Initial Electrocardiogram in Patients with Suspected Ischemic Chest Pain*

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QMI = Q wave infarction; NQMI = non-Q-wave infarction

Newer angiographic, anatomic, and metabolic studies support the view that acute coronary syndromes, which include acute myocardial infarction, unstable angina and ischemic sudden death, are different manifestations of ruptured atheroma with superimposed thrombosis and release of vasoactive substances.1-4 The clinical manifestation of acute coronary syndromes is determined by the size of the atheroma, the extent and duration of the occlusion (partial vs occlusive thrombus), collateral circulation, spontaneous reperfusion, and coronary artery tone.5 It is postulated that a complete occlusion of the infarct-related artery without spontaneous reperfusion or adequate collateral circulation causes a Q wave infarction; an incomplete occlusion, early reperfusion and/or good collateral circulation, a non-Q-wave infarction; and an incomplete and intermittent occlusion, an unstable angina. Because the management of various types of acute coronary syndromes is not uniform, the differentiation between them is important. The ECG, with all its limitations, remains the simplest, inexpensive, readily available noninvasive technique and plays an important role in the diagnosis of the early phases of acute coronary syndromes. In this report, we are reviewing the value of the initial 12-lead ECG in patients with suspected acute coronary syndromes.

ACUTE MYOCARDIAL INFARCTION

The diagnosis of AMI is not difficult in a patient with ischemic chest pain lasting for 30 minutes or longer in whom the initial ECG shows ST segment elevation with or without pathologic Q waves. In patients with ischemic chest pain and repolarization abnormalities, intraventricular conduction defects, or left ventricular hypertrophy, the diagnosis of AMI is less certain and has to be confirmed by elevated serum creatine kinase and its MB fraction (CK-MB) or nuclear perfusion studies.

Based on the ECG pattern, AMI is classified into QMI and NQMI.6 Whether the patient will develop a QMI or NQMI depends on the duration of occlusion and collateral circulation.5

Q-Wave Infarction

In QMI, the abnormal Q waves are usually associated with loss of R waves and typical evolution of the ST segment elevation and T waves. The diagnosis of QMI is further supported by reciprocal ST segment changes. According to Rude et al.,7 in patients with new or presumably new ST segment elevation and Q waves, the incidence of AMI is 82 to 100 percent. Infrequently, pathologic Q waves are a manifestation of transient myocardial ischemia or are due to nonischemic causes (pseudoinfarct).9

Non-Q-Wave Infarction

The ECG manifestations of NQMI are ST segment elevation or depression and T wave changes.6 Because these repolarization abnormalities are not specific for AMI,10 this diagnosis has to be confirmed by non-ECG techniques. Some authors include in NQMI patients without typical ST-T wave changes.11

ST segment Elevation: According to Huey et al.12 and others,13 40 to 60 percent of patients with ST segment elevation only develop a NQMI which should be considered in patients who have less prominent ST segment elevation (0.1 to 0.2 mV), a smaller number of involved leads or a history of prior myocardial infarction.12

ST Segment Depression: The second manifestation of NQMI is ischemic ST segment depression, i.e., horizontal or downsloping ST segment depression more than 0.1 mV in limb and left precordial leads and 0.2 mV in V1-V3 with or without symmetrical T wave inversion of 0.3 mV or more.14 The incidence of
AMI in patients with diffuse or even localized ST segment depression varies between 46 and 56 percent, and therefore, NQMI requires confirmation by elevated CK-MB. Furthermore, according to Boden et al, 15 percent of patients with ST segment depression on the initial ECG develop a QMI.

ST depression in lead V1,4 can also be a manifestation of a posterior AMI. As reported by Boden et al, in 40 percent of patients with ischemic chest pain, ST segment depression in lead V1,4 is due to posterior AMI. This localization of AMI should be suspected if the ST segment depression is horizontal and the T waves are positive. Occasionally, however, the T waves can be biphasic or negative. Patients with posterior MI have a higher CK-MB enzymes than those with anterior NQMI.

T Wave Inversion: The third ECG manifestation of NQMI is T wave inversion. As in other NQMI, the ECG changes should persist for 24 to 48 hours and the diagnosis confirmed by non-ECG methods. According to Granborg et al, only 23 percent of patients with ischemic chest pain and isolated T wave inversion develop AMI.

