UNIT TERMINAL OBJECTIVE

4-2 At the completion of this unit, the EMT-Critical Care Technician student will be able to utilize the assessment findings to formulate a field impression and implement the treatment plan for the patient with hemorrhage or shock.

COGNITIVE OBJECTIVES

At the completion of this unit, the EMT-Critical Care Technician 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 various types and degrees of hemorrhage and shock. (C-1)
4-2.3 Discuss the pathophysiology of hemorrhage and shock. (C-1)
4-2.4 Discuss the assessment findings associated with hemorrhage and shock. (C-1)
4-2.5 Identify the need for intervention and transport of the patient with hemorrhage or shock. (C-1)
4-2.6 Discuss the treatment plan and management of hemorrhage and shock. (C-1)
4-2.7 Discuss the management of external and internal hemorrhage. (C-1)
4-2.8 Differentiate between controlled and uncontrolled hemorrhage. (C-3)
4-2.9 Differentiate between the administration rate and amount of IV fluid in a patient with controlled versus uncontrolled hemorrhage. (C-3)
4-2.10 Relate internal hemorrhage to the pathophysiology of compensated and decompensated hypovolemic shock. (C-3)
4-2.11 Relate internal hemorrhage to the assessment findings of compensated and decompensated hypovolemic shock. (C-3)
4-2.12 Describe the body's physiologic response to changes in perfusion. (C-1)
4-2.13 Describe the effects of decreased perfusion at the capillary level. (C-1)
4-2.14 Discuss the cellular ischemic phase related to hemorrhagic shock. (C-1)
4-2.15 Discuss the capillary stagnation phase related to hypovolemic shock. (C-1)
4-2.16 Discuss the capillary washout phase related to hypovolemic shock. (C-1)
4-2.17 Discuss the assessment findings of hypovolemic shock. (C-1)
4-2.18 Relate pulse pressure changes to perfusion status. (C-3)
4-2.19 Define compensated and decompensated shock. (C-1)
4-2.20 Discuss the pathophysiological changes associated with compensated shock. (C-1)
4-2.21 Discuss the assessment findings associated with compensated shock. (C-1)
4-2.22 Identify the need for intervention and transport of the patient with compensated shock. (C-1)
4-2.23 Discuss the treatment plan and management of compensated shock. (C-1)
4-2.24 Discuss the pathophysiological changes associated with decompensated shock. (C-1)
4-2.25 Discuss the assessment findings associated with decompensated shock. (C-1)
4-2.26 Identify the need for intervention and transport of the patient with decompensated shock. (C-1)
4-2.27 Discuss the treatment plan and management of the patient with decompensated shock. (C-1)
4-2.28 Differentiate between compensated and decompensated shock. (C-3)
4-2.29 Relate external hemorrhage to the pathophysiology of compensated and decompensated hypovolemic shock. (C-3)
4-2.30 Relate external hemorrhage to the assessment findings of compensated and decompensated hypovolemic shock. (C-3)
4-2.31 Differentiate between the normotensive, hypotensive, and profoundly hypotensive patient. (C-3)
4-2.32 Differentiate between the administration of fluid in the normotensive, hypotensive, and profoundly hypotensive patient. (C-3)
4-2.33 Discuss the physiologic changes associated with the pneumatic anti-shock garment (MAST (PASG)). (C-

1)

4-2.34 Discuss the indications and contraindications for the application and inflation of the MAST (PASG). (C-1)
4-2.35 Apply epidemiology to develop prevention strategies for hemorrhage and shock. (C-1)
4-2.36 Integrate the pathophysiological principles to the assessment of a patient with hemorrhage or shock. (C-3)
4-2.37 Synthesize assessment findings and patient history information to form a field impression for the patient with hemorrhage or shock. (C-3)
4-2.38 Develop, execute, and evaluate a treatment plan based on the field impression for the hemorrhage or shock patient. (C-3)
4-2.39 Differentiate between the management of compensated and decompensated shock. (C-3)

AFFECTIVE OBJECTIVES
None identified for this unit.

