EMS Education Standards – Transition Curriculum

Transition Education Guidelines from the old DOT National Standard Curriculum to 2009 EMS Education Standards

EMS Governor’s Advisory Council Training Committee
6/1/2010
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

These materials were developed by the EMS Training Committee, approved by the EMS Governor’s Advisory Council and endorsed by the Arkansas Department of Health, Section of EMS.

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The Committee is pleased to present the following materials for Arkansas Educators to use in updating currently licensed Emergency Medical Service Providers (EMSPs) to the materials in the 2009 EMS Education Standards. They are also thankful to the National Association of State EMS Officials for developing the 2009 National EMS Education Standards Gap Analysis Template which was used in the development of this document. Arkansas Educators are encouraged to access the Gap Analysis Template, the National Scope of Practice Model and the 2009 Education Standards and Instructor Guidelines to enhance the presentation of these materials. A Quick Guide to Resources is provided in the Index for that purpose.

This document is dedicated in memory of David A. Taylor, Sr. Chief, Section of EMS and Trauma Systems who completed the explanation of the CDC Field Triage Decision Scheme: National Trauma Triage Protocol and its use within the Arkansas Trauma System for this document.
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**Items to cover for Basic Transitional Course**

For a Emergency Medical Services Provider educated as an EMT-Basic (based on the 1994 EMT-Basic National Standard Curriculum) and transitioning to the education expectations of the 2009 Emergency Medical Technician (EMT) Education Standards, the following pages present the information needed to educate to the new level.

**Topic – Airway Management, Respiration and Oxygenation**

What’s New?
Anatomy and Physiology – Much more detailed than in the previous 1994 EMT-B National Standard Curriculum.
Respiration- Much more detailed than in the previous 1994 EMT-B National Standard Curriculum.
Artificial Ventilation- Much more detailed than in the previous 1994 EMT-B National Standard Curriculum.

Addition of (this was previously in the curriculum but removed in 1994):
Partial Rebreather Mask, Simple Face Mask, Venturi Mask, Pulse Oximetry, Ventilator AVT), use of oxygen humidifiers.

**Objectives:**
Review Airway Management anatomy and physiology terminology
Review Respiration and the mechanics of ventilation with more emphasis on anatomy and physiology.
Identify Artificial Ventilation devices and know how to use them.

**Declarative:**
(1 Hour)

Define-
- Apnea – absence of breathing – respiratory arrest
- Bronchoconstriction – constriction of the smooth muscle of the bronchi and bronchioles.
- Bronchodilator – a drug that relaxes the smooth muscle of the bronchi and bronchioles and reverses bronchoconstriction
- Hypercarbia – increased carbon dioxide levels in the blood. Also called hypercapnia
- Respiratory Distress – Increased respiratory effort resulting from impaired respiratory function.
- Respiratory Failure – inadequate respiratory rate and /or tidal volume.
- Hypoxemia – decreased oxygen levels in the blood
- Dyspnea – shortness of breath or perceived difficulty in breathing.
- Hypoxia – the absence of sufficient oxygen in the body cells.
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**Chronic Bronchitis** – a disease process that affects primarily the bronchi and bronchioles usually associated with cigarette smoking. Characterized by a productive cough for at least three consecutive months out of the year for at least two consecutive years.

Assessment findings for Chronic Bronchitis
- Typically are overweight
- Chronic cyanotic complexion
- Difficulty in breathing
- Vigorous productive cough
- Coarse rhonchi
- Wheezes and possibly crackles at the bases

**Treatment**: Assist the patient with his meter dosed inhaler if he has one and has not yet used it. Oxygen as needed to keep patient alert. A non-rebreather at 15 liters a minute may be in order or a simple nasal cannula at 2-3 liters. Some protocols allow for use of CPAP in severe cases. Place these patients on a pulse ox and monitor it during care.

**Emphysema** – A permanent disease process that is the destruction of the alveolar walls and distention of the alveolar sacs. More common in Men than women and usually associated with cigarette smoking.

Assessment findings for Emphysema Patients:
- Thin, barrel-chest appearance
- Coughing with little sputum
- Prolonged exhalation
- Diminished breath sounds
- Wheezing and rhonchi on auscultation
- Pursed-lip Breathing
- Extreme difficulty of breathing on minimal exertion
- Pink complexion – “pink puffers”
- Tachypnea – breathing faster than 20 per minute
- Tachycardia – heart rate faster than 100 per minute
- Diaphoresis
- Tripod position
- May be on home oxygen

**Treatment** is similar to Chronic Bronchitis above.

**Asthma** – an increased sensitivity of the lower airways to irritants and allergens, causing bronchospasms. This results in narrowing of the bronchi and swelling of the airway or edema in the lining of the bronchiole.

Assessment findings for Asthma
- Moderate Distress:
- Dyspnea
- Non-productive cough
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- Wheezing on auscultation
- Tachypnea
- Tachycardia
- Anxiety and apprehension
- Possible fever
- Typical allergic signs and symptoms
- Chest tightness
- Inability to sleep
- SpO₂ < 95% before oxygen administration

Severe Distress:
- Extreme fatigue
- Inability to speak
- Cyanosis to the core of the body
- Heart rate > 150 beats per minute or a slow rate
- Quiet or absent breath sound on auscultation of the lungs – silent chest
- Tachypnea (respiratory rate >32 breaths per minute)
- Excessive diaphoresis
- Accessory muscle use (neck, chest, abdomen)
- Confusion
- SpO₂ < 90% with patient on oxygen.

**Treatment of Asthma** - immediately place the patient on oxygen. If severe, assist with ventilations using a BVM. Calm the patient. Use the patient’s inhaler or small volume nebulizer or updraft to administer a beta agonist.

**Pneumonia** – an acute infectious disease caused by bacterium or a virus that affects the lower respiratory tract and causes lung inflammation and fluid or pus-filled alveoli.

Assessment of the pneumonia patient:
- Malaise and decreased appetite
- Fever
- Cough
- Dyspnea
- Tachypnea and tachycardia
- Chest pain on inspiration or coughing
- Decreased chest wall movement and shallow respirations
- Splinting of thorax with arm
- Crackles, localized wheezing, and rhonchi heard on auscultation
- Altered mental status, especially in the elderly
- Diaphoresis
- Cyanosis
- SpO₂ < 95%

**Treatment for Pneumonia** - place on humidified oxygen, consider use of meter dosed inhaler if available or updraft.
Acute Pulmonary Embolism – a sudden blockage of blood flow through a pulmonary artery or one of its branches. The embolism prevents blood from flowing to the lungs.

Assessment of pulmonary embolism patient:
• Sudden onset of unexplained dyspnea
• Signs of difficulty in breathing or respiratory distress
• Sudden onset of sharp, stabbing chest pain
• Cough
• Tachypnea
• Syncope
• Cool, moist skin
• Restlessness anxiety or sense of doom
• Decrease in blood pressure
• Cyanosis
• Distended neck veins
• Crackles
• Fever
• $\text{SPO}_2 < 95$
• Signs of complete circulatory collapse

Treatment: NRB oxygen and be prepared to use BVM if patient’s condition warrants. Immediately transport to appropriate facility.

Acute pulmonary edema – an excessive amount of fluid collects in the spaces between the alveoli and the capillaries disturbing normal gas exchange causing hypoxia.

Assessment of the pulmonary edema patient:
• Dyspnea
• Difficulty in breathing when lying flat (orthopnea)
• Frothy sputum
• Tachycardia, anxiety, apprehensions, confusion
• Tripod position with legs dangling
• Fatigue
• Crackles and possibly wheezing
• Cyanosis
• Pale, moist skin
• Distended neck veins
• Swollen lower extremities
• Cough
• Symptoms of cardiac compromise
• $\text{SPO}_2 < 95$

Treatment: Positive pressure ventilations. Pulse Ox. Use CPAP if available. 100% oxygen.
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**Spontaneous Pneumothorax** – a portion of the visceral pleural lining around the lungs ruptures. This occurs without trauma, hence- spontaneous. This allows air to enter into the pleural cavity disrupting the normally negative pressure and causing the lung to collapse. Known as a ruptured bleb.

Assessment of the spontaneous pneumothorax patient.
- Sudden onset of shortness of breath
- Decreased breath sounds to one side of the chest
- Subcutaneous emphysema
- Tachypnea
- Tachycardia
- Diaphoresis
- Pallor
- Cyanosis
- SPO₂ <95%

**Treatment:** BVM ventilations with minimal tidal volume needed to inflate lungs. Pulse Ox. Use 100% oxygen. Contact ALS unit.

**Epiglotitis** – the epiglottis at the base of the tongue covers the vocal chords when swallowing to prevent aspiration become inflamed along with surrounding structures making it difficult to swallow.

Assessment of the epiglotitis patient
- Dyspnea
- High fever
- Sore throat
- Inability to swallow with drooling
- Anxiety and apprehension
- Tripod position, with jaw jutted forward
- High-pitched inspiratory stridor
- Cyanosis
- Trouble speaking
- SPO₂ <95%

**Treatment:** Do not inspect the airway and mouth as it might cause additional swelling. Place patient on NRB 100% oxygen. Pulse Ox. Reassure patient. Call for ALS back up.

**Pertussis** – commonly known as the whooping cough starts out similar to a cold but progresses within 2 weeks or so to rapid coughing about 15 to 24 episodes in close sequence. The body attempts to expel thick mucus from the airway following a crowing or whooping sound made during inhalation as the patient breaths deeply.

Assessment of the pertussis patient:
- History of upper respiratory infection
- Sneezing, runny nose, low-grade fever
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- General malaise
- Increase in frequency and severity of coughing
- Coughing fits, usually more common at night
- Vomiting
- Inspiratory “whoop” heard at the end of coughing burst
- Possible development of cyanosis during coughing burst
- Lowering pulse ox readings
- Exhaustion from expending energy during coughing bursts
- Trouble speaking and breathing during burst

**Treatment:** BSI. NRB Oxygen Mask with humidified oxygen. Pulse Ox. Keep patient calm and expedite transport to hospital. Consider ALS intercept.

**Cystic Fibrosis** - disease that causes an overabundant production of mucus in the respiratory tree causing excessive mucus to collect. This collection of mucus must be continuously expelled. Repeated lung infections occur causing scarring of the lung tissue and permanent damage to the lung. This all leads to pulmonary failure and death.

Assessment of cystic fibrosis patient:
- Known history of this disease
- Recurrent coughing
- General malaise
- Expectorant of thick mucus during coughing
- Recurrent episodes or history of pneumonia, bronchitis, an sinusitis
- Gastrointestinal complaints that may include diarrhea, and greasy and/or foul smelling bowel movements.
- Abdominal pain from intestinal gas
- Malnutrition or low weight despite a healthy appetite
- Dehydration
- Clubbing of digits
- Trouble speaking and breathing
- Signs of pneumonia

**Treatment:** Relieve respiratory distress by administering oxygen, humidified at 100% NRB or BVM if necessary. In severe cases call for ALS.

**Meter Dose Inhaler or Small Volume Nebulizer**

**Actions:**

**Side Effects:**

**Indications:**
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Contraindication;

Dosage:

**Steps in using a SVN – Small volume Nebulizer**

1) Ensure right patient, right medication, right dose, right route and right date. Determine if the patient is alert enough to use the nebulizer and if any doses have already been administered prior to your arrival.

2) Obtain an order, either on-line or off-line, from medical direction for administration of the medication.

3) Disassemble the medication chamber from the mouthpiece by unscrewing it. While holding the medication reservoir upright, pour in the medication and reassemble the device.

4) Attach the tubing extending from the bottom of the drug reservoir to the nebulizer compressor and turn it on, or attach the tubing to an oxygen tank with the liter flow set to 8 – 10 LPM. You should note the mist coming from the mouthpiece almost immediately.

5) Remove the non-rebreather mask from the patient, instruct the patient to take the nebulizer in his hand and hold it upright. If the patient is unable to hold the device, you may have to do this for the patient, being sure to continuously hold it upright for optimal nebulization of the medication.

6) Have the patient exhale fully.

7) Instruct the patient to place his lips around the mouthpiece of the nebulizer. Another technique is to have the patient open his mouth and place the mouthpiece 1 – 1.5 inches from the front of the lips, estimated by two finger widths.

8) Have the patient begin to slowly and deeply breathe in the mist.

9) Instruct the patient to occasionally (every 2 -3 breaths) hold his breath after inhalation as long as he comfortably can, to assist with medication distribution throughout the respiratory tree.

10) Have the patient exhale normally, and occasionally (every 2 -3 breaths) instruct the patient to cough during exhalation to facilitate removal of any mucus or secretions that may be present.

11) You may need to occasionally shake the nebulizer to dislodge any medication that tends to collect on the sides of the drug reservoir. In about 5 -10 minutes, the misting of medication should cease and the liquid medication you placed in the nebulizer will be gone. Replace the oxygen mask on the patient.

12) Reassess the patient and consult with medical direction if additional doses are needed. If an additional dose is recommended, wait at least 2 minutes between each administration or longer based on the medication being administered or medical direction’s order.

Add:

**Simple face mask**

A simple mask is used to deliver moderate to high concentrations of oxygen. It can deliver from 40% to 60% oxygen at a flow rate of 2.64-3.17 gal (10-12 L) per minute.
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**Venturi Mask**
The venturi mask, also known as an air-entrainment mask, is a medical device to deliver a known oxygen concentration to patients on controlled oxygen therapy. Venturi masks are considered high-flow oxygen therapy devices. This is because venturi masks are able to provide total inspiratory flow at a specified FIO2 to patients therapy.

**Partial Rebreather Mask**
A partial rebreather mask is used to deliver high concentrations of oxygen. It can deliver 70% to 90% oxygen at a flow of 1.58-3.96 gal (6-15 L) per minute.

**Monitoring – Pulse Oximetry;**

*Purpose* for pulse oximetry is to assess oxygenation, assess adequacy of oxygen delivery during positive pressure ventilation, and assess impact of interventions.

The pulse oximetry works by measuring the oxygen saturation on the hemoglobin.

**Technique:**
1) Place the pulse oximetry probe on the finger where the light from the oximeter shines through arterial blood flow.
2) Turn the device on and wait a few seconds for the device to detect the pulse and the reading to appear.
3) If a poor signal is detected some devices may have an error reading or dashed lines. If this happens check the patient for nail polish, or cool extremities.
4) Once a proper reading has occurred, record the reading every 5 minutes in seriously ill patients, every 15 minutes in stable patients.

**How it works:**
The red light and infrared light shines through the tissue and into the blood to a photo sensor on the opposite side of the devise.
The sensor detects the amount of hemoglobin in the blood that is saturated with oxygen and the amount of hemoglobin that is not saturated with oxygen.

The recording of oxygen saturation is recorded as %SpO₂. Normal readings are typically in the upper 90’s. SpO₂ lags behind actual blood concentrations by about one and a half minutes.

**Indications for use of Pulse Oximetry**- This device should be used any time there is concern that oxygen in the blood stream may be affected. Pulse ox is sometimes referred to as the sixth vital sign.

**Limitations** – A good pulse in the extremity being monitored is necessary for the pulse ox to work properly. Any condition that causes poor blood flow to the finger will affect the accuracy of the pulse oximetry device. Cold extremities, shock, low blood pressure, and anemia are a few.

