Introduction

Goals and Protocols
Introduction

• 12 Lead is Standard of Care

• State recognizes need for all EMS to acquire
  • EMT application and transmission
  • Grant for equipment
Acute Myocardial Infarction (AMI)

• Definition:

Death of Heart Tissue due to Obstruction of Blood Flow
AMI - Critical Concept

- AMI is not a single event
- It is a rapidly evolving process

The Goal:
Identify, Intervene, and STOP HEART MUSCLE DEATH
Prehospital Issues

- 1.2 million Americans experience AMI annually
- 400,000 die in the field (ER)
- Most deaths are arrhythmic
- EMS must act with urgency
Deaths from coronary heart disease

Civilization kills. Since 1990, more people have died from coronary heart disease than from any other cause. Unlike stroke, coronary heart disease is a comparable newwonder on the world stage. Variations in death rates are marked: they are lower in populations with short life expectancy.

Heart disease mortality rates are also affected by differences between countries on the major risk factors, especially blood pressure, blood cholesterol, smoking, physical activity and diet. While genetic factors play a part, 80% to 90% of people dying from coronary heart disease have one or more major risk factors that are influenced by lifestyle.

Deaths from coronary heart disease have decreased in North America and many western European countries. This decline has been due to improved prevention, diagnosis, and treatment, in particular reduced cigarette smoking among adults, and lower average levels of blood pressure and blood cholesterol. It is expected that 80% of the future increase in coronary heart disease mortality will occur in developing countries.

Of all coronary heart disease patients who die within 38 days after the onset of symptoms, about two-thirds die before reaching hospital. This highlights not only the need for early recognition of the warning signs of a heart attack, but also the need for prevention.

Despite improvements in survival rates, in the United States one in 4 men and one in 3 women still die within a year of a recent acute heart attack.

Coronary heart disease is now the leading cause of death worldwide. It is on the rise and has become a true pandemic that respects no borders.

Deaths from coronary heart disease compared with other causes
Number of deaths of people aged 15 to 69 years, and 60 years and over 2000

[Graph showing decrease in death rates]

Change of heart
Percentage change in coronary heart disease death rates, in people aged 35 to 74 years, 1989-1998

[Graph showing changes in death rates]
1. Types of cardiovascular disease

The human heart is the size of a fist, but it is the strongest muscle in the human body. The heart starts to beat in the uterus long before birth, usually by 7 to 8 weeks after conception. The average heart beats about 100,000 times daily or about two and a half billion times over a 70-year lifetime.

With every heartbeat, the heart pumps blood around the body. It beats approximately 70 times a minute, although this rate can double during exercise or at times of extreme emotion.

Blood is pumped out from the left chambers of the heart, it is transported through arteries of ever-decreasing size, finally reaching the capillaries in all the tissues, such as the skin and other body organs. Having delivered its oxygen and nutrients and having collected waste products, blood is brought back to the right chambers of the heart through a system of ever-enlarging veins. During the circulation through the liver, waste products are removed.

This remarkable system is vulnerable to breakdown and assault from a variety of factors, many of which can be prevented and treated. Risk factors will be explored on pages 24–43.

Global deaths from CVD

- 8–4 years
- 5–14 years
- 15–29 years
- 30–49 years
- 50–69 years
- 70+ years

Heart disease statistics

- 11.8 million
- 16.5 million
- 22.6 million

2. Coronary heart disease

Disease of the blood vessels supplying the heart muscle.

- Major risk factors: high blood pressure, high blood cholesterol, tobacco use, unhealthy diet, physical inactivity, diabetes, advancing age, and genetic predisposition.
- Other risk factors: smoking, obesity, metabolic syndrome, depression, inflammation, and blood clotting disorders.

3. Rheumatic heart disease

Damage to the heart muscle and heart valves from rheumatic fever, caused by streptococcal bacteria.

4. Congenital heart disease

Malformations of heart structures that are present at birth. They may be caused by genetic factors or by adverse exposures during gestation.

