UNIT TERMINAL OBJECTIVE

1-6 At the completion of this unit, the paramedic student will be able to apply the general concepts of pathophysiology for the assessment and management of emergency patients.

COGNITIVE OBJECTIVES

At the completion of this unit, the paramedic student will be able to:

1-6.1 Discuss cellular adaptation. (C-1)
1-6.2 Describe cellular injury and cellular death. (C-1)
1-6.3 Describe the factors that precipitate disease in the human body. (C-1)
1-6.4 Describe the cellular environment. (C-1)
1-6.5 Discuss analyzing disease risk. (C-1)
1-6.6 Describe environmental risk factors. (C-1)
1-6.7 Discuss combined effects and interaction among risk factors. (C-1)
1-6.8 Describe aging as a risk factor for disease. (C-1)
1-6.9 Discuss familial diseases and associated risk factors. (C-1)
1-6.10 Discuss hypoperfusion. (C-1)
1-6.11 Define cardiogenic, hypovolemic, neurogenic, anaphylactic and septic shock. (C-1)
1-6.12 Describe multiple organ dysfunction syndrome. (C-1)
1-6.13 Define the characteristics of the immune response. (C-1)
1-6.14 Discuss induction of the immune system. (C-1)
1-6.15 Discuss fetal and neonatal immune function. (C-1)
1-6.16 Discuss aging and the immune function in the elderly. (C-1)
1-6.17 Describe the inflammation response. (C-1)
1-6.18 Discuss the role of mast cells as part of the inflammation response. (C-1)
1-6.19 Describe the plasma protein system. (C-1)
1-6.20 Discuss the cellular components of inflammation. (C-1)
1-6.21 Describe the systemic manifestations of the inflammation response. (C-1)
1-6.22 Describe the resolution and repair from inflammation. (C-1)
1-6.23 Discuss the effect of aging on the mechanisms of self-defense. (C-1)
1-6.24 Discuss hypersensitivity. (C-1)
1-6.25 Describe deficiencies in immunity and inflammation. (C-1)
1-6.26 Describe homeostasis as a dynamic steady state. (C-1)
1-6.27 List types of tissue. (C-1)
1-6.28 Describe the systemic manifestations that result from cellular injury. (C-1)
1-6.29 Describe neuroendocrine regulation. (C-1)
1-6.30 Discuss the inter-relationships between stress, coping, and illness. (C-1)

AFFECTIVE OBJECTIVES

At the completion of this unit, the paramedic student will be able to:

1-6.31 Advocate the need to understand and apply the knowledge of pathophysiology to patient assessment and treatment. (A-2)

PSYCHOMOTOR OBJECTIVES

None identified for this unit.

DECLARATIVE

I. Introduction
- A. Correlation of pathophysiology with disease process
  - 1. Cells appear similar to multicellular “social” organism
  - 2. Cells communicate electrochemically - when interrupted disease processes can initiate and advance
  - 3. Knowledge of coordination of specific bodily functions leads to better understanding of the disease process
    - a. Endocrine
    - b. Exocrine
    - c. Other coordinating receptors
      - (1) Chemoreceptors
      - (2) Baroreceptors
      - (3) Adrenergic
      - (4) Others
- B. Correlation of disease process with care provided to patients by paramedics
  - 1. Understanding disease process is important for paramedics to better understand, anticipate, correct, and provide appropriate care
    - a. Once knowledge of physical laws and principles have been gained paramedics can apply these to the mechanisms and complications of disease
    - b. Cells of the immune system and inflammatory responses are found with every type of trauma or disease process

II. Basic cellular review
- A. Major classes of cells - living cells divided into two major divisions
- B. Chief cellular functions
  - 1. Cells become specialized through process of differentiation, or maturation
  - 2. Eventually perform one function or act in concert with other cells to perform a more complex task
- C. Cellular components
  - 1. Structure & function
  - 2. Three main components
- D. Tissue types
  - 1. Epithelial tissue
  - 2. Connective tissue
  - 3. Muscle tissue
  - 4. Nervous tissue

