Cardiovascular Emergencies: Diagnosis and Immediate Management

Royal College of Psychiatrists
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Mental Health and Cardiovascular Disease

• High risk population
  – Psychiatrist may be their only medical contact
• Excess of traditional risk factors
  – Smoking, Recreational drug use
• Psychiatric medication
  – Weight gain and metabolic syndrome
  – Prolonged QTc interval and arrhythmias
  – Autonomic dysfunction
  – Cardiomyopathy ie Clozapine
• Excess mortality
  – SADHART (Sertraline Antidepressant Heart Attack Randomised Trial, APA 2009)
  – Sertraline responders had a 15.6% mortality rate vs 28.4% for non-responders (HR 2.39)
Cardiovascular Emergencies

• Acute Coronary Syndromes
• Pulmonary Embolus
• Left Ventricular Failure
• Ventricular Arrhythmias and Heart Block
Spread of electrical activity through the atria
Atrioventricular node and the bundle of His
The heart in action
The heart in action
ST Segment
ST Segment
Pathological Q Wave

- ≥ 0.04 sec wide
- >25% of R wave
QT interval

- Measure in lead II and V5 and take longest value
- Calculate QTc (Bezett’s formula)

\[
QTc = \frac{QT}{\sqrt{RR}}
\]

- Abnormal QTc >0.45s men; >0.46s women
Chest Pain statistics

• In the UK
  – ~1% of visits to a GP are because of chest pain
  – ~5% of emergency department visits and up to 40% of emergency hospital admissions are related to chest pain
  – 20-40% of the population will experience chest pain in their lifetime
  – 1 in 5 men and 1 in 7 women die from coronary heart disease
Differential diagnosis of chest pain

- Angina, Myocardial infarction
- Pulmonary embolism
- Aortic dissection
- Pericarditis
- Chest Infection, Pneumonia
- Peptic ulcer, Oesophagitis, oesophageal spasm
- Musculoskeletal
Definition of Angina

• Constricting discomfort in the front of the chest, neck, shoulders, jaw or arms
• Precipitated by physical exertion or emotional stress
• Relieved by rest or GTN within about 5 minutes

• Typical angina: All of the above
• Atypical angina: 2 of the above
• Non-anginal chest pain: 1 or none of above
Factors making angina more likely:

- Increasing age
- Male sex
- Cardiovascular risk factors
- History of established CAD (e.g. previous MI, coronary revascularisation)
Angina unlikely if pain is:

- continuous or very prolonged and/or
- unrelated to activity and/or
- brought on by breathing in and/or
- associated with dizziness, palpitations, tingling or difficulty swallowing
HEART score and outcome

HEART score for chest pain patients

<table>
<thead>
<tr>
<th>HEART Score</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
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<td>History</td>
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<td>≤ 45</td>
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<td>Risk factors</td>
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<tr>
<td>≥ 3 factors</td>
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<tr>
<td>1 or 2</td>
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<td>No</td>
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<tr>
<td>Troponin</td>
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<td>≥ 3x</td>
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<td>1 - 3x</td>
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<tr>
<td>≤ normal</td>
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<tr>
<td>Total</td>
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</table>

Risk factors for atherosclerotic disease:
- Hypercholesterolemia
- Cigarette smoking
- Hypertension
- Positive family history
- Diabetes Mellitus
- Obesity (BMI>30)

Low risk: Heart score 0-3, negative Troponins
High risk: Heart score >3, or positive Troponin

HEART score reliably predicts endpoints

<table>
<thead>
<tr>
<th>HEART Score</th>
<th>~ % pts</th>
<th>MACE/n</th>
<th>MACE</th>
<th>Death</th>
<th>Proposed Policy</th>
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<tr>
<td>0-3</td>
<td>32%</td>
<td>38/1993</td>
<td>1.9%</td>
<td>0.05%</td>
<td>Discharge</td>
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<tr>
<td>4-6</td>
<td>51%</td>
<td>413/3136</td>
<td>13%</td>
<td>1.3%</td>
<td>Observation, risk management</td>
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<td>7-10</td>
<td>17%</td>
<td>518/1045</td>
<td>50%</td>
<td>2.8%</td>
<td>Observation, treatment, CAG</td>
</tr>
</tbody>
</table>

