Cardiology

Localisation of Culprit Vessel

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ACUTE MYOCARDIAL INFARCTION

Emergency Decisions

1. Ascertain the time from onset of pain.
2. Evaluate 12-lead ECG for: Type of myocardial infarction (anterior or inferior) ST segment elevation score Q waves. Bundle branch block and hemiblock to identify patients at high risk of dying early; such patients should be managed aggressively.
3. Identify candidates for thrombolytic therapy (ST segment elevation score; how much and in how many leads?).
4. In inferior wall myocardial infarction, record lead V_4R.

Identification of Candidates for Thrombolytic Therapy

In the patient admitted because of acute myocardial infarction, recognition of the area at risk is of utmost importance; its size and location determine aggressiveness of therapy and help in selecting patients most likely to profit from thrombolytic therapy. It is important to rule out patients who will not benefit from thrombolytic therapy, because such patients should not be subjected to the risk of bleeding and the need for blood transfusions associated with thrombolysis. In this section we offer a systematic approach by which those patients with acute myocardial infarction who are most likely to benefit from thrombolytic therapy before or at the time of admission can be quickly identified. Thrombolytic therapy is most beneficial in patients with acute myocardial infarction admitted soon after the onset of chest pain with signs of a large infarction (development of Q waves) or extensive ischemic areas (high ST segment elevation or marked ST depression in several ECG leads).

Time from Onset of Pain

Significant infarction size limitation occurs in patients arriving at the hospital within 2 hours after the onset of chest pain. If thrombolysis is initiated within 1 hour after the onset of chest pain, infarction size is reduced by 50 percent and if initiated between 1 and 2 hours, infarction size is reduced by 30 percent. If patients are admitted between 2 and 4 hours after the onset of pain, infarction size is reduced by only 13 percent; in fact, in a subgroup of these patients there was no limitation of infarction size in spite of the fact that successful reperfusion was achieved. After a delay of more than 4 hours from onset of pain, the value of thrombolytic therapy is less clear. It still seems useful in anterior infarction with a high ST score and Q waves. Recent studies suggest that even at a later stage patients may still benefit from thrombolytic therapy because of a lower incidence of left ventricular dilatation and ventricular arrhythmias. It should be noted that acute pericarditis can be the cause of the subjective symptoms of acute myocardial infarction as well as an ECG pattern with a high ST segment score.

ST Segment Scoring

In both anterior and inferior acute myocardial infarction the greatest reduction of infarction size by thrombolytic therapy is achieved in patients with the largest infarctions. The amount of ST segment elevation and depression and the number of leads in which these changes are present are proportionate to infarction size; the higher the ST segment elevation, the deeper the ST depression, and the more leads showing these changes, the larger the infarction. Patients most likely to profit from thrombolytic therapy can therefore be recognized by using an ST segment scoring system.

Anterior Wall Myocardial Infarction. For ST segment scoring in anterior wall infarction, add the total amount (in millimeters) of ST elevation in the precordial leads (V1-V6). A total of 12 mm or more is a high ST segment score and indicates extensive anterior wall infarction; a low ST segment score in the precordial leads is less than 12 mm.

Inferior Wall Myocardial Infarction. For ST segment scoring in inferior wall infarction, add the total amount (in millimeters) of ST segment elevation in the inferior leads (II, III, and aVF). A total of 7 mm or more is a high ST segment score and indicates extensive inferior wall infarction; a low ST segment score in the inferior leads is less than 7 mm.

Significance of Q Waves

Although in the past Q waves have been thought to indicate myocardial necrosis, it is now known that extensive ischaemia can result in transient Q waves due to conduction delay in the zone under that electrode. It has been shown that significant myocardial salvage by thrombolysis can be accomplished in patients with new pathological Q waves; even after 2 hours the infarction size can be limited by therapy, indicating that large anterior wall infarctions are still evolving at that time. Thus, patients should not be excluded from thrombolytic therapy simply because Q waves are present. When there is little ST segment elevation in anterior wall myocardial infarction (less than 12 mm

