CriticalCareNurse
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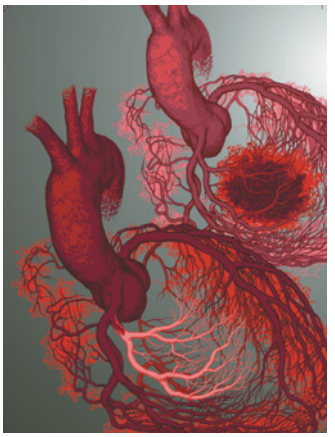
Crit Care Nurse 2002;22:15-26

Published online http://www.cconline.org

Critical Care Nurse is the official peer-reviewed clinical journal of the American Association of Critical-Care Nurses, published bi-monthly by The InnoVision Group 101 Columbia, Aliso Viejo, CA 92656. Telephone: (800) 899-1712, (949) 362-2050, ext. 532. Fax: (949) 362-2049.

AMERICAN ASSOCIATION of CRITICAL-CARE NURSES

Myocardial Injury: Contrasting Infarction and Contusion



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CE

This article has been designated for CE credit. A closed-book, multiple-choice examination follows this article, which tests your knowledge of the following objectives:

1. Discuss and contrast the pathophysiology of myocardial infarction and myocardial contusion
2. Identify diagnostic tests that are used in the diagnosis of myocardial infarction and myocardial contusion
3. Distinguish signs and symptoms associated with myocardial infarction and myocardial contusion

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Acute myocardial infarction (AMI) and myocardial contusion both result in injury to the heart. The underlying pathophysiology, clinical findings, and interventions, however, differ markedly between the 2 disorders. Knowledge of these aspects can assist critical care nurses in facilitating healing, preventing complications, and providing accurate information to patients and patients' families. Understanding the differences between AMI and myocardial contusion is particularly important when caring for a patient who has experienced both problems.

In this article, we present 3 case studies to contrast AMI and myocardial contusion: H.N. with an AMI of the inferior wall, S.B. with a myocardial contusion, and L.R. with both an AMI and a myocardial contusion. Each of these patients is discussed in relation to his or her particular myocardial injury.

MYOCARDIAL INFARCTION

H.N., a 57-year-old man, arrived in the emergency department with "excruciating" chest pain that he rated as 8 on a scale of 0 to 10 in which 0 equals no pain. Triage decisions were based on the patient's history (pain, trigger events, and risk factors), results of initial diagnostic tests (expanded-lead electrocardiography [ECG] and measurement of creatine kinase–MB [CK-MB] level), and findings on physical assessment. We selected H.N.'s case to contrast the differences in pathophysiology between inferior AMI and myocardial contusion and the differences between the two in interventions for changes in hemodynamic status and heart rhythms.

Pathophysiology

Myocardial infarction is due to extended ischemia and, if left untreated, leads to "ultimate cell necrosis."[1] Causes of AMI include thrombus formation, arterial spasm, and embolism; the primary cause is formation of a thrombus in the coronary artery. Although the mechanism of thrombus formation

is not entirely understood, thrombi tend to form on unstabilized, ruptured atherosclerotic plaque. When a thrombus blocks blood flow to the myocardial muscle, muscle damage extends rapidly from endocardium to epicardium. Unless adequate blood supply is restored, necrosis of the myocardium may become complete and irreversible within 3 to 4 hours.[2] Remodeling of the ventricle through expansion and dilatation of the infarct zone may continue for weeks, months, and years after the infarction.[3] Most of the therapy and interventions for AMI are targeted toward restoring blood flow, reducing the risk of reinfarction, reducing oxygen demands on the injured ventricle, and altering dynamics to limit ventricular remodeling.[2,4] Early administration of thrombolytic agents is one of the first-line interventions to establish reperfusion of an occluded artery.[5]

History: Risk Factors, Pain, and Trigger Events

H.N.'s known cardiac risk factors were his 20-year history of hypertension, his elevated cholesterol levels, and his male sex. He stated that he had experienced "some discomfort" in his chest earlier in the morning. In the afternoon while he was vacuuming, the pain increased, and he became diaphoretic and nauseated. He rested until his son came home and took him to the hospital's emergency department, 5 hours after the onset of the severe pain. H.N.'s history is consistent with the experience of AMI. It is not unusual for pain or discomfort to start gradually just before a myocardial infarction, and nausea is often associated with an AMI.[6] Triggering events, such as emotional stress or strenuous physical activity, occur in approximately 50% of persons who experience an AMI.[7]

Initial Diagnostic Tests: 12-Lead ECG, Measurement of CK-MB Levels

H.N.'s initial 12-lead ECG (Figure 1A) showed ST-segment elevations in the inferior leads II, III, and aVF. ST-segment elevation equal to or greater than 0.1 mV in 2 or more contiguous leads is considered indicative of AMI. An expanded ECG, obtained with either 15 or 18 leads, is recommended for all patients with inferior myocardial infarctions to check for associated right ventricular or posterior involvement.[7-9] In this case, a right-sided 12-lead ECG (Figure 1B) showed no right ventricular ischemia or injury.

