Nursing Science

Management of the Patient in the Cardiac Care Unit

Learning objectives

After completing this lecture, the students will be able to:

- **Describe the electrocardiographic (ECG) monitoring in the CCU.**
- **Identify the nursing interventions for inpatient cardiac monitoring.**
- **Explain the most common management for patient with ACS.**

Management of the patient in the cardiac care unit:

Anatomy of the Heart.

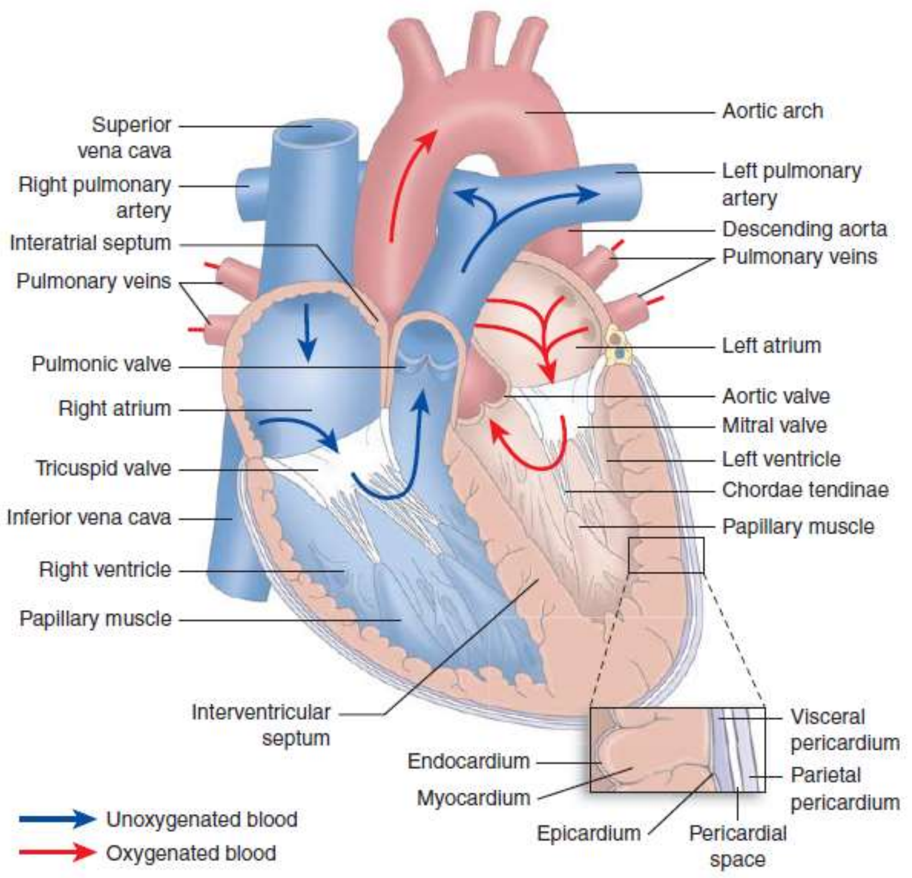


FIGURE 27-1. Structure of the heart. Arrows show course of blood flow through the heart chambers.

Electrocardiographic (ECG) monitoring in the CCU:

- The ECG is a graphic representation of the electrical currents of the heart. The ECG is obtained by placing disposable electrodes in standard positions on the skin of the chest wall and extremities
- Recordings of the electrical current flowing between two electrodes is made on graph paper or displayed on a monitor.
- Several different recordings can be obtained using various electrode combinations, called leads. Simply stated, a lead is a specific view of the electrical activity of heart. The standard ECG is composed of 12 leads or 12 different views

❖ Normal Electrical Conduction:

- The primary pacemaker of the heart is the sinoatrial (SA or sinus) node, located where the superior vena cava enters the right atrium.