III. Alterations in cells and tissues
- A. Cellular adaptation - cells adapt to their environment to avoid and protect themselves from injury; adapted cells are neither normal or injured (they are somewhere between these two states)
  - 1. Cellular adaptations are common and a central part of many disease states
    - a. Early stages of a successful adaptation response may enhance the cell’s function
    - b. Difficult to determine pathological responses versus an extreme adaptation to an excessive functional demand
  - 2. Atrophy

3. Hypertrophy
4. Hyperplasia
5. Dysplasia
6. Metaplasia

B. Cellular injury
   1. Hypoxic injury
      a. Most common cause of cellular injury
      b. May result from
         (1) Decreased amounts of oxygen in the air
         (2) Loss of hemoglobin or hemoglobin function
         (3) Decreased number of red blood cells
         (4) Disease in respiratory or cardiovascular system
         (5) Loss of cytochromes
   2. Chemical injury
      a. Chemical agents causing cellular injury
         (1) Poisons
         (2) Lead
         (3) Carbon monoxide
         (4) Ethanol
         (5) Pharmacological
   3. Infectious injury
      a. Virulence or pathogenicity of microorganisms depends on their ability to survive and reproduce in the human body, where they injure cells and tissues
         (1) Disease producing potential depends upon its ability to
            (a) Invade and destroy cells
            (b) Produce toxins
            (c) Produce hypersensitivity reactions
      b. Bacteria
         (1) Survival and growth depend upon the effectiveness of the body's defense mechanisms and the bacteria's ability to resist the mechanisms
            (a) Coating protects the bacterium from ingestion and destruction by phagocytes and capsules may also function as exotoxins
            (b) Not all virulent extracellular pathogens are encapsulated - mycobacterium tuberculosis can survive and be transported by phagocytes
         (2) Bacteria also produce substances such as enzymes or toxins which can injure or destroy cells
            (a) Toxins are produced by many microorganisms
               i) Exotoxins
               ii) Endotoxins
            (b) Fever is caused by the release of endogenous pyrogens from macrophages or circulating white blood cells that are attracted to the injury site
            (c) Inflammation is one of the body's responses to the presence of bacteria
            (d) Ability to produce hypersensitivity reactions is an important pathogenic mechanism of bacteria toxins
            (e) Bacteremia or septicemia is proliferation of microorganisms in the

blood

c. Viruses
(1) Viral disease are among the most common afflictions seen in humans
(2) Intracellular parasites that take over the control of metabolic machinery of host cells for use to replicate the virus
(3) Protein coat (capsid) encapsulating most viruses allows them to resist phagocytosis
(4) Viral replication occurs within the host cell
(5) Having no organelles, viruses are incapable of metabolism
(6) Causes decreased synthesis of macromolecules vital to the host cell
(7) Viruses do not produce exotoxins or endotoxins
(8) There may be a symbiotic relationships between viruses and normal cells resulting in a persistent unapparent infection
(9) Viruses can evoke a strong immune response but can rapidly produce irreversible and lethal injury in highly susceptible cells (as in AIDS)

4. Immunologic and inflammatory injury
a. Cellular membranes are injured by direct contact with cellular and chemical components of the immune or inflammatory process as in phagocytes (lymphocytes and macrophages) and others such as histamine, antibodies, lymphokines
b. Membrane alterations are associated with rapid leakage of potassium out of the cell and an influx of water
5. Injurious genetic factors
6. Injurious nutritional imbalances
7. Injurious physical agents

C. Manifestations of cellular injury
1. Cellular manifestations
2. Systemic manifestations

D. Cellular death/ necrosis

IV. The cellular environment
A. Distribution of body fluids
1. Intracellular fluid (ICF)
2. Extracellular fluid (ECF)
a. Interstitial fluid
b. Intravascular fluid
c. Other
3. Total body water (TBW)