PSYCHOMOTOR OBJECTIVES
At the completion of this unit, the EMT-Critical Care Technician student will be able to:

4-2.40 Demonstrate the assessment of a patient with signs and symptoms of hypovolemic shock. (P-2)
4-2.41 Demonstrate the management of a patient with signs and symptoms of hypovolemic shock. (P-2)
4-2.42 Demonstrate the assessment of a patient with signs and symptoms of compensated hypovolemic shock. (P-2)
4-2.43 Demonstrate the management of a patient with signs and symptoms of compensated hypovolemic shock. (P-2)
4-2.44 Demonstrate the assessment of a patient with signs and symptoms of decompensated hypovolemic shock. (P-2)
4-2.45 Demonstrate the management of a patient with signs and symptoms of decompensated hypovolemic shock. (P-2)
4-2.46 Demonstrate the assessment of a patient with signs and symptoms of external hemorrhage. (P-2)
4-2.47 Demonstrate the management of a patient with signs and symptoms of external hemorrhage. (P-2)
4-2.48 Demonstrate the assessment of a patient with signs and symptoms of internal hemorrhage. (P-2)
4-2.49 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. Morbidity/ mortality
         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) MAST (PASG)
(b) Apply sterile dressing and pressure bandage
(3) Transport considerations
(4) Psychological support/ communication strategies

II. Pathophysiology, assessment, and management of shock

A. Shock
  1. Epidemiology
    a. Morbidity/ mortality
    b. Prevention strategies
  2. 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 mmHg 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
        (d) Decrease in systolic pressure 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) Failure of compensation to preserve perfusion
(7) Preload decreases
(8) Cardiac output decreases
(9) Myocardial blood supply and oxygenation decrease
(a) Myocardial perfusion decreases
(b) Cardiac output decreases further
(c) Coronary artery perfusion decreases
(d) Myocardial ischemia

(10) 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
(b) Flaccid paralysis distal to injury site
(c) Loss of autonomic function
i) Hypotension
ii) Vasodilatation
iii) Loss of bladder and bowel control
iv) Priapism
v) Loss of thermoregulation
(d) Does not always involve permanent primary injury
(5) Spinal shock
(a) Also called spinal vascular shock
(b) Temporary loss of autonomic function of cord at level of injury which controls cardiovascular function
(c) Presentation includes
i) Loss of sympathetic tone
ii) Relative hypotension
a) Systolic pressure 80 - 100 mmHg
iii) Skin 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 injury
ii) Abdominal injury
iii) Other violent injury
(f) Treatment
i) Focus primarily on volume replacement

e. Assessment
(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

f. Differential shock assessment findings
(1) Shock is assumed to be hypovolemic until proven otherwise
(2) Cardiogenic shock
(a) Differentiated from hypovolemic shock by presence of 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) Dysrhythmia
(3) Distributive shock
(a) Differentiated from hypovolemic shock by presence of one or more of the 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)
(4) Obstructive shock
(a) Differentiated from hypovolemic shock by presence of signs and

symptoms suggestive of
i) Cardiac tamponade
ii) Tension pneumothorax

3. Management
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
(b) Rate of administration
i) External hemorrhage that can be controlled
ii) External hemorrhage that cannot be controlled
iii) Internal hemorrhage
a) Blunt trauma
b) Penetrating trauma
(3) Pneumatic anti-shock garment (MAST (PASG))
(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 **(See SEMAC Advisory on M.A.S.T.)**
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) Rate altering medications
  (3) Distributive shock
    (a) Volume expanders
    (b) Positive cardiac inotropes
    (c) MAST (PASG)
  (4) Obstructive shock
    (a) Volume expanders
  (5) Spinal shock
    (a) Volume expanders

d. Non-pharmacological interventions

e. Transport considerations

Note:

This advisory guideline announces important changes in the *Statewide Basic Life Support Adult and Pediatric Treatment Protocols.* Revised copies of each of the protocols affected by these changes are attached. Revised copies of each of the protocols affected by these changes are also being sent to all emergency medical services agencies statewide. Regional Emergency Medical Advisory Committees, and regional, system, and service medical directors are directed to facilitate use of the revised protocols at the local level, and are further advised to modify local protocols, policies, and procedures accordingly.

