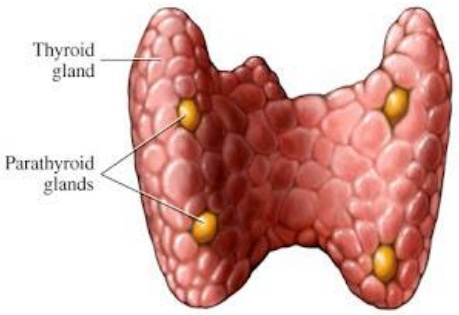
PROBLEMS OF REGULATION

Dr. Leonece Myers, DNP, MSN, CCRN, APRN, FNP-C, FNP-BC

ENDOCRINE

- Hyper/Hypo parathyroidism
- Syndrome of Inappropriate Antidiuretic Hormone
- Diabetes Insipidus
- Cushing Syndrome/Disease
- Addison Disease
- Pheochromocytoma



HYPERPARATHYROIDISM

- Increase secretion of parathyroid hormone (PTH). PTH regulates calcium, phosphate levels, renal tubular reabsorption of calcium, and activation of vitamin D.
- Oversecretion of PTH is associated with increase serum calcium levels

TYPES OF HYPERPARATHYROIDISM

- Primary
- Secondary
- Tertiary

MANIFESTATIONS

- Cardiovascular symptoms
- Neurologic
- Musculoskeletal
- Gastrointestinal
- Renal
- Integumentary

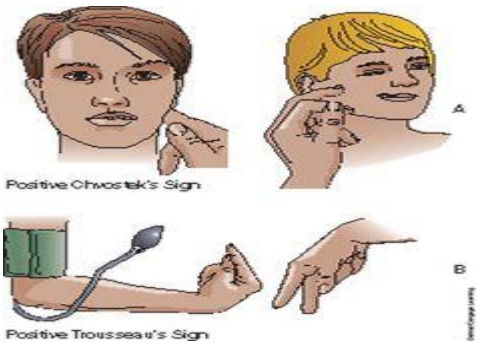
DIAGNOSTIC STUDIES

- PTH
- Serum calcium
- Elevated urine calcium
- Serum phosphate
- Electrolytes (Na/Cl/Cr/amylase)
- Bone density measurements
- X-rays, UTZ, MRI, thalium scan

TREATMENT

Surgical
a. Parathyroidectomy (partial/complete)
b. Autotransplantation

Nonsurgical
a. Monitor electrolytes
b. Diet
c. Phosphorus supplements (Bisphosphonates)
d. Drugs (Estogen, Progestin,Phosphate, Calcimimetic)
e. Ambulation



COMPLICATIONS

- Tetany
- Hemorrhage
- Laryngospasms

HYPERCALCEMIC CRISIS

- Serum calcium level > 15 mg/dL
- Results in neurologic, cardiovascular, and renal symptoms
- Treatment involves rehydration with large volumes
- Diuretics: promote renal excretion of Ca++
- Phosphate therapy
- Cytotoxic agents: Mithramycin
- Calcitonin
- Dialysis

INTERVENTIONS

- I&O
- IV calcium gluconate
- Monitor electrolytes
- Frequent monitoring for Chvostek's and Trousseau's signs.
- Mobility to promote bone calcification

HYPOPARATHYROIDISM

- Inadequate circulating PTH
- Hypocalcemia due to lack f PTH to maintain serum calcium levels.
- Pseudohypoparathyroidism due to resistance of PTH a the cellular level. There is a genetic defect resulting in hypocalcemia in spite of normal or high levels .
- Most common cause is iatrogenic

MANIFESTATIONS

Cardiovascular symptoms
Neurologic
Gastrointestinal
Renal
Musculoskeletal
Integumentary

MANIFESTATIONS

- Numbness and tingling (lips, fingers, feet)
- Tonic spasms of smooth muscles and skeletal
- Dysphagia
- Laryngospasm
- Decreased serum calcium and PTH levels
- Increased serum phosphate

TREATMENT

IV calcium chloride, gluconate, or gluceptate
ECG monitoring
Rebreathing
Lowering pH
Oral calcium supplements (1.5-3 g/day)
Vitamin D
Diet (high calcium)
EKG monitoring during calcium administration

SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE

DR. LEONECE D. MYERS RN, DNP, MSN, CCRN, FNP-BC, FNP-C

MANIFESTATIONS

- Decreased bone density
- Cyst formation
- General weakness
- Hypercalciuria
- Calculi formation
- Osteoporosis
- Fractures

- Overproduction or oversecretion of ADH
- Release of ADH despite normal or low plasma osmolality.
- Excess ADH increases the permeability of the distal tubule and collecting duct, which leads to the reabsorption of water into the circulation.

