UNIT TERMINAL OBJECTIVE

4-2 the completion of this unit, the paramedic student will be able to integrate pathophysiological principles and assessment findings to formulate a field impression and implement the treatment plan for the patient with shock or hemorrhage.

COGNITIVE OBJECTIVES

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

4-2.1 Describe the epidemiology, including the morbidity/ mortality and prevention strategies, for shock and hemorrhage. (C-1)
4-2.2 Discuss the anatomy and physiology of the cardiovascular system. (C-1)
4-2.3 Predict shock and hemorrhage based on mechanism of injury. (C-1)
4-2.4 Discuss the various types and degrees of shock and hemorrhage. (C-1)
4-2.5 Discuss the pathophysiology of hemorrhage and shock. (C-1)
4-2.6 Discuss the assessment findings associated with hemorrhage and shock. (C-1)
4-2.7 Identify the need for intervention and transport of the patient with hemorrhage or shock. (C-1)
4-2.8 Discuss the treatment plan and management of hemorrhage and shock. (C-1)
4-2.9 Discuss the management of external hemorrhage. (C-1)
4-2.10 Differentiate between controlled and uncontrolled hemorrhage. (C-3)
4-2.11 Differentiate between the administration rate and amount of IV fluid in a patient with controlled versus uncontrolled hemorrhage. (C-3)
4-2.12 Relate internal hemorrhage to the pathophysiology of compensated and decompensated hemorrhagic shock. (C-3)
4-2.13 Relate internal hemorrhage to the assessment findings of compensated and decompensated hemorrhagic shock. (C-3)
4-2.14 Discuss the management of internal hemorrhage. (C-1)
4-2.15 Define shock based on aerobic and anaerobic metabolism. (C-1)
4-2.16 Describe the incidence, morbidity, and mortality of shock. (C-1)
4-2.17 Describe the body's physiologic response to changes in perfusion. (C-1)
4-2.18 Describe the effects of decreased perfusion at the capillary level. (C-1)
4-2.19 Discuss the cellular ischemic phase related to hemorrhagic shock. (C-1)
4-2.20 Discuss the capillary stagnation phase related to hemorrhagic shock. (C-1)
4-2.21 Discuss the capillary washout phase related to hemorrhagic shock. (C-1)
4-2.22 Discuss the assessment findings of hemorrhagic shock. (C-1)
4-2.23 Relate pulse pressure changes to perfusion status. (C-3)
4-2.24 Relate orthostatic vital sign changes to perfusion status. (C-3)
4-2.25 Define compensated and decompensated hemorrhagic shock. (C-1)
4-2.26 Discuss the pathophysiological changes associated with compensated shock. (C-1)
4-2.27 Discuss the assessment findings associated with compensated shock. (C-1)
4-2.28 Identify the need for intervention and transport of the patient with compensated shock. (C-1)
4-2.29 Discuss the treatment plan and management of compensated shock. (C-1)
4-2.30 Discuss the pathophysiological changes associated with decompensated shock. (C-1)
4-2.31 Discuss the assessment findings associated with decompensated shock. (C-1)
4-2.32 Identify the need for intervention and transport of the patient with decompensated shock. (C-1)
4-2.33 Discuss the treatment plan and management of the patient with decompensated shock. (C-1)
4-2.34 Differentiate between compensated and decompensated shock. (C-3)
4-2.35 Relate external hemorrhage to the pathophysiology of compensated and decompensated hemorrhagic shock. (C-3)

4-2.36 Relate external hemorrhage to the assessment findings of compensated and decompensated hemorrhagic shock. (C-3)
4-2.37 Differentiate between the normotensive, hypotensive, or profoundly hypotensive patient. (C-3)
4-2.38 Differentiate between the administration of fluid in the normotensive, hypotensive, or profoundly hypotensive patient. (C-3)
4-2.39 Discuss the physiologic changes associated with the pneumatic anti-shock garment (PASG). (C-1)
4-2.40 Discuss the indications and contraindications for the application and inflation of the PASG. (C-1)
4-2.41 Apply epidemiology to develop prevention strategies for hemorrhage and shock. (C-1)
4-2.42 Integrate the pathophysiological principles to the assessment of a patient with hemorrhage or shock. (C-3)
4-2.43 Synthesize assessment findings and patient history information to form a field impression for the patient with hemorrhage or shock. (C-3)
4-2.44 Develop, execute and evaluate a treatment plan based on the field impression for the hemorrhage or shock patient. (C-3)

AFFECTIVE OBJECTIVES
None identified for this unit.