5. Other cardiovascular diseases

- Tumours of the heart: vascular tumours of the brain, disorders of heart valves (cardiomyopathy); heart valve disease; disorders of the lining of the heart.

6. Other factors that can damage the heart and blood vessel system

- Inflammation, drugs, high blood pressure, unhealthy diet, trauma, toxins, and alcohol.

7. Stroke

Strokes are caused by obstruction of the blood supply to the brain. This may result from either blockage (ischaemic stroke) or rupture of a blood vessel (haemorrhagic stroke).

- Major risk factors: high blood pressure, atrial fibrillation, history of heart disease, high blood cholesterol, tobacco use, unhealthy diet, physical inactivity, diabetes, advancing age, and genetic predisposition.
- Other risk factors: smoking, obesity, metabolic syndrome, depression, inflammation, and blood clotting disorders.

8. Aortic aneurysm and dissection

Dilation and rupture of the aorta.

9. Peripheral arterial disease

Disease of the arteries supplying the arms and legs.

10. Deep venous thrombosis (DVT) and pulmonary embolism

Blood clots in the legs, veins, which can dislodge and move to the heart and lungs.

11. Other factors

- Surgery, obesity, cancer, previous episode of DVT, recent childbirth, use of oral contraceptives and hormone replacement therapy, long periods of immobility, for example while travelling, high homocysteine levels in the blood.
Acute Myocardial Infarction (AMI)

*Prehospital Standard of Care -*

- Patient Assessment
- 12 Lead ECG Screening
- PCI/Fibrinolysis Screening
Standard of Care Rationale

- Leading cause of death: Coronary Disease
- Cause of most AMIs: Thrombus
- Reduce time to Rx: Prehospital ECG
- Early Rx: (PCI/Fibrinolysis) Saves Lives

“TIME IS MUSCLE”
The Prehospital ECG in AMI

Although adds Prehospital time:

- Shortens in-hospital treatment time
- Patient’s more likely to receive Fibrinolytics or percutaneous coronary intervention (PCI)
- Significantly reduces patient mortality
Areas of Delay - National Heart Attack Alert Program

- Patient
- Prehospital
- Hospital

GOAL: 30 - 90 Minutes to Treatment
Cardiac Checklist - Key Components

- Focused History
- 12 Lead ECG
- Physical Examination
- Fibrinolytic Criteria
Cardiac Event Protocol

- EMS SYSTEM APPROACH THAT SHOULD ADDRESS:
  - Oxygen - IV - cardiac monitor - vital signs
  - Nitroglycerin
  - Aspirin
  - Pain relief with narcotics
  - Notification of emergency department
  - Rapid transport to emergency department
  - Prehospital screening for PCI or fibrinolytic therapy
  - 12-lead ECG, computer analysis, transmission:
    - to emergency department

- “DOOR-TO-REPERFUSION” TEAM PROTOCOL APPROACH
  - Rapid triage of patients with chest pain
  - Clinical decision maker established (emergency physician, cardiologist, or other)
**ASSESSMENT**

- **Immediate:**
  - Vital signs
  - Oxygen at 4 L/min-Sat<94%
  - Aspirin, if not given prior
  - Start IV
  - Nitroglycerin SL or spray
  - Morphine IV
  - 12-lead ECG (MD review)
  - Focused history & physical
  - Chest X-ray
  - Blood (electrolytes, cardiac markers, coagulation studies)

**TREATMENTS TO CONSIDER if there is evidence of coronary thrombosis plus no reasons for exclusion:**

(some but not all may be appropriate)

- Clopidogrel
- Nitroglycerin IV
- Beta Blockers IV
- Heparin IV
- Ace Inhibitors
- Statins
- Glycoprotein Inhibitors
- Consult as needed
- Decide eligibility for
  - PCI/Fibrinolysis
Pathophysiology of STEMI
Pathophysiology