Another concern is that the pulse oximetry measures saturation of the hemoglobin, if the hemoglobin is saturated with something else like carbon monoxide it will give you a false reading for oxygen saturation.
Ventilator (ATV)

AUTOMATIC TRANSPORT VENTILATOR

I. INTRODUCTION
Use of an Automatic Transport Ventilator requires Medical Control, is at the sole discretion of the base hospital medical director, and must be appropriately documented when used. The medic must be trained in use of specific provider ventilator to be used for transport.

II. INDICATIONS
A. Any patient requiring ventilatory assistance in conjunction with advanced airway adjuncts.
B. Any patient requiring ventilatory assistance in conjunction with basic airway adjuncts.
C. Any patient requiring ventilatory assistance in conjunction with manual airway maintenance.

III. CONTRAINDICATIONS
A. Patients weighing less than 16 Kg. (35 lbs.)
B. Pneumothorax - tension pneumothorax
C. Pulmonary over pressurization syndrome (blast injury, water ascent injury, etc.)

IV. PROCEDURE
A. Determine that a need for the use of the ATV exists.
B. Assure that all tubing is free from kinks.
C. Determine the proper tidal volume setting. This is done by determining the patient ideal weight (approx. weight for any physically fit patient having the same sex, height, frame) and multiplying it by 8-10 ml/kg. Begin with the lowest tidal volume limit.
D. Set Breaths per Minute (BPM) control to rate of 8-15 per minute.
E. Check alarm by occluding the patient valve assembly outlet. The audible pressure limit alarm should sound as the ventilator cycles through the delivery phase.
F. Assess lung compliance and chest rise with a bag valve device. Tidal volume may be adjusted lower if poor lung compliance is found.
G. Attach the patient valve assembly to the airway device or mask used on the patient.
H. Assess the ventilation. Listen for bilateral lung sounds. Observe for proper chest rise . . . this should look normal and be symmetrical.
I. Count the number of complete ventilator cycles for a full minute. The number should be the same as the setting (+/-1).
J. Assess and manage the airway as you normally would for any patient with controlled ventilation.
K. If spontaneous breathing begins, it may be desirable to turn the BPM down as long as patient's spontaneous rate is 10-12 per minute. L. Check oxygen cylinder pressure level frequently. This device will deplete a "D" cylinder rapidly.

V. SPECIAL CONSIDERATIONS
A. Due to COPD, chest rise may not appear full . . . do not increase tidal volume (TV) past upper TV limit.
B. If lung sounds are absent or on one side only: rule out airway obstruction, improper tube placement, or pneumothorax, and check tidal volume ml/bpm settings.
C. If chest expansion is not adequate, the rescuer should slowly increase tidal volume until chest expansion is adequate, or the uppermost limit (for the patient's ideal weight) is reached.
D. If chest appears to over expand, decrease tidal volume.
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**Topic: Cardiovascular/Circulation**

All Levels: EMT, AEMT, & Paramedic

**Notable Content Removed:** Pressure Point for hemorrhage control has been removed and tourniquet application has moved up the decision tree.

Hemorrhage - Tourniquet (15 minutes didactic; 15 minutes laboratory all levels) Comment: no problem

Cognitive Objectives:

- Discuss the need for assessing the patient for external bleeding.
- Differentiate between arterial, venous and capillary bleeding.
- State methods of emergency medical care of external bleeding.

Psychomotor Objectives:

- Demonstrate the techniques for assessing the patient for external bleeding.
- Demonstrate direct pressure as a method of emergency medical care of external bleeding.
- Demonstrate the use of diffuse pressure as a method of emergency medical care for external bleeding.
- Demonstrate the use of tourniquets as a method of emergency medical care of external bleeding.
- Demonstrate the care of the patient exhibiting signs and symptoms of shock (hypoperfusion).
- Declarative (EMS Educational Standards)

Section: Trauma

Subsection: **Bleeding**

I. Pathophysiology
   A. Type of Traumatic Bleeding
      1. External
      2. Arterial
      3. Venous
      4. Capillary
   B. Severity
      1. Volume of blood loss
      2. Rate of blood loss
      3. Age and preexisting health of patient
   C. Physiological response of bleeding
      1. Clotting and clotting disorders
      2. Factors that affect clotting
         a. Movement of injured area
         b. Body temperature
         c. Medications
         d. Removal of bandages
      3. Localized vasoconstriction

II. General Assessment
   A. Mechanism of injury
III. Management Strategies

A. Body Substance Isolation
B. Airway Patency – May be obstructed if unconscious
C. Oxygenation and Ventilation
   1. Pulse oximetry
   2. Apply oxygen
D. Internal and External Bleeding Control
   1. External bleeding
      a. Direct pressure: application of even pressure to an open injury that includes the area just proximal and distal to the injury using a gloved hand and dressings, the wound is covered and firm pressure applied until bleeding is controlled usually effective in capillary and minor venous bleeding in cases of heavier bleeding or major wounds.

      Multiple dressings may be necessary; do not remove existing dressings but apply additional dressings on top of existing dressings in cases of continuing hemorrhage.

      b. Splints
         i. soft
         ii. rigid
         iii. traction splint
         iv. pressure splints
      c. Tourniquet – if severe bleeding is not controlled by direct pressure
         Use as a last resort to control bleeding of an amputated extremity when all other methods of bleeding control have failed.
         Application of a tourniquet can cause permanent damage to nerves, muscles and blood vessels resulting in the loss of an extremity.

Procedures for applying a tourniquet:

1) Use a bandage 4 inches wide and 6 to 8 layers deep.
2) Wrap it around the extremity twice at a point proximal to the bleeding but as distal on the extremity as possible.
3) Tie one knot in the bandage and place a stick or rod on top of the knot and tie the ends of the bandage over the stick in a square knot.
4) Twist the stick until the bleeding stops.
5) Once the bleeding has stopped, secure the stick or rod in position.
6) Notify other emergency personnel who may care for the patient that a tourniquet has been applied.
7) Document the use of a tourniquet and the time applied in the prehospital patient report.

A continuously inflated blood pressure cuff may be used as a tourniquet until bleeding stops. Commercially available tourniquets are available. Follow directions of manufacturer.

Precautions with the use of a tourniquet:

1) Use a wide bandage and secure tightly.
2) Never use wire, rope, a belt, or any other material that may cut into the skin and underlying tissue.
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3) Do not remove or loosen the tourniquet once it is applied unless directed to do so by medical direction.
4) Leave the tourniquet in open view.
5) Do not apply a tourniquet directly over any joint, but as close to the injury as possible.

IV. Orthopedic Trauma
A. General Management
   Control Hemorrhage
   - Internal
   - External
   - Direct pressure
   - Tourniquet (see III., D., 1., c.)
   - Traction splint with fracture

B. Specific Injuries
   - Amputation
     a. Control bleeding of stump
     b. Direct pressure
     c. Tourniquet (see III., D., 1., c.)

   - Soft Tissue Trauma (pp 143)
     General Assessment
     (1) Safety of Environment / Standard Precautions
     (2) Airway Patency
     (3) Respiratory Distress
     (4) Concepts of Open Wound Dressings/Bandaging
       a. Sterile
       b. Non-sterile
       c. Occlusive
       d. Non-occlusive
       e. Wet
       f. Dry
     (5) Tourniquet

   - Complications of dressings/bandages
     (1) Hemorrhage Control
     (2) Pressure dressing
     (3) Tourniquets

   - Associated Injuries
     (1) Airway
     (2) Face
     (3) Neck trauma – increased bleeding

VI. Multi-System Trauma
   - Oxygenation cannot occur when patients are bleeding profusely
   - Stop arterial bleeding rapidly
   - Consider use of tourniquets if severe extremity bleeding cannot be controlled with direct pressure
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Devices to Support Circulation

Active Compression-Decompression CPR
Active compression-decompression CPR (ACD-CPR) is performed with a hand-held device equipped with a suction cup to actively lift the anterior chest during decompression.

ACD-CPR may be considered for use in the in-hospital setting when providers are adequately trained (Class IIb). There is insufficient evidence to recommend for or against the use of ACD-CPR in the prehospital setting (Class Indeterminate).

Impedance Threshold Device
The impedance threshold device (ITD) is a valve that limits air entry into the lungs during chest recoil between chest compressions. It is designed to reduce intrathoracic pressure and enhance venous return to the heart. Recent studies indicate that ITD can be used with an endotracheal tube or with a good face mask seal. Although increased long-term survival rates have not been documented, when the ITD is used by trained personnel as an adjunct to CPR in intubated adult cardiac arrest patients, it can improve hemodynamic parameters and ROSC (Class IIa).

Mechanical Piston Device
The mechanical piston device depresses the sternum via a compressed gas-powered plunger mounted on a backboard. Mechanical piston CPR may be considered for patients in cardiac arrest in circumstances that make manual resuscitation difficult (Class IIb). The device should be programmed to deliver standard CPR based on the 2005 AHA guidelines.

Load-Distributing Band CPR or Vest CPR
The load-distributing band (LDB) is a circumferential chest compression device composed of a pneumatically or electrically actuated constricting band and backboard. Evidence from a case control study of 162 adults (LOE 4)51 documented improvement in survival to the emergency department when LDB-CPR was administered by adequately trained rescue personnel to patients with cardiac arrest in the out-of-hospital setting. LDB-CPR may be considered for use by properly trained personnel as an adjunct to CPR for patients with cardiac arrest in the out-of-hospital or in-hospital setting (Class IIb).

Phased Thoracic-Abdominal Compression-Decompression CPR With a Hand-Held Device

Phased thoracic-abdominal compression-decompression CPR (PTACD-CPR) combines the concepts of IAC-CPR and ACD-CPR. A hand-held device alternates chest compression and abdominal decompression with chest decompression and abdominal compression. There is insufficient evidence to support the use of PTACD-CPR outside the research setting (Class Indeterminate).

References:
National EMS Educational standards
Currents. 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care; volume 112, issue 24 Supplement; December 13, 2005
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

**Topic: Patient Restraint**  (no didactic or laboratory hours assigned)

**Physical restraint**
- Review as necessary
- Removed: using spine boards to “sandwich patients”
- Note: Dangers of positional asphyxiation

**Cognitive Objectives:**
- Define consent and discuss the methods of obtaining consent.
- Differentiate between expressed and implied consent.
- Discuss the implications for the EMT-Basic in patient refusal of transport.
- Discuss the issues of abandonment, negligence, and battery and their implications to the EMT-Basic.
- State the conditions necessary for the EMT-Basic to have a duty to act.
- State the conditions that require an EMT-Basic to notify local law enforcement officials.

**Psychomotor Objectives:**
Demonstrate various techniques to safely restrain a patient with a behavioral problem

**Declarative (EMT)**

**EMT Guidelines:**

**I. Medical Restraint**

A. Use of Force Doctrine
   B. Reasonable Prevention of Harm
      1. Suicidal
      2. Homicidal
      3. Ambulances
      4. Ramps
      5. Winches

C. Medical/Legal & Ethics
   1. Medical restraint -- use of force doctrine
      a. reasonable prevention of harm
         i. suicidal
         ii. homicidal
      b. non-punitive
Psychiatric

II. Agitated Delirium

A. Emergency medical care
   1. Scene size-up, personal safety
   2. Establish rapport
      a. utilize therapeutic interviewing techniques
         1. engage in active listening
         2. supportive and empathetic
         3. limit interruptions
         4. respect patient’s territory, limit physical touch
      b. avoid threatening actions, statements and questions
      c. approach slowly and purposefully
   3. Patient assessment
      a. intellectual functioning
      b. orientation
      c. memory
      d. concentration
      e. judgment
      f. thought content
         1) disordered thoughts
         2) delusions, hallucinations
         3) unusual worries, fears
      g. language
         1) speech pattern and content
         2) garbled or unintelligible
      h. mood
         1) anxiety, depression, elation, agitation
         2) level of alertness, distractibility
            a) appearance, hygiene, dress
            b) psychomotor activity
   4. Calm the patient – do not leave the patient alone, unless unsafe situation; consider need for law enforcement
   5. Restrain if necessary
   6. Transport
   7. If overdose, bring medications or drugs found to medical facility

III. Medical-Legal Considerations

A. Types of Restraints

B. Transporting against Patient Will

Topic: Mechanical CPR devices

(15 minutes didactic; 0 laboratory all levels) Comment: no problem
(Requires additional specialty training)

Cognitive Objective:
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

- Review local EMS mechanical CPR devices.
- Discuss the procedures that must be taken into consideration for standard operations of cardiac arrest.
- Discuss the various mechanical devices that are available and approved by the ILCOR standards.

**Psychomotor Objectives:** Uses assessment information to recognize shock, respiratory failure or arrest, and cardiac arrest based on assessment findings and manages the emergency.

Management (refer to the current American Heart Association guidelines)

Evaluation and appropriate management of cardiac compromise
1. Manual and auto BP
2. Mechanical CPR

**Medication Administration - EMT level - Monte Gagliard**

Medication Administration – EMT Level

Time Frame: 60 minutes

Cognitive Objectives:

- Identify the medications that an EMT can administer/assist patient with medication administration
- Discuss the “five rights” of medication administration
- Explain the indications and contraindications for administration of aspirin, nitroglycerine, epinephrine auto injector, and common inhaled bronchodilator medications.
- Demonstrate effective use of an epinephrine auto-injector
- Demonstrate set up and administration of inhaled bronchodilator medication using a nebulizer
- Identify the signs and symptoms of a severe allergic reaction
- Explain the signs and symptoms associated with ischemic chest pain

**Declarative (New EMT curriculum)**

I Review Five Rights (and Patient Allergies)
   - Right Patient
   - Right Medication
   - Right Dose
   - Right Route

II Aspirin:
   - Actions: Help prevent blood from clotting
   - Indications: Signs and Symptoms of Ischemic Chest Pain
   - Authorized by Medical Direction
   - Patient is able to chew or swallow tablets without endangering airway
   - Patient did not take aspirin prior to arrival of EMT
   - Contraindications:
     - Allergic to aspirin
     - Unable to swallow
     - Patient has GI ulcer and/or recent bleeding or bleeding disorder
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

Already taking aspirin or other anticoagulant (Coumadin, Plavix, etc)
Pregnancy
Recent surgery

Dosage:
- 162-324mg (two to four 81mg chewable tablets)
- Administered only once during patient encounter

Side Effects:
- Nausea/vomiting
- Heartburn
- Cough and Wheezing
- Bleeding

III Nitroglycerin:

Actions: Relaxes blood vessels, Decreases workload of heart
Indications: Patient complains of chest pain
- Patient has prescription for NTG, (non expired)
- Systolic blood pressure greater than 100mmhg
- Authorized by medical direction
- Patient has not recently taken medication for erectile dysfunction

Contraindications:
- Hypotension
- Patient has a head injury
- Patient already taken maximum prescribed dose
- Patient recently taken medication for erectile dysfunction

Dosage:
- One spray or one tablet sublingual (under tongue)
- Per medical direction/protocol: repeat in 5 minutes if less than
- Complete relief and blood pressure remains above 100 systolic. If authorized by medical direction up to a maximum of 3 doses:
  - (emphasize blood pressure assessments before each dose and
  - Ascertaining relief of pain or pressure) Recheck blood pressure 2
- Minutes after each dose.

Side Effects:
- Hypotension (lowers blood pressure)
- Headache
- Dizziness/feel like they may pass out
- *Emphasize trendelenburg, high flow oxygen, if patient becomes hypotensive secondary to NTG

IV. Epinephrine Auto Injector:

Actions: Constricts blood vessels and relax air passages
Indications: Authorization by medical direction (protocols)/and/or assisting patient with their prescribed auto injector
- Signs and Symptoms of severe allergic reaction (anaphylaxis)

Contraindications:
- Caution in patients with cardiac history (medical direction)

Dosage: Prefilled dose in auto injector given in thigh.

