Immune and inflammatory responses protect the body from invading foreign substances. Certain classes of drugs can modify these responses:

a. Antihistamines  
b. Corticosteroids  
c. Immunosuppressants (noncorticosteroid)  
d. Uricosurics

### Antihistamines

Antihistamines primarily act to block histamine effects that occur in an immediate (type I) hypersensitivity reaction, commonly called an allergic reaction.

#### A. HISTAMINE-1 RECEPTOR ANTAGONISTS

**Pharmacodynamics:**

H₁-receptor antagonists compete with histamine for H₁ receptors on effector cells (the cells that cause allergic symptoms), blocking histamine from producing its effects.

**Drug Interactions:**

a. Antihistamines may block or reverse the vasopressor effects of epinephrine, producing vasodilation, increased heart rate, and dangerously low blood pressure.

b. Antihistamines may mask toxic s/sx of ototoxicity associated with aminoglycosides or large doses of salicylates.

c. Antihistamines may increase the sedative and respiratory depressant effects of CNS depressants, such as tranquilizers and alcohol.

d. Loratadine may cause serious cardiac effects when taken with macrolide antibiotics (e.g., erythromycin), fluconazole, ketoconazole, itaconazole, miconazole, cimetidine, ciprofloxacin, and clarithromycin.

**Adverse Reactions:**

The most common adverse reaction to antihistamines (with the exception of fexofenadine and loratadine) is CNS depression. Other CNS reactions include:

- Dizziness
- Fatigue
- Disturbed coordination
- Muscle weakness

**Nursing Responsibilities:**

a. Obtain a history of the patient’s underlying condition before therapy and reassess regularly thereafter.

b. Monitor the patient for adverse reactions and drug interactions.

c. Monitor blood counts during long-term therapy; watch for signs of blood dyscrasia.

d. Evaluate the patient’s and family’s knowledge of drug therapy.

e. Reduce GI distress by giving antihistamines with food.

f. Follow the manufacturer’s guidelines for IV administration.

g. If administering the drug I.M., alternate injection sites to prevent irritation. Give IM injections into large muscles.

h. Provide sugarless gum, hard candy, or ice chips to relieve dry mouth.

i. Increase the patient’s fluid intake (if allowed) or humidify the air to decrease thickened secretions.
j. Notify the prescriber if tolerance is observed because the patient may require a substitute antihistamine.

B. CORTICOSTEROIDS

Natural corticosteroids are hormones produced by the adrenal cortex; most corticosteroid drugs are synthetic forms of these hormones. Natural and synthetic corticosteroids are classified according to their biological activities:

- Glucocorticoids
- Mineralocorticoids

**GLUCOCORTICOIDS**

Glucocorticoids exert anti-inflammatory, metabolic, and immunosuppressant effects.

Prototype Medications:
- Beclomethasone
- Betamethasone
- Cortisone
- Dexamethasone
- Hydrocortisone
- Methylprednisolone
- Prednisolone
- Triamcinolone

**Pharmacodynamics**

Glucocorticoids suppress hypersensitivity and immune responses through a process that isn’t entirely understood. Researchers believe that glucocorticoids inhibit immune responses by:

- Suppressing or preventing cell-mediated immune reactions
- Reducing levels of leukocytes, monocytes and eosinophils
- Decreasing the binding of immunoglobulins to cell surface receptors
- Inhibiting interleukin synthesis

**Drug Interactions**

- Barbiturates, phenytoin, rifampin, and aminoglutethimide may reduce the effects of glucocorticoids.
- Amphotericin B, chlorthalidone, ethacrynic acid, furosemide, and thiazide diuretics may enhance glucocorticoids’ potassium-wasting effects.
- Erythromycin and troleandomycin may increase the effects of glucocorticoids by reducing their metabolism.
- Glucocorticoids reduce the serum concentration and effects of salicylates.
- The risk of peptic ulcers associated with NSAIDS and salicylates is increased when these agents are taken with corticosteroids.
- The response to vaccines and toxoids may be reduced in a patient taking glucocorticoids.
- Estrogen and hormonal contraceptives that contain estrogen increase the effects of glucocorticoids.
- The effects of antidiabetic drugs may be reduced, resulting in increased blood glucose levels.