*MACE = Major Adverse Cardiac Event = Myocardial Infarction, PCI/CABG, all-cause death. Based on N=6174
Acute Coronary Syndromes
Acute Coronary Syndromes

• Unstable Angina (UA)
  – Chest Pain, +/- ECG changes, No cardiac enzyme release

• Non-ST segment elevation myocardial infarction (NSTEMI / NSTEACS)
  – Chest Pain, +/- ECG changes, Cardiac enzyme release

• ST segment elevation myocardial infarction (STEMI)
  – Chest pain, ST segment elevation, Cardiac enzyme release
Diagnosis of MI

Requires the presence of at least two of the following criteria:-

1. Chest pain (clinical manifestation)

2. ECG changes consistent with ischaemia or necrosis

3. Elevation of cardiac markers
Symptoms of MI

Usual distribution of pain with myocardial ischaemia

Less common sites of pain with myocardial ischaemia

Right side

Jaw

Epigastrium

Back
**Signs**

- **General appearance**
  - restless, diaphoretic

- **Heart rate**
  - Increased sympathetic drive, bradycardia due to sinus node dysfunction or AV block

- **Blood pressure**
  - Initially elevated, low with cardiogenic shock or RV infarct

- **Respiration**
  - Increased: pulmonary congestion, anxiety

- **Temperature**
  - Often elevated after infarction
Caution

• Atypical presentation
  – Elderly
  – Females
  – Diabetics
Diagnosis of MI

1. Chest pain (clinical manifestation)

2. ECG changes consistent with ischaemia or necrosis

3. Elevation of cardiac markers
Acute Myocardial Infarction

- ST elevation >2mm in V1-V3 and >1mm in all other leads in >2 contiguous leads\(^1\).
- Infarction can present as Q wave\(^1\).

Evolution of an ST elevation myocardial infarction

A. Onset

B. 15 Minutes

C. > 1 Hour

D. > 24 Hours

E. Days Later

F. Months later
Diagnosis of MI

Requires the presence of at least two of the following criteria:-

1. Chest pain (clinical manifestation)

2. ECG changes consistent with ischaemia or necrosis

3. Elevation of cardiac markers
Elevation of cardiac markers

- LDH
- Total CK
- CK-MB
- Troponin I

Hours from onset of infarction

7x upper limit of normal
Inferior AMI

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<tr>
<th></th>
<th>I</th>
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<th>II</th>
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<th>III</th>
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<td>aVF</td>
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<td>V5</td>
<td>V6</td>
<td>V1</td>
<td>V1</td>
<td>aVR</td>
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</tbody>
</table>

III aVF

II
Antero-septal AMI

- V1
- V2
- V3
- V4
- aVR
- III
- aVL
- aVF
- V1
- V2
- V3
- V4
- V5
- V6
Antero-lateral AMI

<table>
<thead>
<tr>
<th>I</th>
<th>aVR</th>
<th>V1</th>
<th>V4</th>
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<tr>
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</tr>
<tr>
<td>III</td>
<td>aVF</td>
<td>V3</td>
<td>V6</td>
</tr>
</tbody>
</table>

ECG tracing showing characteristic changes in leads I, aVL, and V1-V6.
Posterior AMI V1-V4 Depression
Ischaemia

Normal

ST Depression
Ischaemia
Ischaemia
Initial management of all ACS

- Early ECG, repeat as necessary
- Oxygen if sats <94%
- Aspirin – 300mg
- Analgesia – Opiates (+anti-emetic)
- IV nitrates – hypertension, pulmonary oedema, ongoing pain
- Bloods: Renal function, FBC, clotting profile, Lipid profile, Glucose, Cardiac enzymes (Troponin)
Unstable angina and NSTEMI