- **AMI = Death of Heart Tissue**
  (an evolving process)
- **Coronary Occlusion:**
  atherosclerosis / thrombus
- **Mainly Left Ventricle (LV)**
- **Most of LV supplied by Left Coronary**
Risk Factors - Coronary Artery Disease

- Hyperlipidemia
- Age
- Male sex
- Tobacco use
- Alcohol use
- Hypertension
- Sedentary lifestyle / obesity
- Family history
- Diabetes mellitus
Coronary Arteries

- Right Coronary Artery
- Left Main Coronary Artery
- Circumflex
- Left Anterior Descending
Atherosclerosis (plaque formation)

The background for most infarctions:

ATHEROSCLEROTIC NARROWING
The immediate cause of most infarctions:

THROMBUS FORMATION
The Right Coronary Supplies

- Inferoposterior L.V.
- Posterior Septum
- Right Ventricle
- SA Node, AV Node
Right Coronary - Clinical Correlations

- Inferior/Posterior M.I.
- May Involve R.V.
- Bradycardia
- Heart-Block
  (often transient)
The Left Coronary Supplies

- **Left Anterior Descending**
  - Anterior L.V.
  - Anterior Septum
  - Bundles in Septum
- **Circumflex**
  - Posterolateral L.V.
Left Coronary - Clinical Correlations

- Anterior M.I.
- Bundle Branch Block
- Heart Block
- Posterolateral M.I.
The Conduction System

- **CLINICAL CORRELATIONS**

  - **Bundle Branch Blocks**
    - occur at different sites

  - **Complete Bundle Branch**
    - Blocks may obscure the
    - ECG findings of AMI
History & Physical Exam
The “Chest Pain” of AMI

**Classic:**
- Substantial to left arm
- More than 30 minutes

**Note:**
- Often not in the chest
- Often not described as pain
AMI “Chest Pain” Location
(Typically Diffuse)

Classic: Substernal

Epigastrium
Back
Jaw
Arms
Shoulders
Wrist
AMI “Chest Pain” Character

- Classic: Pressure / Tightness
- Heaviness
- Squeezing
- Aching
- Indigestion
- Distress
- Burning
- Discomfort
- Numbness
Additional History (SAMPLE)

Symptoms (other than pain)
Allergies
Medication
Past history
Last meal
Events prior to episode
Chest Pain Differential

- CARDIOVASCULAR
- PULMONARY
- GASTROINTESTINAL
- MUSCULOSKELETAL
- ANXIETY ATTACK
Chest Pain Differential

CARDIOVASCULAR -

• Infarction
• Angina Pectoris
• Dissecting Thoracic Aneurysm
• Pericarditis

** All effect ECG **
Chest Pain Differential

PULMONARY -

- Embolism
- Pneumothorax
- Pleurisy
- Pneumonia
Chest Pain Differential

GASTROINTESTINAL -

• Cholecystitis
• Pancreatitis
• Peptic Ulcer
• Esophageal
• Hiatal Hernia
Chest Pain Differential

**MUSCULOSKELETAL** -

- Cervical Disc
- Costochondritis

**ANXIETY ATTACK** - (Hyperventilation)
General Physical Exam
(“MACCS”imize)

Mental (AVPU)
Appearance (skin)
Chest (breath sounds)
Cardiovascular (head to toe)
Stroke (focal signs)
Cardiovascular Examination
(Head to Toe)

- Venous Distention
- Heart Sounds (S3, S4)
- Breath Sounds (rales)
- Abdomen (Mimics)
- Radial Pulse
- Peripheral Edema
Elevated Central Venous Pressure
> 15 cm

Normal = 7 cm or less

Sternal angle = 5 cm
Auscultatory Areas - Listen at Apex

- Normal: S1, S2
- Heart Failure: S3, S4
- Systolic murmur
- Pericarditis: rub
Breath Sounds for EMS