B. Aging and distribution of body fluids
1. Birth
2. Infancy
3. Childhood
4. Adulthood
5. Elderly

C. Water movement between ICF and ECF
1. Osmotic forces
2. Role of sodium and potassium

D. Water movement between plasma and interstitial fluid
1. Osmotic forces within capillary bed

2. Starling's hypothesis
3. Role of capillary and membrane permeability

E. Alterations in water movement
   1. Edema
      a. Pathophysiology
         (1) Increased capillary permeability
         (2) Decreased oncotic pressure
         (3) Increased capillary hydrostatic pressure
         (4) Hydrostatic pressure
         (5) Lymphatic vessel obstruction
      b. Clinical manifestations
         (1) Local
         (2) Generalized
      c. Evaluation and treatment

F. Water balance and the role of electrolytes
   1. Water balance
      a. Role of antidiuretic hormone (ADH)
      b. Receptors
         (1) Osmoreceptors
         (2) Volume sensitive receptors
         (3) Baroreceptors
   2. Sodium and chloride balance
      a. Role and function of sodium as a cation
      b. Role and function of chloride as an anion
      c. Hormone regulation by aldosterone and natriuretic hormone
      d. Role of renin-angiotensin system
   3. Alterations in sodium, chloride, and water balance
      a. Isotonic alterations
         (1) Isotonic volume depletions
         (2) Isotonic volume excesses
      b. Hypertonic alterations
         (1) Hypernatremia
         (2) Water deficit
         (3) Hyperchloremia
      c. Hypotonic alterations
         (1) Hyponatremia
         (2) Water excess
         (3) Hypochloremia
   4. Alterations in potassium, calcium, phosphate, and magnesium balance
      a. Potassium
         (1) Hypokalemia
         (2) Hyperkalemia
      b. Calcium and phosphate
         (1) Hypocalcemia
         (2) Hypercalcemia
         (3) Hypophosphatemia
         (4) Hyperphosphatemia
      c. Magnesium

(1) Hypomagnesemia
(2) Hypermagnesemia

G. Acid - base balances
1. Hydrogen ion and pH
2. Buffer systems
a. Carbonic acid-bicarbonate buffering
b. Protein buffering
c. Renal buffering
d. Other buffers
3. Acid-base imbalances
a. Metabolic acidosis
(1) Pathophysiology
(2) Clinical presentation
(3) Evaluation and treatment
b. Metabolic alkalosis (rare)
(1) Pathophysiology
(2) Clinical presentation
(3) Evaluation and treatment
c. Respiratory acidosis
(1) Pathophysiology
(2) Clinical presentation
(3) Evaluation and treatment
d. Respiratory alkalosis
(1) Pathophysiology
(2) Clinical presentation
(3) Evaluation and treatment

V. Genetics and familial diseases
A. Factors causing disease
1. Genetic
2. Environmental
a. Microoganisms and immunologic exposures
b. Personal habits and life-style
c. Chemical substances
d. Physical environment
e. Psychosocial environment
3. Age and gender
a. Accumulative affects of both genetic and environmental factors
b. Life-style, anatomic, or hormonal differences
B. Analyzing disease risk
1. Disease rates
a. Incidence rate
b. Prevalence rate
c. Mortality rate
2. Risk factor analysis
a. Causal risk factors
b. Noncausal risk factors
C. Combined effects and interaction among risk factors

1. Familial disease tendency
2. Aging and age-related disorders

D. Common familial disease and associated risk factors
   1. Immunologic disorders
      a. Allergies
      b. Asthma
      c. Rheumatic fever
   2. Cancer
      a. Breast cancer
      b. Colorectal cancer
      c. Lung cancer
   3. Endocrine disorders
      a. Diabetes mellitus
         (1) Insulin-dependent diabetes mellitus
         (2) Non-insulin dependent diabetes mellitus
   4. Hematologic disorders
      a. Drug-induced hemolytic anemia
      b. Hemophilia
      c. Hematochromatosis
   5. Cardiovascular disorders
      a. Long QT syndrome (autosomal dominant disorder)
      b. Cardiac myopathies
      c. Mitral valve prolapse
      d. Coronary heart disease
         (1) Family history and CHD risk
         (2) Genetic factors and predisposition
      e. Hypertension and stroke
   6. Renal disorders
      a. Gout (uric acid accumulation)
      b. Kidney stones
   7. Gastrointestinal disorders
      a. Malabsorption disorders
         (1) Lactose intolerance
         (2) Ulcerative colitis
         (3) Crohn's disease
      b. Peptic ulcers
      c. Gallstones
      d. Obesity
         (1) Associated disease processes
         (2) Causal risk factors
   8. Neuromuscular disorders
      a. Huntington disease
      b. Muscular dystrophy
      c. Multiple sclerosis
      d. Alzheimer disease
   9. Psychiatric disorders
      a. Schizophrenia
      b. Manic-depressive