ETIOLOGY

- Drugs
- Head Trauma
- Cancer (Small cell lung cancer)
- Metabolic Diseases
- Cerebrovascular Accident
- Systemic Lupus Erythematosus
- Hypothyroidism
- Lung Infections

MANIFESTATIONS

- Serum hypoosmolarity
- Dilutional hyponatremia
- Fluid retention
- Hypochloremia
- Increased intravascular volume
- Concentrated urine
- Normal renal function
- Muscle cramps and weakness

MANIFESTATIONS

- Fatigue
- Thirst
- Low urinary output
- Weight gain
- Low serum sodium < 120 mEq/L
- Cerebral edema
- Seizures, coma

DIAGNOSTIC STUDIES

- Urine osmolality
- Serum osmolality < 280 mOsm/kg
- Serum sodium <134 mEq/L
- Urine specific gravity >1.005

TREATMENT

- Fluid restriction if Na+ > 125 mEq/L
- 800-1000ml/24 hours
- Hypertonic saline solution 3-5%
- Diuretics (Lasix) must have a Na+ of >125 mEq
- **Severe hyponatremia** fluid restriction is 500ml/24 hour (<120 mEq)
- Drugs(demeclocycline, lithium)
- Vasopressin receptor antagonist (Tolvaptan, Conivaptan)

INTERVENTIONS

- Assessment
- Daily weight
- Fluid restriction
- Seizure precautions
- Positioning
- Diet
- I&O

DIABETES INSIPIDUS

- Deficiency of production or secretion of ADH
- Decreased renal response to ADH

- The three D's
  - Diabetes insipidus
  - Decreased ADH
  - Diuresis

CLASSIFICATIONS

- **Central DI** (neurogenic): Occurs when there is an organic lesion that interferes with ADH synthesis or release. *Secondary to intracranial surgery or head trauma.*
- **Nephrogenic DI**: There is adequate AND, but decrease response to ADH by the kidney.
- **Psychogenic DI** (dispogenic): Associated with excessive water intake.

PATHOPHYSIOLOGY

Decreased antidiuretic hormone

↓

Decreased water reabsorption

↓

Decreased intravascular fluid volume

↓

Increase serum osmolality, excessive UOP

ETIOLOGY

- Head trauma
- Brain tumor
- Surgical ablation or irradiation of pituitary
- Infections of the CNS
- Tumors
- Drugs

PHASES OF DIABETES INSIPDUS (Triphasic Pattern)

- Acute Phase : Abrupt onset of polyuria
- Interphase: Urine volume normalizes
- Third phase: Central DI is permanent in 10-14 days. DI from head trauma usually self limiting and improves with treatment. DI following surgery is more likely to be permanent.

MANIFESTATIONS

- Polydipsia
- Polyuria (5-20 liters/day)
- Low specific gravity (<1.005)
- Low urine osmolality (< 100 mOsm/kg)
- Elevated serum osmolality (>295 mOsm/kg)
- Hypernatremia d/t pure water loss

MANIFESTATIONS

- Hypovolemia
- Hypotension
- Tachycardia
- Weight loss
- Shock
- Poor skin turgor
- CNS manifestations
- Constipation

DIAGNOSTIC STUDIES

- Water deprivation test ((Miller-Moses)
- Baseline vital signs, weight, urine & serum osmolality's, and S.G.
- NPO for 8-16 hours
- Hourly, BP, weight, urine osmolality during the test.
- ADH or vasopressin is given either IV or SQ

Water Deprivation Test (Miller-Moses)

- Used to differentiate causes of polyuria, including central DI, nephrogenic DI, SIADH, and psychogenic polydipsia.
- ADH or vasopressin is administered IV or SC, nasally
- Normal/psychogenic: urine & plasma mOsm are normal
- Central DI: increase urine mOsm, decrease UOP
- Nephrogenic: no response