Table 1 : Candidates most likely to profit from thrombolytic therapy

Delay	High ST Segment Elevation Score*	Low ST Segment Elevation Score†
<2 hr	All patients with MI++	Anterior MI with Q waves
2-4 hr	All patients with MI++	
	with Anterior MI with Q waves and without Q waves anterior MI	
	In inferior MI without Q waves	

*>12mm, leads V1-V6, ≥7mm, leads II, III aVF
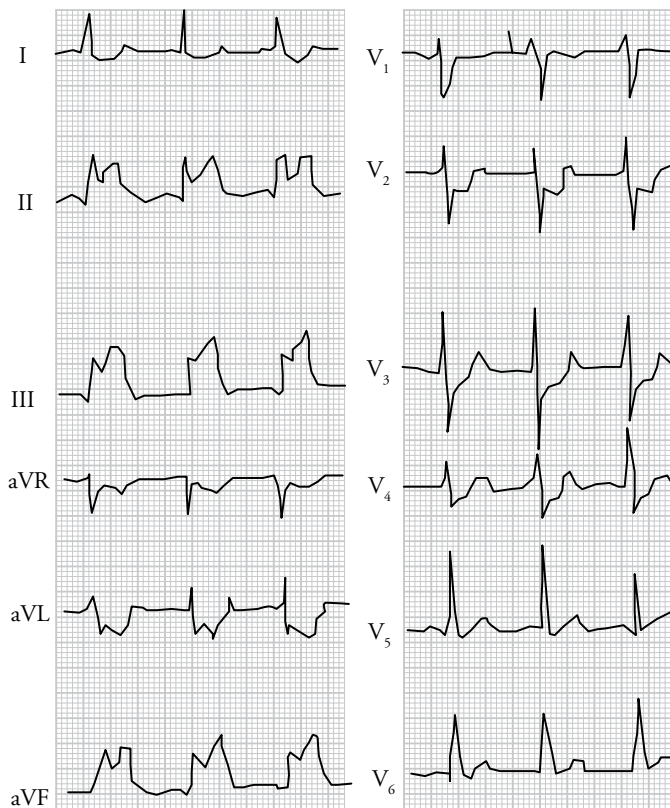†<12 mm, leads V1-V6, ≥7mm, leads II, III aVF
++MI=myocardial infarction

Fig. 1: Acute interior wall myocardial infarction with a high ST score less than 2 hours after the onset of pain. The ST score in leads II, III and aVF is 13 mm; small Q waves are present. This patient is a candidate for thrombolyis. The additional recording of lead V4R should have been of help to identify the coronary artery involved, presence of right ventricular infarction and risk of developing AV nodal block

total in precordial leads), and the absence of Q waves indicates that thrombolysis is not required, spontaneous reperfusion may have already taken place.

Information Needed

1. Time from onset of pain (0-2 hr; 2-4 hr; > 4 hr)
2. Infarction location (anterior or inferoposterior)
3. ST segment elevation score
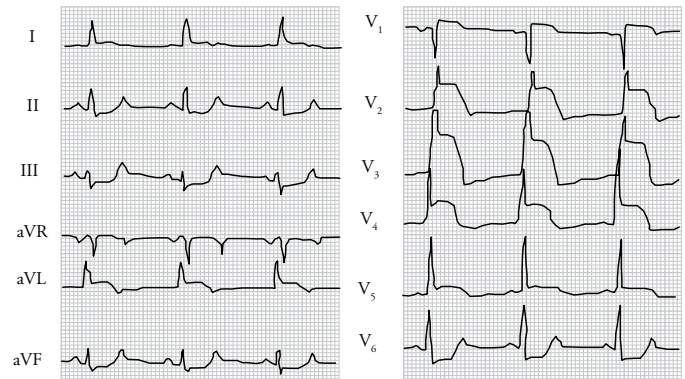4. Presence or absence of Q waves

Fig. 2 : Acute anteroseptal myocardial infarction less than 2 hours after the onset of pain. The ST segment score is high and thrombolytic therapy is indicated.