What is the clinical significance of various ECG patterns in AMI? First, there are important differences between NQMI and QMI. In general, patients with NQMI are usually older, have a higher incidence of previous myocardial infarction, and history of congestive heart failure, as well as having more residual ischemia, lower inhospital mortality, smaller infarct size, and similar or even worse long-term prognosis.

Second, the initial ECG plays an important role in coronary thrombolysis. An abnormal ECG is one of the criteria for thrombolytic therapy and helps to identify patients who will or will not benefit from coronary reperfusion. According to Bar et al, patients who benefited most from coronary thrombolysis were those with prominent ST segment elevation. In contrast, in patients with ST segment depression or previous myocardial infarction, coronary thrombolysis did not improve mortality. However, this finding has not been confirmed by some newer studies; ie, coronary thrombolysis improved mortality regardless of the ECG.

It is possible that in the future, the ECG will play a less important role in candidates for coronary thrombolysis.

Third, there is a difference between patients with NQMI who have ST segment elevation or depression. Willich et al and others showed that patients with ST segment elevation have a better prognosis than those with ST segment depression. The former group has less severe coronary artery disease and better left ventricular function despite a similar or smaller enzymatic infarct size.

Fourth, in patients with isolated T wave changes, the short-term outcome is more favorable than in patients with ST segment depression.

Unstable Angina

The ECG manifestations of unstable angina are transient ST segment depression, ST segment elevation and less frequently, T wave changes. What is the role of the 12-lead ECG in patients with unstable angina? First, patients in whom episodes of chest pain is not associated with ECG changes seem to have less severe coronary artery disease than those with ECG changes. Second, according to some studies, patients with ST segment elevation have less extensive coronary artery disease in comparison to patients with ST segment depression. This finding could explain the higher mortality in the latter. Third, patients with ST segment elevation are more prone to coronary spasm and life-threatening arrhythmias than subjects with ST segment depression. Fourth, the incidence of AMI during the initial hospitalization in patients with ST segment elevation and depression is 12 to 25 and 5 to 7 percent respectively. The early survival rate is high in both groups (>95 percent).

The third ECG manifestation of unstable angina is symmetrical T wave inversion or pseudonormalization of negative T waves. The incidence of T wave changes in unstable angina is between 3 and 16 percent. Because the T waves are influenced by many cardiac and extracardiac factors, it is important to correlate T wave changes with symptoms and other evidence of coronary artery disease. Haines et al compared patients who had unstable angina with and without T wave inversion. As expected, patients with anterior T wave changes had a higher incidence of significant coronary artery disease, AMI, and death in comparison to those with a normal ECG. In patients who underwent coronary bypass surgery, the incidence of AMI and mortality was similar in both groups. According to Granborg et al, the long-term prognosis of isolated ischemic T wave changes regardless of whether they represent NQMI or unstable angina is poor. During 31 month follow-up, 17 percent of patients developed AMI and 24 percent died.

The incidence of AMI and mortality in patients with unstable angina is relatively low, and there is no benefit from early coronary bypass surgery. However, there are patients with unstable angina who have a higher morbidity and mortality and can be identified by typical ECG changes.

First, Gorgels et al showed that ST segment depression in more than eight leads and ST segment elevation in aVR should raise the possibility of left main disease or severe proximal three vessel disease. Consistent with this observation is the ECG in Figure 1 which was recorded in a patient during severe rest pain in whom coronary angiography showed 90 percent proximal left anterior descending artery and left...
circumflex coronary artery stenosis (left main equivalent).

In the second group are patients with impending myocardial infarction who have minimal ST segment elevation and deep T wave inversion in leads V₂ and V₃ and less frequently in other chest leads. Cardiac enzymes are either normal or slightly elevated. According to de Zwaan et al., this ECG finding can be a manifestation of severe proximal stenosis of the LAD and presents an increased risk for the development of extensive anterior myocardial infarction. However, because the ECG does not accurately localize the site

of LAD obstruction, these patients should undergo early coronary angiography.

The limited value of the ECG to localize the site of LAD stenosis is shown in the next two figures. The ECG in Figure 2 was recorded from a patient with 90 percent mid-LAD stenosis 24 hours after a prolonged episode of rest pain. A similar ECG, with slightly less prominent repolarization abnormalities, is from a patient with unstable angina during chest pain who had a 90 percent proximal LAD obstruction (Fig 3).