TREATMENT

Nephrogenic DI

Diet: Low sodium diet (3 g/per day)

Diuretics: Thiazide

Prostaglandin inhibitors: Indomethacin

TREATMENT
Central DI

- Hormonal replacement
  1. Desmopressin acetate (DDAVP)
  2. Pitressin
  3. Diabinese
  4. Tegretol
- Hypotonic saline or Dextrose solution
- Titrate to replace urinary output

INTERVENTIONS

- Fluid replacement
- Accurate I&O's
- Measure urine SG
- Monitor electrolytes
- Daily weights
- Administration of DDAVP
- Assess for signs and symptoms of water intoxication

DISORDERS OF THE ADRENAL CORTEX

- Glucocorticoids: Cortisol
- Mineralocorticoids: Aldosterone
- Androgens

CUSHING SYNDROME

- Clinical abnormalities caused by an excess of corticosteroids, particularly glucocorticoids.

ETIOLOGY

- Iatrogenic administration of exogenous corticosteroids. (Most common cause)
- ACTH-secreting pituitary tumor
- Adrenal cortisol secreting tumor
- Excess secretion of ACTH from carcinoma of the lung or other malignant growth outside the pituitary or adrenal glands

MANIFESTATIONS

- Weight gain
- Truncal obesity
- Moon face
- “Buffalo hump”
- Hyperglycemia
- Protein wasting
- Muscle weakness
- Osteoporosis
- Pathologic c fractures
- Bone and back pain
- Thin fragile skin
- Purplish strae
- Hypervolemia, HTN
- Edema
- Inhibition of immune response
- Na+ and water retention
- Hirsutism
- Menstrual irregularities

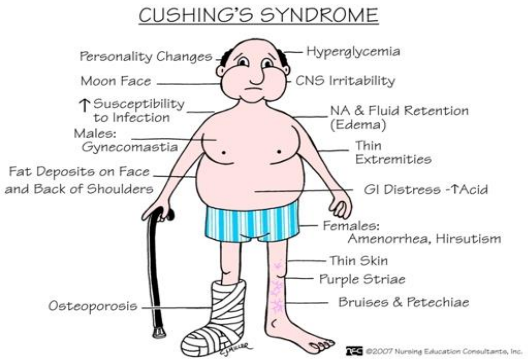
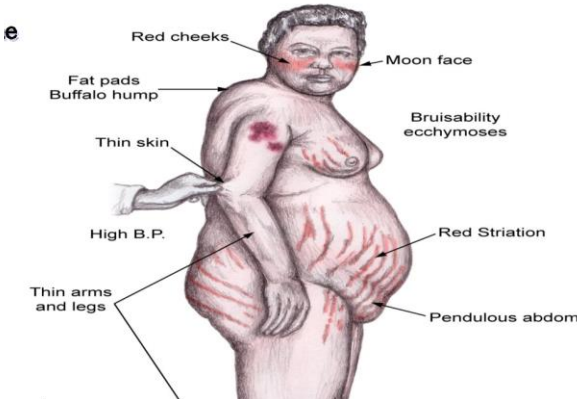
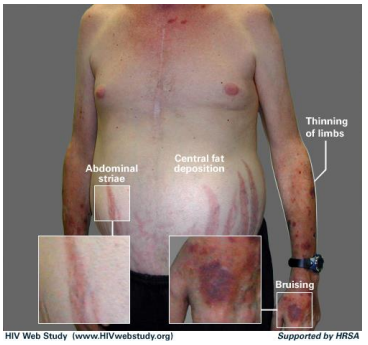


Figure 1) Left and right *A 52-year-old human immunodeficiency virus (HIV)-1-infected man presented with a football-sized mass in the dorsal cervical areal ‘Buffalo hump’). It had existed as a minor area of fullness for several years, but had dramatically increased in size over the preceding year, after the protease inhibitor indinavir was added to his antiviral regimen*





DIAGNOSTIC STUDIES

- 24 hour urine for free cortisol
- Dexamethsone suppression test
- Plasma cortisol
- CT scan, MRI
- Plasma ACTH levels
- CBC, electrolytes
- Eye exam

TREATMENT

Surgery

1. Transsphenoidal for pituitary tumor
2. Adrenalectomy for malignant tumors

Radiation

Drug therapy

1. Mitotane : “Medical adrenalectomy”
2. Ketconazole, Metyrapone, Cytaden

TREATMENT

- Gradual discontinuance of corticosteroids therapy
- Reduction of the corticosteroid dose
- Conversion to an alternate-day regimen