Side Effects: Rapid heart rate
- Elevated blood pressure
- Restlessness
- Headache
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

*Review signs and symptoms of severe allergic reaction: ie: hives, airway and facial edema, low blood pressure
Emphasize importance of early administration in patients with severe allergic reaction
Demonstrate/Student Practice administration with auto injector practice pens.

V. Bronchodilators: (Albuterol, Ipratropium Bromide (Atrovent), Duoneb, Xopenex)
    Actions:  Dilate/relax air passages in bronchial tree
    Indications:  Authorization by medical direction/and/or assisting patient
                 With metered dose inhaler or nebulized breathing treatment.
                 Relief of bronchospasm/respiratory difficulty in patients with a history of asthma, emphysema or bronchitis.
    Contraindications:
                 Allergic to bronchodilators
                 Caution in patients with symptoms of ischemic chest pain
    Dosage:  Per Protocol:  2 metered dose inhalation sprays with 5 min interval between each spray
              Severe asthma:  4 inhalation sprays in succession
              Nebulized treatment: (per protocol) 1 amp of medication in nebulizer delivered with oxygen at 6-7l/min. Medical direction may suggest ½ amp dose in pediatric patients.
              Nebulized Dose may be repeated at 5-15 min intervals per patient response and medical direction.
    Side Effects:  Rapid heart rate
                  Restlessness

Practice set-up and delivery of nebulized inhalation treatments for both adult and pediatric administration.
Emphasize coaching patients inhalation breaths to get full effect of drug
Emphasize that severe asthma and emphysema patients may require back to back nebulized treatments enroute to hospital per medical direction
Demonstrate delivery techniques for pediatric and adult patients unable to hold nebulizer:
venting mask, neb delivered under mask, etc.)

Patient Assessment terminology – EMT level – Jamin Snarr  30 minutes

• terminology  "primary assessment"
• terminology  "secondary assessment"
• terminology  history taking

Medical New assessment terminology – EMT level – Jerry Hutchison

• (neurology)  add stroke updates
• (immunology) term anaphylaxis
• (infectious disease)  MRSA, AIDS, decontamination of unit
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

- (endocrine) pathophysiology - diabetes
- (psychiatric) excited delirium - restraint change
- (toxicology) Poison control info - drugs of abuse
- (respiratory) more depth
- (hematology) sickle cell disease
- (gyn) PID sexually transmitted disease
- (cardiology) A&P aspirin ACS
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

Trauma – 180 minutes

Educational Standards - EMT level – Wesley Gay

NEEDED CHANGES IN DEFINED AREAS  TRAUMA SECTION
The 1994 National Standard Curriculum for Emergency Medical Technicians: Basic defines trauma in Module 5. It teaches bleeding and shock, signs and symptoms, trauma airway management, internal bleeding signs and symptoms, etc. Teaching this is still equally important, but with more detail in certain areas. Listed below is the area of more needed detail and content to be covered.

CHEST TRAUMA: Give the detailed assessment and management of chest injuries. Stress what all can be involved in chest trauma and the potential for long term problems.

Blunt Trauma: Give good examples and use illustration on manikins to show the injury patterns. Compare blunt and penetrating and show organ involvement.

Hemothorax: Give good illustration of blood loss and stages of shock that will be encountered during this. Teach early recognition of breath sounds and patient condition to treat early. Discuss index of suspicion based on MOI.

Pneumothorax: Discuss each type and the causes and treatment by the basic as well as advanced care. Teach to call for advanced help early if this is suspected.

Cardiac Tamponade: Discuss what the causes are and signs of this may be. Discuss treatment if any, and the need for advance care. Discuss heart sounds and what you should expect to hear

Overview- Discussion on the Center for Disease Control Filed Triage Decision.

Scheme- The National Trauma Triage Protocol

Terminology- Fracture being placed back into the vocabulary

Lecture time (30 min)

CHEST TRAUMA

Cognitive Objectives: Discuss the path physiology of the different type of chest trauma management.

Blunt versus penetrating
Hemothorax (describe causes and effects)
Pneumothorax
Simple
Open
Tension
Cardiac Tamponade
Rib Fractures
Flail Segments
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

Commotio cordis

Psychomotor Objective: Demonstrate to the students what is expected to be seen in the all the chest trauma areas. Demonstrate treatment and what is the expected outcome are if treatment are performed correctly.

ABDOMINAL TRAUMA
Cognitive Objective: Discuss in detail about the path physiology of the abdominal area and the seriousness of abdominal trauma. Give examples of the following organs and the effects they can have on the body:

- Solid and hollow organs injuries
- Blunt versus penetrating mechanisms
- Evisceration
- Psychomotor Objective
- General Assessment;
- Mechanism of injury
- Treatment Plan
- BSI
- Airway Patency
- Be aware and treat for hypoperfusion (shock)

ORTHOPEDIC TRAUMA:
Cognitive Objective: Discuss in detail about the path physiology of the different types of fracture. Give examples of the different fractures types and what you can expect to seen. Give example of the following fractures types and complication involved:

- Upper and Lower extremity fractures
- Open fractures
- Closed fractures
- Dislocations
- Sprain/Strain
- Pelvic fractures
- Amputatuions/replantation

Psychomotor Objective: Demonstrate to the students what is expected to be seen the different types of fractures and treatment plan. Give examples of different splinting methods and proper way to perform each.

HEAD, FACE, NECK, AND SPINAL TRAUMA:
Cognitive Objective: Give detailed discussion about neck, eye, oral and brain injuries; emphasize the harm of hyerventatilation in most circumstances. Discuss about the possibility of ICP in head injury patients. Discuss the management and recognition of the following injuries:

- Penetrating neck trauma
- Laryngeotracheal injuries
- Spinal trauma
- Facial fracture
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

Skull fractures
Foreign bodies in eye
Dental trauma

**Psychomotor Objective:** Demonstrate proper treatment and airway management for the entire listed trauma injuries. Demonstrate proper methods of oxygenation and the different types of device that can be used.

**NERVOUS SYSTEM TRAUMA**

**Cognitive Objective:** Discuss the anatomy of the brain and what can happen if nervous system trauma occurs. Emphasize the harm of hyperventilation to patients. Give a detailed disuasion on the importance of a good neurological assessment. Introduce students to the Brain Trauma Foundation website.

**Psychomotor Objective:** Discussion only

**SPECIAL CONSIDERATIONS IN TRAUMA**

**Cognitive Objective:** Discuss the management and assessment of the following situations. Give examples of the following types of trauma situations, and the difference that will be seen. Discuss treatment plans for the following situations:

- Pregnant patients
- Pediatric patients
- Geriatric patients
- Cognitively impaired patients

**Psychomotor Objective:** Discussion only

**ENVIRONMENTAL TRAUMA**

**Cognitive Objective:** Discuss more in depth the on submersion, bite, stings, diving injuries and radiation espouser. Discuss the path physiology, assessment and management of the following injuries

- Near Drowning
- Temperature related illness
- Bites and stings
- Dysbarism
- Electrical injury
- Radiation exposure

**Psychomotor Objective:** Discussion only
CDC Field Triage Decision Scheme: National Trauma Triage Protocol – David Taylor

Topic: Guidelines for Field Triage of Injured Patients

Objectives:

1. Global Impact
2. Reducing the Impact of Injury
3. Roles of Trauma Centers
4. Initiate Treatment of Traumatically Injured Patient
5. Adult Prehospital Triage Criteria & Decision Scheme
6. Pediatric Prehospital Triage Criteria & Decision Scheme
7. Rapid Transport and Contact with the Appropriate medical facility
8. Indications to NOT activate the EMS System
10. Arkansas Trauma Communication Center (ATCC)

Declarative:

1. Global Impact- Burden of Injury

   a. Injury is a major public health problem. Approximately 5 million deaths worldwide are attributed each year to injuries from all causes\(^1\), representing approximately 10% of all deaths\(^\text{i}\).
   b. Millions of persons are disabled either temporary or permanently every year as a result of injuries\(^\text{ii}\).
   c. In the United States, injury is the leading cause of death for persons aged 1-44 years\(^\text{iv}\).

2. Reducing the Impact of Injury

   a. The way to reduce morbidity, mortality, and economic consequences of injuries is to prevent their occurrence\(^\text{v}\). Community involvement and prevention programs must be implemented to target high risk behavior.
   b. Emergency medical services providers must ensure that patients receive prompt and appropriate emergency care at the scene and are transported to a healthcare facility for further evaluation and treatment.
   c. Emergency care of the traumatically injured patient is best accomplished using an inclusive, multi-level trauma care systems approach\(^\text{vi}\).
Roles of Trauma Centers

1. Roles of Trauma Centers
   a. Level I (MAJOR)
      i. Regional resource hospital that is central to trauma care systems
      ii. Provides total care for every aspect of injury, from prevention through rehabilitation
      iii. Maintains resources and personnel for patient care, education, and research (usually in university-based teaching hospital)
      iv. Provides leadership in education, research, and system planning to all hospitals caring for injured patients in the region
   b. Level II: (COMPREHENSIVE)
      i. Provides comprehensive trauma care, regardless of the severity of injury
      ii. Might be most prevalent facility in a community and manage majority of trauma patients or supplement the activity of a Level I trauma center
      iii. Where no Level I trauma center exists, is responsible for education and system leadership
   c. Level III: (GENERAL)
      i. Provides prompt assessment, resuscitation, emergency surgery, and stabilization and arrange transfer to a higher-level facility when necessary
      ii. Maintains continuous general surgery coverage
      iii. Has transfer agreements and standardized treatment protocols to plan for care of injured patients
   d. Level IV: (BASIC)
      i. Rural facility that supplements care within the larger trauma system
      ii. Provides initial evaluation and assessment of injured patients
      iii. Must have 24-hour emergency coverage by a physician
      iv. Has transfer agreement and a good working relationship with the nearest Level I, II, III trauma center

2. Initiate Treatment of Traumatically Injured Patient
   a. Traumatically injured patients will be appropriately assessed using the Prehospital Triage Criteria and Decision Scheme as defined in the Arkansas Trauma Systems Rules and Regulations. Section IV.C. page 15 & 16.
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

b. Basic Life Support interventions (establishment of patient airway, hemorrhage control, spinal immobilization, fracture immobilization, etc) will be initiated by the prehospital provided following local protocols.
c. Treatment during transport shall follow established local protocols.
d. Adult Prehospital Triage Criteria & Decision Scheme (Arkansas Trauma Systems Rules and Regulations)

i. Assess Vital Signs & Level of Consciousness
   If any of the following occurs initiate Rapid Transport and Trauma Treatment Protocol, if not continue assessment and follow local protocol.
   1. Shock: Systolic: Blood pressure of 90 or less with other signs/symptoms of shock
   2. Respiratory Distress: Respiratory rate of 10 or less; or 29 or higher, Evidence of stridor or retractions
   3. Altered Mentation: Glasgow Coma Scale of 13 or less, Trauma Score of 11 or less

ii. Assess Anatomy of Injury
   If any of the following occurs initiate Rapid Transport and Trauma Treatment Protocol, if not continue assessment and follow local protocol.
   1. Penetrating injury to the head/open or depressed skull fracture
   2. Penetrating injury to the neck torso, or groin
   3. Amputation above the wrist or ankle- near or complete amputation
   4. Spinal cord injury with limb paralysis or alteration of SMSs
   5. Flail Chest
   6. Pelvic fracture
   7. Two or more obvious long bone fractures above the elbows or knees
   8. Major burns: 15% or greater
   9. High voltage electrical burns
   10. Severe maxillofacial injuries

iii. Assess Mechanism of Injury
   If any of the following occurs initiate Rapid Transport and Trauma Treatment Protocol, if not continue assessment and follow local protocol.
   1. Speed 40 mph or greater
   2. Vehicle rollover
   3. Death of same vehicle occupant
   4. Pedestrian or pedal cyclist vs. vehicle 20 mph or greater
   5. Falls 20 feet or greater (consider pediatric rules if applicable)
   6. Vehicle deformity 20" or greater
   7. Ejection from moving vehicle
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

8. Motorcycle 20 mph or greater

iv. If none of the above applies, transport to the closest appropriate trauma center which depending on the system may not be the highest level center.

v. Co-morbid Factors

The following factors may compound the severity of injury and shall increase the index of suspicion:

1. Extreme of age: 55 or more
2. Hostile environment (e.g.; extremes of heat or cold)
3. Medical illness (e.g.; COPD, CHF, renal failure)
4. Presence of intoxicants/substance abuse
5. Pregnancy > 20 weeks
6. Anti-coagulation and bleeding disorders
7. EMS provider judgment (For example cases of prolonged extrication)
8. Time sensitive extremity injury (Potential Vascular Injury)
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

Adult Prehospital Triage Criteria & Decision Scheme

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**STEP ONE**

**VITAL SIGNS & LEVEL OF CONSCIOUSNESS**

**YES**

- Initiate Trauma Treatment Protocols

**NO**

**STEP TWO**

**ASSESS ANATOMY OF INJURY**

**YES**

- Transport

**NO**

**STEP THREE**

**ASSESS MECHANISM OF INJURY**

**YES**

- Contact Medical Control

**NO**

**STEP FOUR**

**CO-MORBID FACTORS**

The following factors may compound the severity of injury and shall increase the index of suspicion:

1. Extremes of age: 55 or more

2. Hostile environment (e.g.; extremes of heat or cold)

3. Medical illness (e.g.; COPD, CHF, renal failure)
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

Pediatric Prehospital Pediatric Triage Criteria & Decision Scheme

**STEP ONE**

**VITAL SIGNS & LEVEL OF CONSCIOUSNESS**

- **Respiratory Distress:** Unstable or unmaintainable airway
  - Respiratory Rate < 10 or > 29
- **Shock:**
  - Age specific bradycardia or tachycardia
  - Capillary refill > 3 seconds
  - Systolic Blood Pressure of 80 or less
- **Altered Mentation:** Pediatric GCS of 9 or less

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**STEP TWO**

**ASSESS ANATOMY OF INJURY**

- Penetrating injury to the head/open or depressed skull fracture
- Penetrating injury of the neck torso, or groin
- Amputation above the wrist or ankle
- Spinal cord injury with limb paralysis or alteration of SMC’s

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**STEP THREE**

**ASSESS MECHANISM OF INJURY**

- Speed 40 mph or greater
  - Vehicle deformity 20” or greater
- Vehicle rollover
  - Ejection from moving vehicle

Transport to closest appropriate Trauma center which, depending on the system, may not be the highest level center.

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**STEP FOUR**

**CO-MORBID FACTORS**

The following factors may compound the severity of injury and shall increase the index of suspicion:

1. Extremes of age: 12 or less/55 or more
2. Hostile environment (e.g.; extremes of heat or cold)
3. Medical illness (e.g.; COPD, CHF, renal failure)

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**INITIATE TRAUMA TREATMENT PROTOCOLS**

- FOLLOW
  - TRANSPORT

**CONTACT**

- MEDICAL
- CONTROL
3. Rapid Transport and Contact with the Appropriate medical facility
   a. Patient transport will be initiated by the prehospital care provider following established local protocols.
   b. Contact with the receiving hospital will be made as soon as possible. An accurate description of the incident, injuries, current medical interventions based upon established protocols, and patient status will be relayed to the facility.
   c. Further management guidance will be requested from the receiving hospital medical control as required during transport.

