Acute Coronary Syndrome: Understanding the Spectrum
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Canadian Institute for Health Information
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About CIHI

The Canadian Institute for Health Information (CIHI) collects and analyzes information on health and health care in Canada and makes it publicly available. Canada’s federal, provincial and territorial governments created CIHI as a not-for-profit, independent organization dedicated to forging a common approach to Canadian health information. CIHI’s goal: to provide timely, accurate and comparable information. CIHI’s data and reports inform health policies, support the effective delivery of health services and raise awareness among Canadians of the factors that contribute to good health.
Acknowledgements

This report could not have been completed without the generous support and assistance of staff at participating facilities.
Section 1: Introduction

Acute Coronary Syndrome: Understanding the Spectrum is a self-directed learning package (SLP) addressing the spectrum of diseases falling under the umbrella of acute coronary syndrome (ACS), ranging from unstable angina (UA) to non ST-segment elevation myocardial infarction (NSTEMI) to ST-segment elevation myocardial infarction (STEMI). This SLP includes clinical information to assist the coder in understanding the clinical picture of ACS; a review of the relevant codes using the International Statistical Classification of Diseases and Related Health Problems, 10th Revision, Canada (ICD-10-CA); and the Canadian Classification of Health Interventions (CCI) and applicable directive statements from the Canadian Coding Standards. Case studies are provided to give coders the opportunity to apply the information presented in this package to chart documentation.

Objectives

The main purpose of this SLP is to:

- Provide supplementary information to the Canadian Coding Standard on Acute Coronary Syndrome to ensure a consistent interpretation and application of the directive statements
- Provide information on the clinical picture and treatment protocols for the spectrum of diseases included in ACS
- Inform coders of the changes at subcategory R94.3 Abnormal results of cardiovascular function studies, effective for discharges as of April 1, 2007
- Inform coders of the new mandatory status attributes at 1.IJ.50. ^ ^ Dilation, coronary arteries, effective for discharges as of April 1, 2007
- Facilitate correct code assignment to the new terminology being used to describe acute myocardial infarction (AMI)
Who Should Complete This Self-Learning Package?
This self-learning package is intended for all health information management professionals working with the Discharge Abstract Database (DAD) and/or the National Ambulatory Care Reporting System (NACRS) who are responsible for coding patient records, submitting data to CIHI and/or analyzing clinical health data.

What Will You Need?
Prior to completing this SLP, review the 2007 Canadian Coding Standards for ICD-10-CA and CCI entitled:

- Acute Coronary Syndrome
- Percutaneous Coronary Interventions (PCI)
- Thrombolytic Therapy
- Angina
- Chronic Ischemic Heart Disease

In Appendix A of this document, you will find a glossary of some of the key terms used here.
Section 2: Background

In our lifetime, we have seen significant advances in the prevention, diagnosis and treatment of acute myocardial infarction and acute coronary syndrome. Even the clinical terminology used to describe the spectrum of ACS has changed. For example, the term “ACS” is a fairly recent addition to the medical lexicon. Diagnostically, the high sensitivity of the newer biomarkers enables detection of small areas of myocardial necrosis that may not show up on ECG. Therapeutically, timely reperfusion therapies minimize or avert myocardial necrosis. These changes have contributed to a welcome decline in morbidity and mortality for individuals with ACS.

Learning Outcomes

- Understand that new terminology is being used to describe AMI
- Recognize that, in the ICD-10 classification, the terms “STEMI” and “NSTEMI” are not mutually exclusive with transmural and nontransmural infarction
- Acknowledge the national interest in STEMI and NSTEMI and the coder’s role

2.1 New Terminology

Formerly, AMI was designated by clinicians as being either Q-wave (transmural) or non-Q-wave (nontransmural or subendocardial). AMI is now being clinically designated based on the presence or absence of ST-segment elevation on ECG. This is an important distinction, as it determines the clinical treatment of the patient.

In this document, “Q-wave MI” is used interchangeably with the term “transmural MI,” and “non-Q-wave MI” with the terms “nontransmural MI” and “subendocardial MI.”

2.2 STEMI and NSTEMI in ICD-10

The ability to accurately distinguish between STEMI and NSTEMI is not possible with the current ICD-10 classification system. In October of 2002, CIHI put forward a proposal to the World Health Organization (WHO) to add inclusion terms for STEMI and NSTEMI to the existing categories for transmural and subendocardial myocardial infarction in ICD-10-CA. However, this proposal was withheld because it did not meet the WHO criteria that codes in ICD-10 must be mutually exclusive. As you will see, the term “STEMI” is not always synonymous with transmural MI, and NSTEMI is not always synonymous with subendocardial MI.

The current terminology used by clinicians to describe acute myocardial infarction is STEMI and NSTEMI. These terms have not yet received approval by the WHO for inclusion in ICD-10 or ICD-10-CA at category I21.- Acute myocardial infarction.
2.3 National Interest in STEMI and NSTEMI

Being able to provide statistical information regarding STEMI and NSTEMI will contribute needed insight into the present-day management of AMI in Canada and will lead to changes in diagnosis and treatment and more desirable patient outcomes. To address this need for information, changes have been introduced to both ICD-10-CA and CCI. Subcategory R94.3-\textit{Abnormal results of cardiovascular function studies} has been expanded and new mandatory attributes have been added at rubric 1.IJ.50. ^ ^ \textit{Dilation, coronary arteries}.

Coders across Canada play a pivotal role through the accurate and consistent application of the new codes and standards related to STEMI and NSTEMI.

Section 10: Summary contains additional related information.
Section 3: Acute Coronary Syndrome—What Is It?

Acute coronary syndrome describes a spectrum of conditions, including unstable angina, non ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI). ACS describes the constellation of signs and symptoms compatible with acute myocardial ischemia—chest pain/discomfort/pressure, dizziness/light-headedness, shortness of breath and sweating.

ACS develops when an atheromatous plaque undergoes disruption. Disruption of the plaque causes the formation of a thrombus. The thrombus results in a reduction of blood flow through the affected coronary artery and the patient experiences ischemic pain. The thrombus can completely or partially occlude the coronary artery.

The umbrella term “acute coronary syndrome” is useful in that it groups patients with symptoms consistent with acute myocardial ischemia and is the basis for subsequent established diagnostic and treatment decisions.

Learning Outcomes

- Recognize that the term “acute coronary syndrome” is a catch-all phrase that refers to a spectrum of clinical presentations ranging from unstable angina through NSTEMI and STEMI
- Describe ACS in terms of a continuum of acute events ranging from myocardial ischemia to injury to necrosis
- Explain the ACS schema in the Canadian Coding Standards
- Recognize that patients presenting with ischemic chest pain are quickly divided into two subgroups to ensure that they receive the appropriate treatment protocol

3.1 Pathophysiology of Acute Coronary Syndrome

The lack of blood supply to the heart results in a continuum of acute events ranging from myocardial ischemia to injury to infarction. The most common cause of this diminished blood flow is coronary atherosclerosis—plaque formation within the coronary arteries.

Myocardial ischemia appears at the onset, and the subendocardial region is the first to be affected, since this layer of the heart is farthest from the blood supply. When ischemia is severe it results in injury to the myocardial cells. Simply speaking, subendocardial injury is manifested on ECG by ST-segment depression, and transmural injury is manifested by ST-segment elevation. Myocardial infarction describes necrosis or death of myocardial cells. A myocardial infarction can either be nontransmural (partial thickness) or transmural (full-thickness).
The appearance of pathological Q-waves is the most characteristic ECG finding of transmural infarction. Non-Q-wave infarction is diagnosed in the presence of ST depression and T wave abnormalities.\(^1\)

Elevation of biomarkers is expected in both types of infarction. In the absence of elevated biomarkers, ST and T wave abnormalities are interpreted as due to ischemia or injury rather than infarction.

Non ST-segment elevation (NSTEMI), typically consisting of ST depression or T wave inversion, as well as ST-segment elevation (STEMI), are preliminary findings for which the final outcome can be unstable angina, non-Q-wave infarction, Q-wave infarction or aborted infarction. Thrombus formation secondary to plaque rupture is the common link between these conditions.

Unstable angina corresponds to an acute change in the morphology of a high-risk plaque, with overlying thrombus formation that only partially occludes the vessel and hence results in no more than intermittent ischemia. The thrombus may be quickly broken down by spontaneous lysis; therefore, the symptoms can disappear as quickly as they came.