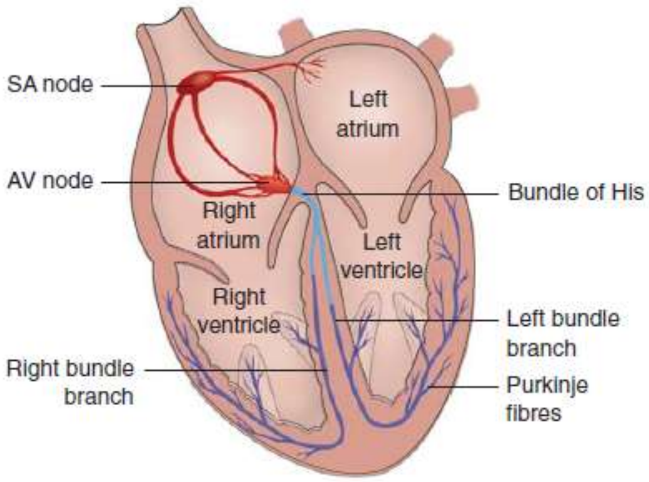


FIGURE 27-3. Cardiac conduction system. AV, atrioventricular; SA, sinoatrial.

- In adults, it usually discharges impulses at a regular rate of 60 to 100 times per minute, the "normal" heart rate.
- The impulse then spreads throughout the atria via the interatrial pathways. These conduction pathways converge and narrow through the atrioventricular (AV) node, slightly delaying the transmission of the impulse to the ventricles
- The electrical stimulation is called depolarization, and the mechanical contraction 4 is called systole. Electrical relaxation is called repolarization, and mechanical 5 relaxation is called diastole.

❖ Waves, Complexes, and Intervals

- The P wave represents the electrical impulse starting in the sinus node and spreading through the atria. P wave represents atrial depolarization. It is normally 2.5 mm or less in height and 0.11 seconds or less in duration.
- The QRS complex represents ventricular depolarization. The QRS complex is normally less than 0.12 seconds in duration.
- The **T wave** represents ventricular repolarization (when the cells regain a negative charge; also called the resting state).
- The **U wave** is thought to represent repolarization of the Purkinje fibers, but it sometimes is seen in patients with hypokalemia (low potassium levels), hypertension, or heart disease. If present, the U wave follows the T wave and is usually smaller than the P wave.
- The **PR interval** is measured from the beginning of the P wave to the beginning of the QRS complex, the PR interval normally ranges from 0.12 to 0.20 seconds in duration.
- The **ST segment,** lasts from the end of the QRS complex to the beginning of the T wave.
- The **QT interval**, which represents the total time for ventricular depolarization and repolarization, is measured from the beginning of the QRS complex to the end of the T wave. The QT interval is usually 0.32 to 0.40 seconds in duration if the heart rate is 65 to 95 bpm

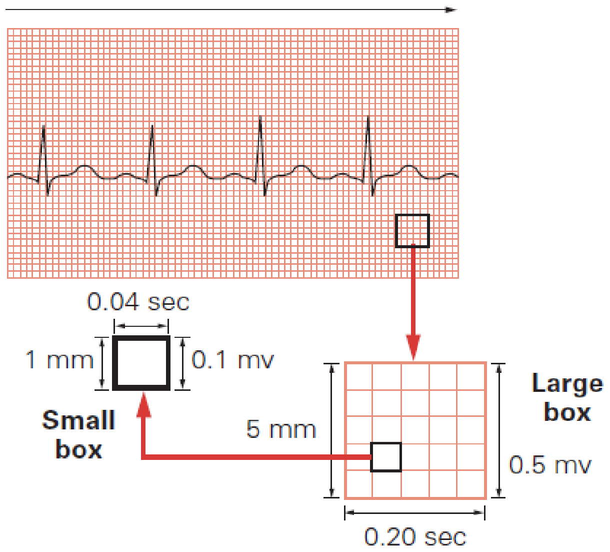


Figure 2.12 ■ ECG graph paper values.