4. Indications to NOT activate the EMS System
   a. Decomposition
   b. Rigor mortis
   c. Normothermic asystole secondary to trauma (as determined by Advanced Life Support providers only; does not apply to Basic Life Support providers.)

5. Trauma Systems Transport Standard/Guidelines
   a. Patient meeting trauma criteria

   Patients who meet the trauma criteria as outlined in the Adult or Pediatric Prehospital Triage Criteria and Decision Scheme shall be transported to a Level I or Level II Facility unless:

      i. The prehospital care provider is unable to establish or maintain an adequate airway or control excessive hemorrhage; in this case, the patient should be transported to the nearest licensed facility to provide the appropriate care:

         1. If transport time to a Level I or Level II Facility is greater than 45 minutes by ground; transport the patient to a closer Level III Facility unless the Section of EMS & Trauma Systems has approved a deviation from these guidelines.
         2. If transport time to a Level I, Level II, Level III Facility is greater than 45 minutes; transport the patient to a closer Level IV Facility unless the Section of EMS & Trauma Systems has approved a deviation from these guidelines.

   b. Override of criteria by Medical control

   Medical control may override the transport requirement outlined in the Adult or Pediatric Prehospital Triage Criteria and Decision Scheme under the following conditions:

      i. The hospital is unable to meet resource standards as defined for its designated Level.
      ii. Multiple patients are involved.
      iii. The patient needs specialized care and is stable.
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

6. Arkansas Trauma Communication Center (ATCC)
   a. Radio Frequencies
      i. Ambulance to ATCC: 155.280 MHz
      ii. Ambulance to hospital: 155.340 MHz
      iii. Cell phone or direct line can also be utilized to communicate with ATCC
   b. Injury Scale
      i. Severe (Red): Patients **meets** criteria outlined in Adult or Pediatric
         Prehospital Triage Criteria and Decision Scheme. Ambulance crew to follow
         local treatment protocol.
         1. Ambulance crew contacts ATCC and provides patient report &
            condition
         2. ATCC provides ambulance service with transport recommendation
            to appropriate trauma center
         3. ATCC will contact receiving trauma center to inform them of
            inbound ambulance traffic
         4. Ambulance will contact receiving trauma center and provide patient
            report and condition
      ii. Moderate (Yellow): Patient **may** meet criteria outlined in Adult or Pediatric
         Prehospital Triage Criteria and Decision Scheme. When in doubt, call ATCC.
         Ambulance crew to follow local treatment protocol.
         1. Ambulance crew contacts ATCC and provides patient report &
            condition
         2. ATCC provides ambulance service with transport recommendation
            to appropriate trauma center
            a. ATCC recommendations:
               i. follow local transport protocol or
               ii. ATCC will direct ambulance crew to appropriate
                  trauma center
         3. If ATCC directs ambulance crew to appropriate trauma center, ATCC
            will contact receiving trauma center to inform them of inbound
            ambulance traffic
         4. Ambulance crew will contact receiving trauma center and provide
            patient report and condition
      iii. Minor (Green): Patient does not meet criteria outlined in Adult or Pediatric
         Prehospital Triage Criteria and Decisions Scheme.
         1. Follow local treatment and transport protocol
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

**Overall LERN Process**

Each Regional Medical Call Center (RMCC) must implement and execute the following LERN process. Procedures and processes will be updated periodically to reflect experience, new knowledge, enhanced technical capabilities, and/or regional preferences. The Vendor must adhere to all processes and procedures promulgated by the LERN Board and/or the LERN State Operations Center (State LOC or LERN LOC). Sequentially, the overall LERN procedure involves the following steps:

7. A call to 911 will be made by an injured patient, which goes to the currently operating 911 centers.
8. 911 will dispatch an EMS unit to the scene of an injured patient.

--------------------------LERN PROCESS BEGINS HERE--------------------------

9. The paramedics will do a quick assessment of the patient at the scene and the trauma status of the patient will be determined as per regional LERN protocol.
10. Based on protocol the injured patient(s) will be designated as a minor injured patient, moderately injured patient or a major injured patient.
11. All major injured patients will be entered into the LERN system by calling the RMCC and giving the history and physical of the patient.
12. Based on this the RMCC will then review the current status of the hospitals in the region and direct the EMS unit to transfer the injured patient to the most appropriate hospital per established medical and transport criteria.
13. Patients that have not sustained major injury have 2 options at this time.
   a. Patients can be transferred to a local area hospital without having to be entered into LERN based on the awareness by the EMS unit of the capacity and capability of the local hospital.
   b. Patients can be entered into LERN by the EMS unit by calling the RMCC and giving the history and physical data about the patient. Because the EMS unit knows that the local area hospital has no capacity and expertise to manage the injury and thus the acuity of the patient exceeds the local area hospital capability, the patient is entered into LERN and the RMCC directs the transfer to the closest and most appropriate facility.
14. Patients that sustain minor injury will not be entered into LERN and are transferred in local area hospitals following already established transfer relationships.
15. If the EMS unit is not sure if the patient meets criteria to enter the LERN system then the EMS unit can still enter the patient into LERN under “EMS Discretionary Status”
16. Discretionary Status allows the EMS unit to take a patient to a hospital with out being directed by the RMCC but if the patient meets the criteria to enter LERN after arriving at the hospital then the hospital can call the RMCC to facilitate the rapid transfer of the patient to the next most appropriate hospital.
17. The EMS Discretionary Status allows for the rapid transfer from one hospital to another by providing all the information to the RMCC during initial assessment.
18. If a patient arrives at a hospital by PRIVATE vehicle and meets criteria to enter the LERN system, then the hospital can call the RMCC and enter the patient into the system and the RMCC facilitates the transfer to the most appropriate facility.

19. If a patient arrives at a hospital by EMS and was designated to be a minor injured patient but the condition of the patient changes and deteriorates becoming a higher level of acuity, then the patient can be entered into LERN by the hospital by calling the RMCC and the RMCC will facilitate the transfer to a more appropriate facility.

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vi Rules and Regulations for Trauma Systems, Section IV, Arkansas, Section of Emergency Medical Services and Trauma Systems; March 1, 2009. Available at www.healthyarkansas.com/ems

vii Centers for Disease Control and Prevention. Guidelines for Field Triage of Injured Patients, MMWR, January 23, 2009; 58:[No.RR-1; page2]

viii Centers for Disease Control and Prevention. Guidelines for Field Triage of Injured Patients, MMWR, January 23, 2009; 58:[No.RR-1; page5]

ix Centers for Disease Control and Prevention. Guidelines for Field Triage of Injured Patients, MMWR, January 23, 2009; 58:[No.RR-1; page5]

x Rules and Regulations for Trauma Systems, Section IV, Arkansas, Section of Emergency Medical Services and Trauma Systems; March 1, 2009. Available at www.healthy.arkansas.gov/ems

xi Rules and Regulations for Trauma Systems, Section IV, Arkansas, Section of Emergency Medical Services and Trauma Systems; March 1, 2009. Available at www.healthy.arkansas.gov/ems
Geriatrics

Objectives

1. Define key terms

2. Summarize age-related anatomical and physiological changes for each of the following systems in the Elderly patient:
   a. Cardiovascular
   b. Respiratory
   c. Musculoskeletal
   d. Renal
   e. Endocrine
   f. Neurological
   g. Gastrointestinal

3. Discuss common cardiac medical emergencies and their treatments found in the elderly population.
   a. Myocardial Infarction, Congestive Heart Failure, Silent Heart Attack and Pulmonary edema
      i. See Cardiac Section for Sign, symptoms and treatment.
      ii. Possible changes in physical assessment
      iii. What different assessment tools will be needed
   b. Pulmonary Embolism
      i. See Respiratory Section for Signs, symptoms and treatment.
      ii. Possible changes in physical assessment
      iii. What different assessment tools will be needed.
   c. Respiratory Changes in the Elderly and medical emergencies they may cause.
      i. Loss of elastic recoil in the chest walls
      ii. Loss of alveoli
      iii. Less O2 and Carbon Dioxide exchanges
      iv. Decrease cough reflex (Pneumonia)
      v. Decrease in the Cilia
   d. Discuss the Signs, symptoms, and treatment of:
      i. Pneumonia
      ii. COPD
      iii. Aspiration Pneumonia

4. Dementia is a chronic, irreversible condition that can be worsened by infection
   a. Discuss the signs and symptoms of dementia
b. Known Reversible causes of dementia
   i. Drug overdose
   ii. Emotional disorder
   iii. Tumors
   iv. Parkinson’s Disease
   v. Huntington’s Disease
   vi. Several Others

c. Discuss the treatment of Dementia

5. Toxicological Emergencies

6. Sensory Changes in the Elderly

7. Hearing impairment

8. Pain Perception

Special Patient Populations

Changes in Defined Areas
Patients with Special Challenges:
   Elder abuse
   Homelessness
   Poverty
   Bariatric
   Technology dependant
   Sensory deficit
   Homecare
   Developmental disabilities

Topic:
Elder Abuse: Defined as when an elderly person is harmed by people the older person knows or with whom they have a relationship, such as a spouse, partner or family member, a friend or neighbor, or people that the older person relies on for services. Many forms of elder abuse are recognized as types of domestic violence or family violence.

Objective
1. Define Key Word
2. Types of Elderly Abuse
   a. Physical
   b. Psychological/emotional
   c. Financial
   d. Sexual;
   e. Neglect
f. Signs:
   i. Depressed
   ii. Will never accept invitations to spend time away from the family and/or caregiver
   iii. Appears afraid to make their own decisions
   iv. Seems to be hiding something about a caregiver
   v. Never seems to have any spending money
   vi. May put off going to the doctor
   vii. Seems to have too many household “accidents”

g. Injury found in elder abuse:
   i. Trauma (see Trauma section)
   ii. Overdose
   iii. Bed sores
   iv. Malnutrition
   v. Decrease in mental status

h. Treatment (See section related to injury)
   i. Know to whom elder abuse should be reported.

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**Topic:**

**Homelessness and poverty:** Homelessness is the condition and social category of people who do not have a regular house or dwelling because they cannot afford, pay for, or are otherwise unable to maintain regular, safe and adequate housing, or they lack “fixed, regular, and adequate nighttime residence. Homeless people are more likely to suffer injuries and medical problems from their lifestyle on the street.

**Objectives:**

1. Define Key Terms
2. Emergencies seen in the homeless person
   a. Poor nutrition
   b. Substance abuse
      i. Signs, symptoms and Treatment (see pharmacology emergencies)
   c. Exposure to severe weather:
      i. Signs, symptoms and Treatment
   d. High exposure to violence (robberies, beatings, sexual abuse)
   e. Little or no medical care

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**Topic:**

**Bariatric:** The field of medicine that offers treatment for the person who is clinically overweight with a comprehensive program including diet, exercise, behavior modification, lifestyle changes and, when indicated, the addition of appetite suppressants and other appropriate medications. Bariatrics also includes research into overweight, its causes, prevention, and treatment.

There are many effects of excess weight on the body systems:

**Objectives:**

1. Discuss the effects of the following in regards to obesity:
   a. Hypertension, coronary artery disease, congestive heart failure and stroke.
      Tx: (see cardiac emergencies)
   b. Sleep apnea, asthma, and COPD:
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Between old DOT NSC and new EMS Education Standards

1. Identify the major objectives and treatment of pregnant patients with:
   a. Preeclampsia
      i. Form of high blood pressure
      ii. Called Toxemia of Pregnancy
      iii. Can develop into eclampsia
   b. Eclampsia
      i. More severe that preeclampsia
      ii. May include seizures
      iii. Generally develops after 20th week
      iv. May develop after delivery
   c. Affect on Infant
      i. Low birth weight
      ii. May need early delivery
      iii. Seizure threatens life of Mother and baby
      iv. Placental abruption
   d. Treatments:
      i. Oxygen
      ii. Position of comfort
      iii. Treatment for shock
      iv. Treatment for seizure
      v. Maternal and infant support

2. Identify symptoms and treatment of Premature Rupture of Placental Membranes (PROM)
   a. Terminology:
      i. PROM (premature rupture of amniotic sac)
      ii. PPROM (preterm premature rupture of membranes)
         i. Before 37 weeks of gestation
         ii. Baby may be born within one week
      iii. Amniotic sac
      iv. Chorioamnionitis (serious infection of placental membranes)
   b. Causes of PROM
      i. Natural weakening of membrane near term
      ii. Force of contractions

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Tx: (See respiratory emergencies)
c. Diabetic emergencies
d. Depression and Suicide
e. Immobility

2. Accommodations and moving for the Bariatric (obese) patient
   a. Airway and breathing
   b. Sitting upright (not supine)
c. O2 need/use

3. Weight concerns
   a. Will the cot hold the patient – maximum cot limits
   b. Need for additional help

Topic: **Pregnant Patients**: Expansion of terminology to use preeclampsia, eclampsia and premature rupture of membranes (may require a lengthy discussion). More detailed discussion on complications.

Objectives:

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iii. Infection of the uterus (PPROM)
iv. Low socioeconomic conditions (no prenatal care)
v. STE (clamydia – gonorrhea)
vi. Previous preterm birth
vii. Vaginal bleeding
viii. Cigarette smoking during pregnancy
ix. Unknown additional causes

c. Complicates as many as 1/3 of premature births

EMS operations
- safely operating ground ambulance
- add lifting and moving

incident management (review location of ICS 100, 200, 700, 800)

MCI- CDC Triage Protocol (see above)

Air medical (same as EMR)

Vehicle extrication (same as EMR)

Hazmat (same as EMR)

MCI from Terror or Disaster (same as EMR)
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Arkansas Department of Health     Section of Emergency Medical Services

EMS Education Standards – Transition Curriculum

Paramedic Level

For a Emergency Medical Services Provider educated as an EMT-Paramedic (based on the 1998 EMT-Paramedic National Standard Curriculum) and transitioning to the education expectations of the 2009 Paramedic Education Standards, the following pages present the information needed to educate to the 2009 Paramedic Educational Standards.

These materials were developed by the EMS Training Committee, approved by the EMS Governor’s Advisory Council and endorsed by the Arkansas Department of Health, Section of EMS.

These materials (EMT and Paramedic) should take approximately 32 hours to cover in the transition course of a total 48 hours. The remaining time can be utilized to cover local material and issues of interest and concern to the Paramedics involved. This course will replace the traditional Paramedic 48 hour refresher course.
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**Topic : Airway/Ventilation/ Oxygenation**

What’s New?
Anatomy and Physiology – Much more detailed than in the previous 1994 EMT-B National Standard Curriculum.
Respiration- Much more detailed than in the previous 1994 EMT-B National Standard Curriculum.
Artificial Ventilation- Much more detailed than in the previous 1994 EMT-B National Standard Curriculum.

Addition of (this was previously in the curriculum but removed in 1994):
Partial Rebreather Mask, Simple Face Mask, Venturi Mask, Pulse Oximetry, Ventilator AVT), use of oxygen humidifiers.