Non-Q-wave infarction is thought to result from persistence of thrombus with greater plaque disruption than in unstable angina. However, occlusion is usually short-lived (less than an hour), and the distal myocardial territory is usually supplied by collaterals; therefore, necrosis is confined to the subendocardium.

Q-wave infarction is believed to develop as a result of larger plaque fissures, when spontaneous thrombolysis, resolution of vasoconstriction and presence of collateral circulation are absent. The result is fixed, persistent and complete thrombotic occlusion, with abrupt cessation of myocardial perfusion lasting more than an hour and resulting in transmural necrosis.\(^2\)

Acute coronary syndrome represents a spectrum of conditions that are all caused by disruption of atherosclerotic plaque.

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3.2 Explanation of Schema in Coding Standards

The schema in the Canadian coding standard entitled *Acute Coronary Syndrome* illustrates the typical flow of diagnostic and treatment events for patients presenting with symptoms of ACS. Patients with ACS may present with or without ST-segment elevation on the ECG.

Of patients with ST-segment elevation, most will ultimately develop Q-wave MI. However, immediate revascularization using either thrombolytic therapy or primary PCI can alter the final outcome or type of MI. Some patients will develop only a non-Q-wave MI, and in others myocardial necrosis may be averted completely.

Patients who present without ST-segment elevation are suffering from either unstable angina or a non ST-segment elevation MI, a distinction that is ultimately made on the presence or absence of a serum cardiac marker (biomarkers) such as CK-MB or a cardiac troponin detected in the blood. Most patients presenting with NSTEMI ultimately develop non-Q-wave MI on the ECG. The remainder of patients presenting without ST-segment elevation will have negative biomarkers; therefore, their diagnosis will be unstable angina.iii

Certain conditions may initially present with symptoms similar to ACS, and biomarkers may even be slightly elevated. Additional investigations may reveal that the symptoms are in fact related to non-ischemic cardiac conditions such as pericarditis or to non-cardiac conditions such as esophagitis or pulmonary embolism.

The schema in the coding standards is not intended to provide direction for code assignment in cases where the documentation is lacking. When documentation is lacking and the coder is not sure of the diagnosis, the coder must seek clarification from the physician or assign a code from the appropriate “unspecified” category.

Patients presenting to hospital with signs and symptoms characteristic of acute cardiac ischemia will quickly be categorized into two broad groups based on ECG findings:

- Those presenting with ST-segment elevation AND
- Those presenting without ST-segment elevation.

Those with ST-segment elevation will be given a working diagnosis of STEMI and those without ST-segment elevation will be given a working diagnosis of NSTEMI. Categorizing patients into these two groups ensures that they receive the appropriate treatment protocols.

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3.3 Classifying Acute Coronary Syndrome

Acute coronary syndrome, when not further described, is classified to I24.9 Acute ischaemic heart disease, unspecified. This is a non-specific code and is used only when no further information or higher knowledge about the condition is known.

A diagnosis of acute coronary syndrome is classified according to the most specific diagnosis documented in the record. R94.3- Abnormal results of cardiovascular function studies is not assigned with I24.9.
Acute Coronary Syndrome: Understanding the Spectrum

Section 4: ECG Changes, Biomarkers and Applying Code R94.3-

When a patient sustains myocardial necrosis, biochemical markers (biomarkers) are released into the blood stream and can be measured using simple blood tests. ECG changes are not always specific for myocardial damage and, in some patients, the ECG may even be normal.

Learning Outcomes

• Explain the significance of ECG changes and elevated biomarkers in ACS
• Correctly apply the new ICD-10-CA codes from subcategory R94.3- Abnormal results of cardiovascular function studies

4.1 ECG Changes in Myocardial Infarction

Most patients with MI have ECG changes. However, the extent of the ECG abnormalities provides only a crude estimate of the magnitude of infarction. The classic evolution of changes, which may occur over a few hours to several days, in a “full blown” infarction is:

Peaked T waves → ST-segment elevation → Q-wave development → T wave inversion

The above information is not provided for the coder to interpret the ECG report. Interpretation of the ECG must be left to the physician.

Only a few ECG patterns have high specificity for infarction. In general, an upwardly concave elevation of the ST-segment is considered diagnostic for acute myocardial injury, with a high degree of specificity. Without acute ST-segment elevation or the development of new Q-waves, no other ECG changes can be considered highly specific. The ECG can even be totally normal.iv

When interpreted in light of the clinical presentation of the patient, electrocardiograms can be invaluable in aiding selection of the most appropriate management.

4.2 Biomarkers

Myocardial necrosis can be recognized by the appearance in the blood of different proteins released into the circulation due to damaged myocytes: myoglobin, cardiac troponins T and I, creatine kinase and lactate dehydrogenase, as well as many others. Myocardial infarction is diagnosed when levels of sensitive and specific biomarkers, such as cardiac troponin and MB fraction of creatine kinase (CKMB), are increased in the clinical setting of acute ischemia. However, although detectable increases of the cardiac biomarkers are indicative of injury to

the myocardium, the elevations are not synonymous with an ischemic mechanism of injury. Therefore, increases do not now and did not in the past mandate a diagnosis of myocardial infarction.v

Do not assume myocardial infarction has occurred based on elevated biomarkers alone. Elevated biomarkers are an indication that myocardial injury has occurred, but the injury is not necessarily due to ischemic causes (such as AMI). In the absence of clinical evidence of ischemia, the clinician will search for other causes of cardiac damage. Non-ischemic conditions that may cause elevated biomarkers include congestive heart failure, pericarditis, myocarditis, contusion, cardiomyopathy, shock, renal insufficiency and pulmonary emboli.

Tests for biomarkers may become positive as early as 4 to 6 hours after the onset of a myocardial infarction and should be abnormal by 8 to 12 hours. Circulating levels of troponins may remain elevated for five to seven days or longer.vi

Troponin, which is more specific than CK-MB for myocardial damage, can identify smaller amounts of myocardial necrosis and is a better predictor of recurrent cardiac events and mortality. Because troponin levels rise sooner than CK-MB and remain elevated longer, troponin has become a critical indicator of myocardial necrosis. Troponin has two forms: cardiac-specific troponin T and cardiac-specific troponin I. Neither is detectable in healthy people. The new guidelines consider values greater than 0.01 ng/ml for either type as elevated and values greater than 0.1 ng/ml as markedly elevated indicating a high risk of death.vii

The above values are not provided for the coder to interpret the significance of biomarker results. Interpretation of the results must be left to the physician.

4.3 Applying R94.3- Abnormal results of cardiovascular function studies

The international version of the ICD does not yet include terminology for STEMI and NSTEMI. In order to maintain international comparability, ICD-10-CA has retained the integrity of category I21 Acute myocardial infarction and category I22 Subsequent myocardial infarction. However, for discharges as of April 1, 2007, subcategory R94.3 Abnormal results of cardiovascular function studies has been expanded to capture working diagnoses of STEMI or NSTEMI. Codes from category I21 are used to capture a final diagnosis recorded as STEMI or NSTEMI.

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In ICD-10-CA, the final diagnosis of AMI is still classified based on the final outcome of Q-wave MI versus non-Q-wave MI. The working diagnosis is captured using the appropriate R code.

**R94.3 Abnormal results of cardiovascular function studies**

**Note:** These expanded codes are valid as of April 1, 2007. For 2006 abstracts, continue to use R94.3.

**R94.30 Electrocardiogram suggestive of ST segment elevation myocardial infarction [STEMI]**

**R94.31 Abnormal cardiovascular function studies (biomarkers or ECG) suggestive of non ST segment elevation myocardial infarction [NSTEMI]**

- ST depression
- T waves

**R94.38 Other and unspecified abnormal results of cardiovascular function studies Abnormal:**

- electrocardiogram [ECG] [EKG] not elsewhere classified
- electrophysiological intracardiac studies
- phonocardiogram
- vectorcardiogram

Use the physician’s documentation of his or her interpretation of the ECG findings and biomarker results (for example, troponin or CK-MB) to select the appropriate fifth digit from R94.3-.

The R code may be based on information from the transferring hospital if available. If the information regarding the working diagnosis is not available, then assign R94.38.

The main problem in NACRS is selected based on the highest degree of knowledge about the condition at the time of discharge from the ED. In the ED, a final diagnosis of STEMI or NSTEMI typically represents a working diagnosis, as the final outcome will likely not be known at the time of discharge from the ED. If the final outcome is known prior to the decision to admit time or transfer time, then it would be appropriate to code AMI or unstable angina as the main problem.