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

4-2.45 Demonstrate the assessment of a patient with signs and symptoms of hemorrhagic shock. (P-2)
4-2.46 Demonstrate the management of a patient with signs and symptoms of hemorrhagic shock. (P-2)
4-2.47 Demonstrate the assessment of a patient with signs and symptoms of compensated hemorrhagic shock. (P-2)
4-2.48 Demonstrate the management of a patient with signs and symptoms of compensated hemorrhagic shock. (P-2)
4-2.49 Demonstrate the assessment of a patient with signs and symptoms of decompensated hemorrhagic shock. (P-2)
4-2.50 Demonstrate the management of a patient with signs and symptoms of decompensated hemorrhagic shock. (P-2)
4-2.51 Demonstrate the assessment of a patient with signs and symptoms of external hemorrhage. (P-2)
4-2.52 Demonstrate the management of a patient with signs and symptoms of external hemorrhage. (P-2)
4-2.53 Demonstrate the assessment of a patient with signs and symptoms of internal hemorrhage. (P-2)
4-2.54 Demonstrate the management of a patient with signs and symptoms of internal hemorrhage. (P-2)

DECLARATIVE

I. Pathophysiology, assessment, and management of hemorrhage
  A. Hemorrhage
    1. Epidemiology
      a. Incidence
      b. Mortality/ morbidity
      c. Prevention strategies
    2. Pathophysiology
      a. Location
        (1) External
          (a) Controlled
          (b) Uncontrolled
        (2) Internal
          (a) Trauma
          (b) Non-trauma
            i) Common sites
            ii) Uncommon sites
          (c) Controlled
          (d) Uncontrolled
      b. Anatomical type
        (1) Arterial
        (2) Venous
        (3) Capillary
      c. Timing
        (1) Acute
        (2) Chronic
      d. Severity
        (1) Amounts of blood loss tolerated by
          (a) Adults
          (b) Children
          (c) Infants
      e. Physiological response to hemorrhage
        (1) Clotting
        (2) Localized vasoconstriction
      f. Stages of hemorrhage
        (1) Stage 1
          (a) Up to 15% intravascular loss
          (b) Compensated by constriction of vascular bed
          (c) Blood pressure maintained
          (d) Normal pulse pressure, respiratory rate, and renal output
          (e) Pallor of the skin
          (f) Central venous pressure low to normal
        (2) Stage 2
          (a) 15-25% intravascular loss
          (b) Cardiac output cannot be maintained by arteriolar constriction
          (c) Reflex tachycardia
          (d) Increased respiratory rate

(e) Blood pressure maintained
(f) Catecholamines increase peripheral resistance
(g) Increased diastolic pressure
(h) Narrow pulse pressure
(i) Diaphoresis from sympathetic stimulation
(j) Renal output almost normal

(3) Stage 3
(a) 25-35% intravascular loss
(b) Classic signs of hypovolemic shock
i) Marked tachycardia
ii) Marked tachypnea
iii) Decreased systolic pressure
iv) 5-15 ml per hour urine output
v) Alteration in mental status
vi) Diaphoresis with cool, pale skin

(4) Stage 4
(a) Loss greater than 35%
(b) Extreme tachycardia
(c) Pronounced tachypnea
(d) Significantly decreased systolic blood pressure
(e) Confusion and lethargy
(f) Skin is diaphoretic, cool, and extremely pale

3. Assessment
a. Bright red blood from wound, mouth, rectum or other orifice
b. Coffee ground appearance of vomitus
c. Melena
d. Hematochezia
e. Dizziness or syncope on sitting or standing
f. Orthostatic hypotension
g. Signs and symptoms of hypovolemic shock