5. Lead V_4R in inferior wall infarction

Table 1 simplifies the identification of candidates most likely to profit from thrombolytic therapy. If the ST segment score is high and the delay less than 2 hours, all patients with myocardial infarction are candidates; if the delay is 2 to 4 hours, the best candidates for thrombolysis are:

1. All patients who have myocardial infarction and do not have Q waves.
2. All patients with anterior wall infarction with Q waves. If the ST score is low, only patients with anterior wall infarction and Q waves will have reduction of infarction size from thrombolysis up to 4 hours after the onset of pain.

Fig. 1 is a 12-lead ECG from a patient with acute inferior wall infarction and was recorded less than 2 hours after the onset of pain. It illustrates a case where use of thrombolytics will result in marked reduction in infarction size. After 2 hours the patients with inferior wall myocardial infarction most likely to profit from thrombolysis are those with high ST segment score and no Q waves.

Fig. 2 is the 12-lead ECG from a patient with acute anteroseptal myocardial infarction recorded less than 2 hours after the onset of pain. The high ST segment score (more than 25 mm) is a clear indication for thrombolytic therapy. Had this patient been seen more than 2 hours from the onset of pain, thrombolysis would still have been indicated.

Fig. 3 is an example of how Q waves, which are present during acute ischaemia, may disappear after successful reperfusion; the figure underscores the fact that Q waves present in the acute stage of myocardial infarction do not necessarily indicate necrotic and unsalvageable myocardial muscle.

Value of Lead V_4R in Acute Inferior Myocardial Infarction

Lead V_4R identifies:

1. The coronary artery occluded
2. Presence or absence of right ventricular infarction
3. Those at risk for atrioventricular (AV) block
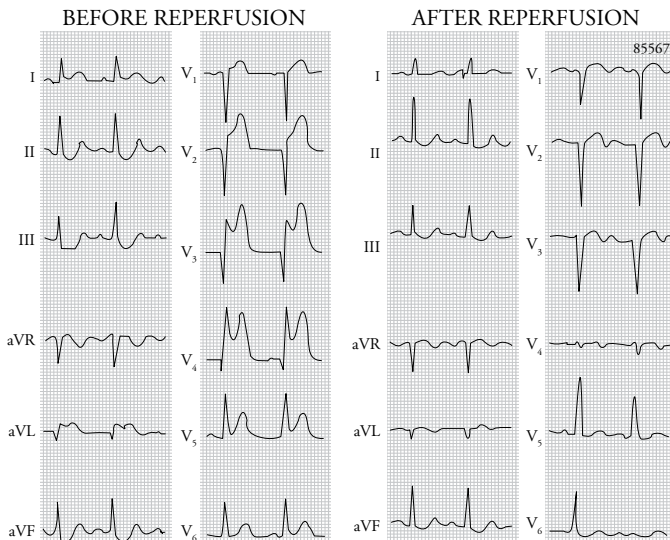4. Those who will profit most from thrombolytic therapy

Fig. 3 : Acute anteroseptal infarction in a patient presenting to the hospital 2 hours after the onset of chest pain. Note the presence of Q waves in leads V2 to V4 (left panel). Following successful thrombolytic therapy these Q waves disappear (right panel), indicating that the tissue was still salvageable.

Table 2 : Features of AV Conduction Disturbances Complicating Acute Myocardial Infarction

Feature	Inferior MI	Anterior MI
Site of Block branches	AV node	Bundle
Artery involved	RCA	LAD
Escape rhythm	Narrow QRS Rate 40-60/minute	Wide QRS Rate< 40/minute
	Dependable	Undependable
Duration of Block	Transient	Transient
Increase in hospital mortality (compared to same infarction location without block)	2.5 times	4 times

Abbreviations: RCA=right coronary artery; LAD=left anterior descending coronary artery; MI=myocardial infarction.

Occlusion Sites

With lead V_4R it is possible to identify the occlusion sites in the setting of acute inferior wall myocardial infarction.

Fig. 4 illustrates that in lead V_4R the ST segment is elevated in proximal right coronary artery occlusion; it is not elevated but coves into a positive T wave in distal right coronary artery occlusion; and for a circumflex artery occlusion the T wave is inverted.

