CMC Certification Review Course:
Handout

Session #: 1
Acute Coronary Syndrome Cardiac Dysrhythmias
Therapeutic Hypothermia

Presented by:
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How to use this module:

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Session 1:
Acute Coronary Syndrome
Cardiac Dysrhythmias
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Acute Coronary Syndrome (ACS)

- Unstable Angina (UA)
  - Definition: Discomfort that occurs when oxygen demand exceeds oxygen supply

Angina

- Classifications
  - Unstable
  - Prinzmetal's

Prinzmetal’s Angina

- Also known as
  - Variant
  - Vasospastic
  - Atypical
- Not exercise-induced
- May not have severe coronary artery disease (CAD)
Prinzmetal’s Angina: Incidence
- 2%-3% of patients having chest pain
- 70%-80% of the patients are male
- More common in Japanese men than Caucasians
- Younger, with average age of 51-57 years
- Most important risk factor is cigarette smoking

Prinzmetal Angina: Symptoms
- Chest Pain
  - Under chest bone
  - Squeezing, constricting, tightness, pressure, or crushing
  - May radiate to neck, jaw, shoulder, or arm
  - Most often occurs at rest
  - Lasts from 5–30 minutes
  - Relieved by nitroglycerin (NTG)
  - Can cause fainting or loss of consciousness

Prinzmetal’s Angina

ST-segment elevation over the indicative leads
Prinzmetal’s Angina: Diagnosis
- Gold standard: coronary angiography with injection of a provocative agent
- Ergonovine, methylergonovine, or acetylcholine

Prinzmetal’s Angina: Treatment
- Nitrates: activate nitric oxide in vessels
- Dihydropyridine calcium channel blockers
  - Nifedipine
  - Nicardipine
  - Nimodipine
  - Felodipine
  - Amlodipine

Unstable Angina: Etiology
- Modifiable Risk Factors
  - Smoking
  - Hypertension (HTN)
  - Obesity
  - Hyperlipidemia
  - Diabetes
  - Sedentary lifestyle
  - Stress
  - Low serum folate
  - Cocaine use
- Nonmodifiable Risk Factors
  - Age
  - Sex
  - Family history
  - Race
Unstable Angina: Diagnosis

- Clinical Symptoms
- Electrocardiogram (ECG)
  - T wave inversion
  - ST-segment depression
- Labs
  - Arterial blood gases (ABGs)
  - Hemoglobin (Hgb) & hematocrit (Hct)
  - Enzymes
    - Troponin
    - Creatine kinase myocardial band (CK-MB)
    - Echocardiogram (echo)

Unstable Angina

Clinical Presentation

- Pain
  - 30 seconds to 30 minutes
- Associated symptoms
  - Shortness of breath
  - Nausea
  - Diaphoresis
  - Numbness or pain
  - Lightheadedness
  - Tachycardia
  - Tachypnea

Cardiac Tests

<table>
<thead>
<tr>
<th>Cardiac Tests</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total CK</td>
<td>36-120 ng/mL</td>
</tr>
<tr>
<td>CK-MB</td>
<td>0-3 ng/mL</td>
</tr>
<tr>
<td>CK-index</td>
<td>0-3</td>
</tr>
<tr>
<td>Troponin</td>
<td>&lt; 0.4 ng/mL</td>
</tr>
</tbody>
</table>
Enzyme Normal Rise Peak Return to Normal
CK 30–210 u/L 3-6 h 24 h 3–4 d
CK-MB < 8 ng/mL 4 h 18–24 h 2–3 d
CK-MB Rise (R) < 3.5
Myoglobin <100 ng/mL 2 h 6–7 h 24 h
Troponin I < 0.1 ng/mL 4–6 h 10–24 h 4 d
Troponin T < 0.5 ng/mL 4–6 h 10–24 h 10 d

Unstable Angina

- Echocardiography
  - Ejection fraction
  - Structural abnormalities
  - Segmental wall motion abnormality (SWMA)
Unstable Angina

- Structural Abnormalities
  - Wall thickening
  - Septum motion problems
  - Valve abnormalities
  - Ventricular aneurysm

Unstable Angina

- SWMA
  - Normal
  - Hypokinetic
  - Akinetic
  - Dyskinetic

Unstable Angina

- SWMA Causes
  - Infarction
  - Ischemia
  - Stunning
  - Hibernating myocardium
  - Apparent
    - Left Bundle Branch Block (LBBB)
    - Epicardial pacing
    - Right ventricular (RV) pressure or volume overload
Unstable Angina

- Treatment
  - Modify risk factors
  - Correct aggravating factors

Unstable Angina

- Treatment
  - Oxygen
  - Aspirin
  - Nitrates
  - β-adrenergic blocking agents
  - Calcium channel blockers
  - Heparin
  - Glycoprotein IIB/IIIA (GPIIB/IIIA) inhibitors

Practice Exam Questions
Question #1 - Answer

Unstable angina symptoms reflect:

A. An imbalance between oxygen supply and demand. The most common etiology is atherosclerosis and a partial occlusion by a thrombi. There is no cell death.
B. The presence of coronary artery emboli
C. Myocardial necrosis
D. Total occlusion of a coronary artery