H.N.'s creatine kinase level, level of isoenzymes (CK-MB), and relative index during the first 24 hours are given in Table 1. His CK-MB levels followed the predictable pattern of an AMI; they increased sharply and declined just as quickly, producing a pattern somewhat like a bell-shaped curve. After an AMI, the CK-MB level is markedly elevated above normal within 4 to 6 hours of the onset of signs and symptoms, maximum levels are reached between 18 and 24 hours, and levels return to normal in 72 hours.[10] If creatine kinase levels do not follow this expected pattern within the first 48 hours, it may be assumed that the person did not have a myocardial infarction. A secondary increase in

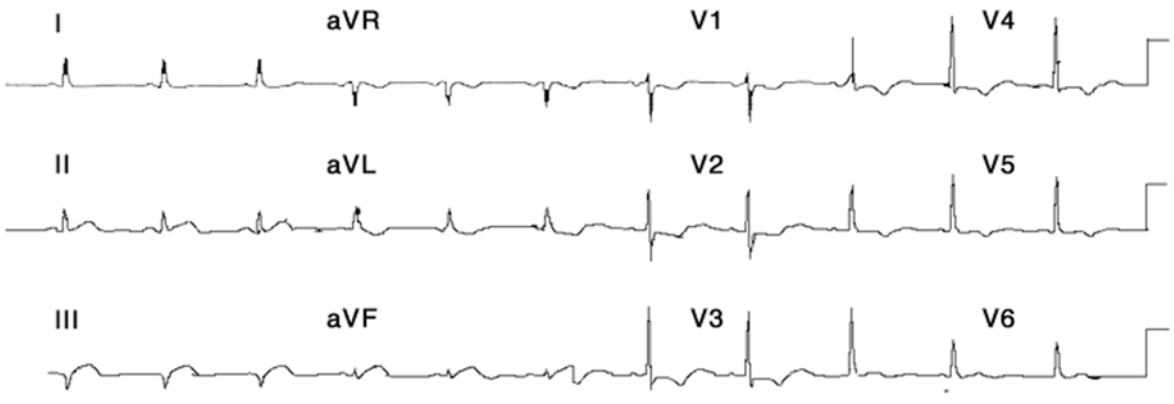

Figure 1A Initial 12-lead electrocardiogram of a patient with acute myocardial infarction.

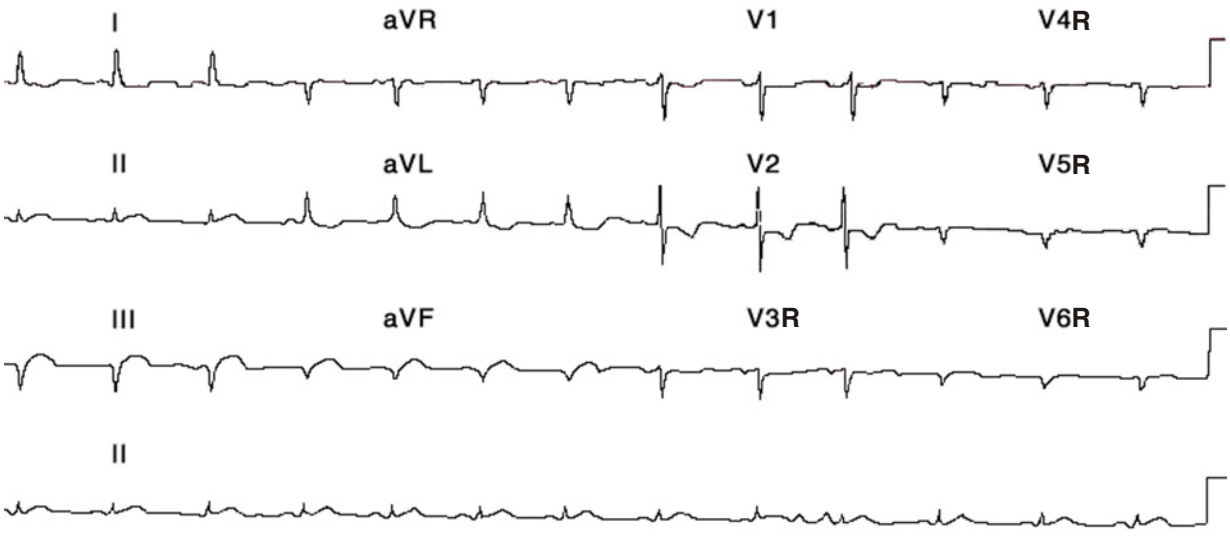

Figure 1B Right sided 12-lead electrocardiogram of a patient with acute myocardial infarction.

CK-MB level on the downward slope of the first increase in CK-MB level indicates a reinfarction.

