyroid Storm

⇒ sepsis
⇒ because of stress preop
⇒ inadequate psychological preparation

Before Surgery

⬆ T_3T_4 ➡ given Tapazole PTU ➡ Euthyroid ➡ good candidate for the underlying infunction

Post Op stress ⟶ T_3T_4 (compensatory) mechanism

3 mos taking PTU ⟶ Euthyroid
(state of normal thyroid function)

S/sx of thyroid crisis:

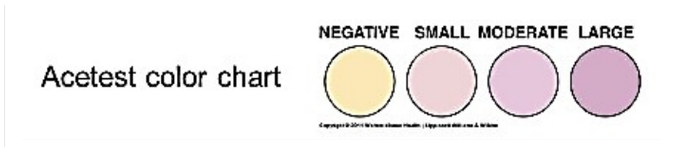
⇒ Tremors
⇒ Palpitation
⇒ Hypertension
⇒ **Early manifestation post op: FEVER w/ Tachycardia**
⇒ Thyroid storm

Management:

⇒ Give antithyroid
⇒ Beta blockers etc.

TESTS FOR DM

1. FPG- Fasting Plasma Glucose / FBS
2. RBS- Random Blood Sugar
3. PPBS- Post prandial blood sugar
✓ NPO 2h after giving diet consisting of atleast 100 gms CHO
4. Hgt- Hemoglucotest (CBG)
5. OGTT- most sensitive test
6. BT, CT
⇒ **Benedict's Test**
⇒ **Clini Test**
} tests for glycosuria
7. Acetest- test for ketonuria (from incomplete breakdown of fats)
⇒ acetest tablet- creamy white, **become PURPLE/LAVENDER** after 2 drops of urine = (+) ketonuria



8. HgbA$_1$C- Glycosylated Hemoglobin
⇒ **Lifespan of RBC-** 120 days/ 3-4 mos
⇒ **gives** control of glucose/ average blood glucose over the past 3 mos

T1DM (IDDM/ Juvenile)

⇒ aka **Brittle/ Labile DM** (unstable/ easily changed)
⇒ **Absolute Deficiency**
⇒ **prone to DKA**

T2DM (NIDDM)

⇒ **Maturity Onset/ Adult Onset**
⇒ Relative Deficiency
⇒ Obese, After age 40
⇒ **prone to HHNKS**
⇒ OHAs- Oral Hypoglycemic Agents
⇒ ff dietary regimen/ routine exercises

Cardinal Symptoms:

❖ **3 P's + Weight Loss + Body Weakness**
⬇
Manifestations results from Tissue Starvation

Manifestation result from failure of cells to utilize cells for energy: **Weakness, Hyperglycemia**

Manifestation that results ⬆ osmolarity secondary to hyperglycemia: **glycosuria, polyuria**

Manifestation that results from ECF Dehydration secondary to hyperosmolarity: **polydipsia**

TYPES OF INSULIN

RA	SA	IA	Slow Acting/ Long Acting
Aspart	Regular Insulin	NPH	Lantus
Lispro	Humulin R	Monotard	Glargine
Humalog	Novolin R	Humulin N	Humulin U
Novolog	Semi-Lente	Lente	Ultra Lente
	Crystalline	Novolin L	PZI
	Zinc	Globin	(Protamine
	MC- Actrapid		Zinc Insulin)
	Velosulin		

Insulin 3x a day- Short Acting
DKA/ Severe Ketoacidosis- Rapid Acting Insulin

INSULIN- IV/SQ, IM
***RA- 10-15 mins**
***SA- 30 mins- 1 hour**
NPH- turbid and cloudy
RI- clear, colorless
***Somogyi-** rebound hyperglycemia
***Dawn Phenomenon**

HYPOGLYCEMIA	DKA
Insulin overdose	Missed insulin dose
Prolong NPO	Infection
Vomiting	Stress
Very long interval the time you serve insulin & the serving of food	
Hunger pangs	Thirst
Double vision	Dim vision
Pallor	Flushed, warmth
Cold, clammy	Cherry red lips

Diaphoretic Temp below normal BP w/in normal Tremors **SYNONYM:** **Hyperinsulinism/ Insulin Shock**	Temp above normal BP is below normal **KUSSMAUL's**, air hunger respiration **ACETONE/ fruity odor of breath** **SYNONYM:** **Diabetic coma/ Hyperglycemia**
MGT: -2-3 slices graham crackers Fruit juice Soft drinks 1 tbsp of sugar 5ml honey/ karo (maple syrup) 4-6 pcs of hard candies	**MGT:** **Administer INSULIN** **RA insulin** **10-20gms CHO** **Epinephrine/ Adrenaline (1:1000) SQ** **Glucagon 1-2 mg IM** **IV of glucose H2O** **SIMPLE DIET:** 45-65% Complex CHO 15-20% CHON: 10-15% *complex so rate of absorption is slower Do regular exercise When exercising, **dec insulin** because during exercise muscles are utilizing glucose for energy

***BEST TIME TO TAKE INSULIN:**

⇒ SA: **60-90 minutes BEFORE MEALS**

Which is an appropriate nursing instruction to a diabetic client who is doing a regular exercises?

⇒ **REMIND THEM TO TAKE SNACKS IN BETWEEN EXERCISES** because exercises can cause hypoglycemia

MEDICAL SURGICAL CONT.
(NEURO CONCEPT)

Reticular Activating System
(Center of Brainstem)
***play a role in alertness, wakefulness and arousal

1. **LOC**

Lesion → **tumor**, **clot**, **abscess**

***lesion of brainstem**

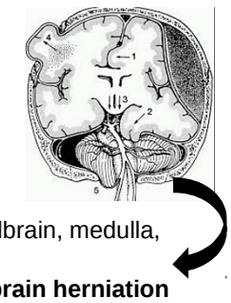
Metabolic Depression → **hypoglycemia**, **hypoxia (restlessness)**, **F&E imbalance**, **Toxic drugs & chemicals**

GLASGOW COMA SCALE

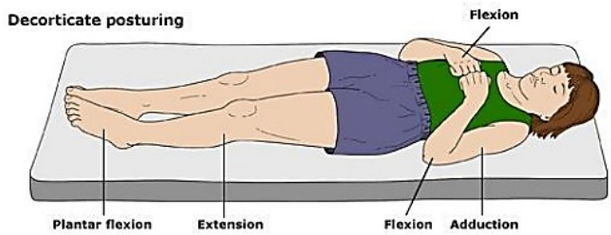
⇒ Eye Opening
⇒ Verbal Response
⇒ Motor Response

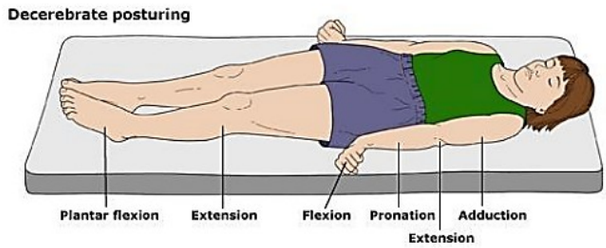
Interpretation

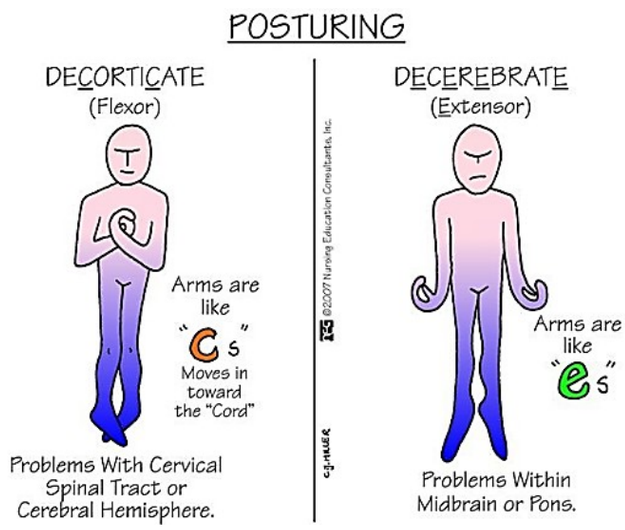
⇒ **7- COMA**
⇒ **3-4- DEEP COMA**
⇒ **5-6- COMA**



