Analysis of the 12-lead electrocardiogram (ECG) reveals crucial data in patients with suspected acute myocardial infarction. Distinguishing ST-segment elevation from other acute coronary syndromes facilitates rapid and appropriate intervention. Clinical research has shown the effectiveness of rapid reperfusion in reducing the amount of myocardial damage and improving outcomes in patients with myocardial infarction and ST-segment elevation. Two reperfusion strategies are recommended in the updated guidelines from the American College of Cardiology and the American Heart Association for patients who have had signs and symptoms of acute myocardial infarction for less than 12 hours: (1) administration of thrombolytic agents less than 30 minutes after admission and (2) arrival in the cardiac catheterization laboratory less than 60 minutes after admission. Accomplishment of this goal requires obtaining a 12-lead ECG within 10 minutes of a patient’s arrival in the emergency department. Rapid recognition of myocardial infarction with ST-segment elevation enables early reperfusion and minimizes the extent of myocardial necrosis.

In this article, case studies are used to compare 12-lead ECGs obtained before and after early percutaneous coronary intervention in patients who have myocardial infarction with ST-segment elevation. The integral role of nurses in rapidly recognizing ECG changes when a patient arrives in the emergency department and after intervention is also discussed.

Twelve-Lead ECGs

ST-segment elevation on 12-lead ECGs occurs with prolonged ischemia and is an early indication of myocardial infarction.

Depolarization of the ventricle occurs when the electrical impulse propagates from the atrioventricular node to the bundle of His and into the Purkinje network of the left and right ventricles. On 12-lead ECGs,
the QRS complex represents depolarization. Normally the QRS complex is 0.04 to 0.10 seconds in duration or width. Q waves, if present, should be less than 0.03 seconds wide and 1 mm or less in depth.3,4

The ST segment begins at the J point located at the end of the QRS complex and continues to the beginning of the T wave. The ST segment signifies the short period between the end of depolarization and the beginning of repolarization. The ST segment is normally even with the isoelectric line of the ECG. Elevation of the ST segment is significant when it is 1 mm or more above the isoelectric line in 2 or more contiguous leads. Contiguous leads are the leads that transmit the electrical activity from a particular area of the myocardium. Leads II, III, and aVF are contiguous leads transmitting the electrical activity from the inferior wall of the heart and represent blood flow through the right coronary artery. The septal and anterior walls of the myocardium receive their blood supply via the left anterior descending coronary artery. The septal wall is represented on ECGs in leads V1 and V2 whereas the anterior wall includes leads V1, V2, V3, and V4. Leads I, aVL, V5, and V6 depict the left lateral wall. The left circumflex coronary artery provides blood supply to this area of the myocardium. The posterior wall, supplied by the right coronary artery, is indirectly represented by reciprocal changes in leads V1, V2, V3, and V4 on standard ECGs.4

The first phase of myocardial infarction, ischemic injury, begins when blood flow through the coronary artery ceases. This phase lasts about 4 hours.5 Reperfusion strateg-ies used during this time prevent extensive necrosis of the involved myocardium.1 ST-segment elevation occurs within the first minutes of myocardial infarction and can persist for hours to several days. ST-segment elevation decreases sooner in patients who receive early reperfusion interventions than in patients who do not receive such interventions.4

Nursing Implications
Emergency Department

Patients coming to the emergency department with signs and symptoms suggestive of acute myocardial infarction require rapid assessment by an emergency department nurse. Meeting the goal of arrival in the cardiac catheterization laboratory within 60 minutes of a patient’s arrival in the emergency department depends on obtaining and interpreting a 12-lead ECG within 10 minutes of the patient’s arrival in the emergency department.5 Rapid identification of acute ST-segment elevation facilitates triage to the cardiac catheterization laboratory.

Other interventions instituted in the emergency department before percutaneous coronary intervention include administration of oxygen, aspirin (if the patient is not allergic), and heparin (with dosage based on the patient’s weight). Aspirin and heparin decrease platelet aggregation and inflammation of the vessel. Glycoprotein IIb/IIIa agents such as eptifibatide (Integrilin) and abciximab (ReoPro) are beneficial in enhancing reperfusion because of their antiplatelet properties that initiate lysis of the thrombus and reduce inflammation.1 Nitroglycerin and morphine may also be administered for pain relief.