### 4.4 Applying the Coding Standards

It is mandatory, for both DAD and NACRS, to code R94.3- as a diagnosis type (3)/Other problem whenever I21.- Acute myocardial infarction or I24.0 Coronary thrombosis not resulting in myocardial infarction appears on an abstract.
Use the physician’s documentation of her or his interpretation of the ECG findings and biomarker results.

When the physician has not documented an interpretation of the ECG findings or biomarker results, then R94.38 Other and unspecified abnormal results of cardiovascular function studies is assigned.

When the discharge diagnosis in the emergency department (ED) is documented as STEMI or NSTEMI, R94.3- is assigned as the main problem.
Section 5: STEMI—What Is It?

Any patient experiencing ST-segment elevation is considered to be undergoing an acute MI and will be evaluated for immediate reperfusion therapy. This subset of patients is said to be “reperfusion eligible.”

Learning Outcomes

- Define the term “STEMI” and recognize that an initial impression of STEMI may have three possible outcomes
- Select the correct ICD-10-CA codes for a final diagnosis of STEMI

5.1 STEMI as a Working Diagnosis

For patients with a working diagnosis of STEMI, every effort will be made to restore normal arterial blood flow as soon as possible with either thrombolytic therapy or PCI. If intervention is employed early enough, myocardial necrosis may be minimized or averted.

When ST-segment elevation is present, the clinician does not have to await the results of biomarkers to confirm that the patient is undergoing a myocardial infarction.

When the working diagnosis is STEMI, there are three possible final outcomes:

1. Most patients will develop a Q-wave MI
2. A few patients will develop only a non-Q-wave MI
3. Some patients who receive prompt reperfusion are known to avoid myocardial necrosis altogether viii aborted or averted MI

5.2 STEMI as a Final Diagnosis

STEMI occurs due to complete and persistent thrombotic occlusion of a major coronary artery. Total thrombotic occlusion occurs most commonly in proximal coronary arteries; its presence has been documented during the first four hours after infarction in more than 85% of patients who present with ST-segment elevation. Most patients who present in this manner subsequently develop Q-waves.ix

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Although most patients with ST-segment elevation will go on to develop a Q-wave myocardial infarction, prompt treatment may minimize or even avert myocardial damage. Therefore, a working diagnosis of STEMI does not necessarily mean that the final outcome is going to be transmural infarction.

### 5.3 Applying the Coding Standards

**DAD Abstract:** When the final diagnosis is stated as STEMI, classify the AMI to acute transmural myocardial infarction (I21.0 to I21.3). **Rationale:** Most patients with ST-segment elevation will ultimately develop a Q-wave MI.

**DAD Abstract:** When the final diagnosis is stated as non-Q-wave, nontransmural or subendocardial MI, but the initial impression is STEMI, classify the MI to subendocardial MI (I21.4-). **Rationale:** A final diagnosis of non-Q-wave, nontransmural or subendocardial MI where the initial presentation was STEMI is indication that prompt revascularization has limited myocardial damage to the subendocardium.

**DAD Abstract:** When a patient presents as STEMI, but myocardial damage is completely averted by prompt revascularization, classify to I24.0 Coronary thrombosis not resulting in myocardial infarction. **Rationale:** Immediate revascularization may completely avert any myocardial necrosis. Look for documentation of averted or aborted MI.

**NACRS Abstract:** When the discharge diagnosis is documented as STEMI, select R94.30 Electrocardiogram, suggestive of ST segment elevation myocardial infarction [STEMI] as the main problem. **Rationale:** In the ED, a discharge diagnosis of STEMI typically represents a working diagnosis or initial impression only. A working diagnosis or initial impression of STEMI is captured by selecting R94.30 rather than a code from category I21.- Acute myocardial infarction.

For reporting to the DAD, unless the physician has somehow indicated that myocardial damage was minimized to the subendocardium or was aborted, it is assumed that the damage was transmural.
Section 6: NSTEMI—What Is It?

The frequency of NSTEMI is increasing because of the use of newer, more sensitive and specific markers, such as troponins, to detect small events. In the past, NSTEMI may have gone unrecognized and was diagnosed as unstable angina.

Learning Outcomes

- Define the term “NSTEMI” and recognize that an initial impression of NSTEMI may have two possible outcomes
- Select the correct ICD-10-CA codes for a final diagnosis of NSTEMI

6.1 NSTEMI as a Working Diagnosis

In patients presenting without ST-segment elevation, it is clinically difficult to distinguish between NSTEMI and UA based on symptoms alone. The differentiating feature is that patients with NSTEMI have abnormal biomarkers, proving that myocardial injury has occurred.

When the working diagnosis is NSTEMI, the physician will have to await results of tests for biomarkers to determine whether the final diagnosis is:

- Non-Q-wave MI
- Unstable angina

For many patients, it takes 6 to 12 hours to complete a series of blood enzyme tests to make this determination.

Very rarely, a NSTEMI may evolve into a transmural MI.

6.2 Unstable Angina

Stable angina refers to chest discomfort that is predictable and has a stable course. Angina becomes unstable when there is a change in frequency and when it occurs during increasingly less physical activity, lasts longer or becomes more severe in nature. In the clinical spectrum of coronary artery disease, the syndrome of unstable angina falls between stable angina and acute myocardial infarction.\(^x\)

Unstable angina may also be referred to as “non ST-segment elevation acute coronary syndrome” (NSTEACS).

R94.3- is not selected when the final outcome is unstable angina.

6.3 NSTEMI as a Final Diagnosis

Most patients presenting with NSTEMI ultimately develop non-Q-wave MI on the ECG.\textsuperscript{xii} Non-Q-wave MI results from incomplete occlusion or spontaneous lysis of a thrombus.\textsuperscript{xii} It often signifies the presence of additional jeopardized myocardium and is associated with higher incidence of reinfarction and recurrent ischemia.

NSTEMI is a myocardial infarction identified by ECG changes, excluding ST-segment elevation, in the presence of elevated cardiac biomarkers. The ECG findings may include changes such as ST depression or T wave inversion, or the ECG may be normal. The high sensitivity of the newer biomarkers enables detection of small areas of myocardial necrosis that may not show up on ECG.

A working diagnosis of NSTEMI does not mean that the final outcome is going to be nontransmural MI. If the biomarkers come back negative, then the physician will diagnose the patient with unstable angina. If biomarkers come back elevated, then the diagnosis will be NSTEMI.

6.4 Applying the Coding Standards

DAD Abstract: When the final diagnosis is stated as NSTEMI, classify the MI to subendocardial MI (I21.4-). \textbf{Rationale:} Most patients without ST-segment elevation ultimately develop non-Q-wave MI.

NACRS Abstract: When the discharge diagnosis is documented as NSTEMI, select R94.31 \textit{Abnormal cardiovascular function studies (biomarkers or ECG) suggestive of non ST-segment elevation myocardial infarction [NSTEMI]} as the main problem. \textbf{Rationale:} In the ED, a discharge diagnosis of NSTEMI typically represents a working diagnosis or initial impression only. A working diagnosis or initial impression of NSTEMI is captured by selecting R94.31 rather than a code from category I21.- \textit{Acute myocardial infarction}.

Section 7: Treatment Protocol for STEMI

Because myocardial damage progresses rapidly during the early hours, efforts during this critical period must be directed toward reducing myocardial oxygen demand and improving coronary blood supply to diminish the extent of myocardial damage. To be most effective, these interventions must be initiated as soon as possible. Thus prompt reperfusion therapy via primary PCI or thrombolytic therapy should be initiated in the absence of contraindications as early as possible in patients with ST-segment elevation acute infarctions.\textsuperscript{xiii}

The benefits of reperfusion therapy during the later phase of AMI (between 6 and 12 hours), although it has a less dramatic effect on improving postinfarction mortality rates, nonetheless have been demonstrated. Patients with ongoing angina, anterior infarctions or persisting ST-segment elevation present greater than six hours after onset of symptoms are high-risk patients who will benefit even from late treatment in the absence of contraindications.\textsuperscript{xiv}

Patients presenting with ST-segment elevation are eligible for immediate reperfusion with either PCI or thrombolytic therapy.

Learning Outcomes

- Recognize that the treatment protocol for STEMI is primary PCI or thrombolytic therapy
- Define primary PCI and select the correct status attribute
- Distinguish between thrombolytic therapy and antithrombotic therapy and select appropriate CCI codes
- Define rescue PCI and select the correct status attribute

7.1 Primary (Direct) Percutaneous Coronary Intervention

When immediate access to cardiac catheterization laboratories is possible, PCI is the treatment of choice in treating STEMI patients.\textsuperscript{xv} A primary PCI is one performed as the first intervention for STEMI within 12 hours of presentation to hospital with no thrombolytic therapy prior to PCI.