LIPID-RICH STABLE ECCENTRIC PLAQUE CAUSING LUMINAL OBSTRUCTION

Normal media

Intraluminal thrombus

Media

Intima

Lumen

Intima

Lipid pool

Intraintimal thrombus
Management of NSTEMI / UA

- Dual antiplatelet therapy
  - Add Ticagrelor or Clopidogrel alongside Aspirin
- Fondaparinux 2.5mg sc 48-72 hours
- Optimal medical therapy
  - Beta blockers, ACE inhibitors, Statins
- Refer to Cardiology asap
  - Angiography +/- revascularisation
Management of STEMI

15 minutes         2 hours         6 hours
% necrosis       0%                50%               90%
Management of STEMI

• Reperfusion therapy
  – The Golden Hour
  – Immediate call to Primary Angioplasty Centre
  – Blue light ambulance transfer
Delay to balloon time of >120 mins?
Early reperfusion is the key!
Thrombolysis

- Heparin
- Thrombolytic
  - Streptokinase: Up to 12 hours (Not if used before)
  - Alteplase: Up to 6 hours
  - Reteplase: Up to 12 hours
  - Tenecteplase: Up to 6 hours, single bolus
- Still Transfer to cardiac centre in case of failed reperfusion and for early angiography
Benefits for Early Diagnosis and Thrombolytic Treatment

Absolute 35-day Mortality Reduction Versus Treatment Delay Per 1000 Patients Treated

- 0-1 hrs: 65/1000
- 1-2 hrs: 37/1000
- 2-3 hrs: 26/1000
- 3-6 hrs: 29/1000
- 6-12 hrs: 18/1000
- 12-24 hrs: 9/1000

Adapted from Boersma et al: Lancet 1996;348:771-75
Pulmonary Embolus
PE – background information

• A PE is where one or more emboli are lodged in and obstruct the pulmonary arterial system.
• The annual incidence of PE is around 3–4 per 10,000 people.
• A PE may be:
  – Provoked – associated with a transient risk factor (e.g. significant immobility, surgery).
  – Unprovoked – no identifiable risk factor or a risk factor that is persistent and not easily correctable (e.g. active cancer).

Based on the CKS topic Pulmonary embolism (June 2013), and NICE guidance (2012a): Venous thromboembolic disease: the management of venous thromboembolic diseases and the role of thrombophilia testing.
Complications

• Mortality
  – Untreated, the risk of death from a PE is high (23–87%)
  – When treated with heparin and anticoagulants the risk of death ranges from 2-6%
  – For a clinically massive PE the risk of death is about 50%
  – PE is the leading cause of maternal deaths in the UK

• Chronic thromboembolic pulmonary hypertension
  – Occurs in 0.5–5% of people with treated PE

Based on the CKS topic Pulmonary embolism (June 2013), and NICE guidance (2012a): Venous thromboembolic disease: the management of venous thromboembolic diseases and the role of thrombophilia testing.
Major risk factors for PE

- Major risk factors include:
  - Deep vein thrombosis (DVT).
  - Previous DVT or pulmonary embolism.
  - Active cancer.
  - Recent surgery, hospitalization, lower limb trauma, or other immobilization (including long-distance sedentary travel).
  - Pregnancy, in particular 6 weeks postpartum.

Based on the CKS topic Pulmonary embolism (June 2013), and NICE guidance (2012a): Venous thromboembolic disease: the management of venous thromboembolic diseases and the role of thrombophilia testing.
When to suspect a PE

- Suspect a PE if the person has:
  - Dyspnoea, tachypnoea, pleuritic chest pain, or features of a DVT.
    - These features are present in 97% of people with PE, but
    - Only 15% of people with a PE have signs of DVT.
  - Other features such as tachycardia, haemoptysis, syncope, hypotension (systolic BP less than 90 mmHg), atrial flutter
  - A risk factor for PE (e.g. previous DVT/PE, pregnant).