Question #2 - Answer

Both UA and non-ST elevation myocardial infarction (NSTEMI) are characterized by:

A. Pathologic Q waves
B. Abnormal wall motion on echo
C. Chest pain or anginal equivalent. Patient symptoms, changes on the ECG, and echo results are similar in UA and NSTEMI patients.
D. A prolonged QT interval

Question #3 - Answer

NSTEMI is differentiated from UA by:

A. Increased serum troponin levels in the presence of ST-segment elevation
B. Positive biomarkers of myocardial necrosis. Patient symptoms, ECG, and echo findings are similar between UA and NSTEMI. The only difference is NSTEMI will have increased troponin levels. Enzymes should be monitored closely when chest pain symptoms are identified.
C. Normal serum troponin levels in the presence of ST-segment depression
D. Negative cardiac biomarkers
Myocardial Infarction (MI)

- Irreversible death or necrosis of myocardial tissue due to inadequate coronary blood supply
- Leads to metabolic changes:
  - Anaerobic metabolism
  - Breakdown of sodium (Na+) - potassium (K+) pump
  - Cellular edema
  - Membrane rupture

MI: Types

- Non-Q wave
  - ST segment elevation, depression, or no change
  - T wave inversion on indicative leads
- Q wave
  - Significant Q wave in presence of acute changes

Clot Formation
MI: Types

- NSTEMI
  - No ST segment elevation
  - Diagnosis made from clinical presentation and enzymes
- ST-elevation myocardial infarction (STEMI)
  - ST segment elevation over the indicative leads

MI: Etiology

- Atherosclerosis
- Coronary artery spasm
- Coronary embolism
- Coronary artery dissection
- MI with normal coronary arteries

MI: Clinical Presentation

- Pain
  - Lasts > 30 minutes
  - Associated symptoms
  - No pain in about 30% of patients experiencing MI
MI: Diagnostics
- 12-lead ECG (18-lead)
- Enzymes
- Echo
- Clinical symptoms

Differential Diagnosis

Differential Diagnosis of Urgent Chest Pain

12-Lead ECG
- UA or NSTEMI
- ST segment
- T wave inversion > 1 mm
- 0.5 mm
- 0.6 – 1.0
- New LBBB
- Evidence of Q wave
- Cardiac Markers
- Nondiagnostic
- STEMI
- ST segment
- 1.0 mm
- Stable Angina
- Noncardiac
- Stable Angina Persistent
- Noncardiac
- Continue ECG monitoring
- Check enzymes
- Continued 8-12 hour Serial and Observation of pain
MI: 12-lead ECG
- Inferior
  - II, III, aVF
- Anterior
  - V2-V6
- Lateral
  - I, aVL, V5-6
- Posterior
  - V1 or V7-9

Left Anterior Descending Artery
- Anterior wall
- Apex
- Lateral wall
- Septum
- Branch
  - Left diagonal
  - Proximal
  - Distal

Circumflex Artery
- Posterior wall
- Lateral wall
- Sinoatrial (SA) node (45%)
- Atrioventricular (AV) node (10%)
- Branch
  - Obtuse marginal
  - Distal
  - Proximal
**Right Coronary Artery**

- Inferior wall
- Posterior wall
- SA node (55%)
- AV node (90%)
- Branches
  - Posterior descending artery (PDA)
  - Acute marginal

**MI: Area of Infarct on ECG**

<table>
<thead>
<tr>
<th>Affected Area</th>
<th>Indicative</th>
<th>Reciprocal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior: RCA</td>
<td>II, III, aVF</td>
<td>I, aVL, V5, 6</td>
</tr>
<tr>
<td>Lateral: Cx</td>
<td>I, aVL, V6</td>
<td>V4–6</td>
</tr>
<tr>
<td>Anterior: LAD</td>
<td>V2–4</td>
<td>II, III, aVF</td>
</tr>
<tr>
<td>Posterior: PDA</td>
<td>None</td>
<td>V1–2</td>
</tr>
<tr>
<td>Anterolateral: LM</td>
<td>I, aVL, V6</td>
<td>II, III, aVF</td>
</tr>
<tr>
<td>Apical: LAD if dominant</td>
<td>V6</td>
<td>None</td>
</tr>
<tr>
<td>Anteroseptal: LAD</td>
<td>V4–6</td>
<td>None</td>
</tr>
<tr>
<td>RV: RCA</td>
<td>V1–3, V5–R</td>
<td>None</td>
</tr>
</tbody>
</table>

**12-Lead ECG**
12-Lead ECG

Practice Exam Questions

Question #4 - Answer
A female patient admits to the hospital with pain between her shoulders and complains of fatigue and nausea. She is very anxious. Your first priority is to:

A. Start the fibrinolytic checklist
B. Request a chest x-ray
C. Request a 12-lead ECG. Within 10 minutes of arrival of a patient with chest pain a 12-lead ECG should be completed and interpreted. Oxygen, NTG, and aspirin should be administered if not contraindicated.
D. Administer heparin
Question #5 - Answer

