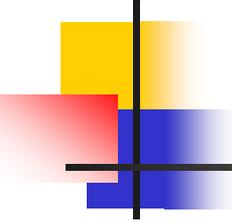


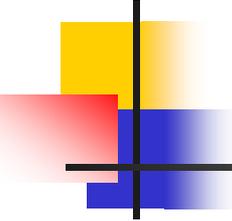
Nursing Care of Patients Immune Disorders

Chapter 53



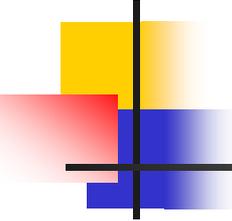
Disorders of Immune System

- Three categories
 - Hypersensitivity reactions
 - Anaphylaxis, hemolytic transfusion reactions, measles, transplant rejections
 - Autoimmune disorders
 - Rheumatoid arthritis, ulcerative colitis, multiple sclerosis
 - Immune deficiencies
 - Hypogammaglobulinemia, acquired immunodeficiency syndrome (AIDS)



Hypersensitivity Reactions

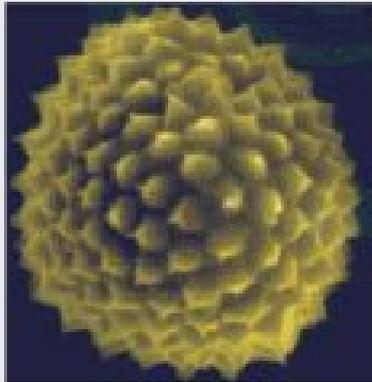
- Injury to body resulting from its exaggerated response
- Classified by way tissue injured
- Type I, II, III, and IV