Monitoring After Percutaneous Coronary Intervention

ECG monitoring after percutaneous coronary intervention is essential for patients who have acute myocardial infarction with ST-segment elevation. With ECG monitoring, acute reclosure of the coronary artery and extension of the infarct can be detected. Patients in whom ST-segment elevation increases in the first 6 to 48 hours after percutaneous coronary intervention may require recatheterization and intervention.5

The ST-Segment Monitoring Practice Guideline International Working Group recommended, in a consensus statement,6 that ideally the lead used for continuous ST-segment monitoring is the lead that was identified during the intervention procedure as the “fingerprint.” The fingerprint is determined by identifying the leads on the 12-lead ECG in which the ST-segment elevation occurred during balloon inflation.7 If the fingerprint cannot be identified on the 12-lead ECG, it can be identified on the 12-lead ECG obtained before the intervention that documents the leads in which ST-segment elevation occurred.7

Continuous ST-segment monitoring should be in the lead that was identified as the fingerprint after percutaneous coronary intervention. Monitors with dual-channel ECG monitoring capabilities can be set up to monitor 2 ECG leads simultaneously. In this instance, the leads chosen should be the ones that revealed the ST-segment elevation. Acute closure of the coronary artery usually occurs within the first 6 hours after percutaneous transluminal coronary angiography (PTCA)
and stenting, so the ST segment should be monitored for at least 6 to 12 hours. Monitoring for 48 hours after intervention provides information to distinguish ischemic chest pain from nonischemic chest pain.6

Blood Flow

Blood flow was described by investigators in the Thrombolysis in Myocardial Infarction (TIMI) trial.1 The TIMI classification system is used to evaluate the flow through the coronary arteries before and after intervention in patients undergoing percutaneous coronary intervention. According to the TIMI flow classification, TIMI flow 0 indicates no flow through a completely blocked artery. TIMI flow 1 indicates that blood flows into the thrombus but not through the thrombus, TIMI flow 2 indicates that blood flows sluggishly through the artery, and TIMI flow 3 indicates that blood flow through the artery is normal.1,2,8 The goal of percutaneous coronary intervention in patients who have acute myocardial infarction with ST-segment elevation is to improve TIMI flow 0 to TIMI flow 3, subsequently decreasing the amount of myocardial necrosis and improving long-term survival.1

Case Studies

The following case studies illustrate decreased ST-segment elevation after reperfusion with PTCA and stent deployment in the lesion. Acute restenosis of a stent is described in case 4.

Case Study 1

Mr P, a 44-year-old man, came to the emergency department within 40 minutes of a sudden onset of chest pain and syncope. Upon arrival, his heart rate was 82/min and blood pressure was 136/79 mm Hg. A 12-lead ECG obtained 4 minutes after his arrival revealed a myocardial infarction of the inferior wall. Mr P’s history included coronary artery disease and PTCA and stenting of his right coronary artery and left circumflex coronary artery in June of the previous year. Mr P was administered aspirin, clopidogrel, and a heparin bolus followed by a weight-based continuous infusion of heparin. The first 12-lead ECG revealed ST-segment elevation in leads II, III, and aVF, consistent with an acute myocardial infarction of the inferior wall (Figure 1). The tall, wide, peaked T waves that accompanied the ST-segment elevation are an early ECG change that occurs in myocardial infarction.9 Small Q waves are normally present in lead III; however, the Q waves in lead III of Mr P’s ECG were more than 1 mm deep. The ST-segment depression and T-wave inversion in leads I, aVL, V1, and V2 were reciprocal changes. Reciprocal changes reflect the electrical activity opposite the area of infarction. They are an early ECG finding and resolve quickly.9 The tall, peaked T waves and reciprocal changes on Mr P’s ECG indicate that he arrived in the emergency department in the very early stages of his myocardial infarction.

Because of his early arrival in the emergency department and the availability of a cardiac catheterization laboratory with cardiac surgery capability, Mr P was a candidate for reperfusion via angioplasty. He arrived in the cardiac catheterization laboratory 42 minutes after arriving in the emergency department. The right coronary artery was 100% blocked at its origin within the previously stented segment and was the lesion causing the problem. PTCA was performed, establishing TIMI flow 3 with a residual stenosis of 0%.

A 12-lead ECG was obtained 14 hours after the procedure in the intensive care unit (Figure 2). The ST-segment elevation had decreased

Figure 1 Case study 1: initial 12-lead electrocardiogram.
to baseline in lead II and approximately 0.5 mm in leads III and aVF. Normally, the ST segment can remain elevated for several hours to several days after an acute myocardial infarction; however, with reperfusion the elevation resolves more quickly.6 The T wave had returned to its normal configuration, and the reciprocal changes in leads I, aVL, V1, and V2 had dissipated. Note the development of a significant Q wave in leads II, III, and aVF. Q waves are considered significant when they are at least 0.03 seconds wide or more than 1 mm deep.3,4,9

Mr P’s peak levels of cardiac markers were as follows: creatine kinase (CK) 4042 U/L (normal, 0-220 U/L), CK-MB 246.5 ng/mL (normal, 0-8.0 ng/mL), CKMB index 6.1 (normal, 0-6.5; calculated as CK-MB level in nanograms per milliliter divided by total CK level in units per liter times 100), and troponin I 392 ng/mL (normal, 0-2.0 ng/mL). On day 4, Mr P was discharged from the hospital with medications including aspirin, captopril, clopidogrel, metoprolol, and simvastatin. He was referred to the outpatient cardiac rehabilitation department for development of an exercise program and education on lifestyle changes to reduce risk factors for subsequent myocardial infarction.