- **Vesicular:** Normal
- **Rales (crackles):** Pulmonary Edema
- **Wheezees (whistles):** Bronchospasm  
  “Cardiac asthma”
- **Friction rub:** Pleuritis  
  (like sandpaper)
Acute Myocardial Infarction
High Risk Profile Patients

- Tachycardia: HR > 100 bpm
- Hypotension: BP < 100 mm Hg
- Pulmonary Edema: rales > 1/2 way up
- Shock: BP < 90 and Pulmonary Edema
- Conduction Abnormalities - AV Block and Intraventricular (BBB)
12 Lead Essentials
Indications for 12 Lead ECG Include:

- “Chest Pain”
- Palpitations/Dysrhythmias
- Shortness of Breath
- Overdoses
- Syncope / Dizziness
- Impending Doom
- Unexplained Sweating or Nausea and Vomiting
CAUTIONS:

- First treat life threatening problems and chest pain
- Do not delay transport of critically ill patients
The 12 Lead ECG Includes

6 Limb Leads (Vertical Plane)
I, II, III, aVR, aVL, aVF

6 Limb Leads (Horizontal Plane)
V1 through V6
**Vertical and Horizontal Planes**

**Limb Leads (Vertical Plane)**

- aVR
- aVL
- aVF

**Chest Leads (Horizontal Plane)**

- V1
- V2
- V3
- V4
- V5
- V6

---

**Center of Heart**

- Zero point

---

**Lateral**

- Inferior

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**Anterior**

- Posterior
Complexes, Intervals, Segments

10.0 mm. = 1.0 mv.

5.0 mm. = 0.2 sec.

1.0 mm. = .04 sec.
Isoelectric Line (reference for ST elevation)
P Wave (atrial depolarization)

P - R Interval
QRS Complex (ventricular depolarization)
T (ventricular repolarization)
J Point  (junction between QRS and ST)

The J Point is not always identifiable
ST Segment  (read for injury - STEMI)
After Rate and Rhythm:

QRS - Is LBBB present?

If answer is no,

Read the ST segments.

ST - Is elevation $\geq 1$ mm. present in at least two contiguous leads?
12 lead application
Limb Lead Placement - 12 Lead ECG

Do Not Place On Torso
Arm Leads Reversed (Negative P in Lead I)
**Chest Electrode Placement - Specifics**

- **V1**: 4th Intercostal Space (ICS), Right Parasternal
- **V2**: 4th ICS, Left Parasternal
- **V3**: Between V2 and V4
- **V4**: 5th ICS, Midclavicular Line (must place prior to V3)
- **V5**: Level = V4, Anterior Axillary Line
- **V6**: Level = V4, Mid Axillary Line
Standardization

Correct (1 mV.)

Under (0.5 mV.)

Over (1.5 mV.)
ECG Changes of STEMI
AMI - ECG Messages

- Remember inferior and anterior
- Worry most about anterior
- Identify significant ST elevation
- STEMI
CLINICAL CORRELATIONS

- BBB may occur at different sites in the conduction system
- BBB may obscure the ECG findings of AMI
## CBBB - Significance

<table>
<thead>
<tr>
<th>QRS ≥ .12 sec. (≥ 3 small boxes)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LBBB</strong> - treat as AMI if symptoms present</td>
</tr>
<tr>
<td><strong>RBBB</strong> - read ST segments Note: may be difficult</td>
</tr>
</tbody>
</table>

When develops during AMI, implies extensive ischemia
Bundle Branch Block - QRS $\geq .12$ sec.

Lead V1 - turn signal:

- QRS down = left
- QRS up = right

Left BBB

Right BBB
Complete Left Bundle Branch Block
Complete Right Bundle Branch Block
After Rate and Rhythm:

QRS - Is LBBB present?

If answer is no,

Read the ST segments.