The terminology currently being used to describe percutaneous transluminal coronary angioplasty (PTCA) is “percutaneous coronary intervention” (PCI). The term “PCI” was initially limited to “PTCA,” but it now also includes techniques such as percutaneous atherectomy, laser angioplasty and implantation of intracoronary stents.

NSTEMI patients are not candidates for primary PCI.

Effective for discharges starting April 1, 2007, new mandatory status attributes have been introduced at rubric 1.IJ.50. "Dilation, coronary arteries." These attributes have been added to distinguish primary PCI from that performed later or following thrombolytic therapy. Information on primary PCI is of special interest to researchers.

The attributes are:

- **N** Primary (performed as the first intervention for STEMI within 12 hours of presentation to hospital—no thrombolytic therapy prior to angioplasty)
- **Z** Other (performed either following thrombolytic therapy or more than 12 hours after presentation to hospital)

In the case of transfer from one facility to another, calculate the time to PCI starting with the time of presentation at the first hospital.

### 7.2 Thrombolytic Therapy

Thrombolytic therapy has become the standard of care for treating STEMI patients when immediate access to PCI is not available. Thrombolytic therapy, also called “thrombolysis,” “fibrinolysis” or “lytics,” involves the administration of clot-busting drugs to dissolve thrombus in the affected coronary artery or arteries and restore blood flow to the heart muscle. Thrombolytic therapy reduces mortality and limits infarct size in patients with AMI associated with ST-segment elevation.

Patients without ST-segment elevation generally have temporary, incomplete or partial occlusions and do not benefit from thrombolytic therapy.

Thrombolytic agents include streptokinase, urokinase, tissue plasminogen activator (tPA), alteplase, retaplase, tenecteplase and TNKase (TNK).

Thrombolytic agents are not to be confused with antithrombotic agents. Thrombolytic agents treat **existing blood clots** by dissolving them, whereas antithrombotic agents reduce blood coagulation or platelet activation to prevent the formation of blood clots.
7.3 Failure of Thrombolytic Therapy and Rescue or Salvage PCI

Despite its expanding use, in some patients with AMI, thrombolytic therapy may be limited by its failure to reperfuse the occluded artery, by recurrent ischemia despite successful initial reperfusion and by hemorrhagic complications. If thrombolytic therapy fails to open the occluded artery, or if there is recurrent ischemia, the patient may undergo rescue PCI.\textsuperscript{xvi}

Rescue PCI is performed as an emergent/urgent intervention in patients with continuing myocardial ischemia after treatment with thrombolytic therapy has failed. The correct status attribute for rescue PCI is \textit{Z Other}.

7.4 Applying the Coding Standards

It is mandatory, for both DAD and NACRS, to code percutaneous coronary interventions.

Status attribute \textit{N Primary} is selected when all three of the following criteria are met:

- The diagnosis is STEMI or Q-wave (transmural) \textbf{AND}
- PCI is performed:
  - Within 12 hours of presentation to hospital (starting with presentation to first hospital) \textbf{AND}
  - Prior to administration of thrombolytic agents

Status attribute \textit{Z Other} is selected when PCI is performed:

- After administration of thrombolytic agents \textbf{OR}
- For NSTEMI or UA \textbf{OR}
- After 12 hours of presentation to hospital

Assign an additional code from rubric \textit{1.L.35. Pharmacotherapy (local), vessels of heart}, mandatory, when drug-eluting stents are inserted during PCI.

It is mandatory, for both DAD and NACRS, to code thrombolytic therapy. If thrombolytic therapy begins in the emergency department and continues into the acute care episode, code the intervention on both the NACRS abstract and the DAD abstract.\textsuperscript{xvii}


Thrombolytic agents administered by intravenous infusion are classified to 1.ZZ.35.HA-C1 Pharmacotherapy, total body using antithrombotic agent and percutaneous approach [intramuscular, intravenous, subcutaneous, intradermal].

Thrombolytic agents that are injected into a coronary artery are classified to 1.II.35.HA-C1 Pharmacotherapy (local), vessels of heart of antithrombotic agent using percutaneous injection approach.
Section 8: Treatment Protocol for NSTEMI

Thrombolytic therapy and primary PCI are not performed in patients with NSTEMI, and these patients are managed similarly to patients with unstable angina.

Learning Outcomes

• Recognize that the treatment protocol for NSTEMI is antithrombotic therapy and PCI for patients with continuing or recurrent ischemia

8.1 Antithrombotic therapy

Patients presenting with an acute myocardial infarction without ST-segment elevation or evolving Q-waves generally do not have persistent thrombotic coronary occlusions. Therefore, these patients are not candidates for thrombolytic therapy, and in general their management should be similar to that of patients with unstable angina. Patients with continuing or recurrent ischemia or very positive stress tests should undergo angiography and PCI when appropriate.xviii

Patients presenting with NSTEMI may be treated with antithrombotics such as heparin to inhibit the coagulation process. Medical management following a myocardial infarction (of any type) may include platelet aggregation inhibitors, ACE inhibitors and acetylsalicylic acid to prevent further atherothrombotic events.

8.2 Applying the Coding Standards

It is optional to capture pharmacotherapy using antithrombotics such as heparin or platelet aggregation inhibitors such as Plavix, ReoPro and Integrisil.

Anticoagulants and platelet aggregation inhibitors are not thrombolytic agents.

Section 9: Diagnosis Typing and Sequencing

In this section we will review some directive statements that are found within Chapter IX, Diseases of the Circulatory System of the Canadian Coding Standards, that pertain to diagnosis typing and sequencing of AMI, CAD and UA.

Learning Outcomes

- Apply correct diagnosis types and sequencing to AMI, UA and Coronary Artery Disease (CAD)

9.1 Acute Myocardial Infarction

AMI within the acute phase (that is, within 28 days) is assigned a comorbid diagnosis type (M), (1), (2), (W), (X) or (Y). Exception: Readmission for subsequent MI.

When a patient is readmitted within four weeks with a subsequent infarction, it is optional to code the original infarction. Rationale: The fact that the patient has had a previous myocardial infarction within the past four weeks is inherent in the code title at I22 Subsequent myocardial infarction. When a code for the original infarction is selected, assign it a diagnosis type (3). Coding the original infarction will also necessitate the assignment of a code from R94.3-.

I22.- (MRDx)
I21.- (3) optional coding
R94.3- (3) mandatory when I21.- is assigned

When the patient has a subsequent MI during the same episode as the original MI, then three codes are required. The original MI is selected as the most responsible diagnosis (MRDx) and the subsequent MI is selected as a type (2) diagnosis. A code from subcategory R94.3- is used once, and its selection is based on the documentation pertaining to the original MI.

I21.- (MRDx)
R94.3- (3) mandatory when I21.- is assigned
I22.- (2) R code is not required to describe the subsequent MI

9.2 Angina

Classify angina as a significant condition only when it is documented as occurring during the current episode of care.

Do not code angina when it progresses to MI. Rationale: UA and MI are a continuum of the same disease process.
9.3 Percutaneous Coronary Intervention (PCI)

For emergent or urgent admissions with any condition in the spectrum of ACS (UA, AMI, STEMI, NSTEMI) with PCI performed during the same admission, select the ACS diagnosis as the MRDx and any mention of coronary artery disease (CAD) as a diagnosis type (1). This applies to in-hospital and out-of-hospital PCI and to both the transferring and receiving hospitals. Rationale: PCI is often a life-saving event; therefore, the focus of care is directed towards the acute ischemic event and secondarily towards the underlying CAD.

ACS spectrum (MRDx)
CAD (1)

When a patient who has an MI that is still in the acute phase (within 28 days) is admitted electively for PCI, the underlying CAD is selected as the MRDx, and the MI is assigned a significant diagnosis type.

CAD (MRDx)
AMI (1)

When an urgent or emergent admission is for a condition within the spectrum of acute coronary syndrome, and the patient undergoes PCI during the same episode of care, the condition within the spectrum of ACS remains the MRDx even for the institution performing the PCI.

9.4 Coronary Artery Bypass Grafting (CABG)

When a CABG is performed for unstable angina due to coronary artery disease, select I25.1 Atherosclerotic heart disease as the MRDx. Unstable angina will be assigned a significant diagnosis type only when documented as occurring during the current episode of care.