DECORTICATE- Spinal cord
DECEREBRATE- Diencephalon (midbrain, medulla, brainstem
***it can be found both in patient with **brain herniation**

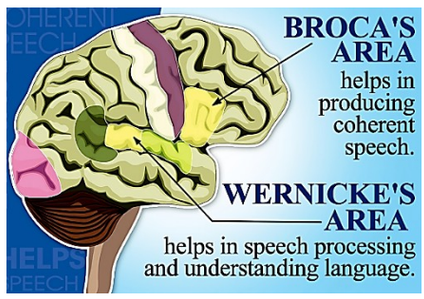






2. SPEECH PROBLEM

⇒ **Motor Speech Center= BROCA's Area**
⇒ **Sensory Speech Center= Wernicke's Area**



***Left Hemisphere- right handed**
*Right/Left Hemisphere- Left handed

Parietal= sensation (pain, pressure, touch, temperature)
= sense of sight

Temporal= hearing, balance, smell, memory
= speech, emotion, attitude, voluntary muscle activity, personality

APHASIA

- ❖ **Motor Expressive**
- ✓ inability to talk, to write (Frontal Lobe affectation)

- ❖ **Sensory Receptive**
- ✓ inability to understand written/ spoken words
  - a. **Visual Receptive-** occipital lobe
  - b. **Auditory Receptive-** temporal lobe

GLOBAL APHASIA → **Broca's** / **Wernicke's**] **affectation**

A. IPSILATERAL- sx on the side of lesion- **abnormal size of pupil**
B. CONTRALATERAL- sx on the opposite side of lesion

Right Sided Lesion/ Tumor= Focal Sx

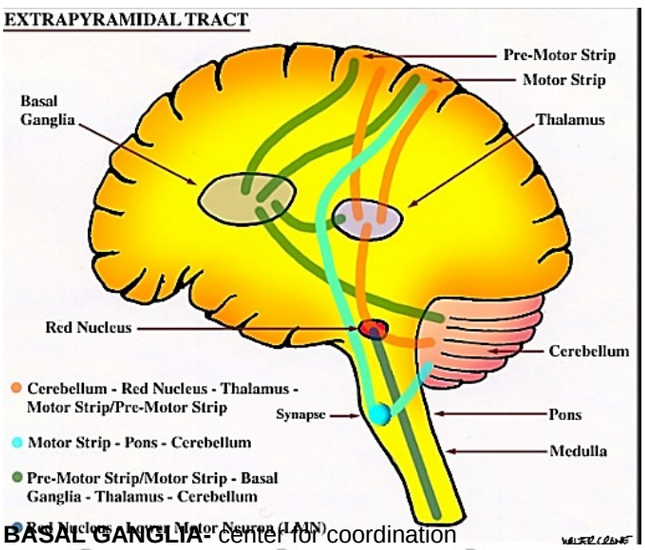


- ⇒ puffiness of right eye lid
- ⇒ drooling of saliva to the right } **IPSILATERAL**
- ⇒ sagging of right shoulder



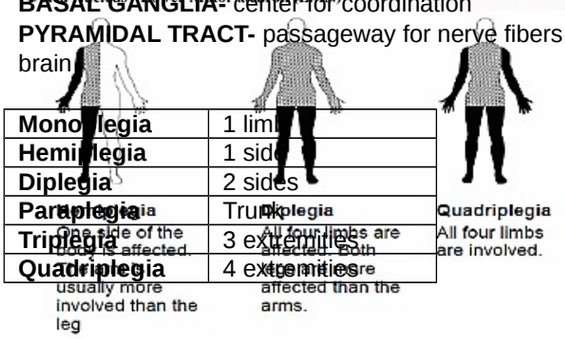
- ⇒ paresis (weakness) } L side } **CONTRALATERAL**
- ⇒ paralysis

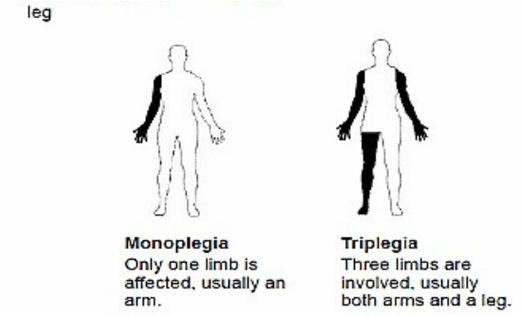
EXTRAPYRAMIDAL TRACT- control course voluntary movement



BASAL GANGLIA- center for coordination
PYRAMIDAL TRACT- passageway for nerve fibers from brain

Monoplegia	1 limb
Hemiplegia	1 side
Diplegia	2 sides
Paraplegia	Trunk
Triplegia	3 extremities
Quadriplegia	4 extremities





***Anisocoria-** unequal pupil size

3. **GAIT (walking)**

- ❖ **ATAXIC-**inability to steady with feet together= d/t lesion in cerebellum *unsteady gait
- ❖ **DYSTONIC-** irregular non-directive movement= Muscle atony
- ❖ **DYSTROPIC (Waddling Gait)-** with feet apart the body weight will move to side
  - **Muscle dystrophy, weakness of pelvic girdles, hip dislocation**
- ❖ **HEMIPHLEGIC-** foot dragging (pyramidal tract affectation) * spastic gait
- ❖ **SCISSORS GAIT-** short, slow steps, legs are alternating/ crossing each other
  - **Spastic Paralysis**
- ❖ **STEPPAGE-** Highly exaggerated steps (lower motor affectation)
  - most common sx of **Foot drop**







ataxic gait **dystonic gait**

ABDUCTOR-extensor
ADDUCTOR- stronger flexor- strongest muscles

TYPES OF ANESTHESIA
⇒ Noxocaine, Tetracaine HCl, Marcaine

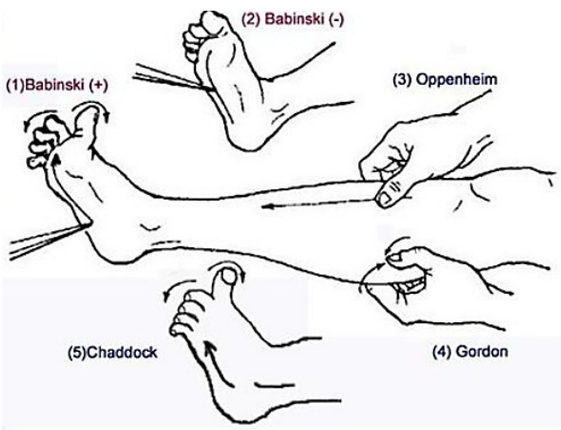
SHOOTING PAIN- during insertion of anesthesia

REFLEXES

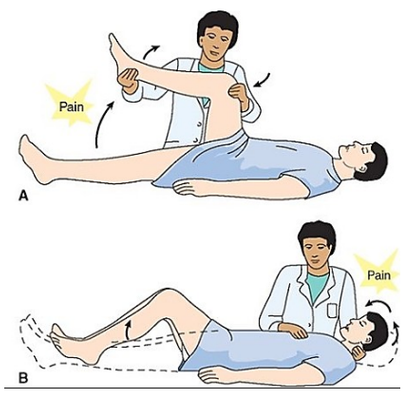
- **Knee Jerk/ Patellar Reflex**
- **Bicep-** arm flexion
- **Tricep-** arm extension
- **Babinski-** stroke the sole of the foot
  - **FLEXION** of toes- normal
  - **EXTENSION** of big toes- abnormal

GORDON's sign- squeeze/ press the calf muscle
⇒ Observe **DORSIFLEXION of BIG TOE-** pathologic

CHADDOCK's sign- stroke inner aspect of leg
⇒ Observe **DORSIFLEXION of BIG TOE-** pathologic