Because of the rapid washout of enzymes from the serum after thrombolytic therapy, CK-MB levels are expected to increase and peak more quickly when thrombolysis-induced reperfusion has been effective.[10] For H.N., treatment with tissue plasminogen activator resulted in successful reperfusion; maximum plasma levels of creatine kinase were reached 10 to 15 hours after the infarction. The relative index, the relationship between the plasma level of CK-MB and the total plasma level of circulating creatine kinase, is useful in ruling out nonmyocardial sources of CK-MB.[11] The index is calculated by dividing CK-MB level (in micrograms per liter) by the total concentration of creatine kinase (in units per liter) and multiplying by 100. A relative index greater than 4.0 indicates myocardial damage.

Measurement of cardiac troponin levels is an additional diagnostic test for AMI. Troponin T and troponin I are released from myocardial cells within 3 to 5 hours after the onset of ischemia. The specificity of troponin I for myocardial damage is very high; levels of troponin T are elevated with myocardial damage but not with skeletal muscle damage.[12] The advantages of using troponin levels are that the troponin level increases earlier and remains elevated longer (up to 14 days) than do CK-MB levels. Troponin levels may be helpful in confirming suspected AMI, diagnosing difficult cases, and triaging patients with chest discomfort, and levels are easily measured at the patient's bedside without specialized laboratory equipment. Troponin levels are not specific and diagnostic for myocardial contusion.[12] In the cases described in this article, troponin levels were not measured because such measurements were not yet standard practice in the facility.

Table 1 Diagnostic levels of creatine kinase and creatine kinase–MB for a patient with myocardial infarction

Hours after event	Creatine kinase, µg/L	Creatine kinase–MB, U/L	Relative index*
5	653	NR	NR
14	2036	254.7	12.5
22	1780	183.4	10.3

*Relative index is calculated by dividing creatine kinase–MB level (in micrograms per liter) by the total concentration of creatine kinase (in units per liter) and multiplying by 100.
NR indicates not reported.

Physical Assessment and Early Interventions

H.N. was diaphoretic. He had no peripheral edema, and his jugular venous distention was normal (<4 cm above his sternal

angle). His heart rate was 70/min, and his blood pressure was 120/80 mm Hg. He had an extra heart sound (S_4), which often occurs soon after AMI because of decreased ventricular compliance and increased end-diastolic pressure in the left ventricle.

H.N. was immediately given 3 L of oxygen by nasal prongs, aspirin, sublingual nitroglycerin, and intravenous morphine. Nitroglycerin, tissue plasminogen activator, and heparin infusions were initiated per hospital protocol, and H.N. was admitted.

After the intravenous infusions of nitroglycerin (30 µg/min) and tissue plasminogen activator, H.N. became hypotensive (blood pressure, 75/60 mm Hg) and bradycardic (heart rate, 40-50/min). This combination of events is most common in patients with volume depletion or inferior AMI and most likely is caused by venodilatation, low ventricular filling, and activation of receptors that trigger a vagal reflex arc and bradycardia. H.N. was given 250 mL of isotonic sodium chloride solution intravenously, and his blood pressure increased to 108/70 mm Hg and his pulse increased to 74/min. Life-threatening arrhythmias, such as heart block and ventricular tachycardia and fibrillation also may occur in AMI. Therefore, ECG monitoring is required immediately after AMI.

Progression and Outcome

Patients with AMI are at risk for arrhythmias and altered cardiac output. H.N. was treated with bed rest to decrease his oxygen demands and was transferred to the critical coronary care unit to be monitored for arrhythmias and hemodynamic status. His vital signs were stable, and he did not have any further documented arrhythmias.

After an AMI, treatment with β-blockers is recommended to reduce myocardial oxygen demands, oppose the action of increased amounts of catecholamines, and provide antiarrhythmic effects.[2] A β-blocker, metoprolol, was prescribed accordingly for H.N., but initially was withheld because of his bradycardia and hypotension. He received his first dose on the second day, when his heart rate and blood pressure had stabilized. He had no further chest pain; his ECGs and CK-MB levels indicated no reinfarction or extension of his AMI. H.N. was eager to learn about preventing further effects of his cardiovascular disease; he stated that the infarction "gave him an opportunity to focus on health." His stay in the hospital was uncomplicated, and he was discharged to home 5 days later.

MYOCARDIAL CONTUSION

S.B., a 53-year-old woman, was admitted to the medical-surgical critical care unit following a motor vehicle injury in which she collided head-on with another car in a highway intersection. The estimated speed of her car was 110 km/h (69 mph). Her car had no air bag, but she was wearing a safety restraint device. S.B. was transported via helicopter to the nearest trauma center, where she was treated for multiple injuries: myocardial contusion, fractured T6 vertebra, basal skull fracture, and pleural effusion. Her heart rate was 118/min, and her blood pressure was 90/70 mm Hg.

Pathophysiology

Myocardial contusion is bruising due to rupture or hemorrhage of small vessels in the myocardium. Direct injury to myocytes may also occur, with rupture of cells and destruction of cell membranes. Cell injury and bruising may be marginal or extensive and can occur across diffuse areas of the myocardium. The right ventricle is at greatest risk in chest trauma, owing to its anterior location under the sternum.