CAD (MRDx)
UA (1) or (3)

When a patient is admitted with AMI and undergoes CABG during the same admission, select I25.1 Atherosclerotic heart disease as the MRDx and assign diagnosis type (1) to the code for myocardial infarction.

CAD (MRDx)
AMI (1)

Whenever a CABG is performed for UA or AMI, select the underlying CAD as the MRDx.
Section 10: Summary

Acute myocardial infarction is now being designated by clinicians based on the presence or absence of ST-segment elevations, a distinction that determines the clinical treatment of the patient. The current terminology to describe AMI is STEMI and NSTEMI. This terminology is not yet included in the international version of the ICD (ICD-10). As an interim solution, ICD-10-CA has expanded subcategory R94.3- Abnormal results of cardiovascular function studies to capture information regarding STEMI and NSTEMI.

This means that if a patient has suffered a myocardial infarction or aborted myocardial infarction, two codes are required. The first code, I21.- or I24.0, will describe the AMI in terms of the final outcome such as Q-wave versus non-Q-wave versus aborted MI. The second code, R94.3, will add information regarding the presence or absence of ST-segment elevations (STEMI versus NSTEMI).

The addition of the R code allows researchers to separate patients that have suffered an MI (or aborted MI) into two subgroups:

- those that had ST-segment elevations present on ECG
- those that did not have ST-segment elevations on ECG

The acute coronary syndrome spectrum concept is a useful framework for developing therapeutic strategies. Patients presenting with persistent ST-segment elevation are candidates for reperfusion therapy (either with thrombolytic therapy or PCI) to restore flow promptly in the occluded epicardial infarct-related artery. Patients presenting without ST-segment elevation are not candidates for immediate reperfusion therapy, but should receive anti-ischemic therapy and PCI therapy where applicable.xix

For research purposes, being able to provide information about STEMI and NSTEMI supplies insight into the acute management of MI in Canada. For example:

- What is the incidence of STEMI versus NSTEMI?
- Are patients receiving appropriate treatment, as per international guidelines, for STEMI and NSTEMI?
- How many patients are receiving PCI as their first intervention?
- Does primary PCI have better outcomes than thrombolytic therapy?
- Do patients have immediate access to facilities that perform PCI?
- Does Canada need more angioplasty facilities so that patient outcomes can be improved?

Of special interest to researchers is the STEMI subset of patients. A recent Canadian study found that:

- Although primary PCI is the preferred mode of reperfusion therapy for STEMI (because if it is performed in a timely manner it has superior outcomes to thrombolysis), less than 10% of patients presenting with STEMI received primary PCI.
- The prompt administration of thrombolytics in STEMI is associated with decreased mortality, yet only 35 to 44% of patients received therapy within the current standard of practice.

The results of this study suggest that the use of evidence-based therapies (such as primary PCI and thrombolyis) is improving but remains lower than optimal.xx

Section 11: Case Studies

You will now have the opportunity to apply what you have learned in this SLP to chart documentation. Assign the correct ICD-10-CA and CCI codes for the following case studies. Record your answers in the space provided.

Case Study #1 DAD

Admitted: June 19, 2006
Discharged: June 22, 2006
Final Diagnosis: Unstable angina
Admitting Diagnosis: Acute coronary syndrome

History of Present Illness: This 59-year-old female with known coronary artery disease presented to the cardiology services of Hospital ABC with complaints of heavy retrosternal chest pain that was only partially relieved with sublingual NitroSpray. The patient’s cardiac risk factors include hypertension, type 2 diabetes, hyperlipidemia, family history and smoking.

Physical Examination: The patient had a blood pressure of 105/50 mmHg, heart rate of 84. Head and neck exam was normal. Her cardiovascular exam demonstrated a normal S1 and S2 and no evidence of extra heart sounds or murmurs. Her JVP was not elevated, and her peripheral pulses were positive. Her chest was clear to auscultation. Her abdomen was obese, but soft and non tender.

Investigations: ECG showed ST depression. Her cardiac enzymes were negative during her stay here in hospital. The patient was admitted for treatment of unstable angina and for further risk stratification.

Summary: This 59-year-old female with known coronary artery disease was admitted to the cardiology service for medical treatment and further investigations into her unstable angina. The patient was not cooperative with regards to the medical team’s desire for further investigations. The risks of her decision were explained to her, but she was steadfast in her decision to not cooperate. The patient, however, was stable during her stay in hospital and was discharged home.
Case Study #1 DAD (cont’d)

Discharge Medications:
Accurpril 40 mg p.o. once daily
Nu-Triazide 50/25 mg p.o. once daily
Nitropatch 0.4 mg q 12 h
Metoprolol 50 mg p.o. b.i.d.
Plavix 75 mg p.o. once daily
Enteric-coated ASA 81 mg p.o. once daily
Lipitor 40 mg p.o. once daily
Omeprazole 20 mg p.o. once daily

My answers:
Case Study #2 DAD

Admitted: May 15, 2006

Discharged: May 18, 2006

Final Diagnosis: Non ST-segment elevation myocardial infarction

History of Present Illness: This is a 45-year-old male admitted to the cardiology service on May 15, 2006. He presented with a clinical picture and subsequent ECG and enzyme documentation of a small ACS event. ECG showed ST depression and troponin was 1.33. A non ST-segment elevation myocardial infarction was documented.

Course in Hospital: This patient was admitted to the cardiology ward with medical management implemented. A subsequent cardiac catheterization documented significant triple-vessel coronary artery disease, and a surgical consultation was obtained. It was determined that proceeding with coronary artery bypass procedure would indeed be appropriate, and this will be scheduled. In the meantime, ongoing medical management will be continued.

No significant past history is identified.

Following satisfactory assessment the patient was discharged home on the morning of May 18, 2006.

Discharge Medications
Enteric coated ASA 81 mg p.o. once daily
Plavix 75 mg p.o. once daily
Metoprolol 25 mg p.o. b.i.d.
Ramipril 5 mg p.o. once daily
Lipitor 40 mg p.o. once daily
Sublingual nitroglycerine spray p.r.n.
Case Study #2 DAD (cont’d)

Catheterization Report:
Indications: NSTEMI
Procedures: Left ventriculogram, selective right coronary angiogram, selective left coronary angiogram
Entry site: Right femoral artery
Conclusion: Triple vessel CAD with the culprit being a chronic occlusion of the RCA, with a large thrombus in the RCA. I discussed the case with Drs. A and B and the patient will be referred for bypass. I did not open the RCA due to the concern for a shower embolus to his microcirculation.

My answers:
Case Study #3 DAD

**Admitted:** June 8, 2006

**Discharged:** June 16, 2006

**Admission Diagnosis:** Inferior myocardial infarction

**Final Diagnosis:** Acute inferior wall ST-segment elevation MI

**History of Present Illness:** This 53-year-old female was air-lifted from her home hospital, ABC, to Hospital XYZ with complaints of severe anginal chest pain radiating to her back beginning at 13:00 today. An ECG done in the peripheral hospital demonstrated ST elevation in her inferior leads and right-sided chest leads as well, and patient was immediately transferred to Hospital XYZ for PCI. On examination in the emergency room, the patient’s vitals were a blood pressure of 102/52, heart rate of 45 beats per minute, respiratory rate of 10 breaths per minute, oxygen saturations appropriate for room air. Patient’s cardiovascular exam demonstrated no extra heart sounds; no murmurs; JVP was not visible; no evidence of peripheral edema. Patient’s chest exam demonstrated some minimal crackles at the bases and a normal abdomen; she was neurologically intact. Her cardiac risk factors included smoking one pack per day for greater than 20 years and diabetes. Patient was admitted to the coronary care unit for emergent PCI.

1. Inferior wall myocardial infarction—The patient underwent emergent PCI, and a stent was inserted to the patient’s RCA. The patient was stable post-procedure and no longer complained of angina. There was no evidence of clinical congestive heart failure. An echocardiogram was performed and the preliminary results demonstrated EF of 45%, inferior wall akinesis consistent with her myocardial infarction and right ventricular hypokinesis. Her angiogram had also shown a normal left main coronary artery, 80% stenosis of her mid LAD, 100% stenosis of her mid left circumflex, 100% of her right coronary artery.