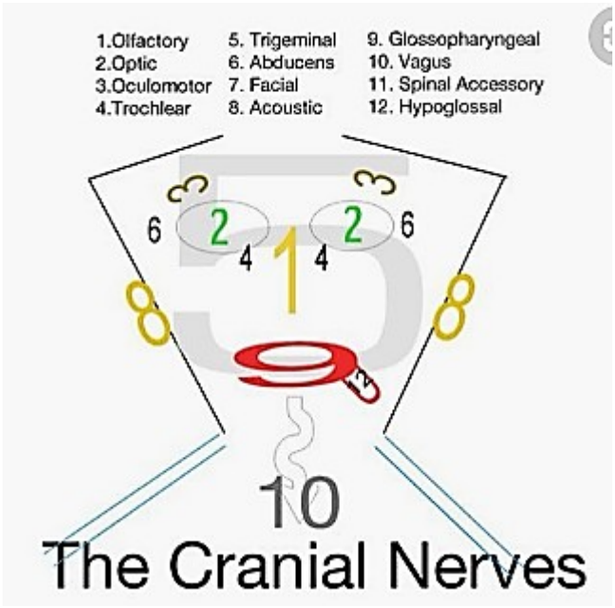
KERNIG's sign- pain & spasm in hamstring muscles
BRUDZINKI's- flex head forward- thigh flexes, ankle, knee



CREMASTERIC reflex- stroke inner thigh
⇒ Observe if TESTES **will rise**/ if scrotum elevate

5. **COORDINATION**
❖ **ROMBERG-** feet together, eyes closed, standing
⇒ if patient fall= **ATAXIA** (disequilibrium)

6. CRANIAL NERVES



Diagnostic Procedures:

1. LUMBAR PUNCTURE (Spinal Tap)
⇒ withdraw CSF fluid
⇒ Administer medication

Preparation
♥ void preprocedure
♥ empty bladder
♥ position client, cleanse
♥ C-position, fetal position
♥ secure consent

⇒ **L3-L4/ L4-L5/ L5-S1-** to prevent injury to spinal nerve (paraplegia)
⇒ **Local Anesthesia: LIDOCAINE**
- **1-2%- colorless**
- **4% aquablue** (topical) ➡ intubation

CSF

⇒ **TRANSPARENCY: CLEAR**
⇒ **COLOR: COLORLESS**
⇒ **AMOUNT: 90-150 mL**

***If its Yellowish CSF (**Xantochromia)-** means you have an **OLD BLOOD CLOT** inside your skull
*yellowish because derived from bilirubin and d/t RBC lysis and Hgb breakdown

CSF is sent to lab to check for level of:
❖ **CHON: N: 15-45 mg%**
❖ **Glucose- N: 50-80mg%** decrease in CNS infiltration / infection
  ✓ **Glycorrhachia-** presence of glucose in CSF
❖ **Chlorides- N: 118-132 mEq/L**
❖ **WBC- N: 0-81mL;** ⬆➡ Pleocytosis (elevated WBC in CSF)
❖ **Gamma globulin (IgG): N: 3-9% Elevated in cases of:**
  ✓ Brain tumor
  ✓ Multiple Sclerosis
  ✓ GBS

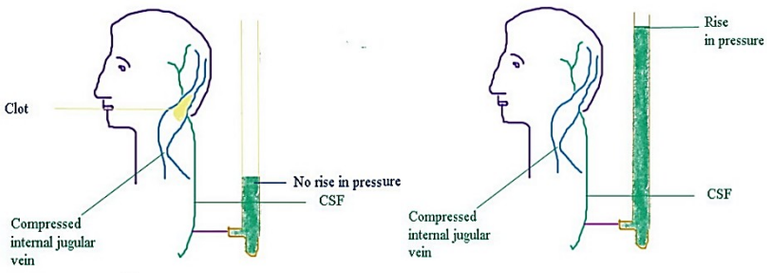
POST LUMBAR POSITION: FLAT ON BED 6-8hrs
❖ to prevent spinal headache d/t leakage of CSF into the subarachnoidal spaces

Latest Trend: Right after pulling spinal needle (L3-L4) put in PRONE POSITION for 30mins- 1hour

- ❖ It is possible CSF will leak from the site of puncture

2. QUECKENSTEDT (Jugular Compression Maneuver)

- ⇒ CSF pressure evaluation while jugular vein is compressed 6-12s
- ⇒ subarachnoidal obstruction
- ⇒ **Spinal Manometric "Dynamic"**- Old Quickenstedt



CSF Pressure

- ✓ cm of fluid
- ✓ mmHg

3. PNEUMOENCEPHALOGRAM

air/O2 ventricles x-ray

- ⇒ How do you prepare? prepare for LP, Dr. will aspirate **20ml of CSF** and then **will introduce 20ml O2**
- ⇒ **O2 will flow to brain ventricle,** it will also outline the ventricles
- ⇒ **Then** dr. will take series of x-ray

4. CEREBRAL ANGIOGRAM

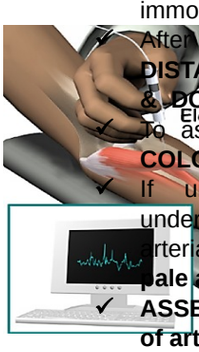
blood vessels x-ray

- ⇒ injection of dye → **conray** / **diodrast** → **into** → carotid / femoral arteries

***synonymous to Arteriogram/ Arteriography**

Nursing Responsiibilities:

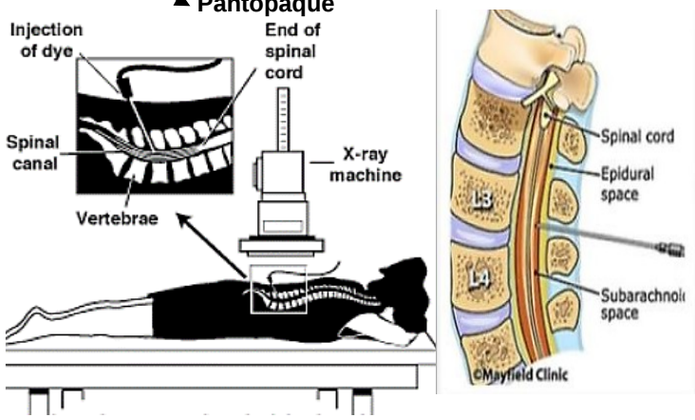
- ⇒ secure consent because there will be a puncture of major vessel
- ⇒ prepare local anesthetic agent
- ⇒ Knowing that there will be bleeding, place an **ICE CAP/COLLAR** around neck to prevent bleeding
- ⇒ since pt will be given dye:
  - ✓ **place client on NPO**
  - ✓ Ask for any allergy to seafoods
  - ✓ Tell client not to be alarmed with flushing of skin & warm sensation & metallic taste in the mouth
  - ✓ **Get a SANDBAG** for femoral arteriography over inguinal area to apply pressure over the femoral artery
  - ✓ Keep **legs in an extended position** & immobilize affected leg
  - ✓ After femoral arteriography, **CHECK PULSE DISTAL to the site of puncture (POPLITEAL & DORSALIS PEDIS)**
  - ✓ To assess for arterial insufficiency: **CHECK COLOR & TEMP of the affected leg**
  - ✓ If unfortunately, the patient who had undergone femoral arteriography experienced arterial occlusion, **The color of leg will be pale and temperature will be cold.**
  - ✓ **ASSESS PALLOR & COLDNESS- indicative of arterial insufficiency**
  - ✓ **Priority Nursing Responsibility to Cerebral Angiogram: ASSESS POPLITEAL PULSE**



*if theres a dye, patient should always be placed on NPO

5. MYELOGRAM

- ⇒ x-ray of subarachnoidal spaces after giving a dye
- ⇒ evaluate abnormalities in SC, its nerve and branches
- ⇒ Dye → **Myodin** / **Pantopaque**



- ⇒ **Method of Injection: Intrathecal/ Intraspinous**

Nursing Responsibilities: same with LP

6. **EEG- ELECTROENCEPHALOGRAM**

- ⇒ measurement of electrical activity of brain

Nursing Responsibilities:

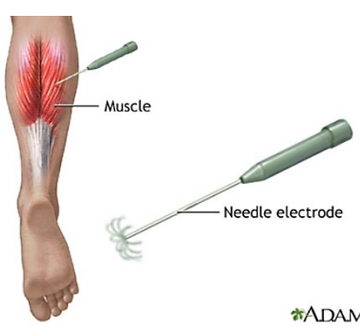
- ⇒ **No depressants** (**anticonvulsants):** 24h prior to surgery
  - ✓ Phenytoin (Dilantin)
  - ✓ Sodium Luminal (Phenobarbital)
  - ✓ Carbamazepine (Tegretol)
  - ✓ Clonazepam (Klonopin)
- ⇒ **No Stimulants** 24h prior to surgery- **caffeine, colas**
- ⇒ Will you place client on NPO prior to EEG? **NO**
- ⇒ Advise a regular diet prior to EEG
  - because state of hypoglycemia will cause **metabolic depression**
  - ⬇
  - **will turn non brain wave activity**
- ⇒ Render hair shampoo to ensure clean skull because electrodes won't stick if hair is oily/sticky
- ⇒ **Prep:** general, w/hold anticonvulsants