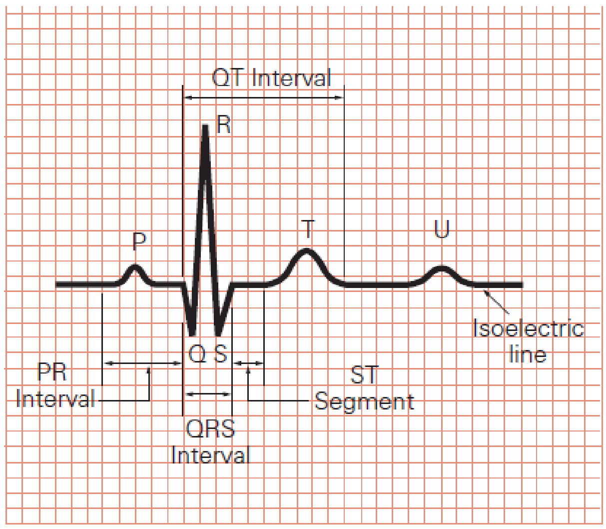


Figure 2.13 ■ The electrical pattern of the cardiac cycle shows waves and intervals.

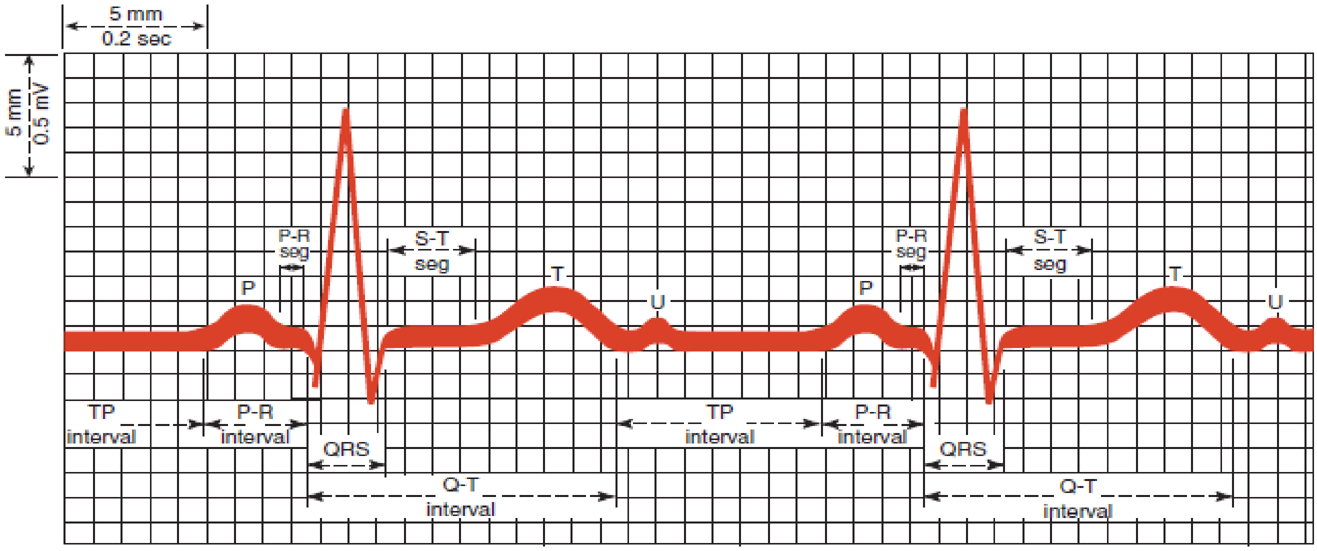


FIGURE 28-3. ECG graph and commonly measured components. Each small box represents 0.04 seconds on the horizontal axis and 1 mm or 0.1 millivolt on the vertical axis. The PR interval is measured from the beginning of the P wave to the beginning of the QRS complex; the QRS complex is measured from the beginning of the Q wave to the end of the S wave; the QT interval is measured from the beginning of the Q wave to the end of the T wave; and the TP interval is measured from the end of the T wave to the beginning of the next P wave.

❖ Determining Heart Rate from the Electrocardiogram

The heart rate can be obtained from the ECG strip by several methods:

- A 1-minute strip contains 300 large boxes and 1,500 small boxes. Therefore, an easy and accurate method of determining heart rate with a regular rhythm is to count the number of small boxes within an RR interval and divide 1,500 by that number. If, for example, there are 10 small boxes between two R waves, the heart rate is 1,500/10, or 150; if there are 25 small boxes, the heart rate is 1,500/25, or 60.
- The method for estimating heart rate, which is usually used when the rhythm is irregular, is to count the number of RR intervals in 6 seconds and multiply that number by 10. The top of the ECG paper is usually marked at 3-second intervals, which is 15 large boxes horizontally.
- The same methods may be used for determining atrial rate, using the PP interval instead of the RR interval.