Case Study 2

Mr T was a 52-year-old man with midsternal heaviness and tightness that radiated down both arms and into his neck and was accompanied by diaphoresis and dyspnea. He arrived in the emergency department 20 minutes after the onset of signs and symptoms. A 12-lead ECG was obtained 6 minutes after his arrival and revealed an acute anteroseptal myocardial infarction (Figure 3). Leads V1, V2, V3, V4, and V5 reflected ST-segment elevation. The ST segment gently sloped upward into the tall, wide T wave, particularly in leads V2, V3, and V4. It was difficult to differentiate the ST segment from the T wave. Reciprocal T-wave inversion was present in leads II and III. Deep, wide Q waves in V1, V2, and V4 indicated infarction of the anteroseptal wall of the myocardium.

Mr T was transported to the cardiac catheterization laboratory for emergent cardiac catheterization and possible intervention 25 minutes after his arrival in the emergency department. The cardiac catheterization revealed a geometrically complex lesion in the proximal left anterior descending coronary artery.
that was completely blocking the vessel. No stenosis was present in the left main, left circumflex, or right coronary arteries. Eptifibatide and heparin infusions were begun before the start of the intervention procedure to inhibit platelet aggregation at the site of the lesion. PTCA was performed and a stent was deployed, reducing the stenosis from 100% to 0% with resultant TIMI flow 3 in the vessel. Mr T continued receiving eptifibatide to prevent acute closure of the vessel and subacute thrombosis of the stent and to facilitate further perfusion of the coronary vessels.

An ECG obtained 3 hours after PTCA and stenting of the left anterior descending coronary artery showed dramatic changes (Figure 4). The inverted T waves that were present in leads II and III were now upright. Deep, wide Q waves remained present in leads V₁, V₂, and V₃, indicating that some myocardial necrosis had occurred. The anterior leads now reflected a smaller, less peaked T wave, and the ST segment was decreasing slightly toward baseline. The ST segment gradually returns to the isoelectric line over several days after myocardial infarction; however, the T-wave inversion may remain for a few weeks. On an ECG obtained 3 days after intervention (Figure 5), the ST segment was closer to baseline but the T wave remained inverted in leads V₁ through V₅. The Q wave will remain indefinitely. ST-segment elevation and T-wave inversion are temporary and return to normal as the myocardium heals.

Peak levels of cardiac markers were CK 3893 U/L (normal, 0-220 U/L) and CK-MB 273.6 ng/mL (normal, 0-8.0 ng/mL), with a CKMB index of 7.0 (normal, 0-6.5). The troponin I level was 98.2 µg/L (normal, 0-2.0 µg/L). Mr T was discharged 3 days after the procedure with medications including aspirin, clopidogrel, metoprolol, and simvastatin. He was referred to the outpatient cardiac rehabilitation department for participation in the smoking cessation program.

Case Study 3
Mrs H was a 55-year-old woman with a history of coronary artery bypass grafting of the left anterior descending artery, the first marginal branch of the left anterior descending artery, and the right coronary artery in 1992 and again in 1994. She came to the emergency department with substernal chest pain and nausea a half hour after onset of signs and symptoms. The initial ECG (Figure 6), obtained 9 minutes after her arrival,
showed ST-segment elevation in the inferior leads and V1 with tall, wide, peaked T waves. T-wave inversion was present in leads I and aVL. Q waves were present in leads V1, V2, and V3, indicating the myocardial infarction documented 9 years before.

Mrs H was taken to the cardiac catheterization laboratory 40 minutes after her arrival in the emergency department. The catheterization revealed a thrombus completely blocking the saphenous vein graft to the right coronary artery. A large amount of the thrombus was removed by mechanical thrombolysis, intracoronary eptifibatide and nitroglycerin were administered, and a stent was deployed in the graft, reducing the occlusion to 0% residual with TIMI flow 3.