***What if done to a client w/ Epilepsy, Convulsion, Seizure?** Don't withhold anti-convulsants prior to EEG

- ❖ sudden stop/ withdrawal will cause seizure attack to occur frequently & for your client to experience status epilepticus

7. **ELECTROMYELOGRAM**

- ⇒ measurement of electrical activities of peripheral muscles done to diagnose dystrophy & peripheral nerve injuries.
- ⇒ measures nerve signal transmission to and through muscles



*In Philippines, no special preparation

- ✓ but explain to them how it is going to be done

- ✓ **where do you attach needles/ wires?** skull, peripheral muscles
- ✓ **Test** is done when normal at rest & when doing activity

*In America, required to secure a consent because you will puncture patient with needles

* Skeletal muscle relaxants such as **Flexeril** can affect test results and should be avoided for at least 2 days before the test.

8. **CT Scan**
- ⇒ scan is a combination of a series of X-ray images taken at different angles
- ⇒ use ionizing radiation

9. MRI
- ⇒ scan that uses magnetic fields and radio waves to produce a detailed image of the body's soft tissues and bones
- ⇒ MRI scan provides better contrast between normal tissue and pathologic tissue

10. Skull X-ray

*scanning w/o contrast- no preparation
w/ contrast- **THALLIUM, TECHNETIUM, NEOHYDRIN- IV**

***All dyes are considered NEPHROTOXIC-** prior to test, will evaluate GFR, serum creatinine, renal function

Serum Creatinine- W/o NPO
- ✓ **Most Definitive Test-** product of Muscle metabolism

BUN- with NPO
- ✓ because it is derived from protein & protein content will influence the results

PARKINSON'S (PARALYSIS AGITANS)

- ⇒ degeneration of basal ganglia d/t ⬆ supply of dopamine

Risk Factors:
- ❖ previous history of encephalitis
- ❖ Head trauma
- ❖ Smoking
- ❖ Hypertension
- ❖ Carbon Monoxide Exposure
- ❖ Use of contraceptive pills

PATHOPHYSIOLOGY

Neuronal degeneration of the substantia nigra of midbrain
⬇
⬇ inhibitory neurotransmitter
⬇
⬇ dopamine ➡ Essential for normal functioning of basal ganglia (center of coordination)
⬇
impairment of the extrapyramidal tract (basal ganglia)
⬇
imbalance in voluntary mvmt
- ⬇ weakness of the M of expression → ⬇ **MASK-LIKE facies (BLANK)**
- ⬇ Impairment of the M responsible for speech
  - ⬇ Drooling of saliva
  - ⬇ Microphonia → ⬇ slow voices, monotonous & poor articulation

TRIAD symptoms

Tremors
⬇
initially felt in upper extremities
experienced at **REST**
disappear w/ any purposeful movement

-**pill rolling movement of thumb against the fingers**
(earliest manifestation)

Rigidity
⬇
both extensor & flexor M
- **Micrographia** (manute illegible handwriting)
- **Cogwheel** (w/ every passive movement of extremities, there will be jerky motion)

Bradykinesia
(slow physical response)
or **dyskinesia** (difficulty in initiating movement)
-from supine to sitting
⬇
"Freezing Phenomenon"
⬇
sudden, brief inability to start movement or continue rhythmic & repeated movement

SHUFFLING/PROPULSIVE GAIT
- ⇒ stiff, rigid, stooped posture

Medical Management:
- ❖ can't give Dopamine, will not cross blood brain barrier
- ❖ **LEVODOPA-** metabolic precursor of dopamine

***enzyme decarboxylase,** will convert Levodopa to dopamine

Examples: Dopaminergic
- ✓ Dopar
- ✓ Larodopa
- ✓ **Carbidopa** (Sinemet)
- ✓ **Amantadine HCl** (symmetrel)
- ✓ **Bromocriptine Mesylate** (Parlodel)- stimulate the substantia nigra to release endogenous dopamine

***What drugs will antagonize effect of dopaminergic?**
- ⇒ **Pyridoxine (B6)**

To relieve Tremors & Rigidity:
- ⇒ **Trihexyphenidyl** (Artane)
- ⇒ **Benztropine Mesylate** (Cogentin)
- ⇒ **Biperidine HCl** (Akineton)

MAOI Inhibitors- prevent breakdown of Dopamine
- ⇒ Eldepryl, Carbex (Selegeline)

Surgical Procedure- done to interrupt nerve fibers pathway
STERIOSTACTIC surgeries- procedure requires use of an electrical stimulator
- ❖ **THALAMOTOMY-** destruction of thalamus
- ❖ **PALLIDOTOMY**- destruction of globus pallidus (located deep inside basal ganglia)

Pertinent Nursing Diagnosis:
- ⇒ Impaired Physical Mobility
  - comprehensive regular exercise program to improve muscle tone and strength
  - prevent contractures
  - give them something to hold on
- ⇒ Impaired Self Esteem
- ⇒ Risk for aspiration
- ⇒ Impaired Verbal Communication
- ⇒ Self-Care Deficit
- ⇒ Risk for Constipation
- ⇒ Risk for Physical Injury

MYASTHENIA GRAVIS

- ❖ autoimmune failure transmission of impulses at the myoneural junction d/t blockage / destruction in AcH receptor

Axon- will release an excitatory neurotransmitter (AcH)
⬇
will bind with AcH receptor of the muscle fibers
⬇
because of attachment of AcH to AcH receptor
⬇

MUSCLE WILL CONTRACT

PATHOPHYSIOLOGY

production of antibodies
↓
will destroy/ block AcH receptors
↓
↓ uptake/ concentration of AcH
↓
↓ # of functioning AcH receptor
↓
Muscle will not contract
↓
will become atonic, weak, paralyzed

EARLY STAGE

- **Ocular M** → **PTOSIS** (drooping of eyelids)
  → **DIPLOPIA**

weak & paralyzed → lingual M ➡ difficulty in swallowing, mastication/ chew
→ facial M ➡ **"SNARLING SMILE" (**teeth are clenched & jaw tends to hang open)

LATE STAGE

- **weakens Respiratory muscles (Diaphragm)**

↓
Respiratory Paralysis --- arrest
↓
State of Myasthenic Crisis

SURGERY:

- **THYMECTOMY**

suprasternal notch (guides u in chest examination) ➡ below is sternum ➡ THYMUS (upper border of sternum)

will originate T lymphocytes ⬅ will not grow w/o a functioning thymus
↓
remove the source of antibodies

THYMOMA- benign tumor of thymus gland
*When you reach adolescence, thymus gland is inactive, **Tonsil, Appendix, spleen** takes the role.