2. Type 2 diabetes—Her blood glucose control was within normal limits in hospital.

**Investigations:**

1. Coronary angiogram with stenting to the right coronary artery.
2. Echocardiogram
Case Study #3 DAD (cont’d)

**Discharge Medications:**
- Plavix 75 mg p.o. once daily
- Enteric coated ASA 81 mg p.o. once daily
- Ramipril 2.5 mg p.o. once daily
- Lipitor 40 mg p.o. q.h.s.
- Pantoloc 40 mg p.o. once daily
- Metformin 500 mg p.o. q.i.d.

**Summary:** This 53-year-old female was admitted to the coronary care unit for management of an inferior wall myocardial infarction. Patient underwent emergent PCI to her right coronary artery. The patient responded well to treatment and was discharged back to her home hospital in stable condition.

**Catheterization Report** June 8, 2006, 15:46
- **Indication:** Inferior wall myocardial infarction.
- **Procedures:** Selective right and left coronary angiogram
  - Balloon angioplasty or right coronary artery
  - Stenting of right coronary artery
- **Entry site:** Right femoral artery
  - Proximal right coronary artery 100% stenosis reduced to normal. Stent deployed.
- **Conclusion:** Successful stenting to the origin of the RCA with a bare metal stent. Reopro was utilized. She will need Plavix for one year and will need to be risk stratified for the triple vessel disease.

**My answers:**
Case Study #4 DAD

**Admitted:** May 8, 2006

**Discharged:** May 11, 2006

**Final Diagnosis:**
Acute inferior ST-segment elevation myocardial infarction with failed thrombolytic therapy. Successful rescue PCI.

**Secondary Diagnosis:** Hyperlipidemia

**Discharge Medications:**
- Clopidogrel 75 mg p.o. once daily for the next 12 months as there were intracoronary stents inserted
- Enteric coated ASA 81 mg p.o. once daily
- Metoprolol 25 mg p.o. bid
- Ramipril 2.5 mg p.o. once daily
- Lipitor 40 mg p.o. at h.s.
- Nitroglycerine spray 0.4 mg sublingual p.r.n.

**Procedures:** On May 8, successful angioplasty and stenting was undertaken to the right coronary artery with two stents placed. On May 9, the patient returned to the catheterization lab for successful stenting to the left anterior descending coronary artery. The echocardiogram performed on May 8 showed a grade II ventricle with an ejection fraction of 48%.

**Course in Hospital:** Patient was received in transfer with an acute inferior wall ST-segment elevation myocardial infarction, having failed thrombolytic therapy. There was no preceding history of coronary artery disease. He had received thrombolytic therapy utilizing TNK at his local hospital. Unfortunately, chest pain persisted and he was transferred to Hospital B for coronary angiography and possible intervention. He went to the cath lab on the day of arrival and he was found to have significant vessel disease with lesions in the RCA and LAD. The left main showed a 20% stenosis. The ostium of the left anterior descending had 30% stenosis and the mid left anterior descending an 80% stenosis with a 60% stenosis thereafter. The left circumflex was normal. The right coronary artery showed an 85% stenosis in the mid right coronary artery. Successful angioplasty and stenting to the right coronary artery was performed with two bare metal stents. He returned to the hemodynamics laboratory for stenting to the distal left anterior descending with a bare metal stent which was also successful the following day. His hospital course was unremarkable. He was doing well and ambulating without symptoms on the ward at the time of discharge.
Case Study #4 DAD (cont’d)

Cardiac Catheterization Report May 8, 2006, 08:33
Indications: Inferior wall myocardial infarction. This patient failed thrombolytic therapy in his local hospital.
Procedures:
Selective right and left coronary angiogram
Percutaneous coronary intervention of right coronary artery
Stenting of right coronary artery
Entry site: Right femoral artery
Right coronary artery
Middle right coronary artery 85% stenosis reduced to normal, stent deployed
Distal right coronary artery 85% stenosis reduced to normal, stent deployed
PTCA balloon used
Conclusion:
Successful angioplasty and stenting to the RCA with two bare metal stents. A significant amount of dye was used for the procedure so flow-limiting lesions in the LAD should be staged in-house.

Cardiac Catheterization Report May 9, 2006, 12:40
Indications: Inferior wall myocardial infarction. This was a staged procedure to the LAD.
Procedures:
Selective left coronary angiogram
PCI of left anterior descending artery
Stenting of left anterior descending artery
Entry site: left femoral artery
Left anterior descending artery
Distal left anterior descending artery 70% stenosis reduced to normal, stent deployed
PTCA balloon used
Conclusion: Successful stenting to the distal LAD with a bare metal stent

My answers:
Case Study #5 NACRS

Registration Date: August 7, 2006, 17:44

Reason for Visit: Unstable angina

Time Seen: 18:30

The patient is a 60-year-old female with history of hypertension and high cholesterol who presents with epigastric discomfort and left arm pain/numbness. Patient started to develop epigastric/LUQ pain around noon today. No dyspnea or palpitations. Also started to develop left arm pain/numbness. Epigastric pain felt like indigestion. She took some Dioval, but it did not help. Went to ED. In ED, she was given ASA, nitroglycerine and morphine for help in controlling pain. History of angina. Able to climb 2 flights of stairs.

Cardiac Risk Factors: Hypertension, high cholesterol, smoker.

On examination: Patient in NAD. JVP not increased. AE = AE crackles in bases bilaterally. No wheeze. ECG shows sinus rhythm with no ST-segment elevations. Inverted Ts.

Physician’s Orders: Admit. Cardiac work-up. Heparin, Plavix, ASA, angiogram when available.

Impression: NSTEMI.

Final Diagnosis: NSTEMI

Discharged: 21:35

My answers:
Case Study #6 NACRS

Registration Date: July 2, 2006, 11:09

Reason for Visit: Confusion/dizziness

Time Seen: 11:25

Patient was walking outside today and experienced a syncopal episode with loss of consciousness for approximately 2 minutes. Nausea and vomiting times 3 afterwards. No chest pain, no shortness of breath associated with this.

Medical History: Diabetes

Physician’s Orders: Admit. Neurological work-up, troponin, CT head, IV heparin

Final Diagnosis: ACS

Discharged: 13:00

My answers:
Case Study #7 NACRS

Registration Date: August 12, 2006, 16:11

Reason for Visit: Chest pain

Time Seen: 16:25

A 45-year-old male with acute onset of chest pain radiating to arms bilaterally approximately 1 hour ago. Seen in clinic, ECG changes, sent here. Still having pain, nausea and diaphoresis.

Cardiac Risk Factors: Overweight, smoked 2 packs per day for 30 years—quit 1 year ago, increased cholesterol.

ECG—ST elevation II, III, aVF

Physician’s Orders: Admit to cath lab then CCU. Heparin, nitroglycerin, Valium, Plavix, ASA

Final Diagnosis: Acute STEMI

Discharge Time: 16:50

My answers:
Case Study #8 NACRS

Registration Date: May 31, 2006, 12:47

Reason for Visit: Chest pain

Physician’s Orders: CCU work-up. Admit.

Final Diagnosis: MI

Discharge Time: 13:23

My answers:
Case Study #9 NACRS

Registration Date: June 8, 2006, 15:17

Reason for Visit: Chest pain

Time Seen: 15:19

Transferred from Hospital A by air ambulance with STEMI inferior AMI. Decreased systolic pressure 60. Decreased heart rate 30 to 40. On examination: ashen. No peripheral pulses. To catheterization lab.

Final Diagnosis: AMI.