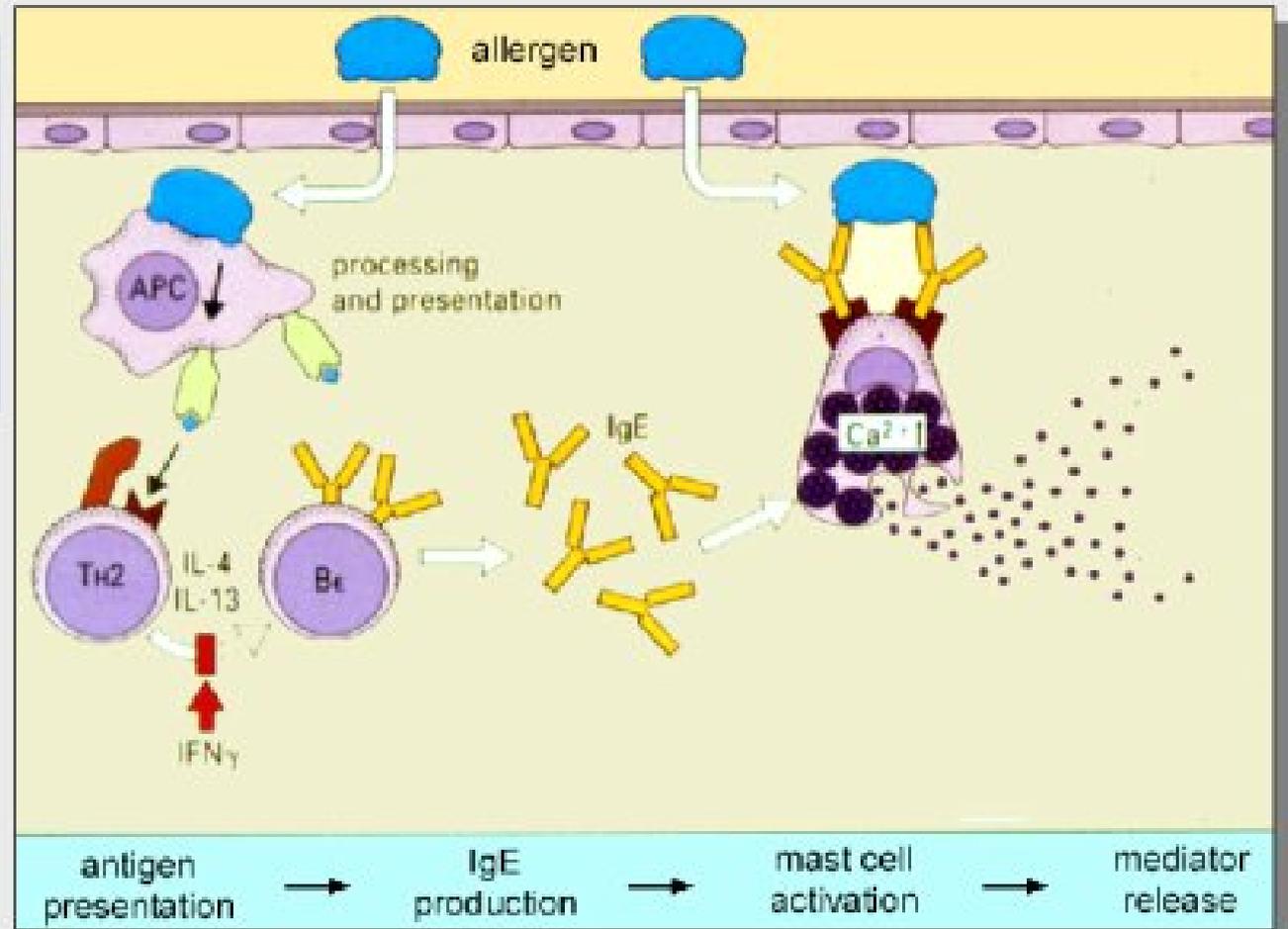
Type I

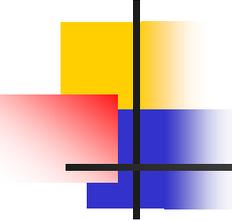
- Anaphylactic reaction
 - Occurs immediately
 - Exposure to specific antigen
 - Mild to severe, life threatening
 - Must have an initial, previous exposure
 - Sensitization
 - Localized or systemic

Sensitization against allergens



pollen



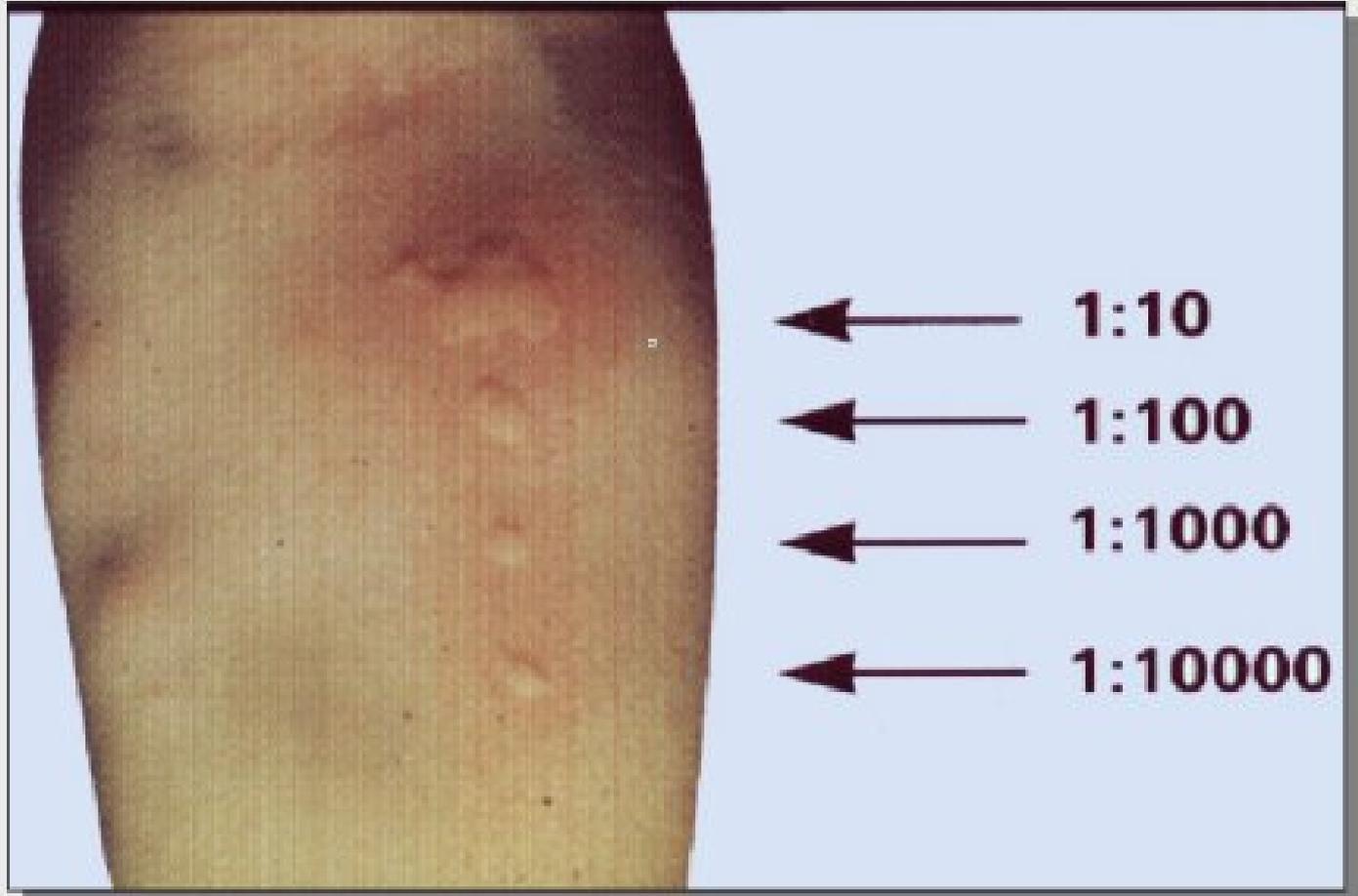


Type I (cont.)