Plasmapheresis/ Plasma Exchange

- ⇒ connected to a separator/machine
- ⇒ will remove antibodies from blood in a client with Myasthenia

Sudden Onset Management:

- ⇒ **GOAL: Establish ARTIFICIAL AIRWAY** (ET/ Tracheostomy tube)
- ⇒ will also receive **Anticholinesterase/ Cholinergics**
  - ★ **Prostigmine/ Neostigmine-** increase concetration of AcH & # of functioning AcH receptor
  - ★ **Pyridostigmine (**Mestinon)

Observe Client for s/sx of **CHOLINERGIC CRISIS**

- ⇒ **opposite of SNS effects**
- ⇒ **Mgt: Anticholinergic/ ATROPINE SO_4/ Scopolamine/** Hyoscine

***Non compliant/ Underdose-** Myasthenic Crisis

***Overdose/ Prolong use of Prostigmine-** Cholinergic Crisis
MYASTHENIC CRISIS: symptoms of Respiratory Distress

DIAGNOSTIC TEST

- **TENSILON TEST** (Edrophonium)- anticholinesterase
  - ⇒ **short acting & very rapid**
  - ⇒ Give 8-10mg per IV
  - ⇒ **30s-1min** evaluate muscle tone and strength
  - ⇒ **If client had a chief complaint of:** facial paralysis, ptosis and disappeared (+) MG
  - ⇒ **Wait for another recurrence of M weakness =** 100% MG

MULTIPLE SCLEROSIS

Predisposing Factors:

- ⇒ autoimmune/ post viral disease
- ⇒ pregnancy & illness
- ⇒ use of toxic drugs & chemicals
- ⇒ Drugs containing **amphetamines** (reducing pills)

PATHOPHYSIOLOGY

demyelination of nerve fibers of brain and SC
↓
replaced with sclerotic patches of necrotic tissue
↓
formation of inflammatory exudate (responsible for inflammatory edema)
↓
Scar/ fibrosis
↓
Failure in transmission of electrical impulses
↓
CHARCOT's TRIAD
Tremors | **Nystagmus/ Strabismus** | **Scanning Speech**


fat like substances ➡ form sheath/ myelin sheath ➡ conduct flow of electrixcal impulses

*Because of Tremors: **SPASTIC/ ATAXIC GAIT**
NYSTAGMUS-movement of eye side to side
STRABISMUS- movement of eye towards the midline
DISCONJUGATE- away from midline
*Nystagmus- involuntary eye movement
*Strabismus- crossed eyes, eyes do not properly align with each other when looking at an object
*disconjugate:



- ⇒ result of **demyelination of CN 3-** Oculomotor
  CN 4- Trochlear
  CN 6- Abducens

CN3- eye accommodation, pupillary constriction
CN4- supply superior oblique muscle of eye
CN6- supply the lateral rectus muscle of eye

SCANNING SPEECH- difficult to pronounce 1st word/ syllable of a statement

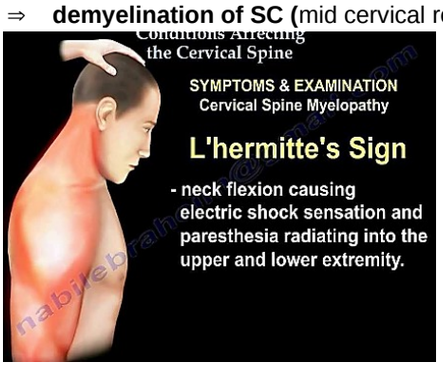
- ⇒ **demyelination of frontal lobe**

Loss sensation for pain, touch, temperature

- ⇒ **demyelination of parietal lobe**

L'hermitte's sign- bends head forward there is an electrical stimulation down the posterior trunk

⇒ **demyelination of SC (**mid cervical reticulae)



Demyelination of the white matter of frontal lobe

- ✓ there will be behavioral manifestation
- ✓ **Euphoria, Apathy** (unconcerned)

Diagnostic test:

- ❖ **LUMBAR PUNCTURE**
  - ➢ significant changes in CSF: **elevated protein & gamma globulin**
- ❖ **MRI**
  - ➢ evaluate extent of demyelination

Management: SYMPTOMATIC, SUPPORTIVE
Cause: Unknown

ABCR of Autoimmune Disorders

A- vonex
B- etaseron
C- opaxone
R- ebif

*prevent proliferation & activation of T cells

***Parkinsons: Incidence:** High in males (Freddie Roach)
***MS: Incidence:** Female 20-30 (alma Moreno)
***GBS: Incidence:** girl, boy sexes

GUILLAIN BARRE SYNDROME

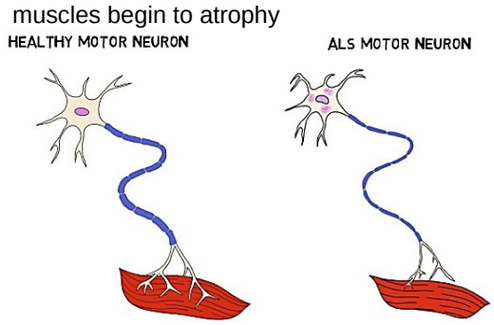
⇒ also a demyelinating DO

1. **Polyradiculoneuritis- CN**
   ⇒ Several cranial nerves causing paralysis: **Ocular, Oropharyngeal & Facial muscles**
2. **Demyelination of peripheral nerves causing**
   - ➢ **PERIPHERAL NEURITIS**
   - ➢ **ASCENDING PARALYSIS**
3. **Autonomic Dysfunction**
   - ➢ result of overexcitation/ underexcitation of both Sympathetic & Parasympathetic nerve fibers

***MS-** demyelinating disease of CNS
***GBS-** demyelinating disease of PNS

AMYOTROPHIC LATERAL SCLEROSIS (Lou Gehrig's)

⇒ degenerative disease
⇒ **degeneration of motor nerves**/ nuclei of anterior horn of SC, brainstem & upper & lower motor neuron
⇒ progressive muscle weakness, resulting in paralysis
⇒ connection between muscle & CNS is blocked, muscles begin to atrophy



*Causes: excessive exposure to **glutamate** may be one of the reasons that nerve cells (motor neurons) die in ALS
*excess of a neurotransmitter called glutamate builds up in the synapse causing eventual nerve cell death and subsequent muscle atrophy of the muscle attached to that nerve
*fasciculation (muscle twitching) & cramping- **early sx**

Upper Motor Neuron	Lower Motor Neuron
⬇ Loss voluntary control ⬇	
Hypertonicity of muscle	Hypotonic
spasticity	Flaccidity
No muscle atrophy	With muscle atrophy
Hyperreflexia	Hyporeflexia/ absent reflexes

Medical Management

- ❖ **Baclofen-** relieve spasticity
- ❖ **RILUZOLE/ RILUTEK- anti-glutamate,** inhibiting release of glutamic acid in CNS & protects neurons
  - prevent further deterioration of motor nerve fibers
  - taken on empty stomach, same time each day

CVA- CEREBROVASCULAR ACCIDENT (Stroke)

- **Other term for Stroke: APOPLEXY**
- ❖ There is cerebral ischemia causing cerebral infarct
- ❖ Cerebral anoxia of more than 4 mins- **irreversible cerebral damage**

2 Basic Pathology:

- ➢ **Occlusive-** embolism, thrombosis
- ♥ **Embolism as result of:** MI, endocarditis, dysrhythmia, fractured bone, malignant cell (CA)
- ♥ **Thrombosis as result of:** DM, atherosclerosis, hypertension, smoking
- ➢ **Hemorrhage**
  - ♥ **as result of:** long standing hypertension, aneurysm (ballooning/outpouching of weak walls of cerebral arteries)

Poorest prognosis
- **Epidural-** bet skull & dura
- **Subdural-** bet dura & arachnoid
- **Subarachnoidal-** arachnoid – pia matter

*arteries pulsate, veins do not for it is superficial

OCCLUSIVE

- ➢ **gradual** onset of sx
- ➢ **TIA (mini stroke)**
- ➢ **Sx:** nuchal/nape pain, paresthesia, lightheadedness, syncope (fainting), transient loss of speech & memory

HEMORRHAGIC

- ➢ **sudden** onset of sx
- ➢ aggravated by stress & physical activity

Ex: your mother is hypertensive for 10 yrs, you had a bitter conversation w/ your mom, you **suddenly** saw your mother experiencing severe unbearable HA to the point of collapsing and losing consciousness

***S/sx:** depend on the site of necrosis
EARLIEST MANIFESTATION OF CVA: associated with ⬆ICP------**RESTLESSNESS**

HIPPUS- alternate dilatation & constriction of pupil

Other signs of ⬆ICP

⇒ projectile vomiting
⇒ papilledema/ choked disc- edematous optic disc
⇒ HA/ **Cephalgia**

LATE MANIFESTATIONS
- **CUSHING's Triad**
  - ♥ **BP-** ↑ **systolic BP & Normal/** ↓ **Diastolic** ↓ **widening pulse**
  - ♥ **PR-** ↓ **P**
  - ♥ **RR-**
  - ♥ **Temp-** ↓ **initially** but ↑ as level of consciousness continuously deteriorate