4. Management
a. Airway and ventilatory support
b. Circulatory support
(1) Bleeding from nose or ears after head trauma
(a) Refrain from applying pressure
(b) Apply loose sterile dressing to protect from infection
(2) Bleeding from other areas
(a) Control bleeding
i) Direct pressure
ii) Elevation if appropriate
iii) Pressure points
iv) Tourniquet
v) Splinting
vi) Packing of large gaping wounds with sterile dressings
vii) PASG
(b) Apply sterile dressing and pressure bandage
(3) Transport considerations
(4) Psychological support/ communication strategies

II. Shock
- A. Epidemiology
  - 1. Mortality/ morbidity
  - 2. Prevention strategies
  - 3. Pathophysiology
    - a. Perfusion depends on cardiac output (CO), systemic vascular resistance (SVR) and transport of oxygen
      - (1) CO = HR X SV
        - (a) HR - heart rate
        - (b) SV - stroke volume
      - (2) BP = CO X SVR
      - (3) Hypoperfusion can result from
        - (a) Inadequate cardiac output
        - (b) Excessive systemic vascular resistance
        - (c) Inability of red blood cells to deliver oxygen to tissues
    - b. Compensation for decreased perfusion
      - (1) Occurrence of event resulting in decreased perfusion, e.g., blood loss, myocardial infarction, loss of vasomotor tone or tension pneumothorax
      - (2) Baroreceptors sense decreased flow and activate vasomotor center
        - (a) Normally stimulated between 60-80 mm Hg systolic (lower in children)
        - (b) Located in carotid sinuses and aortic arch
        - (c) Arterial pressure drop decreases stretch
          - i) Nerve impulse through Vagus and Hering's nerve to glossopharyngeal nerve
          - ii) Impulse transmitted to vasomotor center
          - iii) Frequency of inhibitory impulses decreases
          - iv) Increase in vasomotor activity
          - v) Sympathetic nervous system stimulated
        - (iv) Decrease in systolic less than 80 mmHg stimulates vasomotor center to increase arterial pressure
      - (3) Chemoreceptors are stimulated by decrease in PaO_2 and increase in $PaCO_2$
      - (4) Sympathetic nervous system
      - (5) Adrenal medulla glands secrete epinephrine and norepinephrine
        - (a) Epinephrine
          - i) Alpha 1
            - a) Vasoconstriction
            - b) Increase in peripheral vascular resistance
            - c) Increased afterload from arteriolar constriction
          - ii) Alpha 2 regulated release of alpha 1
          - iii) Beta 1
            - a) Positive chronotropy
            - b) Positive inotropy
            - c) Positive dromotropy
          - iv) Beta 2
            - a) Bronchodilation

b) Gut smooth muscle dilation
(b) Norepinephrine
i) Primarily alpha 1 and alpha 2
a) Vasoconstriction
b) Increase in peripheral vascular resistance
c) Increased afterload from arteriolar constriction
(6) Arginine vasopressin (AVP)
(a) Also known as antidiuretic hormone (ADH)
(b) Released from anterior pituitary gland
(c) Effects
i) Increases free water absorption in distal tubule and collecting ducts of kidney
ii) Decreases urine output
iii) Splanchnic vascular constriction
(7) Renin-angiotensin system
(a) Renin released from kidney arteriole
(b) Renin and angiotensinogen combine in renal arteriole to produce angiotensin I
(c) Angiotensin I converted to angiotensin II by angiotensin converting enzyme
(d) Effects of angiotensin II
i) Potent vasoconstrictor
ii) Sodium reabsorption decreases urine output
iii) Positive inotrope and chronotrope
(8) Aldosterone
(a) Defends fluid volume
(b) Secreted by cells of adrenal cortex in response to stress
(c) Promotes sodium reabsorption and water retention in kidney
(d) Reduces urine output
(9) Insulin
(a) Secretion is diminished by circulating epinephrine
(b) Impaired effect on peripheral tissue
(c) Contributes to hyperglycemia seen following injury and volume loss
(10) Glucagon
(a) Stimulated to be released by epinephrine
(b) Promotes
i) Liver glycogenolysis
ii) Gluconeogenesis
iii) Amino acid uptake for conversion into glucose
iv) Transfer of fatty acid into mitochondria
(11) ACTH (adrenocorticotropic hormone)-cortisol system
(a) ACTH release stimulates the release of cortisol from the adrenal cortex of kidney
(b) Cortisol increases glucose production by inhibiting enzymes that break down glucose
(12) Growth hormone
(a) Secreted by anterior pituitary gland