**Adverse reactions**

Glucocorticoids affect almost all body systems. Their widespread adverse effects include:

- Insomnia
- Increased sodium and water retention
- Increased potassium excretion
- Suppressed immune and inflammatory responses
- Osteoporosis
- Intestinal perforation
- Peptic ulcers
- Impaired wound healing

**Endocrine system reactions may include:**

- DM
- Hyperlipidemia
- Adrenal atrophy
• Hypothalamic-pituitary axis suppression
• Cushingoid s/sx (such as buffalo hump, moonface, and elevated blood glucose levels)

Nursing Responsibilities

a. Assess the patient’s condition before therapy and regularly thereafter.
b. Establish baseline blood pressure, F/E status, and weight; reassess regularly.
c. Watch for depression or psychotic episodes, especially at high doses.
d. Monitor the glucose levels of a patient with diabetes closely; increased insulin may be needed.
e. Monitor the patient’s stress level; dosage adjustment may be needed.
f. Closely monitor the patient for adverse reactions and drug interactions.
g. Evaluate the drug’s effectiveness at regular intervals.
h. Give the drug early in the day to mimic circadian rhythm.
i. Give the drug with food to prevent GI irritation.
j. Take precautions to avoid exposing the patient to infection.
k. Don’t stop the drug abruptly.
l. Avoid prolonged use of corticosteroids, especially in children.
m. Unless contraindicated, offer a low-sodium diet that’s high in potassium and protein. Administer potassium supplements as needed.
n. Evaluate the patient’s and family’s knowledge of drug therapy

Note: if corticosteroids are rapidly withdrawn, abrupt withdrawal symptoms can occur. These include rebound inflammation, fatigue, weakness, arthralgias, fever, dizziness, lethargy, depression, fainting, orthostatic hypotension, dyspnea, anorexia, and hypoglycemia. After long-term therapy, increased stress or abrupt withdrawal may cause acute adrenal insufficiency. Sudden withdrawal after prolonged therapy can be fatal.

MINERALOCORTICOIDs

Mineralocorticoids affect electrolyte and water balance. These drugs include:

• Fludro cortisone acetate-synthetic analogue
• Aldosterone-natural mineralocorticoid

Pharmocodynamics

Fludrocortisone acetate affects F/E balance by acting on the distal renal tubule to increase sodium reabsorption and potassium and hydrogen secretion.

Drug Interactions and Adverse Reactions

The drug interactions associated with mineralocorticoids are similar to those associated with glucocorticoids.

Nursing Responsibilities

a. Assess the patient’s condition before therapy and regularly thereafter.
b. Establish baseline blood pressure, F/E status and weight; reassess regularly.
c. Monitor patient closely for adverse reactions and drug interactions.
d. Evaluate the drug’s effectiveness at regular intervals.
e. Administer the drug as prescribed, and monitor for adverse reactions and drug interactions.
f. Notify the prescriber of severe or persistent adverse reactions.
g. Notify the prescriber if hypertension occurs.
h. Monitor the patient’s electrolyte level; potassium supplements may be needed. Assess for hypokalemia, such as muscle cramps and ECG changes.
i. Evaluate the patient’s and family’s knowledge of drug therapy.
IMMUNOSUPPRESSANTS

Several drugs used for their immunosuppressant effects in patients undergoing allograft transplantation are also used experimentally to treat autoimmune disease. They include:

Azathioprine
Anakinra
Basiliximab
Cyclosporine
Daclizumab
Lymphocyte immune globulin (ATG [equine])
Muromonab-CD3
Mycophenolate mofetil
Sirolimus
Tacrolimus
Thymoglobulin (antithymocyte globulin [rabbit])

Pharmacodynamics

It’s unknown how certain immunosuppressants achieve their desired effects.

For example, the exact mechanisms of action of azathioprine, cyclosporine, and ATG are unknown; however, these drugs may undergo these processes:

- Azathioprine antagonizes the metabolism of the amino acid purine and, therefore, inhibits RNA and DNA structure and synthesis. It may also inhibit coenzyme formation and function.
- Cyclosporine is thought to inhibit helper T cells and suppressor T cells
- ATG may eliminate antigen0reactive T cells in the blood, alter T-cell function, or both.