Myocardial injury due to contusion differs in several respects from injury that occurs in myocardial infarction. Although both types of injury may occur over a large surface area, contusion injury is due to the anatomic location of the injury within the chest and is not related to arterial distribution. Therefore, the injury of contusion is primarily epicardial rather than transmural or endocardial, and distinct ischemic zones do not occur.[13] The cause of injury is external, and the area of injury is far less vulnerable to extension of injury.

Several factors contribute to the decrease in cardiac function that occurs after myocardial contusion. Rupture of small vessels, hemorrhage into the interstitium and around the muscle fibers, and tearing of muscle fibers cause muscle dysfunction and diffuse hypoxia in the area of injury. Impediment of myocardial contractility and blood flow increases the detrimental effects of muscle dysfunction and hypoxia.

In addition, cardiac output may decrease because of valvular dysfunction, decreased preload, and increased afterload. Direct injury of the tricuspid or mitral valve may be caused by the external force and internal compression.

Acute hemorrhage or mechanical ventilation may decrease right ventricular preload. Pulmonary contusion and mechanical ventilation may contribute to increased right ventricular afterload. Adequate left ventricular preload depends on total blood volume and right ventricular function. An extensive sympathetic nervous response to hemorrhage, pain, or shock causes vasoconstriction and increased left ventricular afterload.

History: Mechanism of Injury, Pain, and Kinetic Energy

The mechanism and kinetic energy of the injury are significant factors in determining the risk for myocardial contusion and concomitant injuries. The *mechanism* of injury is the external force that is the cause of the event; the force may be blunt or penetrating. The direction, duration, and area of application of the force are associated with the mechanism of injury. *Kinetic energy* refers to the amount of energy transferred during the event. Myocardial contusion is usually due to blunt and direct injury, such as injuries caused by motor vehicle collisions, falls, or direct blows to the sternum. The primary cause of myocardial contusion is motor vehicle collisions. Wearing a safety restraint device may not prevent contusion; instead, the shoulder harness may increase the propensity for sternal fracture[14] or pulmonary or cardiac contusion but may prevent more severe injuries.[15] The mechanism of injury in S.B.'s case was blunt injury due to compression of the heart between the sternum and the spinal column. The kinetic energy was extensive because of the speed and type of collision.

Because of S.B.'s head injury and decreased level of consciousness, her chest pain could not be assessed accurately. Persons with myocardial contusion may experience sternal pain, which is difficult to differentiate from the pain of infarction. Unlike the pain associated with infarction, the pain associated with contusion is not referred to other sites. Rib fractures, sternal fracture, or pulmonary contusion may also occur and contribute to chest pain. The person may experience shortness of breath (primarily due to concomitant pulmonary contusion), anxiety, and increased oxygen demands. Shortness of breath may occur with the decrease in cardiac output. Nausea may be present because of the stress response.

Initial Diagnostic Tests: 12-Lead ECG, Measurement of CK-MB Levels, Echocardiography

Areas of injury in myocardial contusion may be reflected in diffuse changes in the ST segment or T waves on the ECG. Injury is not necessarily localized, and therefore leads showing changes would not necessarily be contiguous. ECG changes due to contusion are more consistent with ECG changes that occur in pericarditis than with the changes that occur in myocardial infarction. The ST segment may be elevated or depressed. The T wave may be flattened or inverted. Conversely, some patients with myocardial contusion may have no ECG changes indicating either injury or ischemia.[13]

Arrhythmias may also be noted on the continuous or 12-lead ECG. Arrhythmias occur because of increased catecholamine levels, direct damage of the conduction pathways, or hypoxic areas of the myocardium. Most arrhythmias occur within the first 3 days after the injury. The prevalence of arrhythmias after myocardial contusion is correlated with increased age, pulmonary contusion, rib fractures, extrathoracic injuries, and abnormal findings on the initial ECG.[15] Ventricular beats and atrioventricular blocks are the most prevalent arrhythmias other than sinus tachycardia; atrial fibrillation may occur. The right bundle branch is vulnerable to direct injury, and right bundle branch block is more likely than left bundle branch block. S.B.'s ECG changes are consistent with contusion. On day 1, she had a right bundle branch block. On day 3, she had nonspecific flattened T waves (Figure 2).

S.B. had creatine kinase levels measured on days 1 and 2, without the differentiating breakdown of isoenzymes. Troponin levels were not measured because such diagnostic tests were not available at that site. Clinical benefits of measuring the levels of creatine kinase, isoenzymes (CK-MB), or troponin in patients with myocardial contusion have not been demonstrated.[16] It is recommended that measurement of creatine kinase levels be limited to persons in whom AMI is suspected as either the cause or result of chest trauma because creatine kinase levels are nonspecific and unreliable predictors of required treatment or outcome.[17] If enzyme levels are monitored, isoenzymes must be differentiated because most patients have additional injuries that increase the total level of creatine kinase. The timing of the peak in creatine kinase levels is less predictable in myocardial contusion than in AMI, and creatine