❖ Determining heart rhythm from the electrocardiogram:

- The rhythm is often identified at the same time the rate is determined
- The RR interval is used to determine ventricular rhythm, and the PP interval is used to determine atrial rhythm.

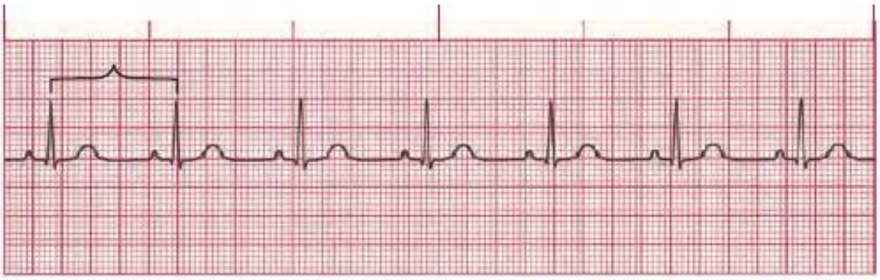


FIGURE 25.11 Rhythm regular: Count the small squares between two of the R waves and divide into 1500: 1500/25 = 60 beats per minute. Alternatively, count the large squares between two of the R waves and divide into 300: 300/5 = 60 beats per minute. Modified from Jones, S. A. (2008). *ECG success: Exercises in ECG interpretation*. Philadelphia: F.A. Davis, with permission.

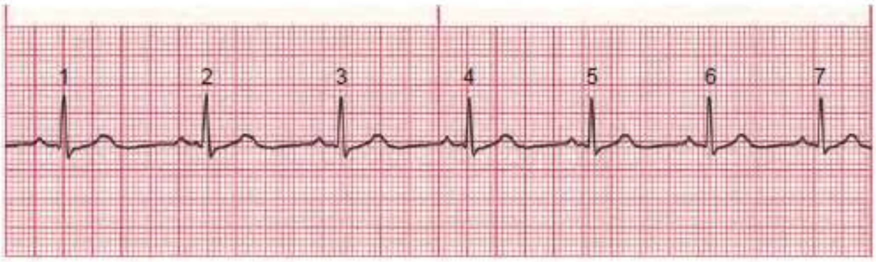


FIGURE 25.12 Rhythm irregular: Counting R waves in a 6-second strip. There are six R waves in this 6-second strip, and 6 × 10 = 60 beats per minute. Modified from Jones, S. A. (2008). *ECG success: Exercises in ECG interpretation*. Philadelphia: F.A. Davis, with permission.

Interventions for Inpatient Cardiac Monitoring

- Continuous ECG monitoring is the standard of care for patients who are at high risk for dysrhythmias. This form of cardiac monitoring detects abnormalities in heart rate and rhythm.
- To minimize false alarms, the ECG recordings must be free of artifact, which is a 24 **abnormal ECG pattern caused by muscular activity, patient movement, electrical interference, or lead cable or electrode malfunction**.
- Artifact can mimic arrhythmias and cause unnecessary false alarms
- Before placing the electrodes and leads on a patient, the nurse must properly prepare the patient's skin **before applying electrodes** and **changing the electrodes every 24 hours**
- During electrode changes, the skin should be assessed for allergic responses to the adhesive or electrode gel
- Excessive hair on the chest wall should be clipped with scissors. The nurse should prepare the skin by rubbing gently with dry gauze until the skin is slightly pink. If the skin is oily, alcohol may be used first. For a diaphoretic patient, a skin protectant should be applied before the electrode secured.