**Topic: Airway:** See Basic section of Gap Curriculum for detailed curriculum

**Supraglottic airway – to include Combitube, King Airway, etc (check what is used in your community.)**

**BIPAP/CPAP**

**Chest tube placement:**

- **Assist placement**
- **Monitor placement – transfer of Patient**

**Cricothyrotomy ;**

A cricothyrotomy (also called thyrocricotomy, cricothyroidotomy, inferior laryngotomy, intercricothyrotomy, coniotomy or emergency airway puncture) is an incision made through the skin and cricothyroid membrane to establish a patent airway during certain life-threatening situations, such as airway obstruction by a foreign body, angioedema, or massive facial trauma. Cricothyrotomy is nearly always performed as a last resort in cases where orotracheal and nasotracheal intubation are impossible or contraindicated. Cricothyrotomy is easier and quicker to perform than tracheotomy, does not require manipulation of the cervical spine, and is associated with fewer complications.[1] However, while cricothyrotomy may be life-saving in extreme circumstances, this technique is only intended to be a temporizing measure until a definitive airway can be established.

**Indications**

- Can’t intubate
- Can’t ventilate
- Severe facial or nasal injuries (that do not allow oral or nasal tracheal intubation)
- Massive midfacial trauma
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- Possible cervical spine trauma preventing adequate ventilation
- Anaphylaxis
- Chemical inhalation injuries

**Contraindications**

- Inability to identify landmarks (cricothyroid membrane)
- Underlying anatomical abnormality (tumor)
- Tracheal transection
- Acute laryngeal disease due to infection or trauma
- Small children under 10 years old (a 12–14 gauge catheter over the needle may be safer).

**Summarized technique**

- With a scalpel, create a 1 cm vertical incision through the skin and the cricothyroid membrane
- Open the hole by inserting the scalpel handle into the wound and rotating 90 degrees or by using a clamp
- Insert a 6 or 7 mm Internal Diameter tracheostomy tube or endotracheal tube
- Inflate the cuff and secure the tube
- Provide ventilation via a bag-valve device with the highest available concentration of oxygen
- Determine if ventilation was successful (bilateral auscultation and observing chest rise and fall)
- No attempt should be made to remove the tracheostomy or endotracheal tube in a prehospital setting

**Cardiovascular:** see EMT section for topics and coverage.

**Medication administration:** see EMT section for topics and coverage.

**IV initiation and fluid maintenance:**

- NOTE: remove umbilical line access
- Access indwelling catheters and implanted ports
- Central line monitoring
- Intraosseous – Adult  review current local device
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**Topic: Morgan Lens**
Access information at: [www.morganlens.com](http://www.morganlens.com).
Call for PowerPoint presentation CD.

**Topic: Glucose meter**

(15 minutes didactic; 15 minutes laboratory) Comment: no problem

**Cognitive Objectives:**
Discuss the signs and symptoms of hypo and Hyperglycemia
Discuss the need to test possible CVA symptoms
Discuss the different types of devices used for testing
Discuss the need to make sure area is clean and dry (if alcohol is used as a disinfectant)

**Psychomotor Objectives:**
Demonstrate the techniques for Cleaning Site
Demonstrate where sample of blood may be taken from
Demonstrate the care of the patient exhibiting signs and symptoms of insulin shock

Declarative (old EMT-B curriculum) did not include this skill

**Summary:** The traditional glucose meter comes with test strips and small needles called lancets. There are many different kinds of these meters, but they all work essentially the same way.

A complete testing kit can be purchased from a pharmacy without a prescription by anyone.

Have all test items within reach before starting -- timing is important. Clean the needle prick area with soap and water or an alcohol swab. Alcohol is the preferred. Completely dry the skin before pricking. Alcohol left on the skin may give a falsely high reading and other cleaning solutions may give a falsely low reading.

Warn your patient that they will feel a stick.

You will prick the patient’s finger with the lancet and place a drop of blood on a special strip. This strip uses a chemical substance to determine the amount of glucose in the
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blood. (Newer monitors can use blood from other areas of the body besides the fingers, reducing discomfort.) The meter displays the blood glucose results as a number on a digital display. There are strips that display a color code to show a range.

**Normal Results**

- Before meals: 70 - 130 milligrams per deciliter (mg/dL)
- After meals: Less than 180 mg/dL

Low levels indicate hypoglycemia. Treat appropriately

If levels are too high, this indicates hyperglycemia. Treat appropriately

**Risks**

There is a slight chance of infection at the puncture site. A small amount of bleeding may occur after the puncture.

**Considerations**

The correct procedure must be followed or the results will not be accurate.

**Topic: Thrombolytic**

Time 30Min review 1hr. class

Cognitive Objective: Understand the indication for thrombolytic therapy.

Cognitive Objective: Understand the contraindications of thrombolytic.

Cognitive Objective: Understand the complications of thrombolytic therapy.

Cognitive Objective: Identify the different thrombolytic drugs.

Special consideration: The Instructor should review the clotting process and the 12 lead ECG results to ensure the student has sufficient knowledge.

**Out-of-Hospital Thrombolytic Therapy**

Currently, prehospital 12-lead ECG programs have been recommended for urban and rural EMS systems. Medical literature supports this recommendation because of its benefits in early diagnosis and earlier treatment. Several studies have documented the ability for trained prehospital professionals to adequately acquire STEMI with 12-lead ECGs.
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Paramedics can provide advance notification to the receiving facility when they encounter an acute coronary syndrome, being able to provide a 12-lead ECG of such patients allows the institution to prepare for reperfusion strategies. It is also recommended that EMS personnel start screening for possible thrombolytic therapy in patients who may be having a STEMI in order to further decrease the time for reperfusion.

For some years, controversy has existed regarding the administration of thrombolytic drugs in the prehospital setting. Previously, out-of-hospital fibrinolysis was only recommended when patient transport time was more than 1 hour. However, today several studies and clinical trails have demonstrated contrary. Out-of-hospital fibrinolysis is safe and reasonable. It can be performed by skilled, trained paramedics, nurses, or physicians under strict protocols. If emergency medical system (EMS) has fibrinolytic capability and the patient qualifies for therapy, prehospital fibrinolysis should be started within 30 minutes of arrival on the scene.²

For EMS systems to implement out-of-hospital thrombolytic programs, several quality standards are required. Protocols must include thrombolytic checklists, 12-lead ECG interpretation and transmission, ACLS-trained personnel, and medical direction must be available at all times. These programs should have an adequate quality evaluation process to evaluate efficacy and safety.

Thrombolytic agents

Thrombolytic agents available today are serine proteases that work by converting plasminogen to the natural fibrinolytic agent plasmin. Plasmin lyses clot by breaking down the fibrinogen and fibrin contained in a clot.

The history of thrombolytic therapy began in 1933 when it was discovered that filtrates of broth cultures of certain strains of Streptococcus bacteria (beta-hemolytic streptococci) could dissolve a fibrin clot.¹ Streptokinase found its initial clinical application in combating fibrinous pleural exudates, hemothorax, and tuberculous meningitis.² In 1958, streptokinase was first used in patients with acute myocardial infarction, and this changed the focus of treatment.

At first, streptokinase infusion produced conflicting results until the Gruppo Italiano per la Sperimentazione della Streptochinasi nell’Infarto Miocardico (GISSI) trial in 1986, which validated streptokinase as an effective therapy and established a fixed protocol for its use in acute myocardial infarction.²

The fibrinolytic potential of human urine was first described in 1947. The active molecule was named urokinase.¹ Unlike streptokinase, urokinase is not antigenic and directly activates plasminogen to form plasmin. Their ability to catalyze the conversion of plasminogen to plasmin is affected only slightly by the presence or absence of local fibrin clot.

Tissue plasminogen activator (tPA) is a naturally occurring fibrinolytic agent found in vascular endothelial cells and is involved in the balance between thrombolyis and thrombogenesis. It exhibits significant fibrin specificity and affinity. At the site of the thrombus, the binding of tPA and plasminogen to the fibrin surface induces a conformational change facilitating the conversion of plasminogen to plasmin and dissolving the clot.²

Fibrinolytics, sometimes referred to as plasminogen activators, are divided into two categories. Fibrin-specific agents such as alteplase, reteplase, and tenecteplase produce limited plasminogen conversion in the absence of fibrin, whereas non–fibrin-specific agents such as streptokinase catalyze systemic fibrinolysis. Streptokinase is indicated for the treatment of acute myocardial infarction, acute massive pulmonary embolism, deep vein thrombosis, arterial thrombosis, and occluded arteriovenous cannulae. Streptokinase is not widely used in the
United States but continues to be used elsewhere because of its lower cost.

Alteplase is the only current lytic agent US Food and Drug Administration (FDA) approved for AMI, acute ischemic stroke, massive pulmonary embolism, and occluded central venous access devices. New agents and new dosing regimens are under constant investigation. A choice of lytic agents must be based upon the results of ongoing clinical trials and upon the clinician’s experience. The most appropriate agent and regimen for each clinical situation will change over time and may differ from patient to patient.

The information presented here is based on clinical and investigational experience as reported in the current literature to the authors’ best knowledge, without respect to FDA approval for a particular indication. Where the literature does not suggest an effective dose for a lytic agent in a particular clinical setting, no dose information is presented. Currently available agents today are alteplase (tPA), reteplase (r-PA), tenecteplase (TNKase), urokinase, prourokinase, anisoylated purified streptokinase activator complex (APSAC), and streptokinase.

**Alteplase**

Alteplase (tPA, Activase) was the first recombinant tissue-type plasminogen activator and is identical to native tissue plasminogen activator. In vivo, tissue-type plasminogen activator is synthesized and made available by cells of the vascular endothelium. It is the physiologic thrombolytic agent responsible for most of the body’s natural efforts to prevent excessive thrombus propagation.

Alteplase is fibrin specific with a plasma half-life of 4-6 minutes. It is the fibrinolytic agent most familiar to emergency departments and is the lytic agent most often used for the treatment of coronary artery thrombosis, pulmonary embolism, and acute ischemic stroke given as an infusion. Alteplase is FDA approved for treatment of ST-elevation myocardial infarction (STEMI), acute ischemic stroke (AIS), acute massive pulmonary embolism, and central venous access devices (CVAD). In theory, alteplase should be effective only at the surface of fibrin clot. In practice, however, a systemic lytic state is seen, with moderate amounts of circulating fibrin degradation products and a substantial risk of systemic bleeding.

Alteplase may be re-administered as necessary, as it is not antigenic and almost never is associated with any allergic manifestations.

Currently, alteplase is the only thrombolytic drug approved for acute ischemic stroke.

**Reteplase**

Reteplase (r-PA, Retavase) is a second-generation recombinant tissue-type plasminogen activator that seems to work more rapidly and to have a lower bleeding risk than the first-generation agent alteplase.

Reteplase is a synthetic nonglycosylated deletion mutein of tissue plasminogen activator containing 355 of the 527 amino acids of native tissue plasminogen activator. The drug is produced in *Escherichia coli* by recombinant techniques. Reteplase does not bind fibrin as tightly as native tissue plasminogen activator, allowing the drug to diffuse more freely through the clot rather than binding only to the surface the way tissue plasminogen activator does. In high concentrations, Reteplase does not compete with plasminogen for fibrin-binding sites, allowing plasminogen at the site of the clot to be transformed into clot-dissolving plasmin. These
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Characteristics help explain the faster clot resolution seen in patients receiving reteplase than in those receiving alteplase.

The modifications also resulted in a molecule with a longer half-life (approximately 18 min) allowing for bolus administration. Reteplase is FDA approved for acute myocardial infarction, and it is administered as two 10 U boluses given 30 minutes apart, each bolus is given over 2 minutes. The result is more convenient administration and faster thrombolysis with reteplase than with alteplase, which is given by a bolus followed by an intravenous (IV) infusion.

Reteplase may be re-administered as necessary, as it is not antigenic and almost never is associated with any allergic manifestations.

**Tenecteplase (TNKase)**

TNKase was approved by the FDA as a fibrinolytic agent in 2000. This drug has a similar mechanism of action as alteplase (tPA). It is the latest thrombolytic agent approved for use in clinical practice. TNKase is currently indicated for the management of acute myocardial infarction (AMI).

Tenecteplase is produced by recombinant DNA technology using Chinese hamster ovary cells. This drug is a 527 amino acid glycoprotein, which sustained several modifications in amino acids molecules. These modifications consist of a substitution of threonine 103 with asparagine, asparagine 117 with glutamine, and a tetra-alanine substitution at amino acids 296-299 in the protease domain. This change permits TNKase to have a longer plasma half-life and more fibrin specificity. Tenecteplase has a half-life ranging initially from 20-24 minutes up to 130 minutes final clearance, most of it by liver metabolism.

Because of the amino acid modifications, TNKase has the advantage for a single bolus administration and decreased bleeding side effects due to high fibrin specificity. The ASSENT-2 trial evaluated the efficacy and safety of tenecteplase compared with alteplase in patients with AMI. Tenecteplase was found noninferior to alteplase in terms of 30-day mortality. Tenecteplase was associated with fewer bleeding complications, major bleeding events (4.66% vs 5.94%), and lower need for blood transfusion (4.25% vs 5.49%; p=.0002). Rates for intracranial hemorrhage were similar (tenecteplase 0.93%, alteplase 0.94%). Follow-up study showed that mortality rates between the two active therapy groups remained similar after one year.

Several clinical trials are in progress seeking new indications for this drug such as in acute ischemic stroke.

**Urokinase**

Urokinase (Abbokinase) is the fibrinolytic agent most familiar to interventional radiologists and the one that has been used most often for peripheral intravascular thrombus and occluded catheters. Recently, urokinase was made available once again from the manufacturer. After some years hold from the market due to manufacturer issues with the FDA, it has been reintroduced. The package insert was revised and, since then, has an indication only for massive pulmonary embolism. During the time this drug was not available, the FDA encouraged the off-label use of reteplase and alteplase for local-regional lysis of venous and arterial thrombus at any location. Currently, this drug is readily used for this purpose in different clinical and interventional settings.

Urokinase is a physiologic thrombolytic agent that is produced in renal parenchymal cells. Unlike streptokinase, urokinase directly cleaves plasminogen to produce plasmin. When purified from human urine, approximately
1500 L of urine are needed to yield enough urokinase to treat a single patient. Urokinase is also commercially available in a form produced by tissue culture, and recombinant DNA techniques have been developed for urokinase production in *E coli* cultures.

In plasma, urokinase has a half-life of approximately 15 minutes. Allergic reactions are rare, and the agent can be administered repeatedly without antigenic problems.

**Prourokinase**

Prourokinase is a new fibrinolytic agent that is currently undergoing clinical trials for a variety of indications. It is a relatively inactive precursor that must be converted to urokinase before it becomes active in vivo. This has handicapped therapeutic exploitation of its fibrin-specific physiological properties.

Researchers have developed a mutant of prourokinase (M5) with even greater plasma stability and which causes faster plasminogen activation and greater fibrin-specific clot lysis than wild-form prourokinase. As with tissue-type plasminogen activator, prourokinase is somewhat clot specific, since the presence of fibrin enhances the conversion of prourokinase to active urokinase by an unknown mechanism.

**Streptokinase**

Streptokinase is the least expensive fibrinolytic agent, but, unfortunately, it is antigenic and produces a high incidence of untoward reactions. This drawback limits the usefulness of streptokinase in the clinical setting. Although other fibrinolytic agents are more popular in developed nations like the United States, streptokinase continues to be widely used in developing nations.

Streptokinase is produced by beta-hemolytic streptococci. Streptokinase by itself is not a plasminogen activator, but it binds with free circulating plasminogen (or with plasmin) to form a complex that can convert additional plasminogen to plasmin. Streptokinase activity is not enhanced in the presence of fibrin.