ST - Is elevation \( \geq 1 \text{ mm.} \)

present in at least two contiguous leads?
Normal Electrocardiogram
### AMI - Locations and Leads

<table>
<thead>
<tr>
<th>LOCATIONS</th>
<th>LEADS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior</td>
<td>II, III, aVF</td>
</tr>
<tr>
<td>Anterior</td>
<td>V1 - V6</td>
</tr>
<tr>
<td>Anteroseptal</td>
<td>V1 – V2</td>
</tr>
<tr>
<td>Lateral</td>
<td>I, aVL, V5, V6</td>
</tr>
</tbody>
</table>
ST-T Wave Changes

- Normal
- T Wave Inversion
- ST Depression
- ST Elevation
- Q Wave / ST Elevation
- Q Wave / Normal ST / Inverted T
Transmural AMI (associated with thrombus)

Ischemia:
- T Inversion

Injury:
- ST Elevation

Infarction:
- Q Wave (Pathologic)
AMI Extent and Progression

- **Subendocardial**
  - ST Segment Depression
  - V5

- **Non Transmural (Non Q Wave)**
  - T Wave Inversion
  - V5

- **Transmural Thrombus Usual**
  - Q Wave and ST Segment Elevation
  - V5
Inferior MI (II, III, aVF)
Anterior MI (V1 - V6)
Anterolateral MI
(I, aVL, V5, V6)
Anterior MI (V1 - V4)
Inferior MI - Initial ECG 

1 of 3
Inferior MI - Discharge ECG (3 of 3)
ECG Mimics of STEMI
ECG Mimics of AMI - History May Help!

- Complete LBBB
- Early Repolarization
- Ventricular Rhythms
- LV Hypertrophy
- Ventricular Pacemakers
- Pericarditis
Complete Left Bundle Branch Block

- Sinus P with wide QRS (> 3 small boxes)
- LBBB and history suggestive of acute MI (Class I recommendation for thrombolysis)
Complete Left Bundle Branch Block
Ventricular Dysrhythmia

- QRS wide and unrelated to P waves
- Cannot interpret ST - T waves
Ventricular Dysrhythmia
Ventricular Pacemaker

- **Spike** followed by wide QRS
- Cannot interpret ST - T waves
A-V Sequential Pacemaker

- Spike followed by P wave
- 2nd spike followed by wide QRS
Early Repolarization

- J and Concave ST elevation V Leads
- Normal variant / young males
Early Repolarization
Left Ventricular Hypertrophy (LVH)

- Tall R waves in leads reflecting L.V. (R wave V5 or V6 > 26 mm)
- ST - T changes due to L.V.H.
Left Ventricular Hypertrophy
Acute Pericarditis

- ST segment elevation most leads
- “Flu” history and atypical chest pain
Pericarditis
STEMI Treatment
The immediate cause of most infarctions: THROMBUS FORMATION
Occluded Coronary Artery - cross section

Atherosclerosis and Thrombus
Left Anterior Descending (LAD)

Totally Occluded by Thrombus
LAD - 15 Minutes After Treatment

Flow Partially Restored
Further Resolution of Thrombus
Immediate Interventions for AMI

Initial Assessment:

**A**irway - ensure patency (prevent aspiration)

**B**reathing - oximeter, O₂

**C**irculation - vital signs, rhythm monitoring, IV
Therapy of AMI

GOAL:

Reduce Infarct Size

METHOD:

- Decrease oxygen demand
- Increase oxygen supply
## Decrease Oxygen Demand

<table>
<thead>
<tr>
<th>DRUG</th>
<th>USE</th>
<th>MECHANISM</th>
<th>CAUTIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitroglycerin</td>
<td>Ischemic pain</td>
<td>May dilate coronary arteries</td>
<td>Headache</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dilates peripheral veins and arteries, reducing heart’s volume and pressure load</td>
<td>Hypotension</td>
</tr>
<tr>
<td>Morphine Sulfate</td>
<td>Severe ischemic pain</td>
<td>Acts on pain sites in brain</td>
<td>Nausea, Vomiting</td>
</tr>
<tr>
<td></td>
<td>Pulmonary Edema</td>
<td></td>
<td>Hypotension</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Respiratory Depression</td>
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</table>
**Increase Oxygen Supply**