CAUSES OF INCREASED ICP

Any condition that will:
1. ↑ **vol of CSF**
   - ✓ Hydrocephalus
   - ✓ Tumor obstructing flow of CSF
2. ↑ **brain tissue bulk/ size**
   - ✓ Meningitis } cerebral edema
   - ✓ Encephalitis
3. **Brain Abscess**
4. **Any factor that will** ↑ **cerebral blood flow will** ↑ **ICP**
   - ✓ coughing
   - ✓ sneezing
   - ✓ straining of stool

*when level of PCO_2 increases- state of **hypercarbia**
***when you retain CO_2 ➡ ↑RR ➡ hypercapnia**
***CO_2** has characteristics of **dilating cerebral blood vessel**
= ↑ **cerebral blood flow**
↓
cerebral congestion
↓
ICP will increase

TO REDUCE ICP:
1. DEXAMETHASONE (decadron)- only steroidal preparation that will cross BBB

2. MANNITOL
***reduce cerebral edema & brain tissue bulk
↓
HYPEROSMOLAR SOLUTION
↓
effect in fluid shifting: **IS- IV**
↓
↑ **renal blood flow & perfusion**
↓
↑ **UO**

3. MECHANICAL VENTILATION/ Ambu bagging
⇒ need to reduce levels of PCO_2 to promote hyperventilation
↓
↑ **RR**
↓
excrete retaining CO2
↓
↓ **ICP**

4. ANTIPLATELET AGGREGATORS
- **ASA- SE** if not bleeding; **prolonged prothrombin time**
- **Aspilet, Ascriptin**
- **Dipyridamole (**Persantine)- coronary vasodilator but for ↑ ICP; thrombolytic
- **Ticlopidine/ Ticlid**
- **Clopidogrel/ Plavix/ Clovix**

***Most common discomfort associated with Aspirin?**
⇒ **GI upset, N/V, abdominal discomfort**
⇒ **Take on a FULL STOMACH**

ANTICOAGULANT
- **Coumadin (PO) = Vit. K = Dx test: PT**
- **Heparin (SQ,IV)= Protamine SO_4= PTT/APTT**

Plasminogen Activator (Thrombolytics)
⇒ Streptokinase, Urokinase, t-PA, Abbokinase, Retaplase
⇒ **Antidote: Aminocaproic Acid/ Amicar**

Plasminogen activator ➡ plasmin ➡ will cause lysis/dissolution of blood clot

BRAIN TUMOR

Classified Accdg to Origin
⇒ from brain tissue- **Glioma**
⇒ from Cranial nerve- **Neuroma**
⇒ From brain covering- **Meningioma**

Accdg to Location
⇒ **Supratentorial**= cerebrum, anterior 2/3 of brain
⇒ **Infratentorial=** cerebellum, brain stem, posterior 1/3 of brain

***EARLIEST INDICATOR/ Manifestation of BRAIN TUMOR? PAPILLEDEMA,** because of tumor compressing optic chiasm

Treatment Modality
SURGERY
- **CRANIECTOMY/Craniotomy**
- **Radiation therapy**

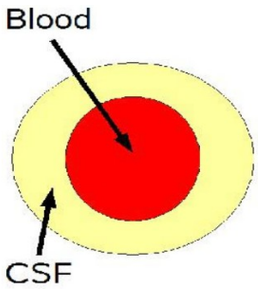
Incision: coronal/ butterfly incision- flap/ burr holes

DON'T's POST CRANIOTOMY:
⇒ don't place client on a shock position (tredelenburg) **can increase ICP**, abdominal contents can compress the diaphragm
⇒ What if a patient went into shock post craniotomy, how will you position? **BACKLYING W/ PILLOW** to prevent aspiration
⇒ If there is no shock, depends on the operation
⇒ To promote venous return after supratentorial surgery: **Elevate head upto 45° (SEMI FOWLER's)**
⇒ After infratentorial surgery to prevent compression of brain stem: **HEAD ELEVATED 10-15°, turn head to 1 side**
⇒ **Post craniectomy,** where will you turn? **Turning to unoperated to prevent compression of brain tissue**
⇒ After surgery there will always be a cerebral edema
⇒ If there is a need to turn the patient to operated side, **you can but not more than 20mins to prevent cerebral ischemia**

DON'T SUCTION YOUR CLIENT
⇒ will increase ICP
⇒ the mere suctioning will trigger cough reflex which can increase ICP
⇒ What if theres a need to suction? can be but through **oropharyngeal suctioning and not more than 10s**
⇒ **Why not nasopharyngeal suctioning?** any trauma to nasal mucosa can lead to CSF leakage

CSF Leakage:
- **HALO SIGN-** pale, yellowish

- ❖ get a gauze, moisten and saturate with the drainage in nose



DO NOT RESTRAIN YOUR CLIENT
- ⇒ client become agitated---⬆ ICP

DO NOT INSERT RECTAL TUBE/ RECTAL THERMOMETER
- ⇒ anything inserted in rectum- can result in vagal stimulation (**Parasympathetic response)**
- ⇒ to prevent severe hypotension & bradycardia

REMIND NOT TO BE CONSTIPATED
- ⇒ **straining** can increase cerebral blood flow----⬆ICP

RADIOTHERAPY
- ❖ **External= Cobalt^{60}/ Tide therapy/ External Beam Radiation/ Linear Acceleration**

Nursing Responsibilities
- ⇒ Explain that it is a **painless** procedure
- ⇒ During exposure there will be **"pencil marking/ ink markings/ tattoo"** made on the skin indicating site to be exposed to cobalt
- ⇒ **Do not expose area to sunlight**
- ⇒ Avoid pressure over the site
- ⇒ Avoid local/cold/warm application over the site
- ⇒ For cleansing: **no soap, just water and pat it, don't rub**
- ⇒ No oil, no cream, no lotion to prevent probability of skin reaction
- ⇒ Observe for s/sx of **radiation sickness/ reaction manifested LOCALLY/SYSTEMICALLY**

LOCALLY
- ⇒ erythema
- ⇒ dryness of skin
- ⇒ loss of skin hair
- ⇒ blister formation
- ⇒ skin desquamation

SYSTEMICALLY
- ⇒ **Pancytopenia:** anemia, leukopenia, thrombocytopenia
- ⇒ **Sterility**

*__Bone Marrow & Gonads__ are very sensitive to radiation and radiation leads bone marrow depression and destruction of gonads

***Which of the ff is indicative of Radiation Reaction?**
- ⇒ **A reaction similar to 1st degree burn**

***IDA-** microcytic, hypoplastic
***Folic Acid Deficiency-** macrocytic

- ❖ **Internal**
  a. **Sealed-** radium seed/ radon seed, Cesium^{137}
  -a form of bead, wire, needle or seed
  b. **Unsealed- RA ^{131}, RA^{125}, RAgold^{198}, RAphosphorus ^{132}**
  -liquid preparation

SEVERAL WAYS:
1. **Implant-** intracavity (radium/radon seed)
2. **Brachytherapy-** intralesion/ intratumors
3. **P.O. (RA ^{131}, RA^{125})**
4. **intraarterial perfusion (RAgold^{198}, RAphosphorus ^{132})**

Nursing Considerations:
- ⇒ Isolate client or place in a private room
- ⇒ Post at door of room- **Radiation sign**
  -nurse should observe **placarding**
- ⇒ Anything in contact with client is radioactive
- ⇒ **All Excreta, urine, vomitus, stool** are all considered contaminated
- ⇒ Be very careful, wear pair of gloves throw directly to toilet bowl **and flush twice or thrice**
- ⇒ For you not to be exposed to Radiation:
  - ♥ **Time-** limit contact for not more than 30 mins
  - ♥ **Distance- 3-6ft** from the site of radiation
  - ♥ **Shielding-** lead apron
- ⇒ Prevent accidental dislodgement of Radium
  - ★ Place on complete bed rest
  - ★ Do not allow to use bed pan
  - ★ Insert indwelling catheter
  - ★ Render an enema prior to insertion of implant
  - ★ Avoid food that will stimulate defecation
    - ✓ high roughage/ fiber, **no fruits and vegetables**
- ⇒ If accidentally removed: **Pick it up w/ a long forcep,** rinse with saline & water & put inside the lead container