VI. Hypoperfusion
- A. Pathogenesis
  - 1. Decreased cardiac output
  - 2. Compensatory mechanisms
    - a. Catecholamine release
      - (1) Epinephrine and norepinephrine
      - (2) Increase in systemic vascular resistance
    - b. Role of aldosterone renin-angiotensin, and ADH
      - (1) Adequate or increased blood volume
      - (2) Vasoconstriction increases systemic blood pressure
    - c. Shift of interstitial fluid
    - d. Splenic discharge
  - 3. Increased preload, stroke volume, and heart rate
    - a. Increased myocardial oxygen demand
    - b. Systemic and pulmonary edema
      - (1) Dyspnea
      - (2) Dusky skin color
      - (3) Low blood pressure
      - (4) Oliguria
      - (5) Impaired mentation
    - c. Decreased cardiac output and ejection fraction
      - (1) Decreased blood pressure
      - (2) Decreased tissue perfusion
      - (3) Impaired cellular metabolism
- B. Types of Shock
  - 1. Cardiogenic shock
    - a. Defined
    - b. Pathophysiology
    - c. Evaluation and treatment
  - 2. Hypovolemic shock
    - a. Defined
    - b. Pathophysiology
    - c. Evaluation and treatment
  - 3. Neurogenic shock
    - a. Defined
    - b. Pathophysiology
    - c. Evaluation and treatment
  - 4. Anaphylactic shock
    - a. Defined
    - b. Pathophysiology
    - c. Evaluation and treatment
  - 5. Septic Shock
    - a. Defined
    - b. Pathophysiology
    - c. Evaluation and treatment
- C. Multiple organ dysfunction syndrome (MODS)
  - 1. Defined

a. Progressive failure of two or more organ systems
b. Occurs after severe illness or injury
c. New diagnosis first described in 1975
d. Mortality rate of 60% - 90%
e. Major cause of death following septic, traumatic, and burn injuries

2. Pathophysiology
a. Injury or endotoxin release
b. Vascular endothelial damage, neuroendocrine response, and release of inflammatory mediators
c. Activation of complement, coagulation, and kallikrein/ kinin systems
d. Massive systemic immune/ inflammatory and coagulation responses
e. Vascular changes
(1) Vasodilation
(2) Increase in capillary permeability
(3) Selective vasoconstriction
(4) Microvascular thrombi
f. Maldistribution of systemic and organ blood flow
g. Hypermetabolism
h. Oxygen supply/ demand imbalance
i. Tissue hypoxia
(1) Tissue hypoperfusion
(2) Exhaustion of fuel supply (i.e. ATP, glucose, etc)
(3) Metabolic failure
(4) Lysosome breakdown
(5) Anaerobic metabolism
(6) Acidosis and impaired cellular function
j. Organ dysfunction
(1) Decreased cardiac function and myocardial depression
(2) Renal failure
(3) Failure of smooth muscle of vascular system
(a) Release of capillary sphincters
(b) Vasodilation

3. Clinical presentation - 24 hours after initial resuscitation
a. Low-grade fever due to inflammatory responses
b. Tachycardia
c. Dyspnea and adult respiratory distress syndrome (ARDS)
d. Altered mental status
e. Hyperdynamic state
f. Hypermetabolic states
g. Renal and liver failure (14 - 21 days)
h. Gastrointestinal and immune collapse (14 - 21 days)
i. Cardiovascular collapse and death (21 - 28 days)

D. Cellular metabolism impairment
1. Oxygen impairment
a. Anaerobic metabolism
b. Increased lactate
c. Metabolic acidosis
d. Decreased oxygen affinity for hemoglobin

e. Decreased ATP
f. Changes in cellular electrolytes
g. Cellular edema
h. Release of lysosomal enzymes