High-Risk Patients

The value of thrombolytic therapy in acute myocardial infarction relates to the area at risk and the delay before instituting such therapy. When thrombolytic therapy is being considered for patients with acute inferior wall myocardial infarction, ECG identification of right ventricular infarction identifies the patient who might require more aggressive therapy when intravenous thrombolytic therapy is not successful. More aggressive therapy
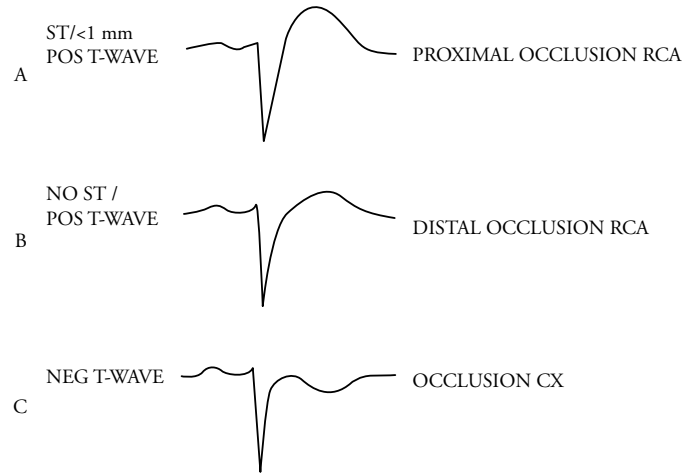

Fig. 4 : Lead V4R in inferior wall myocardial infarction. (A) An elevated ST segment indicates proximal right coronary artery occlusion and right ventricular infarction, and it identifies patients at risk for AD nodal block. (B) An ST segment that coves into a positive T wave, without ST segment elevation, identifies distal right coronary artery occlusion (RCA). (C) An ST segment that slopes into a negative T wave points to circumflex artery (CX) occlusion.

includes percutaneous transluminal coronary angioplasty (PTCA). Approximately 45 percent of patients with acute inferoposterior myocardial infarction have right ventricular involvement, and only 5 to 10 percent of these have the hemodynamic picture of right ventricular infarction (low cardiac output and elevated right-sided pressures).

Right Ventricular Infarction

ST segment elevation of 1 mm or more in lead V_4R has a high sensitivity and specificity for detecting right ventricular infarction, pinpointing the site of occlusion in the proximal right coronary artery, and identifying patients at high risk (45%) of developing AV block. In all patients with inferior wall myocardial infarction the incidence of AV block is 15 percent.

Fig. 5 illustrates the ECG in acute inferoposterior and right ventricular infarction. The ST segment elevation in the inferior leads (II, III, and aVF) indicates inferior wall involvement; the ST segment depression in leads V_1 to V_5 indicates acute posterior wall infarction; and the elevated ST segment in V_4R indicates right ventricular infarction and proximal right coronary artery occlusion.

An Early Sign. The ST segment elevation in lead V4R usually disappears within 10 hours after the onset of pain, and thus it is important to record this lead on admission.

Fig. 6 demonstrates the loss of the telltale ST segment elevation in lead V_4R within the span of 6 hours. A recording of lead V_4R at 11 a.m. shows ST segment elevation consistent with right ventricular infarction and proximal right coronary occlusion. This sign has completely disappeared 6 hours later.