Nursing Responsibilities:

a. Obtain a history of the patient’s immune status before therapy
b. Monitor the drug’s effectiveness by observing the patient for signs of organ rejection therapeutic response usually occurs within 8 weeks.
c. Watch for adverse reactions and drug interactions
d. Monitor Hgb, Hct, and WBC and platelet counts at least once monthly; monitor them more often at the beginning of treatment.
e. Administer the medication as prescribed; reconstitute according to policy and procedure.
f. Monitor the patient’s reaction to the medication and watch for adverse reactions.
g. Monitor WBC counts; the medication may need to be stopped if the patient’s WBC count is less than 3T/mm³. Notify the prescriber.
h. To prevent bleeding, avoid IM injections when the platelet count is below 100T/mm³.
i. Monitor the patient for signs of infection and report fever, sore throat, and malaise because the drug is a potent immunosuppressant.
j. Instruct a female patient to avoid conception during therapy and for 4 months after stopping therapy.
k. Warn the patient that hair thinning may occur.
l. Tell the patient that medication can take up to 12 weeks to be effective.
m. Evaluate the patient’s and family’s knowledge of drug therapy.

URICOSURICS

The 2 major uricosurics are probenecid and sulfinpyrazone. Uricosurics act by increasing uric acid excretion in urine the primary goal in
using them is to prevent or control the frequency of gouty arthritis attacks.

**Pharmacodynamics**

Probenecid and sulfinpyrazone reduce the reabsorption of uric acid at the proximal convoluted tubules of the kidneys. This results in excretion of uric acid in urine, reducing serum urate levels.

**Drug Interactions**

- Many drug interactions, some potentially serious, can occur with uricosuric drugs.
- Probenecid can significantly increase and prolong the effects of cephalosporins, penicillins, and sulfonamides.
- Serum urate levels may be increased when probenecid is taken with antineoplastic drugs.
- Probenecid increases the serum concentration of dapsone, ASA, and methotrexate, causing toxic reactions.
- Sulfinpyrazone increases the effectiveness of warfarin, increasing the risk of bleeding.
- Salicylates reduce the effects of sulfinpyrazone.
- Sulfinpyrazone may increase the effects of oral antidiabetic agents, increasing the risk of hypoglycemia.

**Adverse reactions**

These include uric acid stone formation and blood abnormalities.

**Probenecid**

Additional adverse reactions include:
- H/A
- Anorexia
- N/V
- Hypersensitivity reactions

**Sulfinpyrazone**

Additional adverse reactions include:
- Nausea
- Indigestion
- GI pain
- GI blood loss

**Nursing Responsibilities:**

a. Assess the patient’s condition before therapy and regularly thereafter.
b. Periodically monitor BUN levels and renal function tests in long-term therapy. Note that the drug is ineffective in patients with chronic renal insufficiency (GFR < 30ml/min).
c. Be alert for adverse reactions and drug interactions
d. Monitor the patient’s hydration status if adverse GI reactions occur.
e. Give the medication with milk, food, or antacids to minimize GI distress. Continued disturbances may indicate a need to lower the dosage.
f. Encourage the patient to drink fluids to maintain a minimum daily output of 2L of H2O/day. Sodium bicarbonate or potassium citrate may be needed to alkalize urine. These measures prevent hematuria, renal colic, urate stone development, and costovertebral pain.
g. Begin therapy when an acute attack subsides. Keep in mind that the drug contains no analgesic or anti-inflammatory agents and isn’t useful during acute gout attacks.
h. Be aware that the drug may increase the frequency, severity, and duration of acute gout attacks during the 1st 12 months of therapy. Prophylactic colchicines or another anti-inflammatory is given during the 1st 3-6months.
i. Instruct the patient to avoid drugs that contain aspirin, which may precipitate gout.

j. Tell the patient to avoid alcohol during drug therapy because it increases the urate level.

k. Advise the patient to limit intake of foods high in purine, such as anchovies, liver, sardines, kidneys, sweetbreads, peas and lentils.

l. Evaluate the patient’s and family’s knowledge of drug therapy.

OTHER ANTIGOUT DRUGS

These include allopurinols and colchicine. Allopurinol is used to reduce uric acid production, preventing gouty attacks, and colchicine is used to treat acute gouty attacks.