The principal plasma activity half-life of streptokinase is about 20 minutes, but an unbound fraction (about 15%) has a half-life of 80 minutes. Since it is produced from streptococcal bacteria, it often causes febrile reactions and other allergic problems. It can also cause hypotension that appears to be dose-related. Streptokinase usually cannot be administered safely a second time within 6 months, because it is highly antigenic and results in high levels of antistreptococcal antibodies.

**Anisoylated purified streptokinase activator complex (APSAC)**

APSAC is a complex of streptokinase and plasminogen that does not require free circulating plasminogen to be effective. It has many theoretical benefits over streptokinase but suffers antigenic problems similar to those of the parent compound.

The half-life of APSAC in plasma is somewhere between 40 and 90 minutes.

**Thrombolytic Therapy for Acute Myocardial Infarction**

Thrombolytic therapy is indicated in patients with evidence of ST-segment elevation myocardial infarction (STEMI) or presumably new left bundle branch block (LBBB) presenting within 12 hours of the onset of symptoms if there are no contraindications to fibrinolysis. Patients with STEMI usually have complete occlusion of an epicardial coronary vessel caused by an acute thrombotic obstruction.
Coronary atherosclerosis is a diffuse process with segmental lesions called coronary plaques. The plaque ruptures, exposing the endothelial lining, and allowing prothrombotic enzymes and molecular triggers to mix with the blood. Platelets are activated, and the coagulation cascade is amplified resulting in a thrombus that occludes the vessel, preventing the circulation of oxygenated blood. Irreversible ischemia-induced myocardial necrosis may occur within 20-60 minutes of occlusion. The mainstay of treatment is reperfusion therapy through administration of fibrinolytics (pharmacologic reperfusion) or primary percutaneous coronary intervention (PCI) (mechanical reperfusion).

PCI when performed within 90 minutes of patient arrival has been shown to be superior to fibrinolysis in combined end points of death, stroke, and reinfarction in many studies. The reality is that PCI is not widely available at acute care hospitals. In the United States, of the nearly 5,000 acute care hospitals, 2,200 have catheterization laboratories. Among those, only 1,200 (<25%) are capable of performing PCI.

Fibrinolytic therapy is a proven and effective treatment for the management of acute myocardial infarction (AMI). It is more universally available to patients without contraindications, can be administered by any properly trained health care provider, and can be given in the prehospital setting, reducing the time to treatment. The goal is a door-to-needle time of less than 30 minutes. Every effort must be made to minimize the time to therapy. The efficacy of fibrinolytic therapy declines as the duration of ischemia increases.

Fibrinolytic agents are given in conjunction with antithrombin and antiplatelet agents, which help to maintain vessel patency once the clot has been dissolved.

- **Aspirin** inhibits platelets; the recommended dose is 162-325 mg of chewable aspirin.
- **Clopidogrel** inhibits platelets. In patients younger than 75 years, administer an oral loading dose of 300 mg. The COMMIT-CCS-2 and CLARITY-TIMI 28 trials provided evidence for benefit of adding clopidogrel to aspirin in patients undergoing fibrinolytic therapy. No data are available to guide decision-making regarding an oral loading dose in patients aged 75 years or older. In this group of patients, administer 75 mg per day.
- **Heparins** (unfractionated heparin [UFH] or low molecular weight heparin [LMWH]) inhibit thrombin. For UFH, the recommended dose is an intravenous bolus of 60 U/kg (maximum 4,000 U) followed by an initial infusion of 12 U/kg/h (maximum 1000 U/h) adjusted to maintain aPTT at 1.5-2 times control.
- **LMWH** (enoxaparin) is emerging as an alternative to UFH. Enoxaparin may be administered to patients younger than 75 years; the recommendation is 30 mg IV bolus followed by 1 mg/kg subcutaneous every 12 hours. For patients at least 75 years old, the intravenous bolus is eliminated and the subcutaneous dose is reduced to 0.75 mg/kg every 12 hours. Regardless of age, if the creatinine clearance is less than 30 mL/min, the subcutaneous dose is 1 mg/kg every 24 hours. Enoxaparin appears superior to UFH in the EXTRACT-TIMI 25 trial.

**Contraindications for fibrinolytic use in STEMI**

**Absolute contraindications**

- Prior intracranial hemorrhage (ICH)
- Known structural cerebral vascular lesion
- Known malignant intracranial neoplasm
- Ischemic stroke within 3 months
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Between old DOT NSC and new EMS Education Standards

- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed-head trauma or facial trauma within 3 months

Relative contraindications

- History of chronic, severe, poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (SBP >180 mm Hg or DBP >110 mm Hg)
- Traumatic or prolonged (>10 min) CPR or major surgery less than 3 weeks
- Recent (within 2-4 wk) internal bleeding
- Noncompressible vascular punctures
- For streptokinase/anistreplase - prior exposure (more than 5 d ago) or prior allergic reaction to these agents
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulant (eg, warfarin sodium) that has produced an elevated international normalized ratio (INR) >1.7 or prothrombin time (PT) >15 seconds

Alteplase

Alteplase (tPA) can be administered in an accelerated infusion (1.5 h), 50-mg and 100-mg vials reconstituted with sterile water to 1 mg/mL.

The accelerated infusion of alteplase (tPA) for acute MI is 15 mg IV bolus, followed by 0.75 mg/kg (up to 50 mg) IV over 30 minutes, then 0.5 mg/kg (up to 35 mg) IV over 60 minutes, with a maximum total dose of 100 mg. This is the most common alteplase infusion parameter used for acute myocardial infarction.

Reteplase

Must reconstitute two 10-U vials with sterile water (10 mL) to 1 U/mL. The adult dose of reteplase for acute MI is 2 IV boluses of 10 units each; there is no weight-adjustment. The first 10 U IV bolus is given over 2 minutes; 30 minutes later, the second 10 U IV bolus is given over 2 minutes. Administer normal saline (NS) flush before and after each bolus.

Tenecteplase (TNKase)

To reconstitute, mix the 50-mg vial in 10 mL sterile water (5 mg/mL).

TNKase is administered 30-50 mg IV bolus over 5 seconds. Dosage is calculated based on the patient's weight.

- <60 kg - 30 mg (6 mL)
- ≥60 kg to <70 kg - 35 mg (7 mL)
- ≥70 kg to <80 kg - 40 mg (8 mL)
- ≥80 kg to <90 kg - 45 mg (9 mL)
- ≥90 kg - 50 mg (10 mL)

Streptokinase
The adult dose of streptokinase for AMI is 1.5 million U in 50 mL D5W given IV over 60 minutes. Allergic reactions force the termination of many infusions before a therapeutic dose can be administered.

**APSAC**

The adult dose of anisoylated purified streptokinase activator complex (APSAC) for AMI is 30 U given IV over 2-5 minutes.

**Thrombolytic Therapy for Pulmonary Embolism**

Pulmonary embolism (PE) is a common disorder and an important cause of morbidity and mortality. Pulmonary embolism occurs in approximately 650,000 patients annually in the United States. Among patients who are hemodynamically unstable at presentation, in-hospital mortality reaches 30%.

Pulmonary emboli often arise from thrombi originating in the deep venous system of the lower extremities or pelvis. A blood clot dislodges and is swept into the pulmonary circulation and lodges in a pulmonary artery. If the clot is large enough to obstruct large vessels in the lung, it can cause hemodynamic instability, along with right ventricular failure, and possibly death. Currently, thrombolytic therapy in pulmonary embolism is still controversial.

Pulmonary embolism varies in severity from acute massive pulmonary embolism, acute pulmonary infarction, acute embolism without infarction to multiple emboli. Only patients with acute massive pulmonary embolism, those at the highest risk of immediate death, are eligible for fibrinolytic therapy if no contraindications are present. Other types are treated with anticoagulants or antithrombotic therapy.

Nevertheless, there is a subgroup of patients who present hemodynamically stable but with right ventricular (RV) dysfunction, who might benefit from fibrinolytic therapy due to increased risk of death. Fibrinolytic therapy in these patients (normotensive with RV dysfunction) remains controversial. In this regard, a large trial is needed using contemporary methods and criteria for the inclusion of high-risk normotensive patients.

In addition to generalized, nonspecific symptoms, patients with acute massive pulmonary embolism also present with systemic hypotension, SBP <90 mm Hg or a decrease in systolic arterial pressure of at least 40 mm Hg for at least 15 minutes, and/or cardiogenic shock.

Patients with pulmonary thromboembolism often decompensate suddenly, and, once hemodynamic compromise has developed, the mortality rate is extremely high. When the decision is made to use thrombolysis, the fastest-acting available thrombolytic agent with an acceptable safety and efficacy profile should be chosen. Many centers prefer off-label regimens to the slower on-label regimens that have been approved by the FDA.

UFH should not be given concomitantly with fibrinolytic therapy in acute massive pulmonary embolism. After fibrinolytic therapy, anticoagulation treatment is recommended to prevent recurrent thrombosis. Do not begin heparin until the aPTT has decreased to less than twice the normal control value.

In the worst clinical scenario, pulmonary embolism can cause cardiac arrest. The most common cardiac arrest initial rhythms documented include pulseless electrical activity and asystole. Cardiac arrest in the event of pulmonary embolism has a mortality of about 70%. Recently, numerous case reports state the use of thrombolytic boluses in cardiac arrest due to pulmonary embolism, with apparent heroic results. The clinician’s
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Main goal should focus on avoiding the cardiac arrest and identifying patient candidates for thrombolytic therapy in the event of a pulmonary embolism.

**Reteplase**

Reteplase has not been labeled by the FDA for any indication except acute MI, but it is widely used for acute deep vein thrombosis and pulmonary embolism. The dosing used is the same as that approved for patients with acute MI: 2 IV boluses of 10 U each, administered 30 minutes apart.

**Alteplase**

The FDA-approved regimen for pulmonary thromboembolism is 100 mg as a continuous infusion over 2 hours.

First, administer 15-mg bolus followed by 85 mg over a 2-hour infusion. Heparin drip must be discontinued during alteplase infusion.

Some centers prefer to use an accelerated 90-minute regimen that appears to be faster acting, safer, and more efficacious than the 2-hour infusion. For patients weighing less than 67 kg, the drug is administered as 15 mg IV bolus, followed by 0.75 mg/kg infused over the next 30 minutes (maximum 50 mg) and then 0.50 mg/kg over the next 60 minutes (maximum 35 mg). For patients weighing more than 67 kg, 100 mg is administered as 15 mg IV bolus, followed by 50 mg infused over the next 30 minutes and then 35 mg infused over the next 60 minutes.

**Urokinase**

The FDA-approved regimen is 4,400 U/kg as a loading dose given at a rate of 90 mL/h over a period of 10 minutes, followed by a continuous infusion of 4,400 U/kg/h at a rate of 15 mL/h for 12-24 hours.

**Streptokinase**

The FDA-approved regimen for pulmonary embolism is 250,000 U as a loading dose over 30 minutes, followed by 100,000 U/h over 12-24 hours.

**Thrombolytic Therapy for Deep Vein Thrombosis**

Deep vein thrombosis (DVT) occurs when clots form in the extremities. If pieces of these clots break off and travel to the lungs, pulmonary embolism can occur. The annual incidence of venous thromboembolism (VTE) in the United States is 600,000 cases. Early diagnosis and treatment is crucial to prevent morbidity and mortality. Death from DVT is attributed to massive pulmonary embolism.

The mainstay of initial treatment for DVT is anticoagulation. In selected patients with extensive acute proximal DVT (eg, iliofemoral DVT, upper extremity DVT, symptoms <14 d, good functional status, life expectancy >1 y) with low risk of bleeding, catheter-directed thrombolysis (CDT) may be used to reduce symptoms and post thrombotic morbidity if appropriate resources are available.

Catheter-directed thrombolysis is performed under imaging guidance; the procedure delivers thrombolytic directly to the clot through a catheter inserted in the vein. Intraclot injection of the thrombus with a fibrin-specific thrombolytic agent such as alteplase is an alternative to continuous-infusion and minimizes the duration of systemic exposure to thrombolytic agents.
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Systemic thrombolytic therapy is reserved for selected patients with extensive proximal DVT (eg, symptoms <14 d, good functional status, life expectancy >1 y) who have a low risk of bleeding, to reduce post thrombotic morbidity if catheter-directed thrombolysis is not available.\textsuperscript{15}

**Reteplase**

Non–FDA-approved indication for DVT for lysis of venous thrombus, a catheter-directed infusion of 1 U/h is maintained for 18-36 hours.

**Alteplase**

For lysis of venous thrombus, a catheter-directed infusion of 1-1.5 mg/h for 12-24 hours has been used, it depends on local expertise.

**Urokinase**

The usual systemic dose for deep venous thrombosis is 4,400 U/kg as an IV bolus, followed by a maintenance drip of 4,400 U/kg/h. The drip is continued for 1-3 days, until clinical or laboratory investigations demonstrate thrombus resolution. When available, intrathrombus delivery of urokinase can avoid a systemic lytic state. The dose for this route of administration is a loading dose of 250,000 U IV, followed by an infusion of 500 U/kg/h. If clot lysis is inadequate, the infusion rate can be increased gradually up to 2,000 U/kg/h.

**Streptokinase**

The usual dose regimen for deep venous thrombosis is an IV bolus of 250,000 U followed by a maintenance drip at 100,000 U/h. The drip is continued for 1-3 days, until clinical or laboratory investigation shows thrombus resolution.

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**Thrombolytic Therapy for Blocked Catheters**

*Central venous access devices* (CVADs) are an important component of chronic treatments that require ongoing venous access and regular maintenance. CVADs are subject to malfunctions, such as thrombotic occlusion with an incidence range from 2-40%. Risk factors include type of malignancy, chemotherapy, CVAD, insertion site, and catheter tip.\textsuperscript{20}

Thrombolytic therapy has reopened occluded catheters in 85-90% of episodes, and removal of the catheter is not usually required. Alteplase (tPA), urokinase, and streptokinase have all been used.

Streptokinase is not commonly used because of its antigenic properties and allergic reactions. Urokinase was used until 1999 when the FDA reported the potential for viral contamination and it was removed from distribution. It is available again but does not have an approved FDA indication for clearance of occluded catheters. A randomized trial comparing urokinase (10,000 U) with tPA (2 mg) suggested marked superiority for tPA.\textsuperscript{21}

**Alteplase**
FDA approved for thrombotically occluded CVADs. tPA is available in a 2 mg/2 mL vial, a volume sufficient to fill most catheter lumens. For patients ≥30 kg, 2 mg in 2 mL saline is used. Patients ≤30 kg, fill 100% of the internal lumen volume of the catheter, not to exceed 2 mg in 2 mL saline. It should dwell for 30 minutes to 2 hours and then be withdrawn. The dose may be repeated. If this is unsuccessful, an infusion of tPA 2 mg/50 mL infused over 4 hours may be used.

Urokinase

The dose of urokinase for catheter clearance is 5,000 U in each lumen over 1-2 minutes, leave in the lumen for 1-4 hours, then aspirate: may repeat with 10,000 U in each lumen if 5,000 U fails to clear the catheter. Volume to instill into catheter is equal to the volume of the catheter. For patients undergoing dialysis, 5,000 U is administered in each lumen over 1-2 minutes; leave in the lumen for 1-2 days, then aspirate.

Streptokinase

Slowly instill 250,000 U of streptokinase in 2 mL of solution into each occluded limb of the cannula. Clamp off the cannula limb(s) for 2 hours, and, after treatment, aspirate the contents of the cannula limb(s), flush with saline, and reconnect the cannula.