Fig. 7 shows acute inferoposterior wall infarction in a patient with a downsloping ST segment in lead V4R, indicating a circumflex artery occlusion.
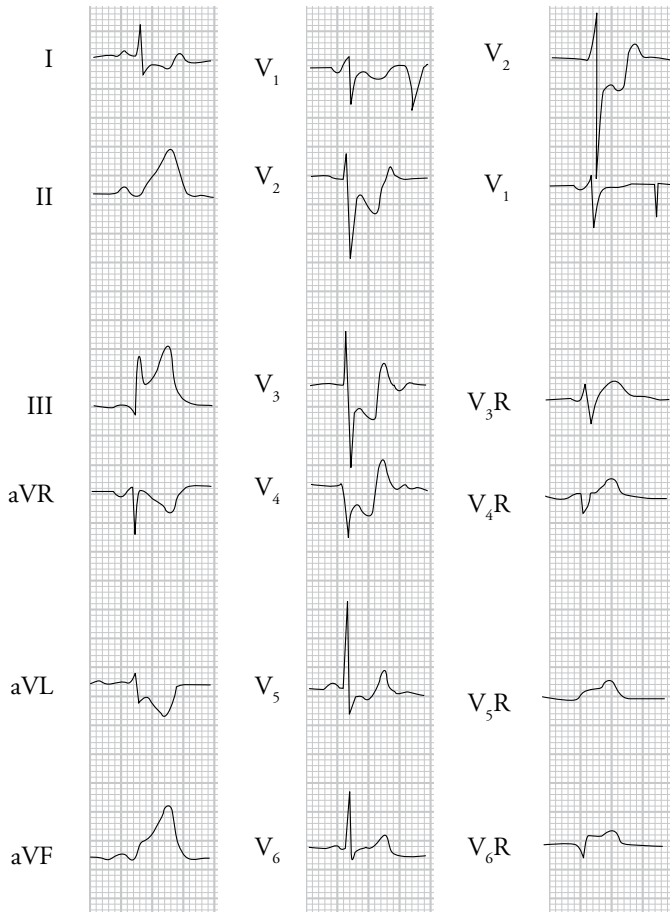
Fig. 5 : Acute inferoposterior wall myocardial infarction and right ventricular infarction. Note the elevated ST segment in lead V4R, indicating an occlusion in the proximal right coronary artery and right ventricular involvement.
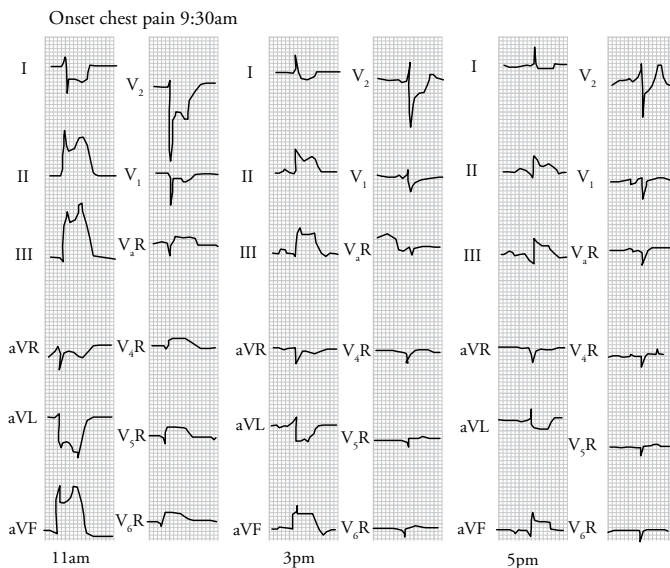

Fig. 6 : Serial tracings from a patient with acute inferoposterior and right ventricular infarction. Note that the diagnostic changes for right ventricular infarction seen in lead V4R have disappeared 7.5 hours after the onset of pain.
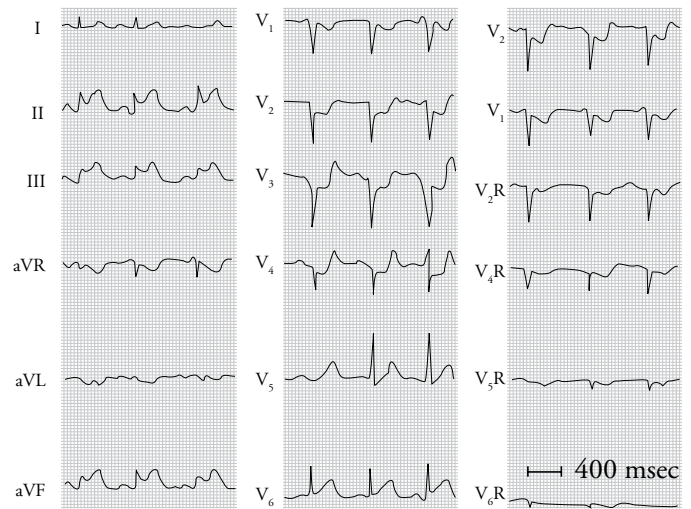

Fig. 7 : Acute inferoposterior wall myocardial infarction due to occlusion in the circumflex artery. Note in lead V4R the downsloping of the ST segment into a negative T wave.