Discharged: 15:30

My answers:
aborted myocardial infarction
Myocardial ischemia not resulting in significant myocardial necrosis due to successful myocardial salvage/preservation. In patients with ST-segment elevation on the ECG, a rapid effective reperfusion therapy improves the likelihood of aborted MI.

acute coronary syndrome (ACS)
Any constellation of symptoms and signs compatible with myocardial (heart muscle) ischemia. It encompasses both unstable angina (UA) and acute myocardial infarction (AMI).

acute myocardial infarction (AMI) or heart attack
Heart muscle necrosis (death) resulting from a severe and sudden cessation of blood flow in a coronary artery (artery supplying blood to the heart muscle), most commonly due to atherosclerotic-thrombotic occlusion. This causes inadequate supply of oxygen and nutrients (ischemia) resulting in the necrosis of myocardial cells (myocytes), which begins to develop 15 to 30 minutes after the occlusion and progresses from the subendocardium (the inner wall of the heart muscle) to the subepicardium (the outer wall of the heart muscle)—this is called the “wave-front phenomenon.” Complete necrosis of myocytes at risk requires at least four to six hours, depending on the presence of collateral blood flow into the ischemic zone, persistent/intermittent coronary artery occlusion and the sensitivity of myocytes.

angina pectoris or angina
Tightness, pressure or pain in the chest due to insufficient blood flow in the heart muscle (myocardial ischemia), resulting most often from a significant narrowing of a coronary artery.

antithrombotics
Drugs that reduce blood coagulation (anticoagulants) or platelet activation (antiplatelet agents) and thus prevent the formation of blood clots.

biomarkers
Proteins released into the circulation due to damaged myocytes. These biomarkers reflect myocardial damage, but do not indicate its mechanism. Cardiac-specific biomarkers include myoglobin, cardiac troponins T and I, creatine kinase and lactate dehydrogenase.
coronary artery disease (CAD)
Any form of pathology of the coronary arteries. Usually it is the narrowing of the coronary artery or arteries resulting from plaque build-up; the process of depositing cholesterol and other fats in the layers on the inner walls is called “arteriosclerosis” or “hardening of the arteries.”

coronary artery bypass graft surgery (CABG)
An open-heart surgical procedure involving replacing the coronary artery or arteries that have narrowed because of plaque build-up with grafts taken from blood vessels elsewhere in the body. It is usually reserved for patients with left mainstream coronary disease, with two or more blocked vessels and/or when PCI/pharmacological therapy is not a treatment option.

fibrinolysis
See thrombolytic therapy

myocyte necrosis
In the context of CAD, refers to the death of heart muscle cells resulting from insufficient blood flow resulting from occlusion (blockage) of coronary artery or arteries.

non-Q-wave myocardial infarction
Myocardial infarction not characterized by abnormal Q-waves.

non ST-segment myocardial infarction (NSTEMI)
A type of myocardial infarction where the ST portion of the QRST part of the ECG is not elevated, but the ECG is either normal or is presenting with normal pattern or abnormal pattern excluding ST-segment elevation.

nontransmural myocardial infarction
A myocardial infarction involving less than the full thickness of the myocardial wall; sometimes used synonymously with subendocardial myocardial infarction.

percutaneous coronary intervention (PCI)
A group of techniques used to improve circulation in the coronary arteries that have narrowed because of plaque build-up (revascularization) or atherosclerotic-thrombotic occlusion (reperfusion). Initially limited to percutaneous transluminal coronary angioplasty or PTCA (“balloon angioplasty”), PCI now includes other techniques (for example, rotational atherectomy, directional atherectomy, extraction atherectomy, laser angioplasty and implantation of intracoronary stents and other catheter devices). PCI is a technique that is being continually refined and modified.
percutaneous transluminal coronary angioplasty (PTCA)
A PCI that consists of inserting a catheter through the blood vessel to the identified coronary artery and inflating and deflating a balloon at the end of the catheter several times to flatten the plaque build-up in the coronary artery (“balloon angioplasty”).

primary PCI (immediate PCI or direct PCI or acute PCI)
PCI performed in patients with myocardial ischemia as emergent/urgent therapy to restore blood flow to the heart muscle (reperfusion therapy), without prior thrombolytic therapy.

Q-wave myocardial infarction
Myocardial infarction characterized by Q-waves that are abnormal either in character or number or both.

rescue PCI or salvage PCI
PCI performed in patients with myocardial ischemia as emergent/urgent therapy following failed thrombolytic therapy.

reperfusion therapy
Treatment aimed at restoring blood flow through acutely occluded (blocked) coronary artery or arteries. The methods include thrombolytic therapy and PCI (primary and rescue). Timely administration of reperfusion therapy is associated with a substantial reduction in AMI patient mortality.

revascularization therapy
See reperfusion therapy

stable angina pectoris
Angina pectoris occurring in attacks of predictable frequency and duration after provocation by circumstances that increase myocardial oxygen demands, such as exercise, emotional stress or excitement, the precipitating circumstances tending to remain constant across episodes.

ST-segment elevation myocardial infarction (STEMI)
A type of myocardial infarction in which the ST portion of the QRST part of the ECG is elevated at least 1 mm above the baseline.

subendocardial myocardial infarction
See nontransmural myocardial infarction
**subsequent myocardial infarction**
Repeat infarction within the acute phase (28 days) of the initial infarction or an extension of the initial infarct occurring within the 28-day period.

**thrombolytic therapy or thrombolysis**
Emergency therapy for patients with STEMI involving the administration of a drug (for example, tPA, rPA or streptokinase) to dissolve the clot in the coronary artery or arteries and restore blood flow to the heart muscle (reperfusion therapy). It is provided pre-hospital (by paramedics) and in-hospital (emergency department, ICU/CCU). The sooner it is initiated, the better the outcome is.

**transmural myocardial infarction**
Myocardial infarction involving the entire thickness of the heart wall starting with the subendocardial layers and progressing through to the epicardial layers.

**troponins**
Biomarkers released from the heart muscle during acute myocardial ischemia reflecting myocardial necrosis. They are highly specific for heart muscle. There is a lag of two to four hours after the onset of symptoms before troponins become detectable in the blood.

**unstable angina**
Angina pectoris occurring unpredictably or suddenly and increasing in severity or frequency; attacks may occur without provocation, such as during sleep or rest, may not respond to nitroglycerin and may be of unusually long duration.
Appendix B: Case Study Answers

This appendix provides the answers to the case studies. Every reasonable effort has been made to ensure accuracy of the answers. Codes have not been selected for conditions that would qualify as optional type (3) diagnoses.

Case Study #1 DAD

I20.0 (M) Unstable angina

**Rationale:** This patient is presenting with ACS. ECG does not show ST-segment elevation and biomarkers are negative, which is an indication to the physician that no myocardial damage has occurred, and the final outcome is unstable angina. R94.3- is not assigned when the final outcome is unstable angina. I24.9 Acute ischaemic heart disease, unspecified is not assigned for this case because this is a non-specific term. For this patient, unstable angina is the specific condition within the spectrum of ACS.

I25.10 (3) Atherosclerotic heart disease of native coronary artery

**Rationale:** CAD is assigned as a type (3) as this was known to be a pre-existing condition and no investigation such as coronary angiogram or treatment such as PCI or CABG was directed towards the CAD. As the documentation has not indicated a past history of CABG, native artery has been selected.

E11.52 (3) Type 2 diabetes mellitus with certain circulatory complications

**Rationale:** It is mandatory to code diabetes, even if just a type (3); .52 is selected based on the use additional code note at both category I20 and I25.

E11.78 (3) Type 2 diabetes mellitus with multiple other complications

**Rationale:** It is mandatory to code E11-.78 whenever more than one complication of diabetes is present. E11.78 qualifies in this case because the patient has two conditions listed under the code separately note at E11.52—unstable angina and CAD.
Case Study #2 DAD

I21.49 (M)  Acute subendocardial myocardial infarction, unspecified site

Rationale: The final outcome for this patient was NSTEMI. In the absence of ST-segment elevation, the positive troponin levels are indication to the physician that the patient has indeed sustained an infarction. A final diagnosis of NSTEMI is classified to subendocardial MI.

R94.31 (3)  Abnormal cardiovascular function studies (biomarkers or ECG) suggestive of non ST segment elevation myocardial infarction [NSTEMI]

Rationale: This code is based on the fact that the physician has documented ST depression and elevated troponin levels.

I25.10 (1)  Atherosclerotic heart disease of native coronary artery (diagnosed on this admission)

Rationale: CAD qualifies as a diagnosis type (1) because it was investigated and diagnosed during this admission. In this case, the MI is the focus of care; therefore, it is the MRDx (most responsible diagnosis).

3.IP.10.VX  Xray, heart with coronary arteries, left heart catheterization with fluoroscopy using (retrograde) percutaneous intra arterial approach

Case Study #3 DAD

I21.1 (M)  Acute transmural myocardial infarction of inferior wall

Rationale: A final diagnosis of STEMI is classified as a transmural MI unless there is evidence on the chart that myocardial damage was minimized to a subendocardial infarction or averted/aborted altogether.

R94.30 (3)  Electrocardiogram suggestive of ST segment elevation myocardial infarction [STEMI]

Rationale: This code is selected based on the physician’s statement that the ECG performed in the peripheral hospital demonstrated ST-segment elevation.

I25.10 (1)  Atherosclerotic heart disease of native coronary artery

Rationale: CAD qualifies as a type (1) in this case because it was investigated and treated during this admission. Since this is an emergent/urgent admission because of the MI, the MI will remain the MRDx.