The third group of patients with unstable angina who are at higher risk for developing extensive anterior myocardial infarction are those with slight increase of CK-MB and intermittent ST segment elevation in leads V1-V5 and occasionally in I and aVL.17

**Intraventricular Conduction Defects**

Twenty percent of patients with AMI have intraventricular conduction defects.8 Patients with AMI and intraventricular conduction defect with exception of left anterior fascicular block, have higher mortality, more advanced coronary artery disease and more impaired left ventricular function.9

The following questions are of clinical significance in patients with ischemic chest pain and intraventricular conduction defects. First is the diagnosis of AMI.8 In patients with left anterior fascicular block, the diagnosis of AMI is not difficult because this conduction abnormality does not interfere with the ST-T wave changes of AMI.40 In contrast, old inferior and less frequently, old anterior MI may be more difficult to diagnose in patients with left anterior fascicular block.40 The recognition of AMI in right bundle branch block is also easy with the exception of a true posterior myocardial infarction which is masked by the conduction defect. The ECG is least helpful in left bundle branch block because in the majority of these patients, the conduction defect prevents the development of Q waves in lead I, aVL and V4-6 and causes ST-T wave changes which can imitate repolarization abnormalities of AMI. In addition, whether the Q waves in the inferior leads are diagnostic for inferior myocardial infarction remains controversial.11,42 From the many suggested ECG criteria for myocardial infarction in LBBB, only the following are highly specific:81 (a) evolutionary ST segment changes; (b) Q waves in the left precordial leads and in I and aVL; (c) late notching of the S wave in lead V1; (d) primary T wave changes (T wave concordant with the QRS complex). However, the low sensitivity of these ECG findings (8 to 30 percent)82 limits their practical diagnostic value. The second question is localization of myocardial infarction. Patients with new right bundle branch block usually have an extensive anterior myocardial infarction in comparison to those with LBBB who can have either anterior or inferior one.85 The third question is whether the intraventricular conduction defect is new or old. According to Ross,86 in 20 to 50 percent of patients, this question remains unanswered.

Figure 4 illustrates the difficulties in differentiating between acute and old myocardial infarction. Upper ECG shows an extensive acute anterior myocardial

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**Figure 3. Symmetrical T wave inversion between 1 and 6 mm in 1, aVL, V2, as well as Q waves in lead 2, 3, and aVF with ST segment elevation consistent with inferior myocardial infarction. The ECG changes in the inferior leads were present for the last two years. This ECG was recorded during chest pain in a patient with unstable angina who had a 90 percent proximal LAD obstruction.**
infarction. Center ECG, which was recorded 14 month later, reveals a left anterior fascicular block with pathologic Q waves in lead 1 and aVL, QRS in V₂ and QS with ST segment elevation in V₄₆ which is consistent with old extensive anterior MI and possible ventricular aneurysm. Finally in the lower ECG, 3.5 years later, there is a LBBB with Q waves in lead 1 and aVL, ST segment elevation in leads V₁₋₄, and a notch in the ascending limb of the S wave in lead V₁. While the rS and ST segment elevation in lead V₁₋₃ can be explained by the LBBB, the RSR and ST segment elevation in lead V₁ raises the possibility of an AMI. The ST segment elevation present on ECG A and B argues against an AMI.

The majority of patients with ischemic chest pain and intraventricular conduction defect, with the exception of left anterior fascicular block, have advanced coronary artery disease, more impaired left ventricular function, and therefore, are of higher risk for life-threatening complications and should be monitored in the coronary care unit. Furthermore, the diagnosis of AMI has to be confirmed or excluded by non-ECG techniques.

**Left Ventricular Hypertrophy**

The next ECG abnormality seen in patients with ischemic chest pain is left ventricular hypertrophy. The relationship between coronary artery disease, myocardial infarction, and ECG in left ventricular hypertrophy is the following. First, patients with left ventricular hypertrophy have a higher incidence of coronary artery disease. Second, the ECG has important limitations in the diagnosis of NQMI because of the similarities between STT wave changes due to left ventricular hypertrophy and myocardial infarction. According to Boden et al, in patients with left ventricular hypertrophy, the diagnosis of NQMI requires 2 mm instead of 1 mm ST segment depression and elevated CK-MB. Third, patients with left ventricular hypertrophy, particularly those with obstructive cardiomyopathy or aortic stenosis, can have ischemic chest pain and pathologic Q waves (pseudoinfarct) which have to be differentiated from AMI. Fourth, patients with left ventricular hypertrophy and ischemic chest pain should be admitted into the coronary care unit because of higher likelihood of development of acute coronary syndromes and life-threatening complications.