- Severest forms
 - Anaphylaxis
 - Urticaria
 - Angioedema
- Scratch test
 - Done to identify specific allergen
 - Tiny amts of common allergens scratched on skin
 - Observed for s/s of reaction
 - Redness, edema, pruritis – if occur- considered local rx



Skin test for allergy



Skin test for allergy



Type I (cont.)

Allergic Rhinitis

- Most common form of allergy
- Perennial
 - s/s throughout year
- Hay fever or seasonal
 - s/s occur seasonally

Type I (cont.)

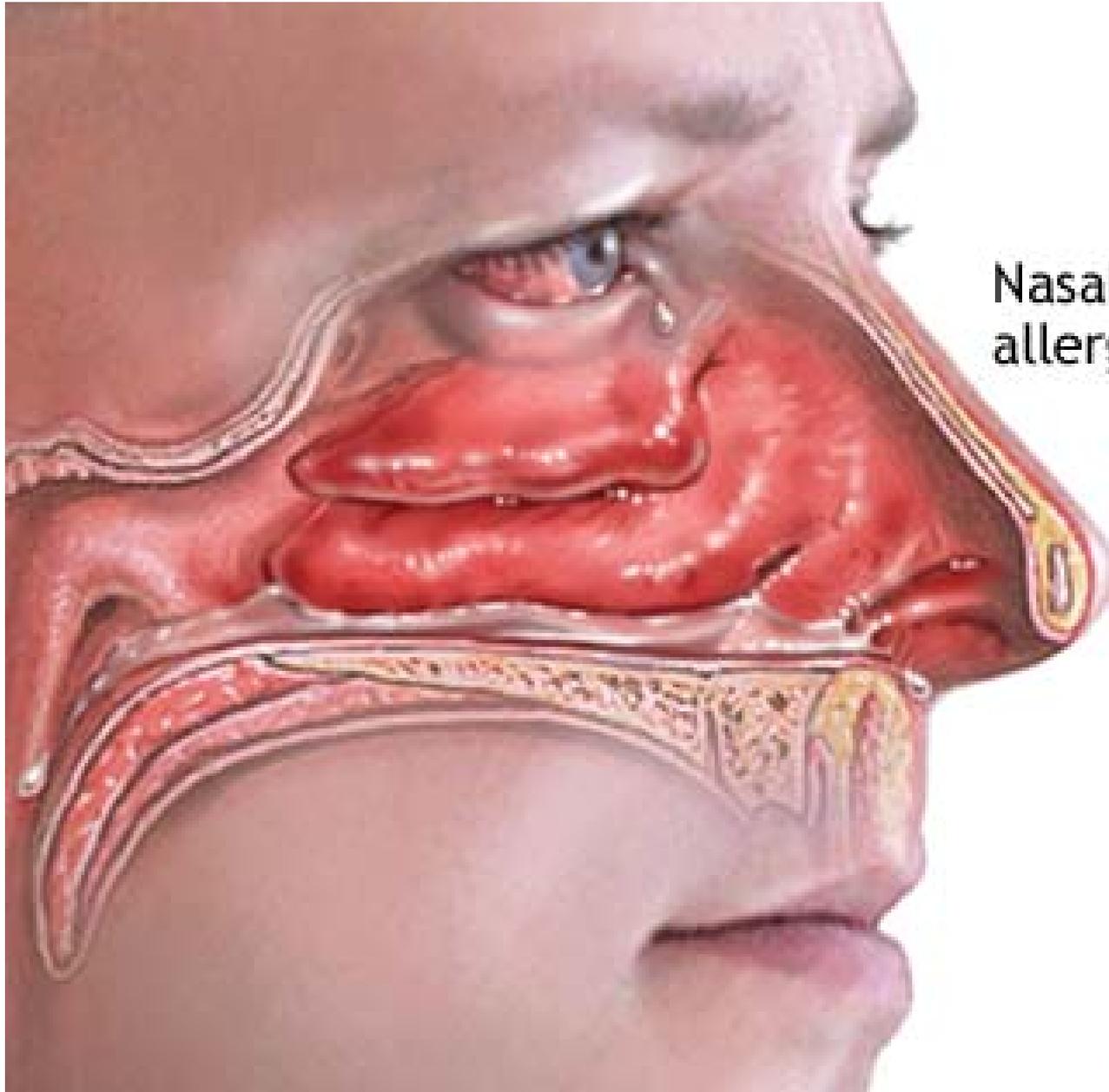
Allergic Rhinitis

■ Pathophysiology

- Result of an antigen-antibody rx
- Ciliary action decreases
- Mucous secretions increase
- Local tissue edema
 - Vasodilation

■ Signs & Symptoms

- Sneezing, nasal itching, runny nose, itchy red eyes
- Pale, cyanotic, edematous nasal mucosa
- Allergic shiners



Nasal cavity:
allergic rhinitis



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Type I (cont.)