2. Impaired glucose use
   a. Increase serum glucose
   b. Catecholamines, cortisol, growth hormone release
   c. Increased gluconeogenesis, gluconeolysis, and lipolysis

VII. Self-defense mechanisms
A. Introduction - lines of defense
1. Anatomic barriers
2. Inflammatory response
3. Immune response
B. Characteristics of the immune response
1. Natural versus acquired immunity
   a. Natural or native immunity
   b. Acquired immunity
      (1) Active acquired immunity
      (2) Passive acquired immunity
2. Primary versus secondary immunity
   a. Primary or initial immune response
   b. Secondary or anamnestic immune response
3. Humoral versus cell-mediated immunity
   a. B-cell lymphocyte
   b. T-cell lymphocyte
C. Induction of the immune response
1. Antigens and immunogens
   a. Antigens
   b. Immunogen
   c. Tolerance
   d. Molecular size
      (1) Larger - proteins, polysaccharides, and nucleic acids
      (2) Smaller - amino acids, monosaccharides, and fatty acids
      (3) Haptens - smaller molecules which become immunogenic
2. Histocompatibility antigens (HLA antigens)
   a. HLA complexes or major histocompatibility complexes (MHC)
   b. Role of HLA antigens
3. Blood group antigens
   a. Rh system
   b. ABO system
D. Humoral immune response
1. B-cell lymphocytes
   a. Formation
      (1) Lymphoid stem cell
      (2) Generation of clonal diversity
      (3) Clonal selection
      (4) Activated B-cell

(a) Immunoglobulin-secreting plasma cells found in blood and secondary lymphoid organs
(b) Memory cells - responsible for long term immunity

2. Immunoglobulins
a. Differences between immunoglobulins and antibodies
b. Structure of immunoglobulin molecules
c. Function of antibodies
(1) Agglutination
(2) Precipitation
(3) Neutralization
(a) Bacterial toxins
(b) Viruses
(c) Opsonization of bacteria
(d) Activation of inflammatory processes
(e) Classes of immunoglobulins
(f) Antibodies as antigens
(4) Isotypic antigens
(5) Allotypic antigens
(6) Idiotypic antigenic determinants
d. Monoclonal antibodies

3. Secretory immune system
a. Mucosal-associated lymphoid tissue
(1) Lacrimal glands
(2) Salivary glands
(3) Bronchial-associated lymphoid tissue
(4) Mammary-associated lymphoid tissue
(5) Gut-associated lymphoid tissue
(6) Genital-associated lymphoid tissue
b. Circulates independently of other lymphocytes
(1) Mucosal-associated lymphoid tissue
(2) Regional lymph nodes
(3) Thoracic duct
(4) Blood
c. One of body's first lines of defense
d. Occurs locally rather than systemically

E. Cell-mediated immune response
1. T-cells
a. Five types of mature T-cells
(1) Memory cells
(2) Td cells or lymphokine-producing cells
(3) Tc cells or cytotoxic cells
(4) Th cells or helper T-cells
(5) Ts cells or suppressor T-cells
b. Proliferation and differentiation

2. Major effects of cell-mediated immune response
a. Cytotoxicity
b. Delayed hypersensitivity
c. Memory

d. Control

F. Cellular interactions in the immune response
1. Cytokines
a. Lymphokines
b. Monokines
2. Antigen processing, presentation, and recognition
a. Antigen degradation
b. Classes of histocompatible antigens (HLA)
c. T-cell receptors
d. Interleukin - 1 (IL-1)
3. T-cell and B-cell differentiation
a. T-cell differentiation
b. B-cell differentiation
c. Control of B and T-cell development

G. Fetal and neonatal immune function
1. Fetal immunological capabilities
a. Immunologic responses
b. Antibody capabilities
2. Antibody levels
a. Umbilical cord blood
b. Neonatal circulation
3. Trophoblasts