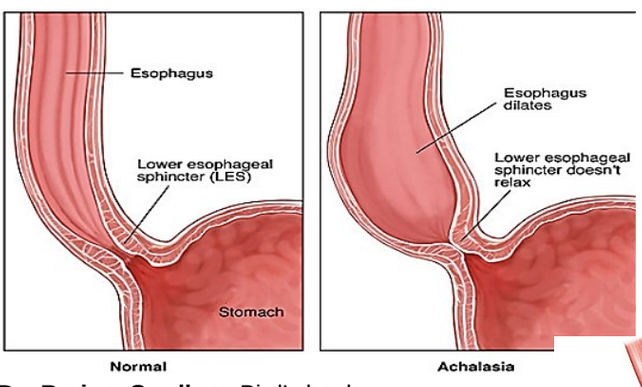
***Lead container** weugh 5-10kg

EFFECTS OF RADIATION in urinary
- ⇒ **Dysuria/ Burning sensation** upon urination may indicate **urethral atrophy** because even the normal cells are destroyed by isotopes inside canal
- ⇒ Cystitis- hematuria
- ⇒ shortening of cervical canal

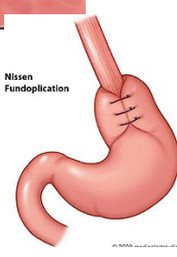
RAI half life: **7-10 days (radioactive isotope)**

DISORDERS AFFECTING DIGESTION & BOWEL ELIMINATION

Achalasia
- ⇒ synonyms: **Aperistalsis, Megaesophagus, Cardiospasm**
- ⇒ **degeneration of mesenteric plexus/ Auerbach plexus**
- ⇒ **deficiency/destruction of postganglionic neurons of the myenteric plexus**
- ⇒ Responsible for Peristaltic Activity: **innervation of myenteric plexus**
- ⇒ Food accumulate at the lower end of esophagus
- ⇒ **Cardiospasm-** failure of cardiac sphincter to relax, to allow food to enter stomach



Dx: **Barium Swallow-** Bird's beak



S/Sx:
- ⇒ Sternal

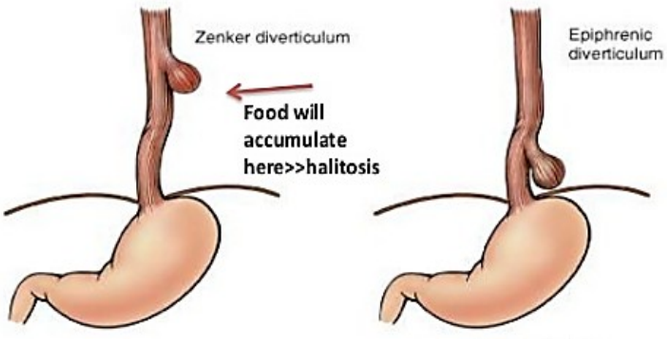
⇒ food accumulating in lower end will undergo decomposition process causing **Halitosis**
⇒ **Dysphagia-** difficulty in swallowing
⇒ **Odynophagia-** pain swallowing
⇒ **Regurgitation (**backflow) of undigested food---- there will be **Heartburn/Pyrosis**

Goal: relax LES
⇒ **Nitrates & CCB**
⇒ **Botulism Toxin**

ESOPHAGEAL DIVERTICULUM

⇒ **outpouching/ ballooning of weak wall of esophagus**

Risk Factors:
⇒ congenital
⇒ Chronic Esophagitis



Zenker/Pulsion Diverticulum- ballooning upper 3rd of esophagus
Traction Diverticulum- middle 3rd of esophagus
Epiphrenic diverticulum- lower 1/3

*s/sx same w/ achalasia
*What do you include in conservative nursing management for client with any esophageal DO?

Conservative Management:
⇒ **Always assume an UPRIGHT position** after eating
⇒ Remind to avoid eating/ drinking **2hrs before resting**
⇒ Because of esophagitis, **avoid irritants, bulky/coarse foods, spicy foods, very hot & cold drinks, caffeine and alcohol**
⇒ Maintain an ideal body weight
⇒ Advise **small frequent meals**
⇒ **For Halitosis: Alkaline oral wash,** mouth care

Specific Nursing Management:
⇒ increase pressure at LES, to increase advise **URECHOLINE** (Betanechol), **DOMPERIDONE (**Motilium)

Only Appropriate Management for Zenker
⇒ **excite esophageal pouch/sac** via central incision

Traction/Epiphrenic
⇒ **esophageal sac removal** via transthoracic incision

Achalasia Surgical Approaches:
❖ **ESOPHAGOMYOTOMY-** opening of esophageal muscle fiber
❖ **CARDIOMYOTOMY/ Heller's Surgery**
❖ **NISSEN FUNDOPLICATION-** the fundus of stomach used to wrap lower end of esophagus

How to meet nutritional needs?
- resort to extra oral feeding via **TPN/ Hyperalimentation –** contains glucose, lipids and amino acids
- **Method of administration: via PERIPHERAL/CENTRAL line (**subclavian vein)

***central if we need to give it more than 10 days**

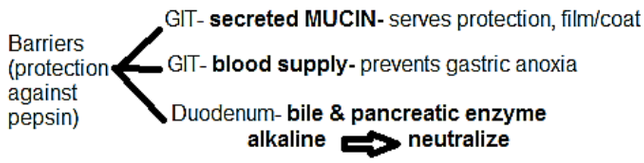
***TPN soln is HYPERTONIC with glucose content >10%**
⇒ regulate accurately the flow rate of TPN
⇒ If fast rate ➡ **HYPERGLYCEMIA**
⬇
there will be **HYPEROSMOLAR DIURESIS**
⬇
Glycosuria, Polyuria
⇒ If very slow rate:
✓ **not giving essential calories needed for day**
✓ you are giving a spoiled TPN solution, TPN is good only for 24hours
▪ To evaluate/assess risk for **HYPEROSMOLAR DIURESIS: CBG monitoring 3x/day**
▪ Monitor I&O, serum electrolytes

***If client is on TPN,** and next follow up is not available, **you can hook/administer D10W** to prevent risk for hypoglycemia
***If using** central line observe for s/sx of **venous thrombosis**
✓ **pain & swelling** at jaw, neck/ shoulder where your TPN catheter is inserted
*Which parameter would best evaluate effectivity of TPN solution? **BODY WEIGHT**

PEPTIC ULCER DISEASE

❖ **autodigestion**-acute hemorrhagic pancreatitis
❖ **erosion of circumscribed in the GIT d/t indigestion of HCl & Pepsin (**digest protein)

HCl & pepsin ➡ erosion ➡ circumscribed GIT



1. Poor Dietary Habit
▪ **Duodenal Ulcer-** common
2. Stress ➡ Parasympathetic ⬆ g. secretion, g. motility
3. Ulcerogenic
4. Gastric Stimulant- coffee
5. **Personality "A"-** highly perfectionist etc.
6. **Age** ⇅ **g. ulcer** = 40y/o, **d. ulcer**= 30-40
7. **Sex** ⇅ **g. ulcer** = both, **d. ulcer =** Male
8. **Smoking-** vasocontrict ----- **Gastric anoxia**
9. H. pylori- release enzyme w/c destroy gastric mucosa
10. **Blood type- "O"-**prone ulcer
"**A"-** cancers to stomach

GASTRIC ULCER	DUODENAL ULCER
Radiating L epigastric	Mid epigastric Radiate right epigastric
30 mins – 2h after meals	2-4h- after meals
Food- aggravate	Food- relieve
Vomit- relieved	Vomit- No
⬇ HCl-Hypochlorydia	⬆ HCl-hyperchlorydia
Hematemesis	Melena

(coffee-ground vomitus)	(black tarry stool)

Buffers → food / antacid

***Avoid RAW vegetables, condiment, coffee**
*emphasize regularity in eating

CHON → potent secretagogue → HCl
Diet: ↓CHON
⇒ to compensate for low CHON, **↑CHO**

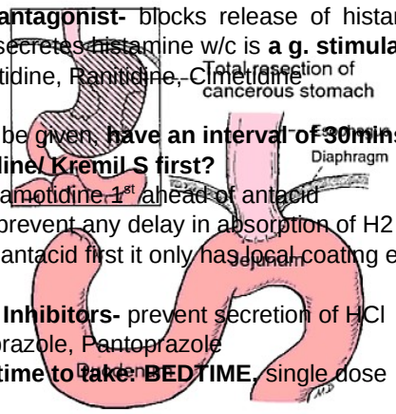
Fat content → stimulate intestinal mucosa → secrete hormone → **ENTEROGASTRONE** → ↓ g. secretion, g. motility