Allergic Rhinitis

- Complications
 - Usually self-limited
 - Sinusitis, nasal polyps, asthma, chronic bronchitis
- Diagnosis
 - Detailed H&P
 - Inspection of nasal passages
 - Skin testing

Many organ are be affected by
“allergy”

The nasohprynx

Nasal polyps



Type I (cont.)

Allergic Rhinitis

- Treatment
 - Elimination of offending allergen
 - Antihistamines/nasal decongestants
 - Corticosteroid sprays if severe s/s
 - Immunotherapy w/debilitating s/s
 - Allergy shots
- Nursing management
 - Education
 - Continued assessment

Type I (cont.)

Atopic Dermatitis

- AKA; Eczema
 - Inflammatory skin response
- Signs & Symptoms
 - Initially
 - Pruritus, edema, extremely dry skin
 - Followed by
 - Blisters that crust over, then become scaled
 - Decreased sweating over these areas
 - Skin becomes thickened



Many organ are be affected by “allergy”

The skin

eczema



Type I (cont.)

Atopic Dermatitis

- Complications
 - Infections
 - Staphylococci
 - Found normally on dermal layer
- Diagnosis
 - No specific tests
 - Detailed H&P

Type I (cont.)

Atopic Dermatitis

- Treatment
 - Focuses on s/s
 - Oil-in-water lubricants
 - Alpha-Keri oil
 - Topical steroids
 - Topical antibiotics if lesions infected
- Nursing care
 - Assessment and documentation
 - Administer meds
 - Education

Type I (cont.)

Anaphylaxis

- Pathophysiology
 - Severe systemic hypersensitivity reaction
 - Widespread histamine release
- Etiology
 - Numerous (see table 53-1, pg 1000)

Type I (cont.)

Anaphylaxis

- Signs & symptoms
 - Bronchial narrowing: Stridor, wheezing, respiratory arrest
 - Hypotension, tachycardia, cardiac arrest
- Complications
 - Respiratory and cardiac arrest
- Diagnosis
 - No specific
 - H & P
 - Allergy testing after pt stable may be considered