An ECG obtained 18 hours after catheterization is shown in Figure 7. The ST-segment elevation that was present before reperfusion was no longer present in lead II, and the T wave was not inverted. The ST segment remained elevated in leads III and aVF with an upright T wave thatwas not as tall or as wide as on the previous ECG. Ischemic changes persisted as ST-segment depression and T-wave inversion in leads I and aVL but less dramatically.

Mrs H was discharged from the hospital on day 3 after intervention. Her peak CK level was 310 U/L (normal, 0-220 U/L), CK-MB level 49.8 ng/mL (normal, 0-8.0 ng/mL), and CKMB index 16.1 (normal, 0-6.5). The troponin I level was 34.1 µg/L (normal, 0-2.0 µg/L). Her discharge medications included aspirin, clopidogrel, metoprolol, and simvastatin.

Case Study 4

Mr D was a 49-year-old man with a history of a previous myocardial infarction and stent placement in the past year. He came to the emergency department with chest pain accompanied by shortness of breath, nausea, and diaphoresis that had started 30 minutes before he arrived. A 12-lead ECG obtained within 3 minutes of his arrival revealed an acute myocardial infarction of the inferior wall. The initial 12-lead ECG (Figure 8) revealed ST-segment elevation in leads II, III, and aVF. Reciprocal changes were seen in leads V1, V2, V3, and V4. No Q waves were present. He was given aspirin, heparin, and morphine. Eptifibatide was ordered; however, Mr D was transported to the cardiac catheterization laboratory before the medication was started.

Mr D arrived in the cardiac catheterization laboratory 30 minutes after his arrival at the emergency department. Selective coronary arteriography revealed an
occluded right coronary artery, 90% obstruction in the mid left anterior descending artery, and an 80% instant restenosis in the proximal left anterior descending coronary artery. Eptifibatide was administered before intervention. PTCA and stenting of the right coronary artery reduced the 100% obstruction to 0% and improved flow to TIMI flow 3.

Mr D was admitted to the intensive care unit, where continuous ST-segment monitoring was used. His chest pain had dissipated after the intervention, and he was maintained on a continuous infusion of eptifibatide. Clopidogrel was administered in the cardiac catheterization laboratory after completion of the intervention.

Three hours after PTCA and stenting of the right coronary artery, Mr D began to complain of severe chest pain. An increase in ST-segment elevation was noted on the monitor. A 12-lead ECG (Figure 9) revealed more severe ST-segment elevation in the inferior leads II, III, and aVF. Reciprocal changes were still present in leads V₁, V₂, V₃, and V₄. A Q wave was now present in leads II, III, and aVF. The Q wave in lead III was greater than 1 mm in depth. Mr D was taken emergently to the cardiac catheterization laboratory for coronary angiography and possible intervention.

![Figure 8](image1.png)
Figure 8 Case study 4: initial 12-lead electrocardiogram.

![Figure 9](image2.png)
Figure 9 Case study 4: 12-lead electrocardiogram during acute closure

![Figure 10](image3.png)
Figure 10 Case study 4: 12-lead electrocardiogram 12 hours after second intervention.
Coronary angiography revealed an acute closure of the right coronary artery just proximal to the previous stent. Mechanical thrombolysis was performed and a stent was deployed at the site of the acute closure.

The 12-lead ECG obtained 12 hours after the second percutaneous coronary intervention is shown in Figure 10. Inverted T waves were discernible in leads II, III, and aVF. Significant Q waves were present in leads III and aVF, indicating infarction in the inferior wall of the myocardium. Less ST depression was evident in leads V₁, V₂, V₃, and V₄.

Mr D was discharged from the hospital 5 days after percutaneous coronary intervention. His peak CK level was 802 U/L (normal, 0-220 U/L), CK-MB level 37.9 (normal, 0-5.0 ng/mL), and CKMB index 4.7 (normal, 0-5.0). The troponin T level was 2.82 µg/L (0-0.03 µg/L). Medications prescribed at discharge included aspirin, clopidogrel, ramipril, and simvastatin. He was referred to the outpatient cardiac rehabilitation department for an exercise program and education on lifestyle modifications.

Conclusion

Critical care nurses have an integral role in assessing 12-lead ECGs for ST-segment elevation. Rapid identification of myocardial infarction with ST-segment elevation and prompt intervention decreases the amount of myocardial necrosis.

References
Case Studies of ST-Segment Elevation Before and After Percutaneous Coronary Intervention in Patients With Acute Myocardial Infarction
Julia McAvoy

Crit Care Nurse 2004;24 32-39
Copyright © 2004 by the American Association of Critical-Care Nurses
Published online http://ccn.aacnjournals.org/

Personal use only. For copyright permission information:
http://ccn.aacnjournals.org/cgi/external_ref?link_type=PERMISSIONDIRECT