H. Aging and the immune response in elderly
1. T-cell function
2. Antibody production

VIII. Inflammation
A. The acute inflammatory response
1. Triggers
a. Lethal cellular injury
b. Non-lethal cellular injury
c. Other microorganisms
2. Response
a. Vascular responses to inflammation
b. Cellular responses to inflammation

B. Mast cells
1. Degranulation of vasoactive amines and chemotactic factors
a. Stimulation of degranulation
(1) Physical injury
(2) Chemical agents
(3) Immunological (IgE-mediated hypersensitivity)
b. Vasoactive amines
(1) Histamine
(2) Serotonin
c. Chemotactic factors
(1) Neutrophil
(2) Eosinophil
2. Synthesis of leukotrienes and prostaglandins

a. Leukotrienes or slow-reacting substances of anaphylaxis (SRS-A)
(1) Composition
(2) Function
b. Prostaglandins
(1) Composition
(2) Function

C. Plasma protein systems
1. Complement system
a. Structure and function
b. Activation
(1) Classic pathway
(2) Alternative pathway
2. Clotting system
a. Structure and function
b. Activation
(1) Extrinsic pathway
(2) Intrinsic pathway
3. Kinin system
a. Structure and function
b. Activation
(1) Plasma kinin cascade
4. Control and interaction of the plasma protein system
a. Reason for control
b. Types of control
(1) Antagonists
(2) Histamine control
(3) Interaction of control processes

D. Cellular components of inflammation
1. Functions of phagocytes
a. Margination
b. Diapedesis
c. Exudation into inflamed tissue
d. Process of phagocytosis
2. Polymorphonuclear neutrophils
a. Predominance in early inflammatory response
b. Role
3. Monocytes and macrophages
a. Monocyte - young macrophage
(1) Structure
(2) Role
b. Macrophages
(1) Structure
(2) Role
4. Eosinophils
a. Structure
b. Role

E. Cellular products
1. Interleukins (ILs)

a. Interleukin - 1
b. Interleukin - 2
2. Lymphokines
a. Production
b. Types and effects
(1) Migration-inhibitory factor
(2) Macrophage-activating factor
3. Interferon
a. Structure
b. Actions and effects

F. Systemic responses of acute inflammation
1. Fever
a. Activation
b. Effects
2. Leukocytosis
a. Activation
b. Effects
3. Increase in circulating plasma proteins or acute-phase reactants
a. Activation
b. Effects

G. Chronic inflammation responses
1. Causes
a. Unsuccessful acute inflammatory response due to foreign body
b. Persistence of infection or antigen
2. Characteristics
a. Persistence of acute inflammation response
b. Neutrophil degranulation and death
c. Lymphocyte activation
d. Fibroblast activation
e. Infiltration (pus)
f. Tissue repair (scar)

H. Local inflammation responses
1. Vascular changes
a. Vasodilation
b. Increased capillary permeability
2. Exudation
a. Functions
b. Compositions

I. Phases of resolution and repair
1. Definitions
a. Regeneration
b. Repair
c. Debridement
d. Primary intention
e. Secondary intention
2. Reconstruction phase
a. Initial wound response
b. Granulation

c. Epithelialization
3. Maturation Phase
a. Completion of contraction, differentiation, and remodeling of scar tissue
b. Disappearance of capillaries from scar tissue
4. Dysfunctional wound healing
a. Dysfunction during the inflammatory response
b. Dysfunction during the reconstruction phase
(1) Impaired collagen synthesis
(2) Impaired epithelialization
(3) Wound disruption
(4) Impaired contraction

J. Aging and self-defense mechanisms
1. Newborn
2. Elderly

IX. Variances in immunity and inflammation
A. Hypersensitivity: allergy, autoimmunity, and isoimmunity
1. Definitions
a. Hypersensitivity
b. Allergy
c. Autoimmunity
d. Isoimmunity
2. Mechanisms of hypersensitivity
a. Immediate versus delayed reactions
b. IgE reactions
(1) Role of IgE
(2) Mechanism of IgE
(3) Clinical indications
(4) Genetic predisposition
(5) IgE-mediated hypersensitivity tests
(6) Desensitization
c. Tissue-specific reactions
(1) Tissue-specific antigens
(2) Mechanisms
d. Immune-complex mediated injury
(1) Mechanisms
(2) Immune-complex disease
e. Cell-mediated tissue destruction
(1) Mechanisms
(2) Clinical instances
3. Targets of hypersensitivity
a. Allergy
(1) Allergens
(2) Neoantigen
b. Autoimmunity
(1) Breakdown of tolerance
(2) Original insult
(3) Genetic factors