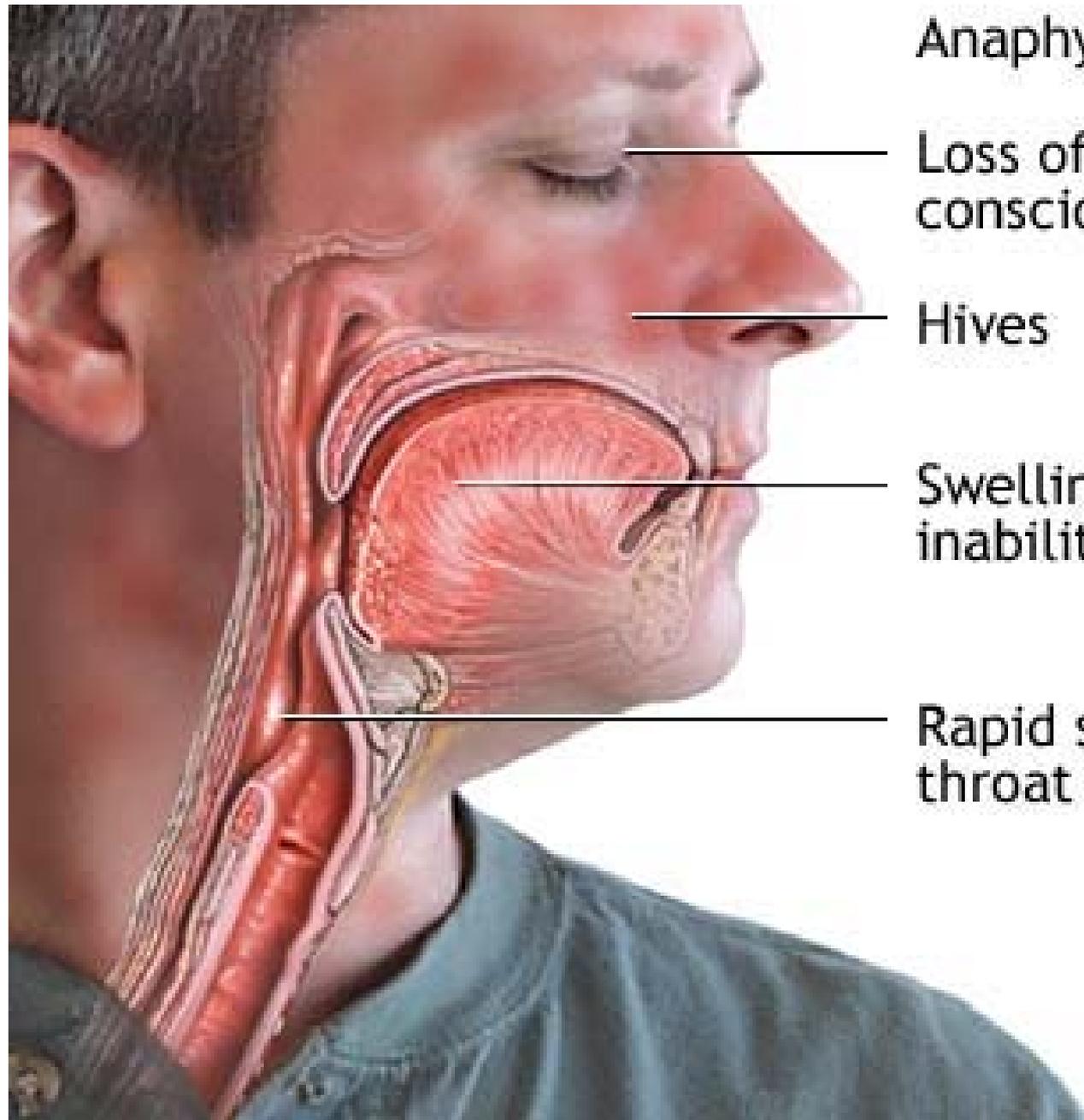
Anaphylaxis

Loss of consciousness

Hives

Swelling of tongue, inability to swallow

Rapid swelling of throat tissues



Type I (cont.)

Anaphylaxis

- Immediate treatment guided by symptoms
 - IV access
 - Oxygen
 - Epinephrine
 - Antihistamines
 - Corticosteroids
 - Vasopressors (ie, Dopamine)
 - Mechanical ventilation

Type I (cont.)

Anaphylaxis

- Nursing care
 - Early recognition and treatment
 - Maintain airway
 - Emotional support
 - Education
 - Avoidance of allergen
 - Anaphylaxis kit or epinephrine pen

Type I (cont.)

Urticaria (Hives)

- Pathophysiology
 - Release of histamine from mast cells
 - Caused by antigen stimulated rx of IgE antibodies
- Etiology
 - Medications, foods, cold, local heat, pressure, stress, others
 - Underlying chronic conditions
 - SLE, lymphoma, hyperthyroidism, cancer, etc.



Type I (cont.)

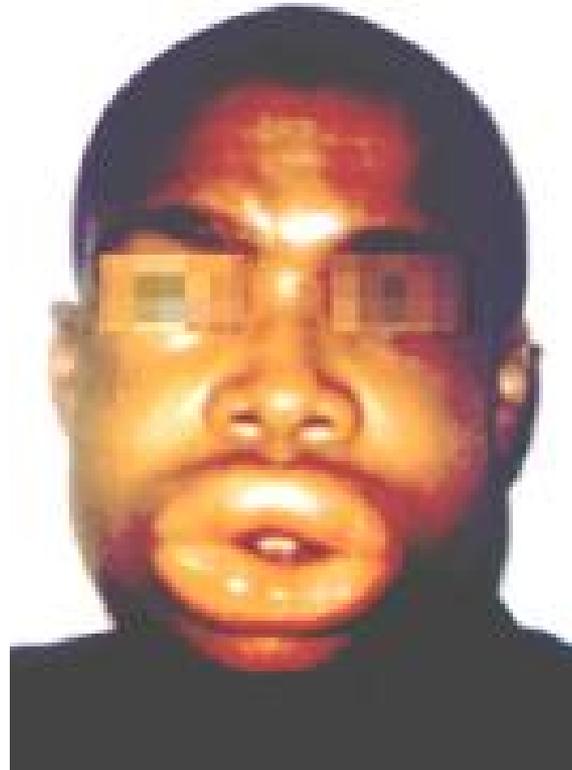
Urticaria (Hives)

- Signs & symptoms
 - Raised, pruritic, nontender, erythematous wheals on skin
 - Usually on trunk and proximal extremities
- Diagnosis
 - H & P
- Treatment
 - Epinephrine
 - Corticosteroids
 - Antihistamines
 - Histamine H₂ blockers
 - Cimetidine or ranitidine

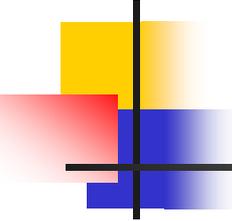
Type I (cont.)