<table>
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<th>DRUG</th>
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<th>MECHANISM</th>
<th>CAUTIONS</th>
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<tbody>
<tr>
<td>Anti-Platelet:</td>
<td>Prevents clot formation</td>
<td>Decreases platelet “stickiness”</td>
<td>Bleeding</td>
</tr>
<tr>
<td>Aspirin</td>
<td></td>
<td></td>
<td>Gastric Ulcers</td>
</tr>
<tr>
<td>Fibrinolysis:</td>
<td>Dissolves the clot</td>
<td>Activates plasmin which lyses the thrombus</td>
<td>Bleeding</td>
</tr>
<tr>
<td>Retavase</td>
<td>occluding coronary artery</td>
<td></td>
<td>Exclusion Criteria for Thrombolytics</td>
</tr>
<tr>
<td><strong>TNKase</strong> Streptokinase</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Fibrinolytics ("Clot busters")

<table>
<thead>
<tr>
<th>AMI Cause:</th>
<th>Thrombus in majority</th>
</tr>
</thead>
<tbody>
<tr>
<td>If Rx Early:</td>
<td>Flow ↑, damage ↓</td>
</tr>
<tr>
<td>Indications:</td>
<td>Persistent symptoms of &lt; 12 hours duration</td>
</tr>
<tr>
<td></td>
<td>ST elevation of STEMI</td>
</tr>
<tr>
<td>Contraindications:</td>
<td>Exclusion criteria</td>
</tr>
</tbody>
</table>
Fibrinolytic Inclusion Criteria

- Persistent symptoms consistent with AMI for > 30 minutes and < 12 hours (chest pain or equivalent)

- ECG changes
  - ST segment elevation 1 mm. in at least 2 contiguous vertical leads, or 2mm. In 2 contiguous horizontal leads
  - QRS is.12 sec. with LBBB (not RBBB)

Inferior - II, III, aVF, Anterior - V1-V6, Lateral - 1, aVL
Fibrinolytic Exclusion Criteria Include:
(Be Sure: BSSSSS)

Bleeding (active or known problems)

Stroke (TIA, brain and spinal surgery)

Streptokinase (previously received - allergy)

Surgery (recent or trauma - traumatic CPR)

Severe Hypertension
Cardiac Event Protocol

- **EMS SYSTEM APPROACH** THAT SHOULD ADDRESS:
  - Oxygen - IV - cardiac monitor - vital signs
  - Nitroglycerin
  - Aspirin
  - Pain relief with narcotics
  - Notification of emergency department
  - Rapid transport to emergency department
  - Prehospital screening for PCI or fibrinolytic therapy
  - 12-lead ECG, computer analysis, transmission:
    - to emergency department

- **“DOOR-TO-REPERFUSION” TEAM PROTOCOL APPROACH**
  - Rapid triage of patients with chest pain
  - Clinical decision maker established (emergency physician, cardiologist, or other)
ASSESSMENT

TREATMENTS TO CONSIDER if there is evidence of coronary thrombosis plus no reasons for exclusion:

(some but not all may be appropriate)

- Clopidogrel
- Nitroglycerin IV
- β-Blockers IV
- Heparin IV
- Ace Inhibitors
- Statins
- Glycoprotein Inhibitors
- Consult as needed
- Decide eligibility for
  - PCI/Fibrinolysis

Immediate:

- Vital signs
- Oxygen at 4 L/min-Sat<94%
- Aspirin, if not given prior
- Start IV
- Nitroglycerin SL or spray
- Morphine IV
- 12-lead ECG (MD review)
- Focused history & physical
- Chest X-ray
- Blood (electrolytes, cardiac markers, coagulation studies)
Precious minutes saved life of Christmas tree grower

Cherri and Gary Trump of Pinecrest Tree Farm in Blue Springs. Gary benefited last summer from a heart attack protocol recently introduced in Beatrice Fire Rescue