Based on the CKS topic Pulmonary embolism (June 2013), and NICE guidance (2012a): Venous thromboembolic disease: the management of venous thromboembolic diseases and the role of thrombophilia testing.
Pulmonary embolus - Investigations

• ABG on air
  – Hypoxia with respiratory alkalosis
• CXR
  – Often normal
• ECG
  – Sinus tachycardia
  – Rarely SI, QIII, TIII, RBBB
  – T wave inversion common
• Bloods
  – Troponin often elevated
  – D-Dimer for negative predictive value only
• Echo
  – May show elevated estimated PA pressure and RV dilatation
• CT-PA
Managing a suspected PE

- Arrange immediate admission if the person is:
  - Pregnant or has given birth within the past 6 weeks.
    - It is not possible to accurately assess the risk of PE in primary care.
  - Severely ill with:
    - Altered level of consciousness.
    - Systolic BP of less than 90 mm Hg.
    - Heart rate of more than 130 beats per minute.
    - Respiratory rate of more than 25 breaths per minute.
    - Oxygen saturation of less than 91%.
    - Temperature of less than 35°C.

Based on the CKS topic Pulmonary embolism (June 2013), and NICE guidance (2012a): Venous thromboembolic disease: the management of venous thromboembolic diseases and the role of thrombophilia testing.
PE Wells Score

– **Score 3 points if:**
  - There are clinical features of a DVT.
  - An alternative diagnosis is less likely than a PE.

– **Score 1.5 points if:**
  - Heart rate is greater than 100 beats per minute.
  - Immobilization for more than 3 days.
  - Surgery in the previous 4 weeks.
  - Previous DVT or PE.

– **Score 1 point if the person has:**
  - Haemoptysis.
  - Cancer.

*Based on the CKS topic Pulmonary embolism (June 2013), and NICE guidance (2012a): Venous thromboembolic disease: the management of venous thromboembolic diseases and the role of thrombophilia testing.*
If PE likely (>4 points)

- Arrange immediate admission for a computed tomography pulmonary angiogram (CTPA), or
- If there will be a delay in the person receiving a CTPA:
  - Give immediate interim low molecular weight heparin (LMWH) or fondaparinux, and
  - Arrange hospital admission.
- Body weight is required to prescribe LMWH or fondaparinux.

Based on the CKS topic Pulmonary embolism (June 2013), and NICE guidance (2012a): Venous thromboembolic disease: the management of venous thromboembolic diseases and the role of thrombophilia testing.
If PE unlikely (4 points or less)

• Arrange a D-dimer test.
• If the D-dimer test is negative, consider an alternative diagnosis.
• If the D-dimer test is positive:
  – Arrange admission to hospital for an immediate CTPA, or
  – If a CTPA cannot be carried out immediately, give LMWH or fondaparinux and arrange hospital admission.

Based on the CKS topic Pulmonary embolism (June 2013), and NICE guidance (2012a): Venous thromboembolic disease: the management of venous thromboembolic diseases and the role of thrombophilia testing.
Treatments in secondary care

• Once a PE has been confirmed in secondary care, most people will be initiated on long term treatment with either:
  – An oral anticoagulant (warfarin or NOAC), or
  – A LMWH.

• LMWH are usually indicated if oral anticoagulants are:
  – Contraindicated, for example pregnancy, or
  – Less preferred, for example cancer

• Thrombolytic therapy or embolectomy may be offered to people who are haemodynamically unstable.