Angioedema

- Pathophysiology
 - Form of urticaria
 - Affects submucosal/subcutaneous tissue rather than skin
- Signs & Symptoms
 - Painless, minimally pruritic
 - Dermal erythematous/subcutaneous eruptions
 - Skin/mucous membrane edema



Company is



Type II

- Cell or substance w/antigen attached to cell membrane
 - Destroyed by IgG or IgM
 - Senses as being a foreign antigen
- May be beneficial
 - Bacteria
- May not be beneficial
 - RBC sensed as foreign

Type II

Hemolytic Transfusion Reaction

- RBCs with antigens foreign to person rapidly lysed
 - Massive amount of cellular debris
 - Occludes blood vessels throughout body
 - Ischemia, necrosis
 - Life threatening

Type II

Hemolytic Transfusion Reaction

- Etiology

- Prior sensitization

- Bacterial or viral infection – occasionally
- Prior blood transfusion or past pregnancy
 - Most common
 - Maternal and fetal Rh factors are different
 - Mother becomes sensitized by the fetal Rh type
 - Affects future fetuses

Type II

Hemolytic Transfusion Reaction

- Signs & Symptoms
 - Sudden onset of low back/chest pain
 - Hypotension
 - Fever, chills
 - Tachycardia, tachypnea, wheezing, dyspnea
 - Urticaria
 - Anxiety, headache, nausea

Type II

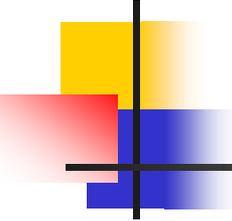
Hemolytic Transfusion Reaction

- Diagnosis
 - Direct Coombs' test
- Prevention
 - Rhogam: Rh₀ (D) negative patients
 - Careful blood transfusion administration

Type II

Hemolytic Transfusion Reaction

- Treatment
 - Depends on severity/organs affected
 - Antihistamines
 - Corticosteroids
 - Epinephrine
 - Diuretics
 - Assist with excretion of cellular debris
 - Prevent occlusion of kidneys



Type III

- Immune complexes formed by antigens and antibodies
 - Usually of the IgG type
 - Pt already sensitized with an initial antigen exposure
 - Reaction occurs on subsequent exposure
- Antigen-antibody complexes form within blood vessels
 - Lead to blood vessel damage
 - Red, edematous skin lesion, hemorrhage, necrosis

Type III

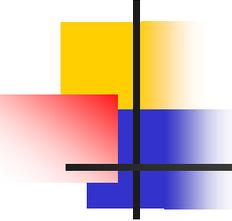
Serum Sickness

- Antigen-antibody complexes cause symptoms of inflammation
- 7 to 10 days after penicillin/sulfonamide
- Signs & Symptoms
 - Severe urticaria and angioedema
- Usually brief and self-limiting condition
 - Can become chronic

Type III

Serum Sickness

- Diagnosis
 - ↑ WBC & ESR, ↓ complement assay
- Medical treatment
 - Antipyretics
 - Antihistamines
 - Epinephrine
 - Corticosteroids – if severe



Type IV

- Delayed reaction
- Sensitized T lymphocyte contacts antigen to which its sensitized
- Cell-mediated immune response
- Necrosis

Type IV

Contact Dermatitis

- Pathophysiology
 - Chemical comes in contact with skin
 - On second exposure, T cells secrete chemicals
- Most common etiologic factors
 - Poison ivy, poison oak, latex rubber

Type IV

Contact Dermatitis

- Signs & symptoms
 - Reddened, pruritic, fragile vesicles
 - Onset usually within hours of exposure
- Diagnosis
 - Appearance of skin lesions/history
- Treatment
 - Symptom control
 - Oral/topical antihistamines or corticosteroids

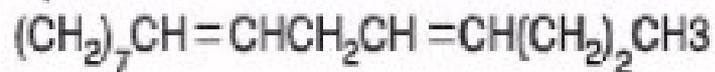
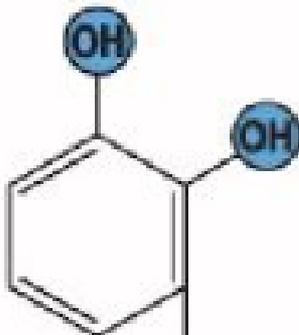
Contact dermatitis reaction to mango sap