The cardiologist reviews the 12-lead ECG, which shows a sinus bradycardia at 54 beats per minute (bpm) with ST-segment elevation in leads II, III, and aVF. The patient is presenting with a (an):

A. Anterior STEMI
B. Lateral STEMI
C. An elevation of the ST-segment in leads II, III, and aVF is consistent with an inferior MI. In the presence of an inferior MI always check for RV involvement.
D. Posterior STEMI

Question #6 - Answer

A patient with a possible STEMI has ongoing chest discomfort. A contraindication to the administration of nitrates for pain is:

A. Use of a phosphodiesterase inhibitor within the past 12 hours. The contraindications to the use of nitrates include BP < 90 mm Hg systolic and PDE inhibitors within 24 to 36 hours.
B. Heart rate 90 bpm
C. Left ventricular (LV) infarct with bibasilar crackles
D. Blood pressure (BP) > 160 mm Hg

MI: Treatment

- Bed rest (1st 24 hours)
- NPO until stable
  - Low cholesterol
  - Low sodium diet
- Patient education
MI: Treatment

- Pharmacologic
  - Oxygen
  - Aspirin
  - Nitrates
  - Morphine
  - Fibrinolytic therapy*

* Previously known as thrombolytic therapy

Fibrinolytic Therapy

- Absolute Contraindications
  - Previous intracerebral hemorrhage (ICH)
  - Structural cerebrovascular lesion
  - Arteriovenous malformation (AVM)
  - Malignant intracranial neoplasm

Fibrinolytic Therapy

- Absolute Contraindications (cont.)
  - Ischemic stroke within 3 months
  - Suspected aortic dissection
  - Active bleeding
  - Severe closed head injury or facial trauma within 3 months
Fibrinolytic Therapy

Relative Contraindications
- Chronic, severe, poorly controlled HTN
- Severe uncontrolled HTN
  - Systolic blood pressure (SBP) > 180 mm Hg
  - Diastolic blood pressure (DBP) > 110 mm Hg
- Ischemic stroke > 3 months previously
- Dementia
- Traumatic or prolonged (> 10 min) cardiopulmonary resuscitation (CPR)

Fibrinolytic Therapy

Relative Contraindications (cont.)
- Major surgery (within last 3 weeks)
- Recent internal bleeding (within 2-4 weeks)
- Noncompressible vascular punctures
- Allergic reaction to these agents
- Pregnancy
- Active peptic ulcer disease
- Current use of anticoagulants

Fibrinolytic Therapy

Treatment
- Reperfusion dysrhythmias
- Stabilization of the ST segment
- Resolution of chest pain
MI Complications
- Acute pulmonary edema
- Hypovolemia
- Low output cardiogenic shock
- Dysrhythmia

Cardiac Catheterization
- Collect blood samples from the heart
- Measure pressure and blood flow in the cardiac chambers and in large arteries around the heart
- Measure oxygen levels in different parts of the heart
- Examine the coronary arteries with fluoroscopy
- Perform a biopsy of the cardiac muscle

Cardiac Catheterization: Considerations
- Allergy to seafood
- Previous reaction to contrast material or iodine
- Current use of sildenafil (Viagra®)
- Pregnancy
Cardiac Catheterization: Indications

- Diagnose or evaluate
  - CAD
  - Congenital heart defects
  - Problems with heart valves
  - Causes of heart failure or cardiomyopathy
- May be used to
  - Repair certain types of heart defects
  - Repair a stenotic heart valve
  - Open blocked coronary arteries of grafts

Cardiac Catheterization: Indications

- May identify heart defects or disease
  - CAD
  - Valve problems
  - Ventricular aneurysms
  - Cardiac enlargement

Cardiac Catheterization: Risks

- Hypotension
- Reaction to the contrast medium
- Stroke
- Trauma to the artery caused by hematoma
Cardiac Catheterization: Complications

- Bleeding
- Infection
- Pain and puncture site
- Soft plastic catheters could damage the blood vessels
- Blood clot formation on catheters and that later blocks blood vessels elsewhere in the body
- Contrast material may cause kidney damage
  - Patients with diabetes

Percutaneous Coronary Intervention (PCI)

- Indications
  - Onset of ACS symptoms occurred more than 3 hours earlier
  - Fibrinolytic therapy is contraindicated
  - Patient is at high risk for developing heart failure
  - STEMI diagnosis is not absolute

PCI: Possible Complications

- Bleeding or hematoma from arterial insertion site
- Decreased peripheral perfusion
- Retroperitoneal bleeding
- Cardiac dysrhythmias
- Coronary spasm or MI
- Acute kidney failure
- Stroke
- Cardiac arrest
PCI: ECG Monitoring
- Select lead(s) based on the artery that had the intervention
  - RCA: Use II, III, or aVF
  - LAD: use V1, 4
  - Left Cx: Use I, aVL, V5, or V6

PCI: Diagnostic Testing
- Lab values
- CK-MB
- Cardiac troponin I or T
- Prothrombin time/international normalized ratio (PT/INR)
- Activated partial thromboplastin time (aPTT)
- Hgb & Hct
- Potassium