ECG SIGNS OF REPERFUSION

Apart from a decrease in ST segment elevation, the accelerated idioventricular rhythm has been shown to be a sign of reperfusion (spontaneous or as a result of thrombolytic therapy) during acute myocardial infarction. Approximately one-half of the patients with reperfusion have an accelerated idioventricular rhythm when the ECG is recorded continuously following thrombolytic therapy. This finding is of practical clinical importance in that it may help to identify both spontaneous and thrombolytic-induced reperfusion in the absence of coronary angiography. As shown by Gorgels, an accelerated idioventricular rhythm after myocardial infarction indicates not only reperfusion, but also myocardial necrosis.

Characteristics of the Reperfusion Arrhythmia

1. Three or more successive ventricular ectopic beats.
2. Rate: 50 to 120 beats per minute.
3. Onset: After a long coupling interval.

An accelerated idioventricular rhythm, as shown in Fig. 8A, begins with a long coupling interval. The first few beats are often fusion beats because the ectopic rhythm emerges when its rate is about the same as that of the sinus rhythm. This type of ventricular rhythm should be distinguished from the one beginning with a short coupling interval that is sometimes called "slow ventricular tachycardia". Fig. 8B which is frequently irregular and occurs during the first 24 hours of infarction, but after the reperfusion phase.

Identification of the Area of Reperfusion

The QRS configuration of the accelerated idioventricular rhythm may be of help in noninvasive identification of the area supplied by the previously occluded vessel using the following clues:

1. Multiple QRS configurations during the accelerated idioventricular rhythm frequently accompany reperfusion of the left anterior descending (LAD) coronary artery; the QRS may be relatively narrow.
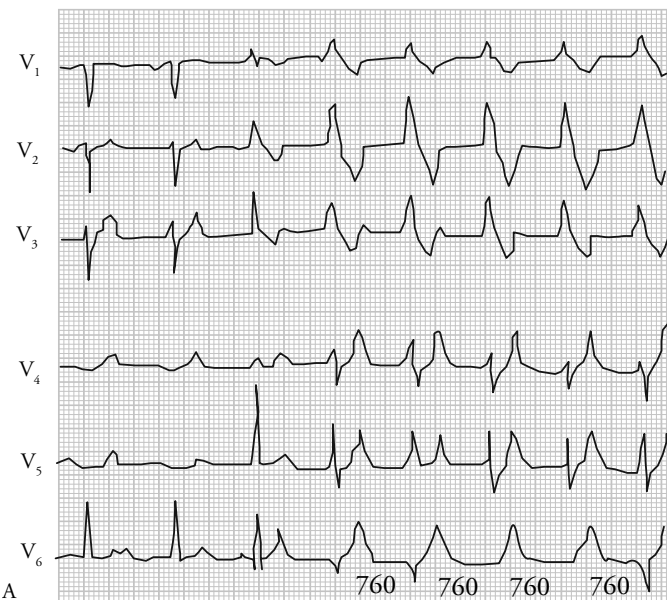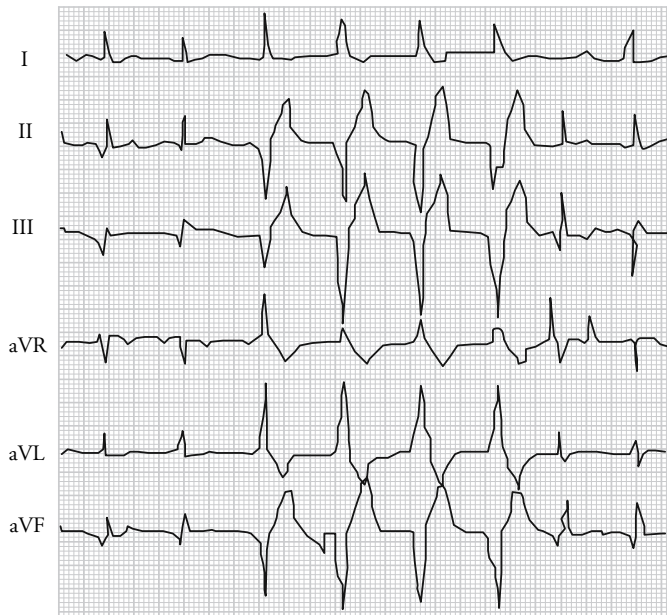2. A V_1-negative configuration excludes a circumflex lesion.