Based on the CKS topic Pulmonary embolism (June 2013), and NICE guidance (2012a): Venous thromboembolic disease: the management of venous thromboembolic diseases and the role of thrombophilia testing.
PE Summary

• The most common source of PE is a DVT in the lower limbs (80%).
• Untreated, the risk of death from a PE is high (23–87%).
• When treated with heparin and anticoagulants the risk of death ranges from 2-6%.
• Arrange immediate admission if the person is:
  – Pregnant or has given birth within the past 6 weeks.
  – Severely ill with, for example altered level of consciousness.
• For every one else use the two-level PE Wells score to assess likelihood of PE. A PE is:
  – Likely if the score is more than 4 points.
  – Unlikely if the score is 4 points or less.
• If PE is likely - arrange immediate admission for a CTPA, or if there will be a delay in the person receiving a CTPA:
  – Give immediate interim low molecular weight heparin (LMWH) or fondaparinux, and arrange hospital admission.
• If PE is unlikely - arrange a D-dimer test, if the D-dimer test is positive:
  – Arrange admission to hospital for an immediate CTPA, or
  – If a CTPA cannot be carried out immediately, give LMWH or fondaparinux and arrange hospital admission.
Heart Failure
Heart Failure

- Heart does not pump enough blood to meet demands of body
- 67,000 admissions per year in England and Wales
- Leading cause of admissions in patients >65yo in UK
- Can present as new onset heart failure or decompensation of chronic heart failure
Broad spectrum of conditions

- Dysfunction due to
  - Muscle damage
  - Valvular dysfunction
  - Arrhythmias
  - Congenital heart disease
  - Other rare causes
- Can effect Left Ventricle, Right Ventricle or Both
- Heart Failure with preserved Ejection Fraction (HFpEF)
  - Diastolic dysfunction
Left Ventricular Failure

- History
  - Breathlessness
- Clinical Examination
  - Coarse crackles in chest, peripheral oedema
- ECG
  - Look for arrhythmias
- CXR
CXR
Further investigations

- BNP < 100ng/litre heart failure unlikely
- BNP > 400ng/litre heart failure very likely
- BNP 100 – 400ng/litre non-specific
  - Age>70, PE, sepsis, COPD, diabetes, liver cirrhosis, eGFR<60ml/min, tachycardia, LVH

- Echocardiography
Initial treatment

• Intravenous diuretics ie Furosemide
  – Bolus ie 40-80mg IV
  – Infusion ie 120-240mg/24hours
• Increase maintenance diuretic dose
• Monitor renal function, weight, urine output
• Consider IV nitrates
  – Ischaemia, hypertension, AR or MR
• Consider IV Diamorphine
  – pulmonary oedema, chest pain, distress
• Admit to level 2 care (HDU/CCU)
Non-pharmacological treatment

• Non-invasive ventilation (CPAP/NIPPV)
  – Pulmonary oedema
  – Severe dyspnoea
  – Acidosis

• Invasive ventilation
  – Respiratory failure
  – Reduced consciousness or physical exhaustion

• Intra-aortic balloon pump

• Left Ventricular Assist Devices or Impella
Treatment after stabilisation

- Beta blockers
  - Bisoprolol, Carvedilol, Metoprolol
- ACE inhibitors or ARII blockers
- Aldosterone antagonist
- Maintenance diuretic therapy
Ventricular arrhythmias and heart block
Heart Block

- 1\textsuperscript{st} degree – prolonged PR interval
- 2\textsuperscript{nd} degree
  - Mobitz Type 1 = Wenckebach
  - Mobitz Type 2
- 3\textsuperscript{rd} degree = Complete heart block
Mobitz type 2

- Intermittent non-conducted P waves *without* progressive prolongation of the PR interval.
- The PR interval in the conducted beats remains constant.
- The P waves ‘march through’ at a constant rate.
Mobitz type 2

- Mobitz II is much more likely than Mobitz I to be associated with haemodynamic compromise, severe bradycardia and progression to 3rd degree heart block.

- Onset of haemodynamic instability may be sudden and unexpected, causing syncope (Stokes-Adams attacks) or sudden cardiac death.

- The risk of asystole is around 35% per year.