Poison ivy / poison oak reaction

active hapten molecule

Pentadecacatechol



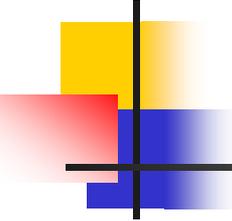




Type IV

Transplant Rejection

- Transplanted living tissue sensed as foreign
- Lymphocytes sensitized immediately after transplant
- Invade transplanted tissue and destroy it
- S/S reflect failure of specific organ or tissue
- Prevention
 - Immunosuppression therapy
- Infection can result in death



Autoimmune Disorders

- Immune system recognizes body's own cell antigens as foreign
- Immune response destroys them
- Influencing factors
 - Viral infections
 - Drugs
 - Cross-reactive antibodies

Autoimmune Disorders

Pernicious Anemia

- Pathophysiology
 - Antibodies against gastric parietal cells and intrinsic factor
 - Vitamin B₁₂ deficiency
 - RBC production decreased
- Etiology
 - Familial tendency
 - Gastric or small bowel resections
 - If vitamin B12 or intrinsic factor not replaced

Autoimmune Disorders

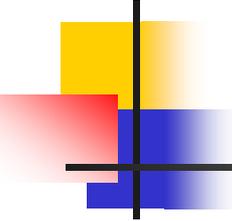
Pernicious Anemia

- Diagnosis
 - Macrocytic (large) RBCs
 - Low vitamin B12 levels
 - Schilling test
 - Gastric secretion analysis
 - Measures HCL levels
 - Will be low or absent
- Treatment
 - Corticosteroids
 - Lifelong vitamin B₁₂ IM
 - Initially weekly – then monthly

Autoimmune Disorders

Pernicious Anemia

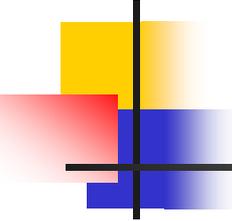
- Nursing management
 - Administer vitamin B12 as prescribed
 - Ambulation w/frequent rest periods
 - Assistance w/ADLs
 - Education
 - Lifelong treatment and follow-up



Autoimmune Disorders

Idiopathic Autoimmune Hemolytic Anemia

- Pathophysiology
 - Autoantibodies attach to RBCs
 - Causes lysis or agglutination (clumping)
 - Lysis - Fragments of destroyed RBCs circulate in blood
 - Agglutination – causes occlusion and ischemia
- Signs & symptoms
 - Vary to mild to severe
 - Fatigue, pallor, hypotension, dyspnea, palpitations, jaundice



Autoimmune Disorders

Idiopathic Autoimmune Hemolytic Anemia

- Diagnosis

- ↓ RBC, Hgb & Hct
- Fragmented RBCs on microscopic exam
- ↑ LDH
 - Due to destroyed RBCs and ischemia

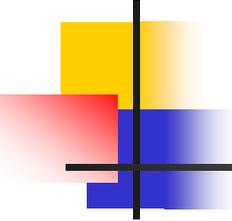
- Treatment

- Immunosuppressive medications & corticosteroids
- Oxygen
- Folic acid
- Blood transfusions & erythrocytapheresis
- Splenectomy

Autoimmune Disorders

Hashimoto's Thyroiditis

- Pathophysiology
 - Auto antibodies for TSH
 - Causes thyroid gland over stimulation
 - Thyromegaly
 - Due to overstimulation, infiltration w/lympocytes and phagocytes → causes inflammation and further enlargement
 - Auto antibodies destroy thyroid
 - Hypothyroidism