**Normal ECG or Nonspecific ST-T Wave Changes**

Finally, in patients with ischemic chest pain, the initial ECG can show minor nonspecific ST-T wave changes or be normal. Among the possible causes of a normal initial ECG in AMI are delayed evolution of the ECG changes, lateral infarction due to occlusion of the left circumflex coronary artery, or small myocardial infarctions.

According to Brush et al and others, in patients with suspected AMI, the initial ECG is a good

![Figure 4](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21620/)
predictor of short-term prognosis and the need for acute interventions (drug therapy, temporary pacemaker or intraaortic balloon). As mentioned, patients with ischemic chest pain and normal ECG or nonspecific ST-T wave changes have a low incidence of AMI, serious complications, and mortality, and therefore, can be admitted into the step down unit. An important limitation of these studies was their retrospective character. Furthermore, the predictive value of the initial ECG was not confirmed by Young et al who showed that the incidence of life-threatening complications, need for interventions and mortality was similar and low regardless of the initial ECG. Similar results were also reported by Weingarten et al.

Another question is whether patients with suspected myocardial infarction and nonspecific repolarization abnormalities have the same prognosis as those with a completely normal ECG. Fesmire and co-workers suggested that patients with nonspecific ST-T wave changes or an abnormal but unchanged ECG have a slightly higher risk than those with completely normal ECG. According to Slater et al, there was no death in 107 patients with AMI and normal ECG and one death in 73 patients who had nonspecific ST-T wave changes.

Conclusions

The review of recent studies dealing with the diagnostic and prognostic significance of the ECG in acute coronary syndromes indicate that the ECG remains an important initial test in patients with AMI or other coronary syndromes. It is of interest, however, that while the ECG is of limited value in the differentiation between transmural and nontransmural myocardial infarction, the different ECG manifestations of acute coronary syndromes are helpful in the assessment of prognosis, severity of coronary artery disease, and degree of myocardial impairment. As suggested by Spodick, the ECG is “trying to tell us something” about the nature of AMI. In patients with QMI, the ECG confirms the diagnosis, while in patients with NQMI, the diagnosis of myocardial infarction is less certain and has to be confirmed by non-ECG methods. In addition, the degree of coronary artery disease is usually less severe in patients with ST segment elevation than in those with ST segment depression. The same probably also applies for unstable angina. Furthermore, the combination of ischemic chest pain and intraventricular conduction defect or left ventricular hypertrophy is highly suggestive of coronary artery disease, left ventricular impairment and higher risk for developing life-threatening complications, and therefore, these patients should be admitted into the coronary care unit. A normal ECG in 10 to 20 percent of patients with documented myocardial infarction is an important limitation of the ECG. However, patients with AMI and normal ECG have good short-term prognosis and whether they can be managed in the intermediate care unit is not completely clear as is the indication for coronary reperfusion.

References

ST-segment depression. Am J Cardiol 1987; 59:782-87
18 Klein LW, Helfant RH. The Q-wave and non-Q wave myocardial infarction: differences and similarities. Prog Cardiovasc Dis 1986; 29:205-20
22 ISIS-2 Collaborative Group. Randomized trial of intravenous streptokinase, oral aspirin, both, or neither among 17,187 cases of suspected acute myocardial infarction. Lancet 1987; 2:549-60
27 Unstable angina pectoris: National cooperative study group to compare surgical and medical therapy: III. Results in patients with S-T segment elevation during pain. Am J Cardiol 1980; 45:819-24
33 Cooperative unstable angina study group. Unstable angina pectoris national cooperative study group to compare surgical and medical therapy: II. In-hospital experience and initial follow-up results in patients with one, two and three vessel disease. Am J Cardiol 1978; 42:839-48
34 Corgels AP, Vos MA, Bar FW, Wellens HJ. An electrocardiographic pattern, characteristic for extensive myocardial ischemia. Circulation 1986; (suppl II):422
37 Boden WE, Bough EW, Benham I, Shulman RS. Unstable angina with episodic ST segment elevation and minimal creatinine kinase release culminating in extensive, recurrent infarction. J Am Coll Cardiol 1983; 2:11-20
44 Stark ME, Vacek JL. The initial electrocardiogram during admission for myocardial infarction: use as a predictor of clinical course and facility utilization. Arch Intern Med 1987; 147:843-46