- The electrodes on the patient may need to be replaced or moved to areas that are less affected by movement and rotation of electrode placement will reduce the risk for skin breakdown
- Improper positioning of electrodes and lead can result in artifact.
- Two leads should be selected that provide the best tracing for arrhythmia monitoring. These are usually lead II and chest lead V1.
- An effort should be made to individualize the ECG alarm parameters to meet the 9 patient's monitoring needs. For example, if the patient has atrial fibrillation, it is appropriate to turn off the irregular heart rate alarm.
- Similarly, the bradycardia and tachycardia alarms should be adjusted, slightly below or above the patient's underlying heart rate.
- A patient should never be connected to monitoring equipment that has not been thoroughly cleaned between patients.
- Never placed electrodes over implanted device, open wounds, or inflamed skin.
- Electrodes should be removed once monitoring is discontinued and skin cleansed to remove excess electrode gel and adhesive.

Most Common Heart Diseases:

- The term **acute coronary syndrome** (ACS) is used to encompass the continuum of CAD, such as unstable angina pectoris and MI.
- Acute coronary syndrome is caused by a sequence of inflammatory processes mediated by activated macrophages, plaque rupture, tissue factor expressions, and platelet activation leading to thrombus formation and coronary vessel occlusion.
- The terms coronary occlusion and heart attack are used synonymously.
- This syndrome encompasses the spectrum of unstable angina, non–ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI).

❖ Pathophysiology of unstable angina

- Angina is usually caused by atherosclerotic disease. Almost invariably, angina is associated with an obstruction of a major coronary artery.
- Symptoms of angina usually occur when an artery is narrowed by at least 60% to 70%.
- Rest does not decrease the chest pain of unstable angina. This pain may even occur when the patient is at rest. The episodes of chest pain with unstable angina increase in frequency and severity, placing the patient at risk for myocardial damage or sudden death.

❖ Pathophysiology of myocardial infarction (MI):

- MI is usually caused by reduced blood flow in a coronary artery due to rupture of an atherosclerotic plaque and subsequent occlusion of the artery by a thrombus.
- MI includes vasospasm (sudden constriction or narrowing) of a coronary artery, decreased oxygen supply (e.g., from acute blood loss, anemia, or low blood pressure), and increased demand for oxygen (e.g., from a rapid heart rate, thyrotoxicosis, or ingestion of cocaine).
- Imbalance exists between myocardial oxygen supply and demand
- The area of infarction develops over minutes to hours.

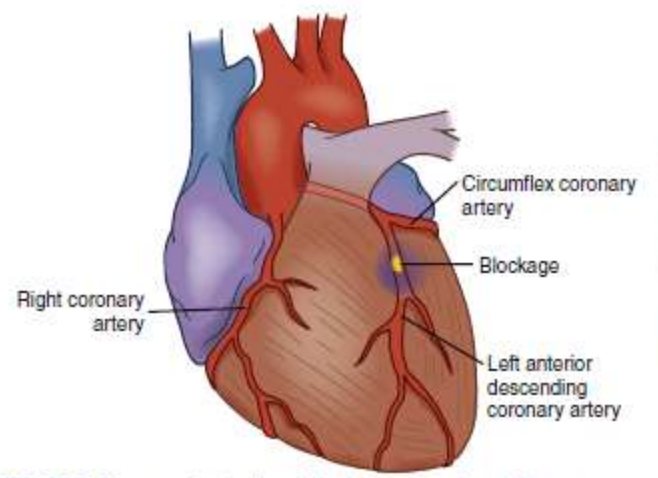
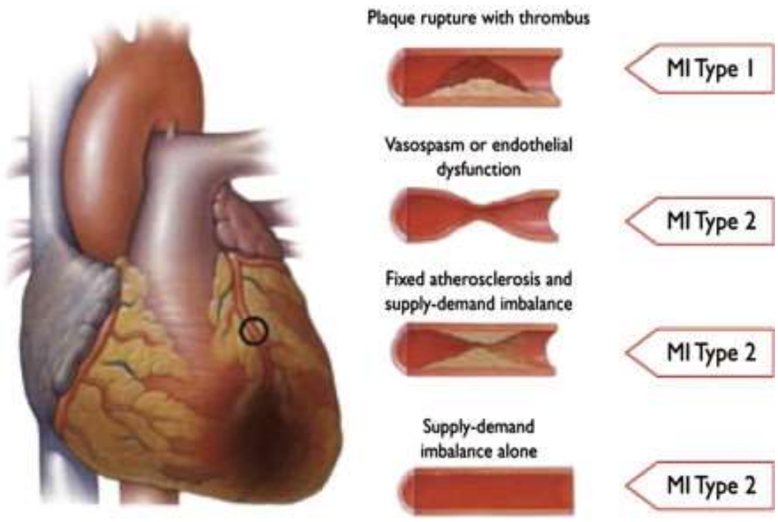