Pharmacodynamics

Allopurinol and its metabolite oxypurinol inhibit xanthine oxidase, the enzyme responsible for the production of uric acid. By reducing uric acid formation, allopurinol eliminates the hazards of hyperuricuria.

Drug interactions

Colchicine doesn’t interact significantly with other drugs. When allopurinol is used with other drugs, the resulting interactions can be serious.

- Allopurinol potentiates the effect of oral anticoagulants.
- Allopurinol increases the serum concentrations of mercaptopurine and azathioprine, increasing the risk of toxicity.
- ACE inhibitors increase the risk of hypersensitivity reactions to allopurinol.
- Allopurinol increases serum theophylline levels.
- The risk of bone marrow depression increases when cyclophosphamide is taken with allopurinol.

Nursing Responsibilities:

a. Assess the patient’s condition before therapy and regularly thereafter.

b. Assess the patient’s uric acid level, joint stiffness, and pain before and during therapy. Optimal benefits may require 2-6 weeks of therapy.

c. Monitor the patient’s CBC and hepatic and renal function at the start of therapy and periodically during therapy.

d. Be alert for adverse reactions and drug interactions.

e. Monitor the patient’s fluid I&O. Daily urine output of at least 2L and maintenance of neutral or slightly alkaline urine are desirable.

f. Give medications with meals or immediately after to minimize GI distress.

g. Encourage the patient to drink fluids while taking the drug, unless contraindicated.

h. Notify the prescriber if renal insufficiency occurs during treatment; this usually warrants a dose reduction.

i. Give colchicines with allopurinol, if ordered. This combination prophylactically treats acute gout attacks that may occur in the first 6 weeks of therapy.

j. Advise the patient to refrain from driving or performing hazardous tasks requiring mental alertness until the CNS effects of the drug are known.

k. Advise the patient taking allopurinol for treatment of recurrent calcium oxalate stones to reduce intake of animal protein, sodium, refined sugars, oxalate-rich foods, and calcium.

l. Stop the drug at the first sign of a rash, which may precede severe hypersensitivity or other adverse reaction. A rash is more common in
patients taking diuretics and those with renal disorders. Tell the patient to report all adverse reactions immediately.
m. Tell the patient to avoid alcohol during drug therapy because it increases urate level.

**COMMONLY USED VACCINES**

What is a vaccine?

- A vaccine is a biological preparation that improves immunity to a particular disease.
- A vaccine typically contains an agent that resembles a disease-causing microorganism, and is often made from weakened or killed forms of the microbe or its toxins. The agent stimulates the body's immune system to recognize the agent as foreign, destroy it, and "remember" it, so that the immune system can more easily recognize and destroy any of these microorganisms that it later encounters.
- Artificially acquired active immunity

Two types according to Purpose:
- a. Prophylactic – to prevent or ameliorate the effects of future infections
- b. Therapeutic

Types according to Source:

**Killed**
contains killed, but previously virulent, microorganisms that have been destroyed with chemicals or heat.

Examples: influenza vaccine, cholera vaccine, polio vaccine, hepatitis A vaccine, and rabies vaccine.

**Attenuated**
contain live, attenuated microorganisms. Many of these are live viruses that have been cultivated under conditions that disable their virulent properties, or which use closely-related but less dangerous organisms to produce a broad immune response.

Examples include the viral diseases yellow fever, measles, rubella, and mumps and the bacterial disease typhoid.

**Toxoid**
made from inactivated toxic compounds that cause illness rather than the micro-organism.

Examples: tetanus and diphtheria.

**Subunit**
Protein subunit – rather than introducing an inactivated or attenuated micro-organism to an immune system

Examples: Influenza, human Papilloma virus and Hepatitis B virus vaccine

**Conjugate**
certain bacteria have polysaccharide outer coats that are poorly immunogenic. By linking these outer coats to proteins (e.g. toxins), the immune system can be led to recognize the polysaccharide as if it were a protein antigen.

Example: the *Haemophilus influenzae* type B vaccine.

**Valence**
- A monovalent vaccine is designed to immunize against a single antigen or single microorganism.
- A multivalent or polyvalent vaccine is designed to immunize against two or more strains of the same microorganism, or against two or more microorganisms.
Note: In certain cases a monovalent vaccine may be preferable for rapidly developing a strong immune response.

**Commonly Used Vaccines in the Philippines:**

*(please see separate sheet)*