***Allowed fat: PUFA**
⇒ Those antacids containing:
⇒ **Al OH, Ca Carbonate= CONSTIPATION**
⇒ **Mg Oxide, Mg OH= LAXATIVE**

***When is the best time to take an antacid?
⇒ Old- an hour after a meal
⇒ **Take it an hour after a meal-** for food is about to leave stomach
⇒ Or **In between meals**
7AM ——— Antacid ——— **12NN**
10
⇒ **At night-** when level of HCl at its peak

Responsibilities when taking an Antacid:
⇒ **To be effective:** Take 1 tbsp of antacid / 2-3 tab
⇒ **Always follow it with water,** without water it will simply coat the gastric mucosa

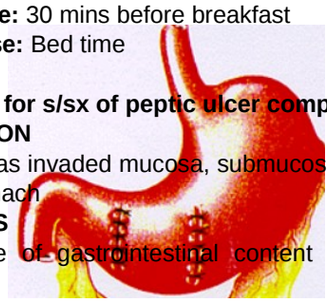
Medical Mgt
H2 receptor antagonist- blocks release of histamine by parietal cells (secretes histamine w/c is **a g. stimulant)**
⇒ Famotidine, Ranitidine, Cimetidine



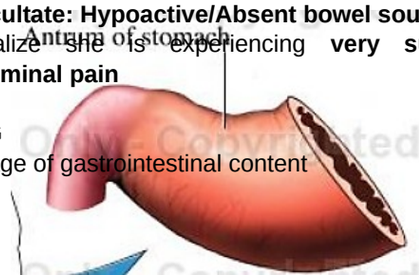
*If 2 drugs will be given, **have an interval of 30mins-1hour**
***Oral Famotidine/ Kremil S first?**
✓ Give famotidine 1st ahead of antacid
✓ it will prevent any delay in absorption of H2 blocker
✓ bec if antacid first it only has local coating effect

Proton Pump Inhibitors- prevent secretion of HCl
⇒ Omeprazole, Pantoprazole
⇒ **Best time to take: BEDTIME,** single dose

If **BID**
- **1st dose:** 30 mins before breakfast
- **2nd dose:** Bed time



Observe client for s/sx of peptic ulcer complications
1. PERFORATION
➢ ulcer has invaded mucosa, submucosa, muscularis of stomach
2. PERITONITIS
➢ leakage of gastrointestinal content to peritoneal cavity
S/sx:
- **Inspection: DISTENDED ABDOMEN**
- **Percussion:** Board-like Rigid Abdomen
- **Auscultate: Hypoactive/Absent bowel sounds**
- Verbalize she is experiencing **very sudden abdominal pain**

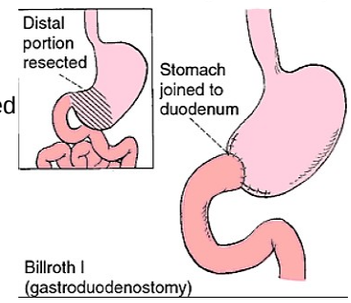


3. BLEEDING
➢ leakage of gastrointestinal content
➢ ↓ **BP,** rapid, weak, feeble & thready PR, rapid but shallow RR, cold, clammy, diaphoretic
➢ hematemesis, melena

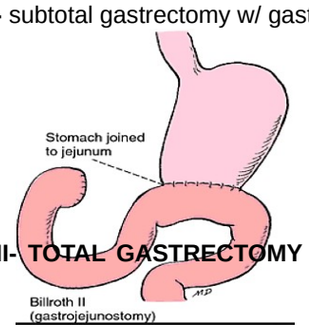
4. PYLORIC OBSTRUCTION
⇒ scarring/ fibrosis
⇒ fibrotic tissue develop around pyloric opening causing pyloric stenosis
⇒ devomiting of undigested food
⇒ sx of fluid & electrolyte imbalances
⇒ Epigastric fullness

5. INTRACTABLE ULCER
⇒ ulcer not responding to any form of medical & nursing management

ONLY MANAGEMENT:
❖ **GASTRIC SURGERY**
1. BILLROTH I- subtotal gastrectomy w/ gastroduodenostomy remove distal 3rd of atoma & remaining portion is anastomosed to duodenum



2. **BILLROTH 2-** subtotal gastrectomy w/ gastrojejunostomy



3. **BILLROTH III- TOTAL GASTRECTOMY w/** esophago-jejunostomy

remove entire stomach

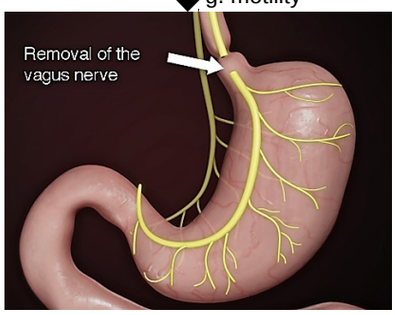
4. **GASTRORRHAPHY-** suturing of a perforated stomach

5. **ANTRECTOMY-** remove antrum (between fundus & pylorus)
❖ Antrum secretes the hormone **GASTRIN** which stimulates HCl production

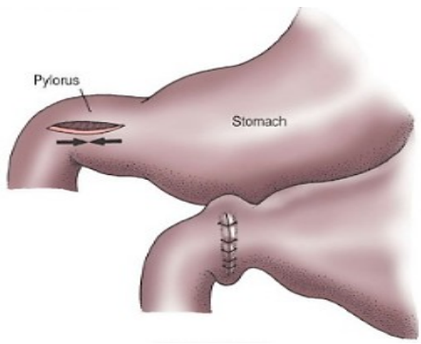
6. **VAGOTOMY-**remove part of vagus nerve/ vagal stimulation

g. secretion
g. motility

Parasympathetic
g. secretion
g. motility



7. **PYLOROPLASTY-** reconstructive surgery done to enlarge pyloric opening



ANTICHOLINERGIC: dec gastric emptying
- ⇒ **PROPANTHALINE Bromide**
- ⇒ **PROBANTHINE**
- ⇒ **Bentyl**

Buscopan- decrease/ delay gastric emptying

2. **MARGINAL ULCER**
- ⇒ HCl is coming contact w/ site of anastomosis

3. PERNICIOUS ANEMIA

reduced size of stomach
⬇
reduced gastric mucosa
⬇
reduced production of intrinsic factor
⬇
malabsorption of Vitamin B12

4. MALNUTRITION

decrease size of stomach
⬇
reduced gastric capacity
⬇
malabsorption of different nutrients

WHEN ARE THEY INDICATED:
- ⇒ For Peritonitis- **Billroth 1, Billroth 2, Gastrorrhapy**
- ⇒ Pyloric Obstruction- **Pyloroplasty**
- ⇒ Cancer of Stomach- **Billroth 3**
- ⇒ Intractable Ulcer- reduce lvl of acic: **Antrectomy, Vagotomy**

Complications Specific to Gastric Surgery

1. DUMPING SYNDROME- rapid passage of hyperosmolar solution into jejunum causing local & systemic effect
- a. **Local-** jejunum distend- causing hypermotility, diarrhea, vomiting, abdominal cramps
- b. **Systemic-** food enters jejunum is hyperosmolar- will attract fluid- causing fluid to move from IV to jejunum
  ***nawalan fluid sa IV---shock like sx (hypovolemic shock)**

***How will you prevent dumping syndrome? LIE DOWN AFTER EATING**
- ⇒ Eat in a recumbent/ semi recumbent position

Complication true for all surgery
- ⇒ **Hypostatic Pneumonia**
- ⇒ Atelectasis
- ⇒ CV: thrombophlebitis, DVT
- ⇒ GI: paralytic ileus d/t anesthesia
- ⇒ GU: bladder atony causing urinary retention
- ⇒ Wound: bleeding, infection, dehiscence, evisceration

Another way:
- ⇒ take fluids in between meals but not with meals
- ⇒ Take a **DRY MEAL** (nakakabilaok)
- ⇒ No sweet/ food rich in CHO bec
  *emptying time of CHO- **1-2h after eating**
  *****emptying time of CHON- **2-4h after eating**