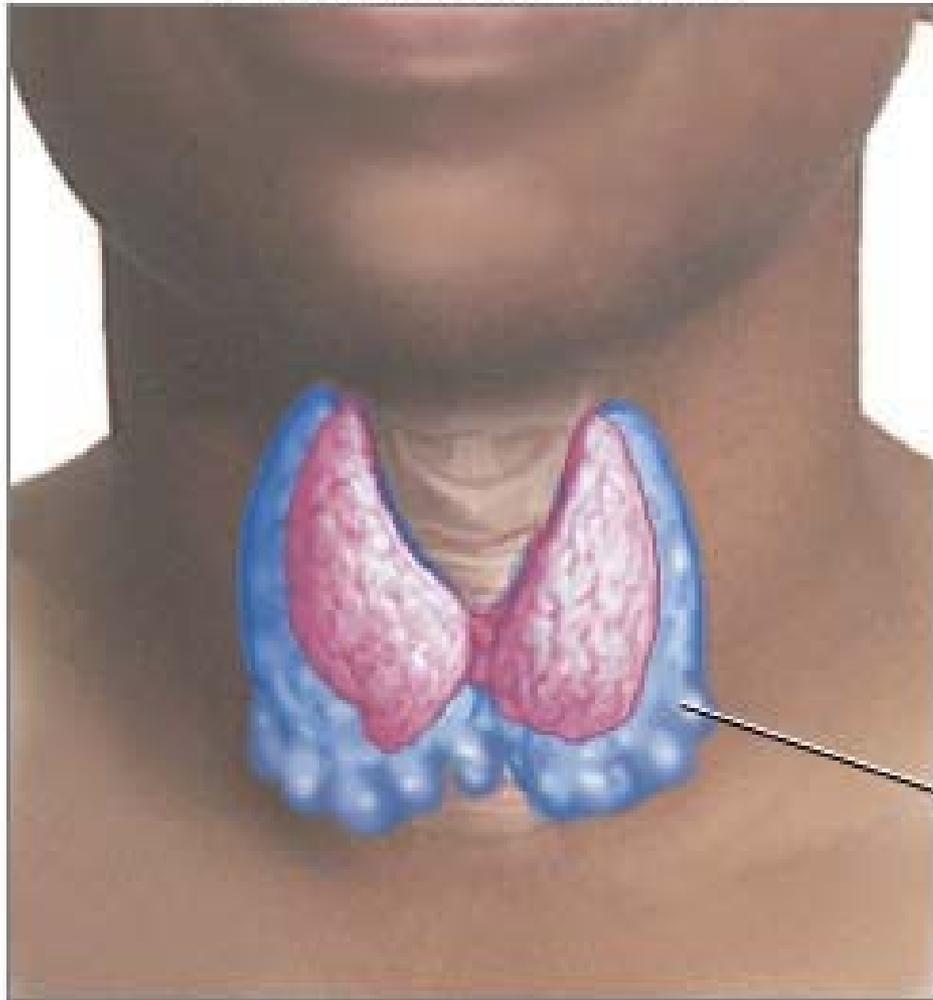


Autoimmune Disorders

Hashimoto's Thyroiditis

- Etiology
 - Unknown
 - More common
 - Women by 8 times
 - 30 – 50 years old
 - Down syndrome and Turner's syndrome
- Signs & symptoms
 - Initially same as hyperthyroidism
 - Then progress to hypothyroidism
 - Goiter

Hashimoto's disease



Enlarged, inflamed
hypofunctioning
thyroid (goiter)

Autoimmune Disorders

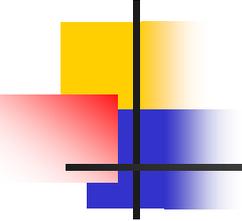
Hashimoto's Thyroiditis

- Diagnosis

- Immunofluorescent assay
- ↑ TSH, ↓ T3 & T4
- Thyroid scan

- Treatment

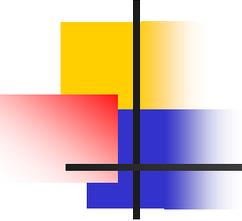
- Lifelong thyroxine replacement therapy



Autoimmune Disorders

Ankylosing Spondylitis

- Pathophysiology
 - Chronic progressive inflammatory disease of sacroiliac, costovertebral, large peripheral joints
- Etiology
 - Familial tendency
 - Affects men more frequently

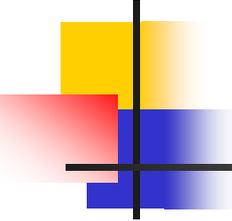


Autoimmune Disorders

Ankylosing Spondylitis

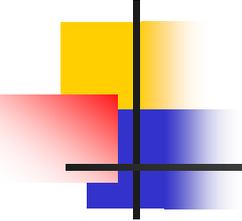
- Signs & symptoms
 - Lower back stiffness, pain, lordosis, kyphosis, spasms, fatigue, anorexia, weight loss
- Treatment
 - No cure, supportive care, surgery
- Education: ROM frequently





Immune Deficiencies

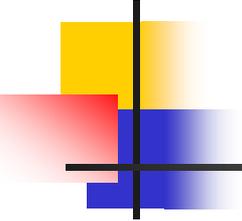
- One or more components of immune system completely absent or deficient
 - Inability to elicit or sustain an adequate immune response
 - Unable to fight off an infectious agent



Immune Deficiencies

Hypogammaglobulinemia

- Pathophysiology
 - Absence or deficiency of one or more of 5 immunoglobulins from defective B cell function
 - Prone to infections
- Etiology
 - Hereditary congenital disorder
 - More common in males
 - Usually have a normal lifespan
 - Acquired after childhood from unknown cause



Immune Deficiencies

Hypogammaglobulinemia

- Signs & symptoms
 - Asymptomatic until 6 months
 - When maternal immunoglobulins are gone
 - Recurrent infections
 - Especially staph & strep
- Treatment
 - Minimizing infections
 - IgG injections
 - Fresh frozen plasma replaces IgM
 - IgA cannot be replaced
 - Increases risk of pulmonary infections