c. Isoimmunity
(1) Transient neonatal diseases
(2) Transplant rejections and transfusion reactions
4. Autoimmune and isoimmune diseases
a. Grave's disease
b. Rheumatoid arthritis
c. Myasthenia gravis
d. Immune thrombocytopenic purpura
e. Isoimmune neutropenia
f. Systemic lupus erythematosus (SLE)
g. Rh and ABO isoimmunization

B. Immunity and inflammation deficiencies
1. Congenital immune deficiencies
2. Acquired deficiencies
a. Nutritional deficiencies
b. Iatrogenic deficiencies
c. Deficiencies caused by trauma
d. Deficiencies caused by stress
e. AIDS
3. Replacement therapies for immune deficiencies
a. Gamma globulin therapy
b. Transplantation and transfusion
c. Gene therapy

X. Stress and disease
A. Concepts of stress
1. Triad of manifestations
2. General adaptation syndrome (Selye)
a. Alarm stage
b. Resistance or adaptation stage
c. Exhaustion stage
d. Definition of physiological stress
3. Psychologic mediators and specificity
a. Psychologic factors effects on physiological responses to stress
b. Pituitary gland and adrenal cortex sensitivity to emotional, psychologic and social influences
4. Homeostasis as a dynamic steady state
a. Definitions
(1) Dynamic steady state
(2) Turnover
b. Reaction of body to stressors

B. Stress responses
1. Psychoneuroimmunologic response
a. Interaction of consciousness, brain and central nervous system, and the body's defense mechanisms
b. Stress response
2. Neuroendocrine regulation
a. Catecholamines

(1) Components
  (a) Epinephrine
  (b) Norepinephrine
(2) Physiologic actions of alpha and beta receptors
  (a) $Alpha_1$
  (b) $Alpha_2$
  (c) $Beta_1$
  (d) $Beta_2$
(3) Physiologic effects of catecholamines
  (a) Brain
  (b) Cardiovascular
  (c) Pulmonary
  (d) Muscle
  (e) Liver
  (f) Adipose Tissue
  (g) Skin
  (h) Skeleton
  (i) G.I. and G.U. systems
  (j) Lymphoid tissue

b. Cortisol
(1) Source
(2) Primary effects of cortisol
  (a) Stimulation of glucogenesis
  (b) Formation of glycogen
  (c) Cortisol effects on cell-mediated immunity
(3) Other physiologic effects of cortisol
  (a) Protein metabolism
  (b) Digestive function
  (c) Urinary function
  (d) Connective tissue function
  (e) Muscle function
  (f) Bone function
  (g) Vascular system and myocardial function
  (h) Central nervous system function

c. Other hormones
(1) Endorphins
(2) Growth hormone
(3) Prolactin
(4) Testosterone

d. Role of the immune system
(1) Interaction of immune, nervous, and endocrine systems during a stress response
(2) Influence of stress response on immune system
(3) Relationship between stress and immune-related conditions and diseases
  (a) Cardiovascular
  (b) Muscles
  (c) Connective tissue
  (d) Pulmonary system

(e) Immune system
(f) G.I. system
(g) G.U. system
(h) Skin
(i) Endocrine system
(j) Central nervous system

C. Stress, coping, and illness interrelationships
  1. Stress as interdependent processes
    a. Definition of physiologic stress and psychologic distress
    b. Effects of psychologic distress
    c. Relationship between distress and immune dysfunction
  2. Potential stress effects on
    a. Healthy individuals
      (1) Ineffective coping
      (2) Effective coping
    b. Symptomatic individuals
      (1) Ineffective coping
      (2) Effective coping
    c. Medical interventions
      (1) Ineffective coping
      (2) Effective coping

REFERENCE

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