PCI: Sheath Removal Site Complications
- Bleeding
- Hematoma
- Pseudoaneurysm
- Retroperitoneal bleed
PCI: Reocclusion Symptoms (Early or Late)

- Chest pain
- ECG ST-segment changes
- Shortness of breath
- Diaphoresis
- Nausea

Coronary Artery Bypass Graft (CABG): Indications

- LM artery stenosis of 50% or greater
- Proximal 3-vessel disease (> 50% stenosis of 3 main coronary arteries)
- Multivessel disease with:
  - Decreased LV function
  - UA
  - Chronic stable angina that is lifestyle limiting

CABG: Relative Contraindications

- Lack of adequate conduit
- Coronary arteries distal to the stenosis < 1–1.5 mm
- Severe aortic sclerosis
- Severe LV failure
- Coexisting pulmonary, renal, carotid, and peripheral vascular disease may significantly increase the risk of surgery by predisposing to complications during postoperative period
CABG: Postoperative Complications

Cardiovascular
- Low Cardiac Output Syndrome
  - Decreased preload
  - Increased afterload
  - Cardiac tamponade
  - Myocardial depression
- Dysrhythmias
  - Atrial dysrhythmias in 20%–40% of patients
  - Occur anytime during the first 2–3 days postop with peak incidence days 3–5

CABG: Postoperative Complications

Cardiovascular
- Atrial fibrillation
  - Increased circulating catecholamine
  - Electrolyte or metabolic imbalances
  - Atrial volume and pressure overload
  - Myocardial ischemia or infarction
  - Alterations in autonomic nervous system tone

CABG: Postoperative Complications

Cardiovascular
- Atrial fibrillation
  - If rate exceeds 100, slow rate
    - Diltiazem (Cardizem)
    - Digoxin (Lanoxin)
    - Beta blocker
CABG: Postoperative Complications

- Excessive Bleeding
  - Coagulopathy
  - Uncontrolled HTN
  - Inadequate hemostasis

- Pulmonary
  - Mild pulmonary complications are common after cardiac surgery
  - Increased capillary permeability
  - Increased pulmonary vascular resistance (PVR)
  - Intrapulmonary aggregation of leukocytes and platelets
  - Severe pulmonary dysfunction uncommon
  - May be related to preexisting lung disease
  - Noncardiac pulmonary edema (2-3 days postop)
  - Atelectasis
  - Pneumothorax
  - Phrenic nerve damage
  - Pulmonary emboli

- Renal
  - Reduced glomerular filtration rate (GFR) and renal blood flow
  - 0.1%-0.7% develop acute renal failure
  - Patients with oliguric renal failure after cardiac surgery have a mortality rate of 70%-100%
  - Cardiopulmonary bypass (CPB) reduces GFR by 25%-75%
  - Renal artery vasconstriction
  - Hypothermia
  - Loss of pulsatile perfusion
  - Anesthesia
  - Radiocath
  - Nephrotoxic drugs
CABG: Postoperative Complications

- Neurologic: central nervous system (CNS) dysfunction
  - Long CPB times
  - Perioperative hypotension
  - Preoperative neurological disease
  - Carotid artery disease
  - Cerebral ischemia
  - Cerebral infarction
  - Emboli

CABG: Postoperative Complications

- Postcardiotomy delirium (2–5 days postop)
  - Mild confusion
  - Somnolence
  - Agitation
  - Hallucinations

CABG: Late Postoperative Complications

- Postpericardiotomy syndrome (pericarditis)
- Cardiac tamponade
- Wound infection
Question #7 - Answer

When the patient has bleeding from the catheter insertion site, the most appropriate initial nursing action is to:

A. Call the code team
B. Change the patient’s position
C. Discontinue the anticoagulant
D. Apply direct manual pressure. The most important intervention is to apply direct pressure. The patient’s position should be no greater than 30° head of bed (HOB) elevation. The anticoagulants should be maintained to ensure patency of the coronary vessel.

Question #8 - Answer

After the PCI you are monitoring for evidence of reocclusion. The most likely sign would be:

A. Tachycardia
B. T wave elevation
C. Widened QRS complex
D. ST segment depression. Signs of reocclusion include chest pain, shortness of breath, diaphoresis, and nausea. The ST segment can depress or elevate, and the T wave will invert.
Question #9 - Answer

The emergency treatment for reocclusion is:

A. Heparin
B. Sublingual NTG. The first would be to administer oxygen. A vasodilating drug would then be given. This would include NTG or a calcium channel blocker.
C. Dipyridamole (Persantine)
D. Diazepam (Valium)
Sinus Tachycardia
- Causes
  - Anxiety
  - Exercise
  - Fever
  - Shock

Causes
- Drugs
- Hyperthyroidism
- Electrolyte abnormalities

Sinus Bradycardia
- Causes
  - Well-trained athlete
  - Drugs
  - Hypothyroidism
  - Aging
  - MI