Thrombolytic Therapy for Acute Ischemic Stroke

Stroke is the leading cause of long-term disability and the third leading cause of death in the United States. Approximately 700,000 new cases occur annually in the United States, of which 85% are ischemic and the remainder are hemorrhagic. Among patients with ischemic strokes, 8-12% die within 30 days. Intravenous thrombolytic therapy for acute ischemic stroke is now generally accepted.

The US Food and Drug Administration (FDA) approved the use of intravenous tPA in 1996, partly on the basis of the results of the NINDS rtPA Stroke Study. The primary end point was neurological improvement at 24 hours. Favorable outcomes were achieved in 31-50% of patients treated with rtPA compared with 20-38% of patients given placebo. The major risk of treatment was symptomatic intracranial hemorrhage, which occurred in 6.4% of patients treated with rtPA and in 0.6% of patients given placebo.

Other intravenously administered thrombolytics agents have been considered for treatment of patients with acute ischemic stroke. Clinical trials of streptokinase were halted prematurely because high rates of hemorrhage; therefore, this agent should not be used. Tenecteplase appears promising as an effective thrombolytic agent with fewer bleeding complications, but future trials are needed to compare the effect of tenecteplase on neurological outcome and safety compared with tPA. Desmoteplase has been tested in a pilot study but was stopped because of lack of efficacy in the desmoteplase-treated patients.

Alteplase

Alteplase (tPA) is the only drug approved by the FDA for use in acute ischemic stroke with well-established time of symptom onset (<3 h). Currently, several clinical trials are running with third-generation thrombolytic drugs in order to evaluate their efficacy and safety in stroke.

Patients must arrive preferably to an institution with a stroke center. Time of symptom onset must be well established (<3 h), and the patient must be presenting with a measurable neurologic deficit. Stroke severity must be assessed with NIH stroke scale (maximum score 42). Patients with a score above 22 are considered
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High risk for hemorrhagic conversion due to the probability of a large infarcted area. Patients with a score less than 4 have only minor neurologic deficits, for which thrombolytic therapy is not indicated. High-risk patients often have early CT scan changes showing a large area of edema or mass effect.

Despite the increased risk of hemorrhage in patients with a massive stroke, fibrinolysis remains indicated whenever other exclusion criteria are absent, because the potential benefit is tremendous in this population of patients, who almost always have a dismal outcome if therapy is withheld. Inclusion and exclusion criteria must be reviewed before administration of thrombolytic. Be aware of subarachnoid hemorrhages that present early without CT scan findings.

**Contraindications for alteplase**

*Absolute contraindications*

- History or evidence of intracranial hemorrhage
- Clinical presentation suggestive of subarachnoid hemorrhage
- Known arteriovenous malformation
- Systolic blood pressure (SBP) >185 mm Hg or diastolic blood pressure (DBP) >110 mm Hg despite repeated measurements and treatment
- Seizure with postictal residual neurologic impairment
- Platelet count <100,000/mm$^3$
- Prothrombin time (PT) >15 or INR >1.7
- Active internal bleeding or acute trauma (fracture)
- Head trauma or stroke in the previous 3 months
- Arterial puncture at a noncompressible site within 1 week

*Relative contraindications*

- Suspected acute pericarditis
- Rapidly improving stroke symptoms
- Myocardial infarction in the previous 3 months
- Glucose level <50 mg/dL or >400 mg/dL

If no contraindications, start 2 peripheral IV lines, one for alteplase infusion and the second one to manage any complication that may occur. The recommended dose of alteplase for acute ischemic stroke is 0.9 mg/kg (maximum of 90 mg) infused over 60 minutes, with 10% of the total dose administered as an initial IV bolus over 1 minute.$^{32}$

The patient must be admitted to a critical care area in order to provide frequent neurologic assessments and blood pressure and cardiovascular monitoring. The clinician must be ready to recognize and manage possible complications mentioned below. The effectiveness of thrombolytic therapy in stroke is strictly associated to strict patient selection within the inclusion and exclusion criteria.

No adjunctive therapies should be given with alteplase for the management of acute ischemic stroke. Anticoagulants and antiplatelet agents may increase the risk of bleeding complications and are not recommended within 24 hours of alteplase administration.
Alteplase is a safe and effective treatment for carefully selected stroke patients presenting within 3 hours of symptoms onset.\textsuperscript{23,27,28} The benefit is higher if tPA is given earlier and is attributed to rescuing the area of ischemic penumbra. Although risks are associated with its use, these risks, in appropriate patients, do not outweigh the benefits.

A recent randomized study evaluated the efficacy and safety of alteplase administered between 3 and 4.5 hours after the onset of an ischemic stroke in more than 800 patients. The primary end point was disability at 90 days, and the secondary end point was global outcome analysis of four neurologic and disability scores combined.\textsuperscript{30}

In this study, intravenous alteplase given 3-4.5 hours after the onset of symptoms significantly improved clinical outcomes in patients with acute ischemic stroke. The incidence of intracranial hemorrhage was higher in the alteplase group than in the placebo group, but mortality rates did not differ between the groups.\textsuperscript{30} Alteplase remains safe when given at 3-4.5 hours after ischemic stroke, offering an opportunity for patients who cannot be treated within the standard 3-hour time frame. Early treatment remains essential, and patients should be treated with tPA as soon as possible to maximize the benefit. The 3-hour time window for alteplase is based on findings from the NINDS trials, which reported that the benefits of tPA decrease as the duration of occlusion increases.\textsuperscript{23}

An alternative regimen to systemic thrombolysis is to give a lower dose of local intra-arterial thrombolysis. No drugs currently are approved by the FDA for intra-arterial treatment of acute ischemic stroke, and such therapy is not standard. Intra-arterial thrombolysis is an option for treatment of selected patients who can be treated within 3-6 hours after symptoms onset due to occlusion of the middle cerebral artery (MCA) and who are not otherwise candidates for intravenous tPA.\textsuperscript{27,28}

**Thrombolytic Therapy for Peripheral Arterial Disease**

Peripheral arterial disease (PAD) is a common manifestation of atherosclerosis and may present as an obstruction of arterial blood flow to an extremity.

Low-dose intra-arterial thrombolytic therapy is being used for acute arterial occlusions. Primary fibrinolysis is the initial treatment of choice for many patients with acute peripheral arterial occlusions. The ability to perform catheter-directed thrombolysis with subsequent angioplasty and stenting has reduced the need for arterial surgery in many settings.

Patients with limb-threatening ischemia are not candidates for local fibrinolysis. Usually, it takes between 6 and 72 hours to achieve clot lysis. These patients require emergent embolectomy. Catheter-directed thrombolysis is reserved for patients with non–life-threatening limb ischemia due to in situ thrombosis of less than 14 days.\textsuperscript{31,32,33,34} Consider that patients with thrombosis for more than 30 days are not likely to respond to local fibrinolysis.

Initially, streptokinase was the most widely used agent but later was replaced by urokinase and alteplase (tPA). Other drugs that have been studied include prourokinase (not currently available), reteplase, and tenecteplase. The optimal dosage and concentration of reteplase, alteplase, and tenecteplase are still under investigation.\textsuperscript{34}

**Reteplase**

0.5 U/h by intra-arterial infusion
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Alteplase

Standard regimen: 0.05-0.1 mg/kg/h intra-arterially

High-dose regimen: 3 doses of 5 mg over 30 min, then 3.5 mg/h for up to 4 h

Urokinase

4,000 U/min until initial recanalization, then 1,000-2,000 U/min until complete lysis, all given intra-arterially

Streptokinase

5,000-10,000 U/h intra-arterially

Thrombolytic Therapy Complications

Complications of thrombolytic therapy include hemorrhage, allergic reactions, embolism, stroke, and reperfusion arrhythmias, among others. Clinicians must be prepared to handle such complications in a timely manner. The most feared complication of fibrinolysis is intracranial hemorrhage, but serious hemorrhagic complications can occur from bleeding at any site in the body.

Risk factors for hemorrhagic complications include increasing age, elevated pulse pressure, uncontrolled hypertension, recent stroke or surgery, the presence of a bleeding diathesis, and severe congestive heart failure.

Overdoses of fibrinolytic agents can cause severe hemorrhagic complications. Overdose most often occurs when a full dose of a fibrinolytic agent is given to a small patient with a low body weight.

In patients receiving fibrinolysis for AMI, the overall incidence of hemorrhagic complications is about 10%, and the incidence of intracranial hemorrhage is about 0.8%. In patients receiving fibrinolysis for acute ischemic stroke, the incidence of intracranial hemorrhage is higher, approximately 6%.

Patients receiving thrombolytic therapy for acute ischemic stroke must have constant neurologic and cardiovascular reevaluation. Blood pressure checks must be every 15 minutes for 2 hours, then every 30 minutes for 6 hours and finally every hour for 16 hours. Strict blood pressure monitoring is essential during and after thrombolytic treatment in order to prevent complications. If a patient has signs of neurologic deterioration, stop thrombolytic therapy and obtain an emergent CT scan. Consider immediate expert consultation.

If a patient who was treated with fibrinolytic medications develops serious bleeding complications, the first step is cessation of the fibrinolytic agent and any anticoagulation therapy. Supportive therapy should be instituted. This often includes volume repletion and transfusion of blood factors. When possible, direct pressure should be used to control bleeding. If the patient has also been receiving heparin, protamine sulfate may be used to reverse the heparin effect.

Aminocaproic acid (Amicar) is a specific antidote to fibrinolytic agents. In adults, 4-5 g of aminocaproic acid in 250 mL of diluent is administered by infusion during the first hour of treatment, followed by a continuing infusion at the rate of 4 mL (1 g) per hour in 50 mL of diluent. The infusion would be continued for about 8 hours or until
the bleeding situation has been controlled.  Fresh frozen plasma and/or cryoprecipitate may be used to replenish fibrin and clotting factors.

Aminocaproic acid should not be given unless hemorrhage is life threatening, because it inhibits intrinsic fibrinolytic activity and can precipitate runaway thrombosis with end-organ damage at many sites. The drug worsens disseminated intravascular coagulation, including that associated with heparin-induced thrombocytopenia.

**Thrombolytic Therapy in Cardiac Arrest**

Several reports of adult patients have documented successful resuscitation after thrombolytic administration during cardiac arrest. In most case reports, acute pulmonary embolism or acute myocardial infarction were the suspected cause; these patients failed initially to standard CPR guidelines and ACLS protocols.  Active CPR is clearly not a contraindication for thrombolytic therapy. Today, evidence to support the routine use of thrombolytic drugs during cardiac arrest is not sufficient. The clinician may consider it on a case-by-case basis.

http://medscape.com/

**Topic: Anatomy and Physiology**

**Notable Content added:**

**Understanding of the normal values of CBC**

Topic: Understanding of the normal values of CBC (15 minutes didactic; 0 minutes laboratory)

Comment: no problem (Requires no additional specialty training)

**Cognitive Objective:**

Recognize the abnormal blood values.
Understand the pathophysiology of the abnormal values.

**Psychomotor Objectives:**

Given a simulated patient demonstrate the understanding of normal blood values

Declarative (EMS Educational Standards)

**XII. Circulatory**

**A. Blood**

1. The paramedic should have an understanding of blood values mean along with knowledge of the normal values in a complete blood count-

2. A major portion of the complete blood count is
   a. white blood cells
   b. red blood cells
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c. platelets

3. Normal values include:
   a. **White blood cell count (WBC)**
      Normal 4,300 and 10,800 cells per cubic millimeter (cmm)
      a. granulocytes
      b. lymphocytes
      c. Monocytes
      d. Eosinophils
      e. Basophils
   
   b. **Red cell count (RBC)**
      Normal 4.2 to 5.9 million cells/cmm
   
   c. **Hemoglobin (Hb).**
      Normal Men 13 to 18 grams per deciliter
      Normal Women 12 to 16 grams per deciliter
   
   d. **Hematocrit (Hct).**
      Normal Men 45% to 52%
      Normal Women 37% to 48%
   
   e. **Platelet count.**
      Normal range varies slightly between laboratories but is in the range of
      150,000 to 400,000/ cmm (150 to 400 x 109/liter)

**Topic: Blood Chemistry**

There was a question within the Training Committee about the purpose for Blood Chemistry knowledge for paramedics as they would not be doing any in the field. Danny Bercher contacted Kenny Navarro, Assistant Professor, University of Texas Southwestern Medical Center, who had been on the committee that developed the Gap Analysis document that the Arkansas EMS Training Committee worked from. The question was what was intended with covering “Blood Chemistry analysis” in the educational gap. His reply was:

> With respect to blood chemistry, we primarily meant that medics would need to know the purpose, indications, limitations and procedure for using a 1. Glucometer and 2. Cardiac biomarkers (point of care tests)

> We also intended for medics to have enough cognitive knowledge to understand other blood analyses such as 1. CHEM-7 2. BNP 3. Arterial blood gases (ABGs) even though they might not ever perform the tests in the field.
Finally, in order to meet the standard in the future, training programs should include any additional monitoring devices that are recognized as the "standard of care" in the out-of-hospital setting.

**Time should take around 2 hours for all**

- Cognitive Objective: Understanding of the meaning of lab results from a Chem. 7 test
- Cognitive Objective: Understanding of the meaning of the Blood gas analysis
- Cognitive objective: Understanding of the BNP (B-type Natriuretic Peptide)
- Cognitive Objective: Understanding of Cardiac Markers
Normal Values: Chem. 7

A battery of blood chemistry tests; the seven parts of a Chem 7 are sodium, potassium, chloride, bicarbonate, blood urea nitrogen (BUN), creatinine, and glucose.

Definition

CHEM-7 is a group of blood tests that provides information about your body's metabolism. The test is commonly called a basic metabolic panel.

The measured chemicals with their normal reference intervals are:

- **BUN** (blood urea nitrogen): 7 to 20 mg/dl
- **Serum chloride**: 101 to 111 mmol/L
- **CO₂ (carbon dioxide)**: 20 to 29 mmol/L
- **Creatinine**: .8 to 1.4 mg/dl
- **Glucose test**: 64 to 128 mg/dl
- **Serum potassium**: 3.7 to 5.2 mEq/L
- **Serum sodium**: 136 to 144 mEq/L

Key to abbreviations:

- L = liter
- dl = deciliter = 0.1 liter
- mg = milligram
- mmol = millimole
- mEq = milliequivalents

What abnormal results mean:

See the individual tests.

What the risks are:
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- Excessive bleeding
- Fainting or feeling light-headed
- Hematoma (blood accumulating under the skin)
- Infection (a slight risk any time the skin is broken)
- Multiple punctures to locate veins

**BUN**  This test determines the level of urea nitrogen in the blood.

Urea nitrogen is produced when proteins are metabolized or broken down. Elevated levels of blood urea nitrogen (BUN) can be a sign of kidney disease, liver disease or dehydration.

Normal values for the total amount of chloride in the blood range from 98 to 106 mEq/L (milliequivalents per liter).