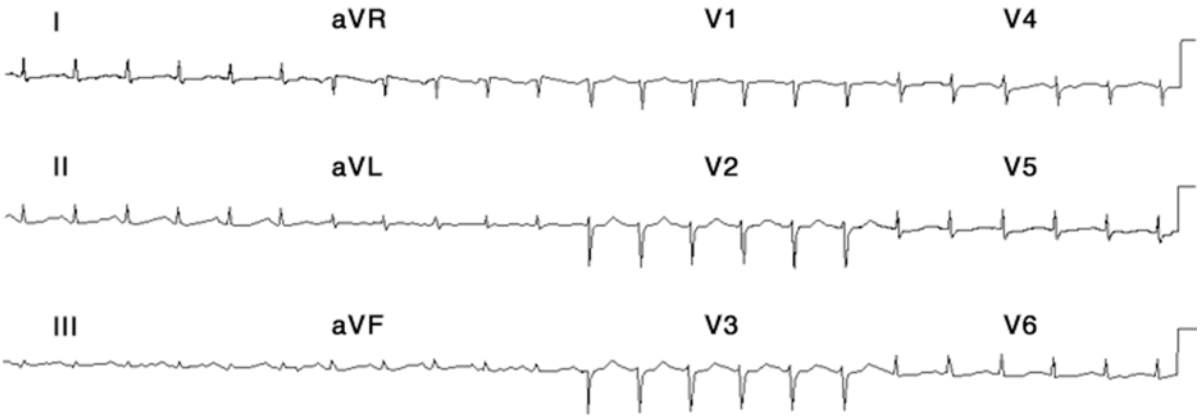

Figure 2 12-Lead electrocardiogram obtained on day 3 in a patient with myocardial contusion.

kinase levels may peak within the first 8 hours after injury or continue to increase over time. Troponin levels are not sensitive or diagnostic for myocardial contusion.[18]

S.B.'s initial echocardiogram showed normal left ventricular function and normal valve function. She had a small pericardial effusion and decreases in right ventricular and left posterior and lateral wall motion. Like CK-MB levels, findings on echocardiograms are not reliable indicators of contusion or potential adverse effects.[19]

Physical Assessment and Early Interventions

Initial and ongoing assessment is essential for patients with myocardial injury. For example, findings on assessment contribute to differentiating between potential causes of low blood pressure related to coronary injury or hemorrhage. S.B.'s score on the Glasgow Coma Scale was 4 of 15. Her heart rate was 40/min to 60/min; she had no palpable pulses and an inaudible blood pressure. She had a systolic murmur on the left sternal border at the fourth intercostal space. Breath sounds to the left lower lobe were absent, bruising was evident over the left upper quadrant, and subcutaneous emphysema was present from the clavicle to the sixth rib. In the emergency department, needle decompression of the lung was performed and a chest tube was inserted; 150 mL of sanguineous fluid was drained. S.B. was intubated and was treated with pressure-cycled ventilation, with a fraction of inspired oxygen of 0.50. Atropine (0.6 mg) was given for the bradycardia, and her heart rate increased to 75/min. Her blood pressure was 70/50 mm Hg. Isotonic sodium chloride solution (2000 mL) was given, and infusions of epinephrine (0.2 µg/kg per minute) and dopamine (4 µg/kg per minute) were initiated to increase blood pressure and perfusion. S.B. had a left-sided thoracotomy and creation of a pericardial window and was admitted to the medical-surgical critical care unit.

Progression and Outcome

On admission to the critical care unit, S.B.'s inotropic and vasoactive medications were changed from dopamine and epinephrine to norepinephrine (0.2 µg/kg per minute) and dobutamine (12.5 µg/kg per minute). On day 2, the norepinephrine was discontinued and her blood pressure was maintained with dobutamine, 1 L of lactated Ringer's solution, and 500 mL of 5% albumin. S.B. remained in right bundle branch block and alternated between sinus rhythm and atrial fibrillation. On day 3, she converted from right bundle branch block to continuous normal sinus rhythm. Her blood pressure was 115/80 mm Hg, and the dobutamine was discontinued. She was treated with mechanical ventilation for an additional 10 days until resolution of the pulmonary contusion and head injury.

Generally, patients with myocardial contusion respond to fluid and inotropic therapy, such as administration of dobutamine. Resolution of cardiac output

usually occurs within 3 days of treatment, and blood pressure is restored.[13,15] If hemodynamic instability increases or recurs, severe right ventricular failure or tamponade must be considered. S.B.'s myocardial contusion and right bundle branch block contributed to her loss of cardiac function. Her pulmonary and brain injuries, although not the focus of this article, were of greater importance to her long-term recovery than was the myocardial contusion.

MYOCARDIAL CONTUSION AND INFARCTION

L.R., a 54-year-old man, was admitted to the trauma critical care unit after a motor vehicle collision with a lamp pole. Because of the history, particularly the mechanism of injury, the initial considerations for cardiac injury were limited to myocardial contusion. L.R. was tachycardic with decreased blood pressure. A cardiologist was consulted to evaluate L.R.'s cardiac status on the basis of L.R.'s history, results of diagnostic tests, and findings on physical assessment.