FIG. 36.9 Diagram of occlusion of the left anterior descending coronary artery, resulting in a myocardial infarction.



Clinical Manifestations:

- Chest pain that occurs suddenly and continues despite rest and medication
- Patients may present with a combination of symptoms, including chest pain, shortness of breath, indigestion, nausea, and anxiety, cool, pale, and moist skin
- Their heart rate and respiratory rate may be faster than normal
- In many cases, the signs and symptoms of MI cannot be distinguished from those of unstable angina.

Assessment and diagnostic findings

- The diagnosis of MI is generally based on the presenting symptoms
- The ECG provides information that assists in diagnosing acute MI. It should be obtained within 10 minutes from the time a patient reports pain or arrives in the emergency department.

- This change in the ST segment in two contiguous leads is a key diagnostic indicator for MI, classified as non–ST-elevated MI (depressed or normal ST) and ST-elevated MI.
- The injured myocardial cells depolarize normally but repolarize more rapidly than normal cells, causing rise ST segment
- Q waves develop within 1 to 3 days because there is no depolarization current conducted from necrotic tissue. The lead system then views the flow of current from other parts of the heart.

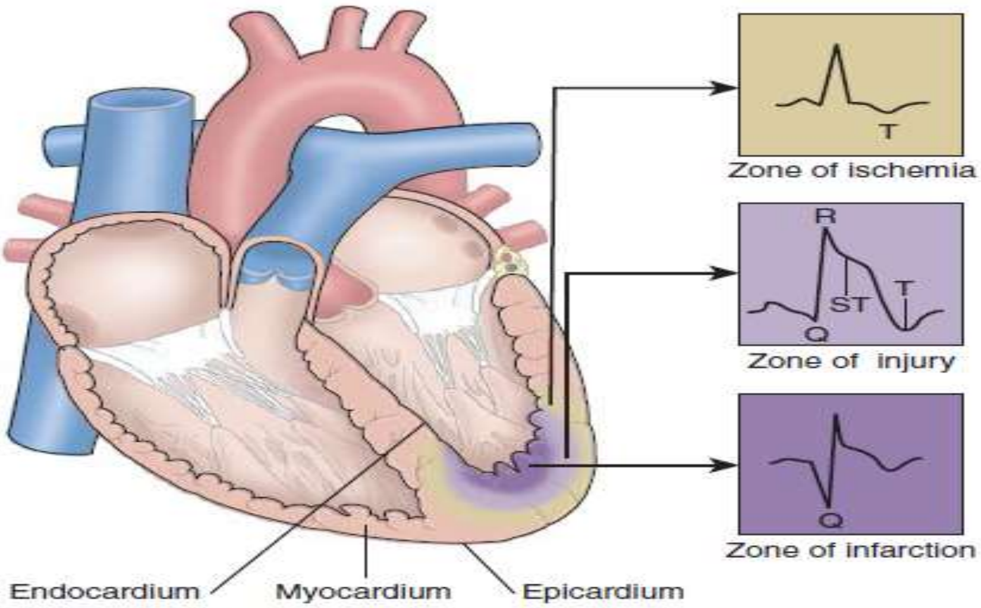


FIGURE 29-5. Effects of ischemia, injury, and infarction on an electrocardiogram recording. Ischemia causes inversion of the T wave because of altered repolarization. Cardiac muscle injury causes elevation of the ST segment and tall, symmetrical T waves. Later, Q waves develop because of the absence of depolarization current from the necrotic tissue and opposing currents from other parts of the heart.