Paroxysmal Atrial Tachycardia (PAT)
- Causes
  - Wolff-Parkinson-White (WPW) syndrome
  - Mitral valve prolapse
  - Ischemic coronary artery disease (CAD)
  - Excessive use of drugs, cigarettes, caffeine, alcohol
Atrial Flutter

- Causes
  - Atrial stretching or enlargement
  - MI
  - Congestive heart failure (CHF)

Atrial Fibrillation (AF)

- Causes
  - Fibrotic changes of aging
  - MI
  - Valvular heart disease
  - Digitalis
  - Post open heart surgery

Premature Ventricular Contractions (PVCs)

- Causes
  - Acute MI
  - Pulmonary diseases
  - Electrolyte imbalance
  - Metabolic instability
  - Drug abuse
Ventricular Tachycardia (VT)

- Causes
  - Acute MI
  - WPW
  - Lactic acidosis
  - Electrolyte disturbances
  - Drug toxicities

Ventricular Fibrillation (VF)

- Causes
  - Acute MI
  - Acidosis
  - Deteriorating VT
  - Electrolyte disturbances

Heart Blocks

- Causes
  - Acute MI
  - CHF
  - Drugs
    - Beta-blocker
Treatment
- Follow current Advanced Cardiovascular Life Support (ACLS) guidelines

Sudden Cardiac Death (SCD)
- Largest cause of natural death in the United States
- 325,000 deaths/year
- One-half of all heart deaths
- Adults mid-30s to mid-40s
- Men 2x more likely than women

Malignant Ventricular Dysrhythmias
- #1 cause of death in the 1st 48 hours after MI
- Similar incidence in NSTEMI and STEMI
- Predictors include:
  - Increased white blood cell (WBC) count
  - Hypokalemia
Malignant Ventricular Dysrhythmias
- Mechanism in SCD
  - Acute coronary thrombosis
  - Primary ventricular dysrhythmia
- Strong predictors of mortality
  - Killip class
  - Creatine kinase (CK) and cardiac troponin
  - LV function

Sudden Cardiac Death
- Treatment
  - CPR
  - Defibrillation
SCD: Risk Factors

- 75% are linked to a previous MI
- Higher during first 6 months after MI
- CAD
  - 80% of SCD cases linked to CAD
  - Risk factors for CAD:
    - Smoking
    - Family history of cardiovascular disease
    - High cholesterol
    - Enlarged heart

Risk factors for CAD:

- Smoking
- Family history of cardiovascular disease
- High cholesterol
- Enlarged heart

SCD: Risk Factors

- Ejection fraction (EF) < 40% combined with VT
- Prior episode of sudden cardiac arrest
- Family history of SCD
- Family history
  - Long QT syndrome
  - WPW
- VT or VF after ACS
- Congenital heart defects
- Syncope

Cardiac Dysrhythmias

SCD: Risk Factors

- Heart failure
  - 6x-9x more likely to experience ventricular dysrhythmias
- Obesity
- Dilated and hypertrophic cardiomyopathy
- Diabetes
- Recreational drug use
  - Cocaine
  - Amphetamines

Cardiac Dysrhythmias

Cardiac Dysrhythmias
SCD in Athletes

- < 35 years of age
  - Congenital heart defects
- > 35 years of age
  - CAD

SCD: Screening

- Physical exam
- Family history
- Exercise stress test
  - Men > 40
  - Women > 50
- Screening every 2 years
  - If history, screening annually

SCD: Reducing Risk Factors

- Stop smoking
- Weight loss
- Regular exercise
- Follow a low-fat diet
- Manage diabetes
- Manage other health conditions
SCD: Reducing Risk – Medications
- Angiotensin-converting enzyme (ACE) inhibitors
- β-blockers
- Calcium channel blockers
- Antidyssrhythmics
- Statins

SCD: Interventions
- Implantable cardioverter-defibrillator (ICD)
  - Preventive
- Interventional procedures or surgery
  - Angioplasty
  - Coronary artery bypass graft (CABG)
  - Ablation

Pacemaker
- A battery-powered device that delivers an electrical stimulus to the myocardium resulting in mechanical contraction

Cardiac Dysrhythmias

Asynchronous or fixed
- Regardless of intrinsic rate

Demand
- Preset rate and will pace if intrinsic rate drops below that preset rate

AV sequential
- Paces in both the atrium and ventricle

Types of Pacing

Conduction disorders
- Symptomatic second-degree heart block
- Symptomatic third-degree heart block

Rate disorders
- Asystole
- Symptomatic bradycardia
- Sick sinus syndrome

Prophylaxis
- Post surgery
- Augment cardiac output (CO)

Indications for Temporary Pacing
Undersensing

- Pacemaker does not sense patient’s intrinsic beats and discharges an impulse regardless of the patient’s own rhythm
- Appears on ECG as pacer spikes that are inappropriate
  - Within the QRS
  - After the QRS
- Pacing regardless of what patient’s own ECG is doing
  - Must decrease the number representing sensitivity on pacer, which will increase sensitivity

Undersensing: Causes

- Battery depletion
- Decreased QRS voltage
- Fusion beat
- Dislodged/fractured lead
- Inappropriate sensitivity setting

Undersensing: Interventions

- Increase sensitivity—lower millivolt (mV)—thus allowing the pacemaker to more readily see the intrinsic cardiac activity (P wave or R wave)
- Correct the underlying problem
- If epicardial pacing (make sure you have 2 leads capable of pacing), may switch the pacing lead and reassess sensitivity threshold
Pacing Example
- What happens here?