COMPLICATIONS

- Cardiovascular disease
- Diabetes Mellitus
- Infection
- Thromboembolic phenomena (polycythemia)
- Changes in appearance (Hisutism, gynecomastia)

INTERVENTIONS

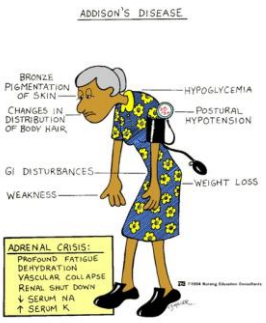
- Monitoring vital signs
- Daily weights
- Monitor glucose
- Emotional support
- Control HTN, hyperglycemia, hypokalemia
- Diet (high protein)

POSTOPERATIVE

- Monitor for postoperative hemorrhage
- Monitor BP, fluid balance and electrolyte
- High doses of corticosteroids (SoluCortef)
- Monitor for infection
- Monitor I&O
- Monitor urine cortisol levels

EDUCATION

- Medical alert bracelet
- Avoid exposure to extremes of temperatures, infections, and emotional disturbances
- Adjustment corticosteroid replacement therapy
- Lifetime replacement



ADRENOCORTICAL INSUFFICIENCY

- Hypofunction of the adrenal cortex
- Primary: Addison's disease
- Secondary: Lack of ACTH secretion, may be due to pituitary disease or suppression of the hypothalamic-pituitary axis.

ADDISON's DISEASE

The most common cause is due to an autoimmune response.

Adrenal tissue is destroyed by antibodies against the patient own adrenal cortex.

ETIOLOGY

- Infarction
- Fungal infections
- Acquired Immunodeficiency Syndrome
- Metastatic cancer
- Adrenal hemorrhage (anticoagulant therapy)
- Chemotherapy
- Ketoconazole therapy
- Bilateral adrenalectomy

MANIFESTATIONS

- Weight loss
- Hyperpigmentation
- Vitilligo, alopecia
- Hypotension
- Hyperkalemia
- Hyponatremia
- Anorexia
- Fatigability
- Diarrhea
- Irritability
- Depression
- Decreased axillary and pubic hair
- Decrease muscle size and tone
- Shock
- Anemia

DIAGNOSTIC STUDIES

- ACTH-stimulation test
- Electrolytes
- Glucose
- CBC
- BUN, creatinine
- EKG (Peaked T waves)
- CT scan, MRI

TREATMENT

- Replacement therapy (Hydrocortisone)
- During stress need to increase glucocorticoid (cortisol) must be increased to prevent Addisonian crisis.
- Mineralocorticoid replacement (Floinef)
- Increase salt intake
- Patient education

MANIFESTATION OF ADDISONIAN CRISIS

- Hypotension
- Tachycardia
- Dehydration
- Hyponatemia, hyperkalemia, hypoglycemia
- Fever, confusion
- Weakness
- Shock
- Circulatory collapse

COMPLICATIONS ADDISONIAN CRISIS

- A life threatening emergency caused by insufficient adrenocortical hormones or a sharp decrease in these hormone.
- Triggered by stress
- After adrenal surgery
- Pituitary gland destruction
- Sudden withdrawal of corticosteroid hormone replacement therapy.

TREATMENT OF ADDISONIAN CRISIS

- High dose hydrocortisone replacement
- Large volumes of normal saline and D5W
- VS q 30 minutes-4 hour for first 24 hrs
- I&O
- Daily weights
- Protect from light and environmental extremes

COMPLICATIONS OF CORTICOSTEROIDS

- Hypertension
- Suppression of inflammation and immune response
- Inhibit the antibody response to vaccines.
- Predisposition to PUD
- Mood and behavior changes
- Redistribution of fat
- Healing delayed
- Osteoporosis

QUESTION

The best time to take corticosteroids for replacement purposes is:

a. once a day at bedtime.
b. every other day on awakening.
c. on arising and in the late afternoon.
d. at consistent intervals every 6 to 8 hours.