E11.52 (3)  Type 2 diabetes mellitus with certain circulatory complications

Rationale: It is mandatory to code diabetes, even if just a type (3); .52 is selected based on the use additional code note at both category I20 and I25.
**E11.78 (3)**  *Type 2 diabetes mellitus with multiple other complications*

**Rationale:** It is mandatory to code E1-.78 whenever more than one complication of diabetes is present. E11.78 qualifies in this case because the patient has two conditions listed under the code separately note at E11.52: AMI and CAD.

1. **I.J.50.GQ-OA  Dilation, coronary arteries using percutaneous transluminal approach and balloon dilator with (endovascular) stent (insertion)**
   
   Mandatory status = N  primary
   
   Mandatory extent = 1  One coronary artery

   **Rationale:** This is a primary PCI because it is being performed 1) for STEMI; 2) within 12 hours of presentation to Hospital A; and 3) without prior administration of thrombolytic therapy.

3. **I.P.10.VX  Xray, heart with coronary arteries, left heart catheterization with fluoroscopy using (retrograde) percutaneous intra arterial approach**

**Case Study #4 DAD**

**I21.1 (M)**  *Acute transmural myocardial infarction of inferior wall*

**Rationale:** A final diagnosis of STEMI is classified to transmural MI. Even though this patient was transferred emergently for PCI and CAD (stenosis of coronary artery) was noted during the intervention, MI remains the MRDx.

**R94.30 (3)**  *Electrocardiogram suggestive of ST segment elevation myocardial infarction [STEMI]*

**Rationale:** STEMI was confirmed at the transferring hospital. The fact that the patient received thrombolytic therapy is also an indication that this MI was a STEMI.

**I25.10 (1)**  *Atherosclerotic heart disease of native coronary artery*

**Rationale:** CAD qualifies as a type (1) in this case because it was investigated and treated during this admission.

**Operative Episode #1 May 8, 2006**

1. **I.J.50.GQ-OA  Dilation, coronary arteries using percutaneous transluminal approach and balloon dilator with (endovascular) stent (insertion)**

   Mandatory status = Z  Other
   
   Mandatory extent = 1  One coronary artery

   **Rationale:** This patient received thrombolytic therapy prior to the PCI; therefore, this is not a primary PCI and the correct status attribute is Z Other. Two dilations were performed on the right coronary artery; therefore, the correct extent attribute is 1.
3.IP.10.VX Xray, heart with coronary arteries, left heart catheterization with fluoroscopy using (retrograde) percutaneous intra arterial approach

Operative Episode #2 May 9, 2006
1.IJ.50.GQ-OA Dilation, coronary arteries using percutaneous transluminal approach and balloon dilator with (endovascular) stent (insertion)
Mandatory status = Z Other
Mandatory extent = 1 One coronary artery

3.IP.10.VX Xray, heart with coronary arteries, left heart catheterization with fluoroscopy using (retrograde) percutaneous intra arterial approach

Case Study #5 NACRS
R94.31 (MP) Abnormal cardiovascular function studies (biomarkers or ECG) suggestive of non ST-segment elevation myocardial infarction [NSTEMI]
Rationale: The main problem in NACRS is selected based on the highest degree of knowledge about the condition at the time of discharge from the ED. In ED, a final diagnosis of NSTEMI typically represents a working diagnosis, as the physician will have to await the results of biomarkers before confirming this diagnosis. If results of biomarkers are known to be elevated prior to the decision-to-admit time, then it would be appropriate to capture I21.4- Acute subendocardial myocardial infarction as the main problem and R94.31 as an other problem.

Case Study #6 NACRS
I24.9 (MP) Acute ischaemic heart disease, unspecified
Rationale: The highest degree of knowledge about the condition is ACS; therefore, for this case it is appropriate to select I24.9 as the main problem. R94.3- is not applied with I24.9.
E14.52 (OP) Unspecified diabetes mellitus with certain circulatory complications
Rationale: It is always mandatory to capture diabetes. .52 is selected based on the use additional code note at category I24. Documentation does not provide information regarding the type of diabetes; therefore, it is appropriate to select E14.- Unspecified diabetes mellitus.
Case Study #7 NACRS

R94.30 (MP) Electrocardiogram suggestive of ST-segment elevation myocardial infarction [STEMI]

Rationale: The main problem in NACRS is selected based on the highest degree of knowledge about the condition at the time of discharge from the ED. In the ED, a final diagnosis of STEMI typically represents a working diagnosis, as the final outcome (transmural, subendocardial or averted MI) will likely not be known at the time of discharge from the ED. If the physician has confirmed a transmural or Q-wave MI prior to the decision-to-admit time, then it would be appropriate to select I21.- to I21.3- as the main problem and R94.30 as an other problem.

Case Study #8 NACRS

I21.9 (MP) Acute myocardial infarction, unspecified

Rationale: The physician has documented the final diagnosis as myocardial infarction; therefore, it is appropriate to select I21.- as the main problem. Documentation does not support the selection of transmural versus subendocardial; therefore, .9 unspecified has been selected.

R94.38 (OP) Other and unspecified abnormal results of cardiovascular function studies

Rationale: A code from subcategory R94.3- is mandatory with codes from category I21.-. There is no documentation of the physician’s interpretation of ECG findings or biomarkers; therefore, .38 is selected.

Case Study #9 NACRS

I21.9 (MP) Acute myocardial infarction, unspecified

Rationale: STEMI inferior MI was diagnosed at the transferring hospital and the physician has documented the diagnosis as AMI; therefore, it is correct to capture the main problem with a code from category I21.- for this case. Since we do not know what the final outcome is at this point (transmural, nontransmural or aborted MI), the correct fourth digit is .9 unspecified.

R94.30 (OP) Electrocardiogram suggestive of ST-segment elevation myocardial infarction [STEMI]
Appendix C: Bibliography


Jokovic, A., Health Services Research and Canadian Institute for Health Information, Toronto.
Email communication, August 22, 2006.


This evaluation form will be used to assess the quality of the product, to provide feedback to the developer(s) and to identify the future educational needs of CIHI clients. Please take a few minutes to fill it out.

Product name: ____________________________________________________________
Date: ________________________________

Client Information

My title: ________________________________________________________________

My department/area of responsibility: (please check one)
- Administration
- Ambulatory care services
- Decision support
- Diagnostic services
- Finance
- Other (please specify): ____________________________

My organization/facility: (please check one)
- Acute care facility
- Community health centre
- Consulting firm
- Continuing care facility
- Government
- Other (please specify): ____________________________

Education Content and Organization

Please provide the following information by circling your choice. Refer to the scale below.

SA = Strongly agree  A = Agree  D = Disagree  SD = Strongly disagree

1. Session design and materials:
   a) The material presented was relevant to my needs. SA A D SD
   b) The amount of material was appropriate for the program. SA A D SD
      If Disagree or Strongly disagree, please indicate whether there was:
      - Too much material OR Not enough material
   c) The SLP has adequately prepared me and I feel confident about the material SA A D SD

2. Delivery:
   a) This method of presenting the information (SLP) was appropriate. SA A D SD
      If Disagree or Strongly disagree, please indicate what you think would be a more effective and/or appropriate delivery method:
      - Audio presentation
      - Online learning
      - Group discussion
      - Instructor led training
      - Webconference
      - Video presentation
      - Other: ____________________________
3. **Program promotion:**
   a) The promotional material sent for this SLP provided sufficient information for me to consider the product.  
   b) How did you initially learn about this SLP?  
      - Flyer  
      - Internet/e-mail  
      - Manager/colleague  
      - Other (specify)  

4. **Learning experience:**
   a) The SLP content met my expectations.  
   b) I learned valuable skills and/or information.  
   c) I would recommend this product to others.  
      If *Disagree* or *Strongly disagree* please indicate why:  

5. **Please indicate the rating that best reflects your overall evaluation of this product:**  
   - Excellent  
   - Good  
   - Fair  
   - Poor  

6. **What aspect of the product was most valuable to you?**

7. **What aspect of the product could have been strengthened?**
   - How?  

8. **Please list the topics you would like to see CIHI offer as education programs.**

Would you like to be contacted by CIHI Education staff to discuss any of your comments or concerns?  
   - Yes  
   - Name:  
   - Facility:  
   - Phone no.:  
   - No  

Please provide us with your e-mail address if you would like to receive future information and/or brochures from CIHI via the internet.  
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Thank you for taking the time to fill out this evaluation form.  

Please return this form by fax to 613-789-2114