Inhibition of the pacemaker by events the pacemaker should ignore
- Electromagnetic interference (EMI)
- T waves
- Myopotential

Oversensing
Oversensing
- Pacemaker senses artifact or other electrical activity as intrinsic cardiac activity and does not fire
- Appears on the ECG as the absence of a pacemaker spike with the absence of the patient’s intrinsic ventricular activity

Oversensing: Causes
- Myopotential inhibition
- EMI
- T waves outside of refractory period
- Dislodged/fractured lead
- Inappropriate sensitivity setting

Oversensing: Interventions
- Check pacemaker connections
- Decrease sensitivity
  - Pacemaker is seeing all kinds of activity as ventricular
  - Decreasing the sensitivity will increase the number on the pacemaker setting so the pacemaker can’t see all the extra electrical activity
Pacemaker Capture
- Milliamps (mA) on the pacemaker
- Immediate depolarization of cardiac muscle following an electrical stimulus

Failure to Capture
- The pacemaker impulse fails to depolarize the atria/ventricles
- Appears on the ECG strip as a pacemaker spike not followed by a P wave or QRS complex (mechanical contraction)

Failure to Capture: Etiology
- Fluid status changes
- Pericardial effusion
- Electrolyte or metabolic abnormalities
- Medications
- Tissue inflammation, fibrosis, or necrosis
- Generator battery failure
- Low pulse generator
- Development of endothelial sheaths
- Disconnection, dislodgement, or fracture of leads
Failure to Capture: Interventions

- Ensure connections are secure
- Recheck pacing threshold; may need to increase the output (mA)
- Turn patient
- Replace pacemaker battery
- Replace pacemaker

Loss of Capture

Practice Exam Questions
A. The exact mechanism for response to magnesium sulfate is not known. There are several causes of torsades including hypomagnesemia, hypokalemia, and hypocalcemia. Many drugs, such as haloperidol, can lead to torsades. In reference to magnesium, a deficiency may enhance early repolarization and predispose the patient to develop this dysrhythmia. Class I antidysrhythmics such as lidocaine may precipitate the onset of torsades and other ventricular dysrhythmias. The magnesium dose generally given is 1–2 gm.

B. 2nd degree AV block
C. Sinus tachycardia
D. Atrial fibrillation

Which of the following dysrhythmias responds to magnesium sulfate?

A. Administer 300 mg amiodarone intravenous (IV) push
B. Establish IV access and sedation for electrical cardioversion
C. Obtain 12-lead ECG
D. Perform immediate electrical cardioversion

A 57-year-old woman has palpitations, chest discomfort, and tachycardia. The monitor shows a regular wide complex QRS at a rate of 180 beats/minute. She becomes diaphoretic with a blood pressure (BP) of 80/60. The priority intervention would be to:

A. Administer 300 mg amiodarone intravenous (IV) push
B. Establish IV access and sedation for electrical cardioversion
C. Obtain 12-lead ECG
D. Perform immediate electrical cardioversion

This rhythm strip shows:

A. Undersensing
B. Oversensing
C. Failure to capture
D. 100% AV paced. The rhythm is regular. There is an atrial pacemaker spike followed by a P wave and a ventricular pacing spike followed by a QRS.
Question #4 - Answer

This rhythm strip shows:

A. Ventricular paced with undersensing. The pacer spike is followed by a QRS complex; however, the rate is irregular. The pacer sensed the artifact at the arrow as an intrinsic beat and inhibited the pacer firing. Decrease the sensitivity.

B. Ventricular paced with oversensing

C. AV paced with undersensing

D. AV paced with oversensing

Question #5 - Answer

This rhythm strip shows:

A. Undersensing. The first 5 beats are ventricular paced. The fifth is a pacer spike without capture. The sixth beat is an intrinsic beat. The last 2 beats are ventricular paced again. Have the patient turn or cough. Check the battery.

B. Oversensing

C. Failure to capture

D. 100% AV paced

Question #6 - Answer

This rhythm strip shows:

A. Ventricular paced with undersensing

B. Ventricular paced with oversensing

C. AV paced with undersensing. The pacer is functioning properly for the first 2 complexes. The patient has a PAC. The pacemaker did not sense this and discharged a ventricular pacemaker spike as scheduled right in the middle of the T wave. So, the pacemaker undersensed the patient’s intrinsic rhythm.