Fig.8a : Accelerated idioventricular rhythm - a reperfusion arrhythmia in a patient with acute inferior wall infarction. Note the long coupling interval.
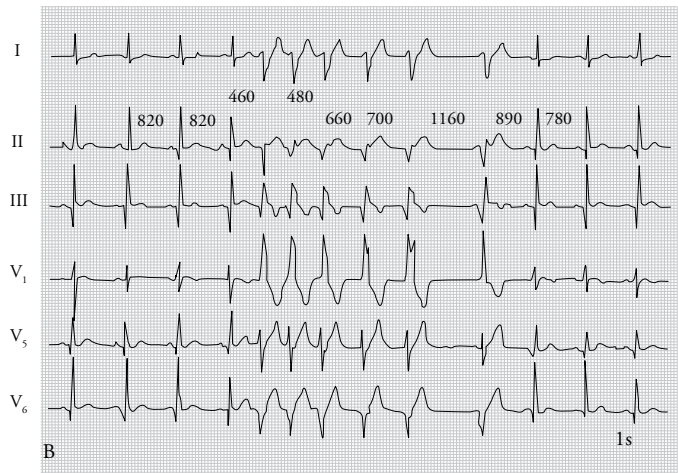

Fig.8b : Continued (B) Example of a slow ventricular tachycardia. Note the shorter coupling interval and the irregular rhythm.
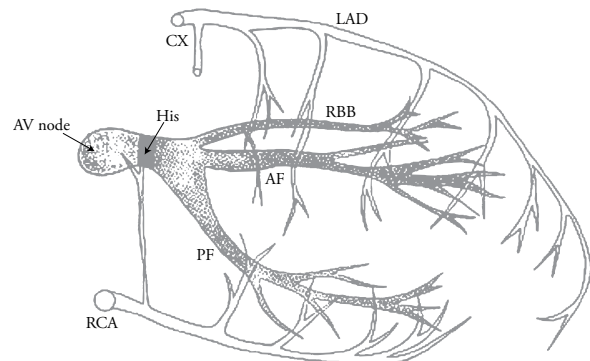

Fig. 9 : The trifascicular intraventricular conduction system. Note that the right buyndle branch (RBB) and the superior division of the left bundle branch (AF) are both anterior structures and therefore are vulnerable in anteroseptal myocardial infarction. The posterior division of the left bundle branch (PF) is broad and supplied by both the left anterior descending (LAD) and the right coronary artery (RCA).

3. An electrical axis between 0 and 180 degrees virtually rules out the right coronary artery as the infarct artery.

CONDUCTION BLOCKS IN ACUTE MYOCARDIAL INFARCTION

The emergence of conduction disturbances between atrium and ventricle in the acute phase of myocardial infarction is of great prognostic and therapeutic significance and should therefore be recognized immediately. The site of block in the AV conduction system (AV node or bundle branches) is related to which coronary artery is occluded during myocardial infarction.

Anatomy of the AV Conduction System

As shown in Fig. 9, the AV conduction system consists of the AV node, the bundle of His, and the specialized intraventricular conduction system. The latter consists mainly of three fascicles in the right bundle branch and the left bundle branch with its two main divisions (anterior and posterior fascicles). The posterior fascicle is usually broad and short, and the right bundle branch and the left anterior fascicle are long and thin.

Blood Supply to the Conduction System

As illustrated in Fig. 9, in 90 percent of people the right coronary artery, by way of its posterior descending branch, perfuses the posterior one-third of the interventricular conduction system. The AV nodal branch is the main blood supply to the AV node and proximal part of the bundle of His. In some people the AV nodal artery also supplies the distal portion of the His bundle and the proximal bundle branches. The LAD coronary artery and its branches supply the anterior wall of the heart and the anterior two-thirds of the septum. In most hearts, the first septal perforator of the LAD is the main blood supply to the distal part of the bundle of His and the proximal bundle branches.