  (1) Indications for rapid transport
  (2) Indications for transport to a trauma center
  (3) Considerations for air medical transportation

f. Psychological support/ communication strategies

III. SEMAC Advisory 97-04: Medical Anti-Shock Trousers

Medical Anti-Shock Trousers

Current *Statewide Basic Life Support Adult and Pediatric Treatment Protocols* stipulate that Medical Anti-Shock Trousers (MAST), also known as the Pneumatic Anti-Shock Garment (PASG), should be inflated if the systolic blood pressure is below 90 mm Hg in adults or below 70 mm Hg in children and signs of inadequate perfusion are present, if MAST (PASG) are available. The State Emergency Medical Advisory Committee has reviewed these protocols, and concludes, on the basis of recent scientific evidence, that prehospital MAST (PASG) use in New York State should be considered only in adult major blunt trauma with severe hypotension (systolic blood pressure < 50 mm Hg) and hypotension (systolic blood pressure < 90 mm Hg) associated with unstable pelvic fracture.

In 1989, Mattox et al, in a prospective randomized study of 911 adult trauma patients, mostly with penetrating injuries, found that MAST (PASG) use was associated with longer scene times, and worsened the survival of adult patients with systolic hypotension (BP < 90 mm Hg) as well as those with primary thoracic injuries who presented in traumatic cardiac arrest. In 1992, Cooper et al, in a retrospective study of the efficacy of MAST (PASG) use in 436 pediatric trauma patients, mostly with blunt injuries, from the National Pediatric Trauma Registry who presented in hypotensive shock, found similar results. In 1993, Cayten et al reported the results of a retrospective study of MAST (PASG) use in 629 hypotensive adult trauma patients which concurred with Mattox's findings, although they were able to demonstrate a small but statistically significant survival advantage in severe hypotension (BP < 50 mm Hg). While there have been no prospective studies and no published trauma registry data in support of MAST (PASG) use for hypotension associated with unstable pelvic fractures, retrospective reviews and cases reports consistently support MAST (PASG) use in such circumstances.

In 1997, O'Connor et al performed a collective review of the scientific literature as an evaluation of MAST (PASG) in various clinical settings. On the basis of this review, Domeier et al developed a position paper on use of MAST (PASG) for the National Association of EMS Physicians, the Summary Recommendations from which, as they pertain to trauma, are summarized below.

MAST (PASG) are "usually indicated, useful, and effective" (Class I evidence) for:

None.

MAST (PASG) are "acceptable, of uncertain efficacy, [although the] weight of evidence favors usefulness and efficacy" (Class IIa evidence) for:

"Hypotension due to suspected pelvic fracture;
Severe traumatic hypotension (palpable pulse, blood pressure not obtainable). *"

MAST (PASG) are "acceptable, of uncertain efficacy, may be helpful, probably not harmful" (Class IIb evidence) for:

"Penetrating abdominal injury;
Lower extremity hemorrhage (otherwise uncontrolled); *
Pelvic fracture without hypotension; *
Spinal shock. *"

MAST (PASG) are "inappropriate, not indicated, may be harmful" (Class III evidence) for:

"Adjunct to CPR;
Diaphragmatic rupture;
Penetrating thoracic injury;
Pulmonary edema;
To splint fractures of the lower extremities;
Extremity trauma;
Abdominal evisceration;
Acute myocardial infarction;
Cardiac tamponade;
Cardiogenic shock;
Gravid uterus."

* Data from controlled trials not available. Recommendation based on other evidence.

The literature cited supports the conclusion that the role of MAST (PASG) in the prehospital emergency medical care of adult and pediatric patients is extremely limited. The State Emergency Medical Advisory committee agrees with the National Association of EMS Physicians that the weight of the evidence favors the usefulness and efficacy of MAST (PASG) only for adult major blunt trauma with severe hypotension (systolic blood pressure < 50 mm Hg) and hypotension (systolic blood pressure < 90 mm Hg) associated with unstable pelvic fracture, a position which is consistent with the 1997 Edition of the Advanced Trauma Life Support Course of the American College of Surgeons.

The State Emergency Medical Advisory Committee (SEMAC) therefore recommends their use under these circumstances, although Regional Emergency Medical Advisory Committees (REMAC) may prescribe their use under other circumstances to address specific local conditions. The *Statewide Basic Life Support Adult and Pediatric Treatment Protocols* are being modified to reflect this change, and Regional Emergency Medical Advisory Committees, and regional, system, and service medical directors are advised to modify local protocols, policies, and procedures accordingly.

<u>Selected References</u>

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