History: Pain, Trigger Events, Risk Factors, Kinetic Energy, and Mechanism of Injury

At the scene of injury, L.R. stated that he had sternal chest pain on inspiration. The pain did not refer to other sites and was not associated with nausea. Pain assessment was compromised because L.R. was intoxicated with alcohol, and it was not determined if he had experienced chest pain before the collision. L.R. had no history of coronary artery disease. His known risk factors were his male sex and his family history; his wife indicated that L.R.'s father had died at the age of 57 of a myocardial infarction. L.R. was traveling at an estimated speed of more than 115 km/h (72 mph) and was not wearing a safety restraint device. He was partially thrown through the windshield and was trapped. Extrication time was prolonged (30 minutes).

Initial Diagnostic Tests: 12-Lead ECG, Measurement of CK-MB Levels, Radiography, Echocardiography

L.R.'s initial 12-lead ECG (Figure 3) showed significant ST-segment elevation in the anterior and lateral leads, with hyperacute T waves in the inferior leads. Creatine kinase and CK-MB levels were elevated (Table 2). These findings are more consistent with AMI than with contusion.

Additional blood tests included a complete blood cell count, measurement of ethanol and electrolyte levels, and arterial blood gas analysis. L.R.'s ethanol level was 24 mmol/L (110 mg/dL). Chronic use of alcohol was suspected on the basis of his magnesium level of 0.3 mmol/L (0.73 mg/dL), his calcium level of 1.68 mmol/L (6.7 mg/dL),[20] his lipase level of 230 U/L (230 mU/mL),[21] and his acidosis.[22] L.R. had both respiratory and metabolic acidosis, with an initial pH of 7.13. With 10 L of oxygen administered via a

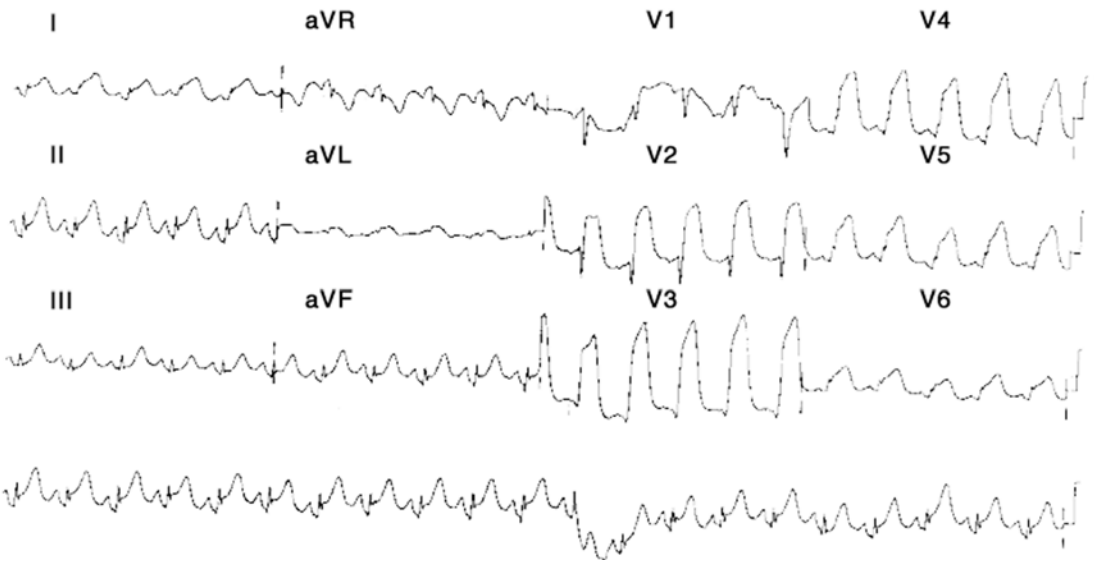

Figure 3 12-Lead electrocardiogram of a patient with both myocardial infarction and contusion.

Table 2 Diagnostic levels of creatine kinase and creatine kinase–MB for a patient with myocardial contusion and infarction

Hours after event	Creatine kinase, µg/L	Creatine kinase–MB, U/L	Relative index*
4	800	34.3	4.3
18	4162	512.0	12.3
22	4002	455.8	11.4

*Relative index is calculated by dividing creatine kinase–MB level (in micrograms per liter) by the total concentration of creatine kinase (in units per liter) and multiplying by 100.

nonrebreathing mask, L.R.'s Pao_2 was 75 mm Hg, oxygen saturation was 88%, and hemoglobin level was 168 g/L.

A chest radiograph showed fractured ribs (ribs 4 to 8) and a large pneumothorax on the left side. These injuries are consistent with the nature of the collision, the high speed, and the sudden hard impact that propelled L.R. against the steering column.

Significant changes in cardiac function were noted on L.R.'s echocardiogram, with akinesis and dyskinesis across the septum and the anterior and lateral walls. These changes in left ventricular function are consistent with his ECG changes, creatine kinase levels, and the occurrence of a large AMI. Mild mitral regurgitation, mild aortic insufficiency, and trivial tricuspid regurgitation also occurred. The combination of these valvular echocardiographic findings is more consistent with direct trauma than with ischemia from coronary thrombus.