D. AV paced with oversensing
Therapeutic Hypothermia

Cardiac Arrest: Epidemiology
- Out-of-Hospital Cardiac Arrest
  - 64% of all arrests
  - 2%–9% survive to discharge
  - 1/3 of survivors have irreversible cognitive dysfunction
- In-Hospital Cardiac Arrest
  - 36% of all arrests
  - 18% survival to discharge

Phases of Therapeutic Hypothermia
- Induction
  - Goal is to get patient to target body temperature as quickly as possible
  - Use ice packs, iced lavage, rapid cold fluid infusion, noninvasive cooling devices, or an intravascular catheter that circulates cold fluid in a closed loop within a large vein
  - Sedation and neuromuscular blockade (NMB) when the cooling process begins, to prevent shivering
Cold Diuresis
- Results from increased venous return stemming from vasoconstriction, decreased antidiuretic hormone levels, and tubular dysfunction
- Leads to increased urine output — up to several liters in 1–2 hours

Induction Phase

Maintenance Phase
- Controlling the patient’s temperature within the target range is crucial
  - Usually 32°C to 34°C
  - Can last up to 24 hours from the time the target temperature is reached

Rewarming Phase
- Temperature control remains important during rewarming
- Warming the patient too quickly or allowing continued shivering causes dangerous electrolyte shifts, leading to potentially lethal dysrhythmias
- Controlled rewarming of 0.15°C to 0.5°C per hour is recommended
- Electrolytes shift out of the cells back into the serum during rewarming
  - Frequent electrolyte monitoring is needed during this phase to prevent critically elevated levels
Rewarming Phase

- Slow controlled rewarming allows the kidneys to excrete excess potassium, preventing hyperkalemia.
- May become hypoglycemic during rewarming as the insulin resistance of earlier hypothermia phases diminishes.
- Careful fluid monitoring during rewarming is crucial because of the vasodilation that accompanies a rise in body temperature:
  - Volume replacement may be needed to prevent fluid deficit and hypotension.

Identification of Eligible Patients

- Comatose survivors after out-of-hospital cardiac arrest with a primary rhythm of VT/VF regardless of presence of shock.
- Hypothermia should be considered for non-VF rhythms and in-hospital cardiac arrest.
- < 60 minutes CPR prior to return of spontaneous circulation (ROSC).
- Prearrest Glasgow coma scale (GCS) = 15 or independent activities of daily living (ADLs).

Ineligible Patients

- Written do not resuscitate (DNR)/do not intubate (DNI).
- Cognitive status severely impaired before arrest.
- Underlying coagulopathy or bleeding disorder.
- Other known reason for coma/arrest (e.g., septic shock, severe acidosis, trauma, etc.).
Ineligible Patients

- Questionable head injury or head computed tomography (CT) with mass or hemorrhage
- Unstable cardiac rhythms not terminated during initial management
- Core body temperature below 30°C before initiation of therapy
- Length of downtime > 60 minutes
- Time elapsed from ROSC ≥ 12 hours

Cooling Induction

If eligible, page team “ice”
- Similar to STEMI team
- Prior to cooling
  - Intubate patient
  - Insert arterial pressure monitoring line
  - Insert central venous catheter (CVC) or central venous oxygen saturation (ScvO₂) catheter
  - Insert temperature-sensing urinary catheter
  - Sedate with IV midazolam and fentanyl
  - Paralyze with cisatracurium (Nimbex®) to prevent shivering

Cooling Induction

- Target temperature and duration
  - 32°C to 34°C for 24 hours after reaching the target
  - Goal = 6 hours to target temperature
- Methods of induction
  - Ice-cold lactated ringers (LR) or normal saline (NS)
  - 30 mL/kg with pressure bags via large bore cannula
  - Avoid in patients with pulmonary edema or severely reduced LV systolic function
  - Combine with cooling device
Hypothermia and STEMI

- Proceed to catheterization lab for PCI with continuation/induction of cooling
- Continuous temperature measurement must be provided
- Follow American College of Cardiology (ACC)/American Heart Association (AHA) guidelines

Therapeutic Hypothermia

Maintenance

- Maintain temperature at 32°C to 34°C for 24 hours after reaching the target
- Check water level of cooling device
- Refill with distilled water if needed
- Nursing to monitor temperature, mean arterial pressure (MAP), CVP, ScvO₂ hourly
- If pulmonary artery (PA) catheter in place, also monitor systemic vascular resistance (SVR) and cardiac index (CI)
- Labs every 6 hours
  - Basic metabolic profile (BMP)
  - Complete blood count (CBC)
  - Troponin/CK/K-MB
  - ABGs — temperature corrected

Therapeutic Hypothermia

Maintenance: Side Effect Monitoring

- Bradycardia has higher risk of temperature < 30°C
- Lidocaine if recurrent VT/VF
- Closely monitor for infection
  - No evidence of prophylactic antibiotics despite higher rates of sepsis and pneumonia
- Closely monitor for electrolyte imbalance
- Potentially higher bleeding complications after PCI
  - Platelet function unaltered by hypothermia
**Maintenance: Side Effect Monitoring**

- Altered drug action and metabolism
  - Reduces systemic clearance of cytochrome P450 metabolized drugs 7%–22% per °C
- Paralyze to prevent shivering
- Neurological checks every 2 hours — monitor for:
  - Decerebrate or decorticate posturing
  - Change in pupil symmetry
  - Seizure activity