QUESTION

The nurse would expect to fine which of the following manifestation in Cushing syndrome?

a. Hypotension
b. Decrease axillary and pubic hair
c. Bronzed hyperpigmentation of the skin
d. Purplish red striae on the abdomen

QUESTION

Which of the following statements demonstrates lock of knowledge for a client diagnosed with Addison's disease?

a. " I double my dose of hydrocortisone if I am experiencing moderate stress."

b. " If I take too much of my medication I have vomiting and diarrhea, so I reduce the dosage."

c. " I will notify my physician if my BP is out of range."

PHEOCHROMOCYTOMA

- A catecholamine-secreting neoplasm associated with hyperfunction of the adrenal medulla.
- A rare condition characterized by a tumor of the adrenal medulla that produces excessive catecholamines (epinephrine, norepinephrine)
- 90% tumors are benign

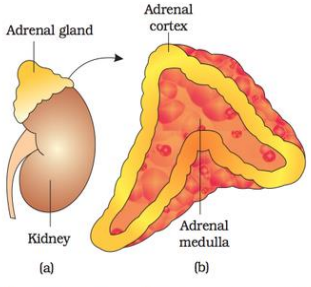


Figure 22.4 Diagrammatic representation of : (a) Adrenal gland on kidney (b) Section showing two parts of adrenal gland

Differences between Epinephrine and Norepinephrine

Epinephrine >> norepinephrine – in terms of cardiac stimulation leading to greater cardiac output (β stimulation).

- Epinephrine < norepinephrine – in terms of constriction of blood vessels – leading to increased peripheral resistance – increased arterial pressure.
- Epinephrine >> norepinephrine –in terms of increasing metabolism Epi = 5-10 x Norepinephrine

MANIFESTATIONS

- Hypertension
- Hypermetabolism
- Postural hypotension in a hypertensive patient
- Hyperglycemia (polyuria)
- Headaches and visual disturbances
- Tremors, pallor, perspiration, face flushing
- Diarrhea, vomiting, abdominal pain
- Emotional changes, psychotic behavior

DIAGNOSTIC STUDIES

- VMA and metanephrine
- Elevated epinephrine and norepinephrine in serum and urine
- CT scan and MRI of the adrenal gland
- Clonidine suppression test (used to distinguish essential HTN from phenochromocytoma)

TREATMENT

- Alpha-adrenergic blocking agents (Retgitine,
- Phenoxybenzamine, Terazosin, Prazosin)

- Catecholamine synthesis inhibitor (Metyrosine)
- Calcium channel blocker (Cardene)

- Surgery : adrenalectomy

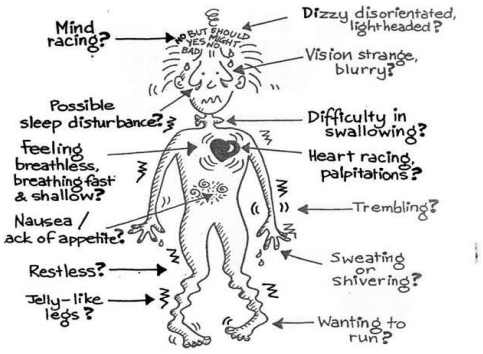
INTERVENTIONS

- Bed rest, elevate HOB 45 degrees
- Calm quiet environment
- Eliminate stimulants(caffeine)
- Sedatives to promote relaxation
- Encourage oral fluids and maintain IV infusion
- Monitor vital sign, ECG, Neurologic status
- I&O, labs (BUN, creatinine, and glucose)

QUESTION

- After an adrenalectomy for pheochromocytoma, the client is most likely to experience:

a. hypokalemia
b. hyperglycemia
c. marked sodium and water retention
d. marked fluctuations in the blood pressure



Signs	Symptoms
Hypertension++++	Headache++++
-Paroxysmal+++	Palpitations++++
-Sustained++	Anxiety+++
Tachycardia+++	Nervousness+++
Reflex Bradycardia+++	Tremulousness++
Sweating++++	Weakness++
Hyperglycemia++	Fatigue++
Postural Hypotension+	Nausea and vomiting+
Pallor++	Faintness+
Flushing+	Visual disturbances+
Weight loss+	Paraesthesias+
Decreased peristalsis+	Bowel disturbances+
Tachypnea+	Pain abdomen+

The signs (++++) – (+) depicts the occurrence of signs and symptoms in decreasing frequencies in patients with phaeochromocytoma