- Mobitz II mandates admission for cardiac monitoring, consideration of backup temporary pacing and ultimately insertion of a permanent pacemaker.
3\textsuperscript{rd} degree (complete) heart block

- Typically the patient will have severe bradycardia with independent atrial and ventricular rates, i.e. AV dissociation.
Treatment for Heart Block

• Withdraw negatively chronotropic drugs
  – Beta blockers, Diltiazem, Verapamil, Digoxin
• If haemodynamically compromised
  – Atropine 600mcg boluses (Max 300mg)
  – Isoprenaline infusion
  – External (Transcutaneous) pacing
  – Temporary venous pacing
  – Permanent pacemaker if no reversible cause
Complete heart block

- Patients with third degree heart block are at high risk of ventricular standstill and sudden cardiac death.

- They require urgent admission for cardiac monitoring, backup temporary pacing and usually insertion of a permanent pacemaker.

- May resolve if acute underlying cause treated ie Inferior STEMI, severe hypothyroidism, drugs
QTc prolongation

• Marker of arrhythmic risk
• Torsade de pointes
  – May lead to syncope, dizziness, ventricular fibrillation, sudden death
• Can occur with all antipsychotic drugs
  – Pimozide
  – Thioridazine
  – Sertindole
  – Zotepine
  – IV Haloperidol
Torsades de Pointes
Torsades de Pointes

- Polymorphic ventricular tachycardia
- Usually paroxysmal
- QRS complexes twist around the isoelectric line
- Correct electrolyte disturbance
  - Hypokalaemia, Hypomagnesaemia
- Withdraw drugs causing prolonged QTc
- Magnesium IV 1-2g IV over 10-15 minutes
Ventricular Tachycardia
Immediate management

- If haemodynamically tolerated
  - Correct electrolytes (K+ >4, Mg)
  - Beta blockers
  - Amiodarone
  - IV Lidocaine if refractory
  - Overdrive pacing
  - Consider cardioversion

- If haemodynamically unstable
  - DC Cardiovert (Remember synchronise button)
Steps for synchronized cardioversion

- Turn on defibrillator
- Attach monitor leads to the patient
- Ensure proper display of the patient’s rhythm (lead II)
- Press ‘sync’ control button
- Look for markers on R waves
- If no markers, adjust monitor gain until markers on each R
Ventricular Fibrillation
Adult resuscitation guidelines

**Resuscitation Council (UK) 2015**

**Adult Basic Life Support**

Unresponsive and not breathing normally

- Call 999 and ask for an ambulance
- 30 Chest compressions
- 2 Rescue breaths
- Continue CPR 30:2
- As soon as AED arrives switch it on and follow instructions

**Adult Advanced Life Support**

Unresponsive and not breathing normally

- Call resuscitation team
- CPR 30:2 Attach defibrillator/monitor Minimise interruptions

**Shockable (VF/Pulseless VT)**

- 1 Shock Minimise interruptions
- Immediately resume CPR for 2 min Minimise interruptions

**Return of spontaneous circulation**

**Immediate post cardiac arrest treatment**
- Use ABCDE approach
- Aim for SpO2 of 94-98%
- Aim for normal PaCO2
- 12-lead ECG
- Treat precipitating cause
- Targeted temperature management

**Non-shockable (PEA/Asystole)**

- Immediately resume CPR for 2 min Minimise interruptions

**During CPR**
- Ensure high quality chest compressions
- Minimise interruptions to compressions
- Give oxygen
- Use waveform capnography
- Continuous compressions when advanced airway in place
- Vascular access (intravenous or intraosseous)
- Give adrenaline every 3-5 min
- Give amiodarone after 3 shocks

**Treat Reversible Causes**
- Hypoxia
- Hypovolaemia
- Hypo- or hyperkalaemia/metabolic
- Hypothermia
- Thrombosis - coronary or pulmonary
- Tension pneumothorax
- Tachypnoea – cardiac
- Toxins

**Consider**
- Ultrasound imaging
- Mechanical chest compressions to facilitate transfer/treatment
- Coronary angiography and percutaneous coronary intervention
- Extracorporeal CPR

**Assess rhythm**
Any Questions?