**Rewarming**

- Goal is to rewar over 6–8 hours
- Using cooling device — increase warming setting by 0.5°C every 1–2 hours
- Discontinue when patient reaches 36°C
- Maintain normothermia (36.5°C–37.5°C)
  - Up to 72 hours after cardiac arrest

**Adverse Effects: Dysrhythmias**

- Bradycardia, AV blocks, AF and VF may occur
- Hypothermia may render atropine ineffective in bradycardia
  - Transcutaneous or transvenous pacing can be used to treat symptomatic bradycardia
  - Other ACLS protocols can be used effectively
Adverse Effects: Insulin Resistance

- Hypothermia causes insulin resistance, commonly leading to hyperglycemia
- Monitor glucose levels
- Administer insulin as ordered

Adverse Effects: Shivering

- Shivering increases metabolic activity and enhances oxygen consumption and rewarming
- Adequate sedation and counter-warming of extremities helps control shivering

Shivering Assessment

<table>
<thead>
<tr>
<th>Score</th>
<th>Type of Shivering</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>None</td>
<td>No shivering is detected on palpation of the masseter, neck, or chest muscles</td>
</tr>
<tr>
<td>1</td>
<td>Mild</td>
<td>Shivering localized to the neck and thorax only</td>
</tr>
<tr>
<td>2</td>
<td>Moderate</td>
<td>Shivering involves gross movement of the upper extremities (in addition to neck and thorax)</td>
</tr>
<tr>
<td>3</td>
<td>Severe</td>
<td>Shivering involves gross movements of the trunk and upper &amp; lower extremities</td>
</tr>
</tbody>
</table>

Medications to Control Shivering
- Neuromuscular blockers
- Meperidine
- Fentanyl
- Magnesium sulfate
- Propofol
- Dexmedetomidine
- Buspirone

Therapeutic Hypothermia

Adverse Effects: Coagulation Problems
- Mild platelet dysfunction may arise during hypothermia, increasing the bleeding risk
- Most patients don’t exhibit bleeding problems other than oozing from central or other invasive lines
- If bleeding problems occur, increase the target temperature on the cooling device from 33°C to 34°C, which alleviates most platelet dysfunction
- Blood products may be given
- Monitor Hgb, PT, aPTT

Therapeutic Hypothermia

Adverse Effects: Pain and Sedation Management
- How to assess for discomfort in a comatose patient who's cold
- Hypothermia diminishes the body’s ability to respond to stimulation
- Bispectral index (BIS) monitors found to be less reliable in hypothermic than normothermic patients
- Continuous electroencephalographic (EEG) monitoring can be used to monitor the sedation level and detect seizures

Therapeutic Hypothermia
Adverse Effects: NMB Monitoring

- In hypothermia, intracellular electrolyte shifts and the cold itself can make train-of-four (TOF) monitoring less accurate
- Cooling slows nerve conduction, so TOF monitoring at temperatures used for therapeutic hypothermia isn’t reliable

Adverse Effects: Drug Metabolism

- Reduced clearance of some drugs commonly used in intensive care patients undergoing therapeutic hypothermia
  - Epinephrine, norepinephrine, morphine, fentanyl, propofol, midazolam, barbiturates, rocuronium, vecuronium, phenytoin, nitrates, and certain β-blockers
- Hypothermia-induced changes in volume and renal function may play a part in drug metabolism

Adverse Effects: Inflammatory Response

- Therapeutic hypothermia suppresses the inflammatory response, increasing the risk of infection
  - Scrupulous hand hygiene
  - Provide meticulous care to prevent hospital-associated infections (HAIs)
    - Catheter-related bloodstream infections (CRBSIs)
    - Ventilator-associated pneumonia (VAP)
    - Catheter-associated urinary tract infections (CAUTIs)
Practice Exam Questions

Therapeutic Hypothermia

Question #1 - Answer

The mechanism that most likely places the patient at risk for hyperkalemia is:

A. Overcooling
B. Overtreatment of hypokalemia during the cooling phase
C. Undertreatment of hypokalemia during the cooling phase
D. Cold diuresis

Question #2 - Answer

Administration of 100% oxygen (O₂) during the 1st hour following cardiac arrest can harm the patient by:

A. Increasing the risk of dysrhythmias
B. Causing oxidative stress to post-ischemic neurons
C. Harm ing the cerebrum during reperfusion
D. Decreasing renal function during reperfusion
**Question #3 - Answer**

Which patient is a candidate for induction of hypothermia after cardiac arrest?

A. Patient with ROSC who is responsive and alert  
B. Patient with life-threatening dysrhythmias  
C. Pregnant patient with ROSC within 60 minutes  
D. Witnessed cardiac arrest with ROSC and downtime < 15 minutes

**Question #4 - Answer**

The signs and symptoms of shivering that should be monitored during therapeutic hypothermia include:

A. Increase in venous oxygen saturation  
B. Decrease in respiratory rate  
C. Palpation of muscle fasciculation on face or chest  
D. Development of paronychia