(b) Early effects of growth hormone
i) Promotes uptake of glucose and amino acids in muscle
ii) Stimulates protein synthesis
(13) Failure of compensation to preserve perfusion
(14) Preload decreases
(15) Cardiac output decreases
(16) Myocardial blood supply and oxygenation decrease
(a) Myocardial perfusion decreases
(b) Cardiac output decreases further
(c) Coronary artery perfusion decreases
(d) Myocardial ischemia
(17) Capillary and cellular changes
(a) Ischemia
i) Minimal blood flow to capillaries
ii) Cells go from aerobic to anaerobic metabolism
(b) Stagnation
(c) Precapillary sphincter relaxes in response to
a) Lactic acid
b) Vasomotor center failure
c) Increased carbon dioxide
i) Postcapillary sphincters remain constricted
ii) Capillaries engorge with fluid
iii) Anaerobic metabolism continues, increasing lactic acid production
a) Aggregation of red blood cells and formation of microemboli
b) Potent vasodilator
c) Destroys capillary cell membrane
iv) Plasma leaks from capillaries
v) Interstitial fluid increases
a) Distance from capillary to cell increases
b) Oxygen transport decreases secondary to increased capillary-cell distance
vi) Myocardial toxin factor released by ischemic pancreas
(d) Washout
i) Postcapillary sphincter relaxes
ii) Hydrogen, potassium, carbon dioxide, thrombosed - erythrocytes wash out
iii) Metabolic acidosis results
iv) Cardiac output drops further

c. Stages of shock
(1) Compensated or nonprogressive
(a) Characterized by signs and symptoms of early shock
(b) Arterial blood pressure is normal or high
(c) Treatment at this stage will typically result in recovery
(2) Decompensated or progressive
(a) Characterized by signs and symptoms of late shock
(b) Arterial blood pressure is abnormally low

(c) Treatment at this stage will sometimes result in recovery
(3) Irreversible
(a) Characterized by signs and symptoms of late shock
(b) Arterial blood pressure is abnormally low
(c) Even aggressive treatment at this stage does not result in recovery
d. Etiologic classifications
(1) Hypovolemic
(a) Hemorrhage
(b) Plasma loss
(c) Fluid and electrolyte loss
(d) Endocrine
(2) Distributive (vasogenic)
(a) Increased venous capacitance
(b) Low resistance, vasodilation
(3) Cardiogenic
(a) Myocardial insufficiency
(b) Filling or outflow obstruction (obstructive)
(4) Spinal neurogenic shock
(a) Refers to temporary loss of all types of spinal cord function distal to injury
i) Flaccid paralysis distal to injury site
ii) Loss of bladder and bowel control
iii) Priapism
iv) Loss of thermoregulation
(b) Does not always involve permanent primary injury
(5) Spinal shock
(a) Also called spinal vascular shock
(b) Temporary loss of the autonomic function of the cord at the level of injury which controls cardiovascular function
(c) Presentations includes
i) Loss of sympathetic tone
ii) Relative hypotension
a) Systolic pressure 80 - 100 mmHg
iii) Skin is pink, warm and dry
a) Due to cutaneous vasodilation
iv) Relative bradycardia
(d) Occurrence is rare
(e) Shock presentation is usually the result of hidden volume loss
i) Chest injuries
ii) Abdominal injuries
iii) Other violent injuries
(f) Treatment
i) Focus primarily on volume replacement
4. Assessment - hypovolemic shock due to hemorrhage
(1) Early or compensated
(a) Tachycardia
(b) Pale, cool skin