Physical Assessment and Early Interventions

L.R. was tachycardic with a heart rate of 127/min and had a decreased blood pressure of 95/60 mm Hg. Tachycardia is a common sign in a variety of situations. Potential contributing factors for L.R. included the stress of the motor vehicle collision, hypovolemia related to bleeding, and the presence of an anterior AMI. Tachycardia is a consequence of high levels of circulating catecholamines and sensitized β-receptors present after ischemia.[23]

L.R. was intoxicated with alcohol and was inconsistently coherent, a situation that impeded an accurate neurological assessment. His heart sounds were distant and quiet, with no audible extra sounds or murmur. Estimated blood loss through the abdominal injuries and scalp lacerations was 1000 to 1200 mL. Phenylephrine (0.45 µg/kg per minute) and a bolus of 500 mL of lactated Ringer's solution were administered to maintain blood pressure. L.R.'s respiratory rate stayed at 42/min. He had a left-sided flail chest and decreased breath sounds throughout, with minimal breath sounds in the left lower lobe of the lung. Bruising over the sternum was extensive, extending from the clavicle to the diaphragm. L.R. was intubated and treated with assist-control ventilation, with a fraction of inspired oxygen of 0.40 and a tidal volume of 600 mL. His spontaneous respirations decreased to 30/min. Two chest tubes were inserted on the left side; both had air leaks and sanguineous drainage. L.R. was immediately taken to surgery, where the scalp lacerations were repaired. An investigative laparotomy indicated multiple lacerations of the spleen and liver, which had ceased to bleed. Chronic pancreatitis was evident.

Progression and Outcome

The phenylephrine infusion was titrated up to 2.0 µg/kg per minute to achieve a mean arterial pressure of 65 mm Hg. Phenylephrine has a potential vasoconstrictive effect on the coronary artery. Nitroglycerin (80 µg/min) was added to prevent constriction of the coronary arteries.

L.R. had a single run of atrial fibrillation, but the rhythm converted spontaneously to sinus rhythm. Other than tachycardia, no other arrhythmias were noted. L.R. was examined by a cardiologist, and a large anterolateral AMI was diagnosed. It is unknown whether the AMI was the cause of or a result of the motor vehicle collision. Motor vehicle collisions and other traumatic events sometimes occur if the person experiencing an AMI loses consciousness or becomes sufficiently distracted by the discomfort caused by the AMI. Myocardial infarction with subsequent thrombus formation in the injured artery may occur at the time of chest trauma when either direct or indirect injury of the coronary artery occurs.

On day 2, auscultation of his heart sounds revealed an S_4. The ECG showed Q waves in the anterolateral leads. L.R. had frequent episodes of tachycardia. He reported chest pain with coughing and moving. No ECG changes occurred with the chest

pain, which most likely was due to the pulmonary and thoracic injuries. Administration of morphine via a patient-controlled pump was started, with a minimum rate of 2 mg/h and a maximum rate of 4 mg/h.

A pulmonary artery catheter was inserted; the initial findings were central venous pressure, 12 mm Hg; pulmonary wedge pressure, 16 mm Hg; stroke volume, 76 mL; systemic vascular resistance index, 1650; and cardiac index, 3.6. (Systemic vascular resistance index was calculated as systemic vascular resistance in dynes times seconds per centimeters to the fifth power divided by body surface area in square meters. Cardiac index was calculated as cardiac output in liters per minute divided by body surface area in square meters.) L.R.'s mean arterial pressure was 60 to 65 mm Hg. His systemic vascular resistance index decreased to 1370; phenylephrine administration was increased to 3.0 µg/kg per minute, and nitroglycerin was discontinued. The reason for the low systemic vascular resistance index was unknown.

On day 3, L.R.'s tachycardia increased; his heart rate was sustained at 90/min to 125/min. Intravenous metoprolol (5 mg) was given to oppose the elevated catecholamine levels, thereby reducing heart rate and myocardial oxygen demands. Mean arterial pressure was 72 to 78 mm Hg, and the systemic vascular resistance index was 1890. Urine output was less than 20 mL/h. L.R.'s normal serum levels of creatinine and urea were not consistent with renal dysfunction; therefore, fluid balance and medications were evaluated. Phenylephrine can constrict renal blood vessels and decrease urine output; consequently, the concentration of this drug was titrated down and administration was discontinued. L.R.'s urine output improved over the course of day 3.

On day 4, L.R. began treatment with captopril, an angiotensin-converting enzyme inhibitor and potent vasodilator. The reduction of afterload with angiotensin-converting enzyme inhibitors has potential beneficial effects on limiting ventricular remodeling after a large infarction, reducing the size of the infarct,[24] and lessening the risk of sudden death and heart failure.[2] On day 5, L.R. was extubated, started taking aspirin and metoprolol, and was transferred out of the critical care unit.