Laboratory Tests

- Laboratory tests called cardiac biomarkers are used to diagnose an MI.
- These markers, specifically serum cardiac enzymes and troponin, are important in the diagnosis of MI. When cardiac cells die, their intracellular enzymes are released into circulation. The increase in serum cardiac markers that occurs after cellular death. CK and troponin are typically measured to diagnose an MI
- An increase in the level of troponin in the serum can be detected within a few hours during acute MI. It remains elevated for a long period, often as long as 3 weeks, and it therefore can be used to detect recent myocardial damage.
- Elevated CK-MB assessed by mass assay is an indicator of acute MI; its level begins to increase within a few hours and peaks within 24 hours of an MI.

- The myoglobin level starts to increase within 1 to 3 hours and peaks within 12 hours after the onset of symptoms. An increase in myoglobin is not very specific in indicating an acute cardiac event; however, negative results are an excellent parameter for ruling out an acute MI.

Medical Management:

✓ **The goal of medical management is:**

- To minimize myocardial damage
- Preserving myocardial function, and
- Prevent complications.

✓ **Medical Management of patient with ACS:**

- Obtain 12-lead electrocardiogram to be read within 10 minutes.
- Obtain laboratory blood specimens of cardiac biomarkers, including troponin.
- **Begin routine medical interventions:**
  1. **Supplemental oxygen**
  2. **Nitroglycerin**
  3. **Morphine**
  4. **Aspirin 162 to 325 mg**
  5. **Beta-blocker**
  6. **Angiotensin-converting enzyme inhibitor within 24 hours**
  7. **Anticoagulation with heparin and platelet inhibitors. 13**
  8. **Statin.**

✓ **Angiotensin-Converting Enzyme (ACE) Inhibitors:**

- Angiotensin-converting enzyme (ACE) inhibitors prevent the conversion of angiotensin I to angiotensin II. In the absence of angiotensin II, the blood pressure decreases and the kidneys excrete sodium and fluid (diuresis), decreasing the oxygen demand of the heart.
- It is important to ensure that the patient is not hypotensive, hyponatremic, hypovolemic, or hyperkalemic before administering ACE inhibitors
- Blood pressure, urine output, and serum sodium, potassium, and creatinine levels need to be monitored closely.

✓ **Administration of Fibrinolytics & anticoagulants for patient with ACS:**

- Fibrinolytics is to dissolve and lyse the thrombus in a coronary artery (thrombolysis), allowing blood to flow through the coronary artery again (reperfusion), minimizing the size of the infarction, and preserving ventricular function. Hospitals monitor their ability to administer these medications within 30 minutes from the time the patient arrives in the emergency department. Eg., alteplase (Activase, tissue plasminogen activator (t-PA).

- Aspirin and unfractionated heparin may be used with t-PA to prevent another clot from forming at the same lesion site.

✓ Pain management for patients with ACS

- Morphine sulfate was administered in IV boluses to reduce pain and anxiety. It reduces preload and afterload, which decreases the workload of the heart also relaxes bronchioles to enhance oxygenation
- The cardiovascular response to morphine is monitored carefully, particularly the blood pressure, which can decrease, and the respiratory rate, which can be depressed.
- Ensure physical rest: use of the bed side commode with assistance; back rest elevated to promote comfort;
- Assist with alternative pain relief measures (positioning, and relaxation).
- Obtain ECG. 10
- Administer O2 on need.

References

1. Harding, M., Kwong, J., Roberts, D., Hagler, D., &Reinisch, C., (2023). LEWIS'S Medical-Surgical Nursing in Canada Assessment and Management of Clinical Problems. Fifth edition. Elsevier Canada Pp: 715-925.

2. Smeltzer, C., Bare, G., Hinkle, L., & Cheever, H., (2016). Brunner and Suddath's textbook of Canadian medical-surgical nursing.3th edition. Wolters Kluwer, Lippincott Williams and Wilkins China., Pp 717- 863

3. Williams, L., and Hopper, P. (2015). Understanding medical surgical nursing. Fifth edition. F.A. Davis Company. USA. Pp 433-519