Chloride can become too high because of conditions including:

- Dehydration
- Conditions causing excessive urination
- Severe vomiting or diarrhea
- Severe burns

Many things can cause the chloride to become too low including:

- Diuretics, also called water pills
- Kidney disease
- Uncontrolled diabetes
- Congestive heart failure
- Cirrhosis of the liver
- Very high protein, triglycerides, or glucose in the blood

The normal value for CO2 in the blood is 23 to 29 mEq/L. Most of the carbon dioxide in the blood is in the form of bicarbonate. Either term can be used. The lungs and kidneys control the amount of bicarbonate in the blood. This keeps the blood from becoming too acidic. In order for a person to live, he or she must have neither too much acid nor too much base in the blood.
Carbon dioxide or bicarbonate can be **too high** or **too low** in the following conditions:

- Kidney problems
- Breathing or lung problems
- Poisonings or drug overdoses
- Severe diarrhea
- Uncontrolled diabetes
- Severe dehydration

**What do the test results mean?**

The normal level of creatinine varies slightly based on age, body size, and sex. The level also changes during pregnancy. However, the healthy range is usually between 0.5 and 1.4 milligrams per deciliter (mg/dL).

A decreased value for this test is rarely a concern. It can occur with decreased muscle mass, such as in elderly people. Conditions such as muscular dystrophy, which is an inherited defect in muscles, can cause a low value for this test. Pregnancy may also cause a low value.

A high value for this test can occur for many reasons. Some of these reasons are described in the sections that follow.

**Decreased blood flow to the kidneys**

The blood flow to the kidneys can be decreased by the following conditions or events.

- severe dehydration
- massive blood loss
- congestive heart failure
- blockage in the kidney arteries, called renal artery stenosis

**Kidney damage or failure**
The kidneys can be damaged by a number of conditions, including:

- high blood pressure
- diabetes
- an inherited condition called polycystic kidney disease
- a high blood calcium level, called hypercalcemia
- a cancer called multiple myeloma
- autoimmune diseases, which are conditions in which a person's immune system attacks his or her own body

Other conditions can also cause an elevated value for this test:

- blockage of urine flow, which commonly occurs with enlargement of the prostate, also called benign prostatic hyperplasia
- taking certain medicines, such as captopril or non-steroidal anti-inflammatory drugs, which are called NSAIDs

What do the test results mean?

The normal blood glucose levels also vary depending on:

- which test was performed
- whether a person was fasting before the test
- whether any special dietary or glucose substances were given during testing

Increased levels of blood glucose, a condition known as hyperglycemia, may be caused by the following:

- acromegaly, a condition that causes elongation of the bones of the limbs and head
- Cushing syndrome, a condition in which the level of the hormone cortisol is too high and causes fatigue, weakness, protein loss, swelling, and diabetes mellitus, which is also called DM
- diabetes mellitus
- diuretics, also known as water pills
- gestational diabetes, or diabetes that develops during pregnancy
- inadequate therapy for diabetes mellitus
• infection in the pancreas, known as pancreatitis
• kidney failure, such as chronic renal failure
• liver disease, such as cirrhosis
• pheochromocytoma, which is a noncancerous tumor that causes an increase in certain chemicals that can cause high blood pressure
• steroid medicines, such as prednisone
• stress response, including infection, severe burns, or surgery

Decreased levels of blood glucose, a condition known as hypoglycemia, may be caused by the following:

• Addison disease, a condition in which there is a decreased amount of the adrenocorticol hormone
• blood loss
• extensive liver disease
• hypopituitarism, a condition in which the pituitary gland does not release enough hormone
• hypothyroidism, a condition in which too little thyroid hormone is present in the blood
• insulin overdose
• insulinoma, which is a tumor in the pancreas that causes too much insulin to be produced
• malabsorption, or inadequate absorption of nutrients from the stomach or intestines
• starvation

What do the test results mean?

The normal value for potassium in the blood is 3.5 to 5.1 mEq/liter. The potassium has to remain in this range for the heart to function properly.

Potassium can become too high because of conditions including:

• Kidney problems
• Dehydration
• Technical problems with the blood specimen
Many things can cause the potassium to become too low including:

- Diuretics, also called water pills
- Severe vomiting or diarrhea
- Cirrhosis of the liver

**What do the test results mean?**

The normal value for sodium in the blood is 136 to 146 mEq/liter. Sodium must stay in this range for all the cells in the body to function properly.

Sodium can become too high because of conditions including:

- Dehydration
- Conditions causing excessive urination
- Severe vomiting or diarrhea
- Severe burns

Many things can cause the sodium to become too low including:

- Diuretics, also called water pills
- Kidney disease
- Uncontrolled diabetes
- Congestive heart failure
- Cirrhosis of the liver
- Very high protein, triglycerides, or glucose in the blood

**Cardiac**

What do the test results mean?

If a heart attack has taken place, the amount of troponin I in the blood will start to rise within the first 4 to 6 hours. Almost half of the time, it will be elevated before the fourth hour. This blood test usually becomes positive earlier than other blood tests that are used to diagnose a heart attack. This test is very important to ensure the earliest treatment of a heart attack. The normal value for troponin I is less than 0.7 mg/ml.

**What do the test results mean?**
Normal levels of CPK in the blood range from about 35 to 190 units per liter.

Abnormally high levels of CPK may indicate the following.

- alcoholism
- brain trauma
- convulsions or seizures
- delirium tremens, or severe withdrawal from alcohol
- dermatomyositis, a disease in which the immune system attacks the muscles and the skin
- electric shock
- heart attack

- muscular dystrophy
- polymyositis, a disease in which the immune system attacks the muscles
- lung damage
- severe muscle breakdown from prolonged or strenuous exercise

Blood Gas Analysis

Definition
Blood gas analysis, also called arterial blood gas (ABG) analysis, is a test which measures the amounts of oxygen and carbon dioxide in the blood, as well as the acidity (pH) of the blood.

Purpose
An ABG analysis evaluates how effectively the lungs are delivering oxygen to the blood and how efficiently they are eliminating carbon dioxide from it. The test also indicates how well the lungs and kidneys are interacting to maintain normal blood pH (acid-base balance). Blood gas studies are usually done to assess respiratory disease and other conditions that may affect the lungs, and to manage patients receiving oxygen therapy (respiratory therapy). In addition, the acid-base component of the test provides information on kidney function.

Description
Blood gas analysis is performed on blood from an artery. It measures the partial pressures of oxygen and carbon dioxide in the blood, as well as oxygen content, oxygen saturation, bicarbonate content, and blood pH.
Oxygen in the lungs is carried to the tissues through the bloodstream, but only a small amount of this oxygen can actually dissolve in arterial blood. How much dissolves depends on the partial pressure of the oxygen (the pressure that the gas exerts on the walls of the arteries). Therefore, testing the partial pressure of oxygen is actually measuring how much oxygen the lungs are delivering to the blood. Carbon dioxide is released into the blood as a by-product of cell metabolism. The partial carbon dioxide pressure indicates how well the lungs are eliminating this carbon dioxide.

The remainder of oxygen that is not dissolved in the blood combines with hemoglobin, a protein—iron compound found in the red blood cells. The oxygen content measurement in an ABG analysis indicates how much oxygen is combined with the hemoglobin. A related value is the oxygen saturation, which compares the amount of oxygen actually combined with hemoglobin to the total amount of oxygen that the hemoglobin is capable of combining with.

**Key terms**

**Acid-base balance** — The condition that exists when the body's carbonic acid-bicarbonate buffer system is in equilibrium, helping to maintain the blood pH at a normal level of 7.35-7.45.

**Hemoglobin** — A protein—iron compound in red blood cells that functions primarily in carrying oxygen from the lungs to the tissues of the body.

**pH** — A measure of the acidity of a solution. Normal blood pH ranges from 7.35-7.45.

Carbon dioxide dissolves more readily in the blood than oxygen does, primarily forming bicarbonate and smaller amounts of carbonic acid. When present in normal amounts, the ratio of carbonic acid to bicarbonate creates an acid-base balance in the blood, helping to keep the pH at a level where the body's cellular functions are most efficient. The lungs and kidneys both participate in maintaining the carbonic acid-bicarbonate balance. The lungs control the carbonic acid level and the kidneys regulate the bicarbonate. If either organ is not functioning properly, an acid-base imbalance can result. Determination of bicarbonate and pH levels, then, aids in diagnosing the cause of abnormal blood gas values.

**The procedure**

The blood sample is obtained by arterial puncture (usually in the wrist, although it could be in the groin or arm) or from an arterial line already in place. If a puncture is needed, the skin over the artery is cleaned with an antiseptic. A technician then collects the blood with a small sterile needle attached to a disposable syringe. The patient may feel a brief throbbing or cramping at the site of the puncture. After the blood is drawn, the sample must be transported to the laboratory as soon as possible for analysis.
Preparation
There are no special preparations. Patients have no restrictions on drinking or eating before the test. If the patient is receiving oxygen, the oxygen concentration must remain the same for 20 minutes before the test; if the test is to be taken without oxygen, the gas must be turned off for 20 minutes before the test is taken. The patient should breathe normally during the test.

Aftercare
After the blood has been taken, the technician or the patient applies pressure to the puncture site for 10-15 minutes to stop the bleeding, and then places a dressing over the puncture. The patient should rest quietly while applying the pressure to the puncture site. Health care workers will observe the patient for signs of bleeding or circulation problems.

Risks
Risks are very low when the test is done correctly. Risks include bleeding or bruising at the site, or delayed bleeding from the site. Very rarely, there may be a problem with circulation in the puncture area.

Normal results
Normal blood gas values are as follows:

- partial pressure of oxygen (PaO₂): 75-100 mm Hg
- partial pressure of carbon dioxide (PaCO₂): 35-45 mm Hg
- oxygen content (O₂CT): 15-23%
- oxygen saturation (SaO₂): 94-100%
- bicarbonate (HCO₃): 22-26 mEq/liter
- pH: 7.35-7.45

Abnormal results
Values that differ from those listed above may indicate respiratory, metabolic, or kidney disease. These results also may be abnormal if the patient has experienced trauma that may affect breathing (especially head and neck injuries). Disorders, such as anemia, that affect the oxygen-carrying capacity of blood, can produce an abnormally low oxygen content value.

BNP
BNP assay is a 15-minute blood test that is highly sensitive and fairly specific for diagnosing heart failure and is useful in evaluating suspected heart failure in outpatients and in emergency rooms. Other uses include screening for left ventricular dysfunction and predicting outcomes in patients with an established diagnosis of heart failure or myocardial infarction. BNP, a cardiac
Educational recommendations for “filling the gap”
Between old DOT NSC and new EMS Education Standards

neurohormone, was first discovered in the brain of pigs. In humans, the main source of BNP is the ventricles of the heart. BNP is secreted in response to increased ventricular volume and pressure. Circulating BNP levels increase in proportion to the severity of the disorder, and is detectable with minimal clinical symptoms. With a negative predictive value of greater than 95%, a normal BNP level can help exclude heart failure from other causes.

The natriuretic peptides, which include atrial natriuretic peptide (ANP) and BNP, help regulate blood pressure and fluid balance by counter balancing the renin angiotensin system. Whereas renin and angiotensin raise blood pressure, decrease urine output and cause vasoconstriction, the natriuretic peptides have the opposite effects. Both ANP and BNP increase excretion of sodium and water by increasing glomerular filtration and inhibiting renal sodium resorption (4). They also decrease secretion of aldosterone and renin and cause vasodilatation, reducing blood pressure and extracellular fluid volume.

B-type natriuretic peptide (BNP) is a 32 amino acid polypeptide containing a 17 amino acid ring structure common to all natriuretic peptides (5). Unlike A-type (ANP), whose major storage sites include both the atria and ventricles, the major source of plasma BNP is cardiac ventricles, suggesting that BNP may be a more sensitive and specific indicator of ventricular disorders than other natriuretic peptides (6). Patients with symptomatic left ventricular systolic dysfunction have increased BNP levels that correlate with New York Heart Association classification (Table 1). BNP is an independent, significant predictor of high left ventricular end-diastolic pressure in patients with CHF.

Cardiac markers
Cardiac biomarkers should be measured in all patients who present with chest discomfort consistent with acute coronary syndrome (ACS).

- Cardiac Troponins T and I are the preferred markers for myocardial injury because they are more sensitive and more specific than the creatine kinase MB isoenzyme (CK-MB), aspartate aminotransferase (AST) or lactate dehydrogenase (LAD), which have much lower specificity to cardiac muscle.
- Patients with negative cardiac biomarkers within 6 hours of the onset of symptoms that are consistent with ACS should have biomarkers re-measured in the time frame of 8 to 12 hours after the onset of symptoms.¹
- Elevations of cardiac enzyme levels should be interpreted in the context of clinical and ECG findings.
- Peaks circulating enzyme levels tend to occur earlier and are often higher following successful thrombolytic therapy.

Indications for measurement of cardiac enzymes
Any patient presenting with a possible acute coronary syndrome
- Routinely following **percutaneous coronary intervention (PCI)**
- Routinely following surgical revascularisation (**CABG**)

**Troponins T and I**

- Troponin is a contractile protein that normally is not found in serum. It is released only when myocardial necrosis occurs.\(^2\)
- Cardiac troponins T and I are highly sensitive and specific for cardiac damage. Troponin I and T are of equal clinical value.\(^3\)
- Serum levels increase within 3-12 hours from the onset of chest pain, peak at 24-48 hours, and return to baseline over 5-14 days.
- Troponin levels may not be detectable for six hours after the onset of myocardial cell injury. The most sensitive early marker for myocardial infarction is CK-MB followed by myoglobin.
- Troponin levels should be measured at presentation and again twelve hours after the onset of symptoms. When there is uncertainty regarding the time of symptom onset, troponin should be measured at twelve hours after the presentation.\(^3\)
- The risk of death from an acute coronary syndrome is directly related to troponin level and patients with no detectable troponins have a good short-term prognosis.
- Elevated troponin levels can occur in patients without an acute coronary syndrome and are associated with adverse outcomes in many other clinical situations, including **congestive heart failure**, sepsis, acute **pulmonary embolism** and **chronic renal failure**.\(^3\)

**Creatine kinase**

- Myocardial muscle creatine kinase (CK-MB) is found mainly in the heart.
- CK-MB levels increase within 3-12 hours of onset of chest pain, reach peak values within 24 hours, and return to baseline after 48-72 hours.
- Sensitivity and specificity are not as high as for troponin levels.

**Lactate dehydrogenase**

- Serum lactate dehydrogenase (LAD) level rises above the reference range within 24 hours of a myocardial infarction, reaches a peak within 3-6 days, and returns to the baseline within 8-12 days.

**Myoglobin levels**

- Myoglobin is found in cardiac and skeletal muscle.
It is released more rapidly from infarcted myocardium than troponin and CK-MB and may be detected as early as 2 hours after an acute myocardial infarction.

Myoglobin has high sensitivity but poor specificity. It may be useful for the early detection of myocardial infarction.

**Natriuretic peptides**

Studies in several types of acute coronary syndromes have shown that elevated levels of natriuretic peptides, e.g. B-type natriuretic peptide (BNP), are independently associated with adverse outcomes, especially mortality.4

**Other blood test findings following myocardial infarction**

**Leucocytosis** may be seen within several hours after an acute myocardial infarction. It peaks in 2-4 days and returns to normal levels within 1 week.

- Patients without biochemical evidence of myocardial necrosis but with elevated **CRP** level are at increased risk of a subsequent ischemic event.
- **Erythrocyte sedimentation rate** (ESR) rises above reference range values within 3 days and may remain elevated for weeks.

- California Association for Medical Laboratory Technology Distance Learning Program
- **American College of Cardiology, American Heart Association**; Guidelines for the management of patients with unstable angina/non ST-Elevation myocardial infarction. 2007.