(c) Diaphoresis
(d) Level of consciousness
i) Normal
ii) Anxious or apprehensive
(e) Blood pressure maintained
(f) Narrow pulse pressure
i) Pulse pressure is the difference between the systolic and diastolic pressures, i.e., pulse pressure = systolic - diastolic
ii) Pulse pressure reflects the tone of the arterial system and is more sensitive to changes in perfusion than the systolic or diastolic alone
(g) Positive orthostatic tilt test
(h) Dry mucosa
(i) Complaints of thirst
(j) Weakness
(k) Possible delay of capillary refill

(2) Late or progressive
(a) Extreme tachycardia
(b) Extreme pale, cool skin
(c) Diaphoresis
(d) Significant decrease in level of consciousness
(e) Hypotension
(f) Dry mucosa
(g) Nausea
(h) Cyanosis with white waxy looking skin

a. Differential shock assessment findings
(1) Shock is assumed to be hypovolemic until proven otherwise
(2) Cardiogenic shock
(a) Differentiated from hypovolemic shock by one or more of the following
i) Chief complaint (chest pain, dyspnea, tachycardia)
ii) Heart rate (bradycardia or excessive tachycardia)
iii) Signs of congestive heart failure (jugular vein distention, rales)
iv) Dysrhythmias
(b) Distributive shock
(c) Differentiated from hypovolemic shock by presence of one or more of following
i) Mechanism that suggests vasodilation, e.g., spinal cord injury, drug overdose, sepsis, anaphylaxis
ii) Warm, flushed skin, especially in dependent areas
iii) Lack of tachycardia response (not reliable, though, since significant number of hypovolemic patients never become tachycardic)
(d)) Obstructive shock
i) Differentiated from hypovolemic shock by presence of signs and symptoms suggestive of

ii) Cardiac tamponade
iii) Tension pneumothorax

5. Management/ treatment plan
   a. Airway and ventilatory support
      (1) Ventilate and suction as necessary
      (2) Administer high concentration oxygen
      (3) Reduce increased intrathoracic pressure in tension pneumothorax
   b. Circulatory support
      (1) Hemorrhage control
      (2) Intravenous volume expanders
         (a) Types
            i) Isotonic solutions
            ii) Hypertonic solutions
            iii) Synthetic solutions
            iv) Blood and blood products
            v) Experimental solutions
            vi) Blood substitutes
         (b) Rate of administration
            i) External hemorrhage that can be controlled
            ii) External hemorrhage that can not be controlled
            iii) Internal hemorrhage
               a) Blunt trauma
               b) Penetrating trauma
      (3) Pneumatic anti-shock garment
         (a) Effects
            i) Increased arterial blood pressure above garment
            ii) Increased systemic vascular resistance
            iii) Immobilization of pelvis and possibly lower extremities
            iv) Increased intra-abdominal pressure
         (b) Mechanism
            i) Increases systemic vascular resistance through direct compression of tissues and blood vessels
            ii) Negligible autotransfusion effect
         (c) Indications
            i) Hypoperfusion with unstable pelvis
            ii) Conditions of decreased SVR not corrected by other means
            iii) As approved locally, other conditions characterized by hypoperfusion with hypotension
            iv) Research studies
         (d) Contraindications
            i) Advanced pregnancy (no inflation of abdominal compartment)
            ii) Object impaled in abdomen or evisceration (no inflation of abdominal compartment)
            iii) Ruptured diaphragm
            iv) Cardiogenic shock
            v) Pulmonary edema

(4) Needle chest decompression of tension pneumothorax to improve impaired cardiac output
(5) Recognize the need for expeditious transport of suspected cardiac tamponade for pericardiocentesis

c. Pharmacological interventions
(1) Hypovolemic shock
(a) Volume expanders
(2) Cardiogenic shock
(a) Volume expanders
(b) Positive cardiac inotropes
(c) Vasoconstrictor
(d) Rate altering medications
(3) Distributive shock
(a) Volume expanders
(b) Positive cardiac inotropes
(c) Vasoconstriction
(d) PASG
(4) Obstructive shock
(a) Volume expanders
(5) Spinal shock
(a) Volume expanders

d. Psychological support/communication strategies
e. Transport considerations
(1) Indications for rapid transport
(2) Indications for transport to a trauma center
(3) Considerations for air medical transportation

III. Integration