The cause of L.R.'s AMI is not known; however, he did have permanent myocardial damage. After L.R. was discharged from the trauma intensive care unit, his medical care was assumed by the cardiologist. The medication regimen of aspirin, captopril, and metoprolol was consistent with recommended management for myocardial infarction. L.R. required further surgery to repair his pelvis but was required to wait 6 weeks to allow his cardiac status to improve. It was recommended to L.R. that he complete the cardiac rehabilitation program after his surgery. Before discharge, L.R. was taught about risk factors, management of chest discomfort, activity, and medications. In addition to cardiac risks factors, L.R.'s drinking and driving place him at risk for additional future traumatic injury, and he should have follow-up or counseling about his use of alcohol.

SUMMARY

Patients experiencing myocardial injury require immediate and thoughtful nursing care. The pathophysiology, assessment findings, and interventions for myocardial contusion and infarction have some similarities and some differences. AMI is localized injury due to decreased or absent perfusion, whereas myocardial contusion results from both cellular injury due to the mechanism of traumatic injury and cellular injury due to alterations in perfusion. Chest pain, pressure, and discomfort are expected symptoms with each type of injury, but vary. Diagnostic tests for AMI, including ECGs showing changes in contiguous leads and blood tests indicating elevated enzyme levels (CK-MB and troponin), are more specific and accurate than are tests for myocardial contusion. Understanding each type of myocardial injury can enable nurses to provide rapid, efficient, appropriate, and safe care. With each type of injury, the focus is on improving oxygenation and cardiac perfusion while protecting cardiac work and myocardial oxygen consumption. This focus includes preventing complications, facilitating healing, and providing accurate information to patients and patients' families.

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AMERICAN ASSOCIATION of CRITICAL-CARE NURSES

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CE Test Form

Myocardial Injury: Contrasting Infarction and Contusion

Objectives:

1. Discuss and contrast the pathophysiology of myocardial infarction and myocardial contusion
2. Identify diagnostic tests that are used in the diagnosis of myocardial infarction and myocardial contusion
3. Distinguish signs and symptoms associated with myocardial infarction and myocardial contusion

1. ❑a	2. ❑a	3. ❑a	4. ❑a	5. ❑a	6. ❑a	7. ❑a	8. ❑a	9. ❑a	10. ❑a	11. ❑a
❑b	❑b	❑b	❑b	❑b	❑b	❑b	❑b	❑b	❑b	❑b
❑c	❑c	❑c	❑c	❑c	❑c	❑c	❑c	❑c	❑c	❑c
❑d	❑d	❑d	❑d	❑d	❑d	❑d	❑d	❑d	❑d	❑d

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	Agree	Neutral	Disagree
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The level of difficulty of this test was:
❑ easy ❑ medium ❑ difficult
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CE Test Questions

Myocardial Injury: Contrasting Infarction and Contusion

1. Which one of the following has been identified as the primary cause of an acute myocardial infarction?
   a. Arterial spasm
   b. Coronary artery blockage
   c. Embolism
   d. Thrombus formation

2. When a myocardial infarction occurs, necrosis of the myocardium may be complete and irreversible within what time frame?
   a. Less than 60 minutes
   b. 1 hour
   c. 1 to 2 hours
   d. 3 to 4 hours

3. Which one of the following is recommended to check for associated right ventricular or posterior involvement in patients with inferior myocardial infarctions?
   a. Echocardiogram
   b. Stress testing
   c. Expanded electrocardiography (ECG) with 15 or18 leads
   d. Creatine kinase–MB (CK-MB) levels for 72 hours

4. After a myocardial infarction, when are maximum levels of elevation of CK-MB reached?
   a. 4 to 6 hours
   b. 8 to 12 hours
   c. 12 to 18 hours
   d. 18 to 24 hours

5. What does a secondary increase in CK-MB level on the downward slope of the first increase in CK-MB level indicate?
   a. Reinfarction
   b. Left ventricular failure
   c. Acute myocardial infarction
   d. Myocardial contusion

6. What value of the relative index indicates myocardial infarction?
   a. 0.5 to 1.0
   b. 1.0 to 2.0
   c. 3.0 to 4.0
   d. Greater than 4.0

7. Which one of the following diagnostic tests is not specific and diagnostic for myocardial contusion?
   a. CK-MB
   b. 12-lead ECG
   c. Troponin levels
   d. Echocardiography

8. Which one of the following cardiac structures is at greatest risk in chest trauma for myocardial contusion?
   a. Left ventricle
   b. Right ventricle
   c. Aortic arch
   d. Right atrium

9. What is the primary cause of myocardial contusions?
   a. Falls
   b. Motor vehicle collisions
   c. Bicycle accidents
   d. Motorcycle accidents

10. In a myocardial contusion, most arrhythmias occur within what period of time after the injury?
    a. The first day
    b. The second day
    c. The third day
    d. 5 to 7 days

11. ECG changes due to contusion are most consistent with the changes that occur in which one of the following myocardial conditions?
    a. Myocardial infarction
    b. Pericarditis
    c. Right bundle branch block
    d. Tamponade