AV Nodal Block

Clinical implications

The right coronary artery usually supplies the AV node, and obstruction of that artery causes inferior myocardial infarction,
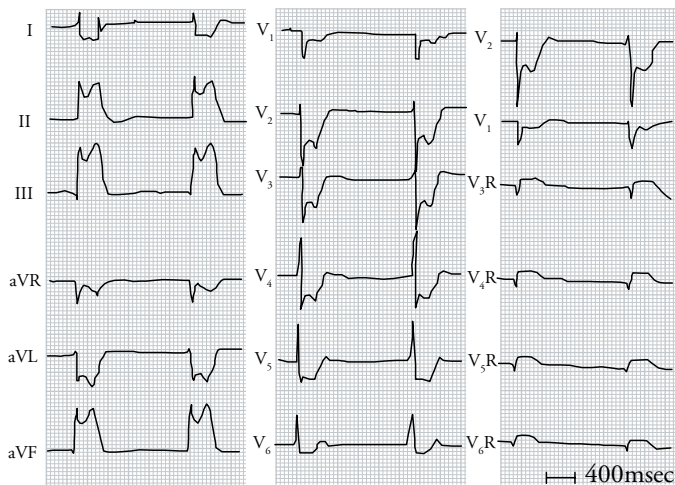
Fig. 10 : Example of complete AV nodal block in a patient with an acute inferoposterior myocardial infarction and right ventricular involvement.

frequently leading to AV nodal conduction disturbances. In acute inferior wall infarction, ST segment elevation in lead V_4R identifies patients having proximal obstruction in the right coronary artery and an almost 50 percent chance of developing high-degree (second-degree of more) AV nodal block (Fig. 10).

Prognosis

When high-degree AV nodal block occurs in acute inferior myocardial infarction (which is the case in 15 percent of the patients) the in-hospital mortality rate is two and one-halftimes that of inferior wall infarction without high-degree AV block. This increased mortality is probably the result of the proximal location of the obstruction in the right coronary artery leading to a large inferior wall infarction with right ventricular involvement. These AV nodal conduction disturbances are transient, usually disappearing after a few days; in exceptional cases, they may last for a few weeks. The escape pacemaker during complete AV nodal block is located just below the AV node, and it usually produces an acceptable and dependable rhythm at a rate of 40 to 60 beats per minute.

Treatment

Atropine or temporary transvenous pacing is indicated in case of Adams-Strokes attacks, a low ventricular rate accompanied by congestive failure, and/or bradycardia-dependent ventricular arrhythmias.

Bundle Branch Block and Hemiblock

Clinical Implications

The development of bundle branch block and hemi block during the acute phase of myocardial infarction indicates extensive anterior wall infarction, because such conduction problems indicate an occlusion proximally in the LAD coronary artery. Before the introduction of thrombolytic therapy this complication occurred in 10 percent of patients with an acute anterior wall myocardial infarction. The inhospital mortality was around 55 percent, which is four times higher than that in patients not developing bundle branch block. Although exact figures are not yet available, the clinical impression is that following the introduction of thrombolytic therapy, the incidence of bundle branch block after acute anterior wall myocardial infarction decreased.

Prognosis

When anterior wall myocardial infarction is complicated by bundle branch block and hemiblock, early death occurs because of pump failure and ventricular tachycardia or fibrillation. Death from pump failure occurs within a few days. If the patient survives the critical early days, there is a 30 percent chance that sustained ventricular tachycardia or fibrillation will develop 1 to 2 weeks later. Lie and associates have shown that in this clinical setting (anterior wall myocardial infarction complicated by bundle branch block and hemiblock) there is a high chance of developing complete AV block, which, if the patient survives, is usually transient and requires only a temporary pacemaker.

Treatment

The finding of bundle branch block as a complication of anterior wall infarction calls for aggressive treatment. Intravenous thrombolytic therapy is given; when it is not successful, the patient is taken to the cardiac catheterization laboratory to receive intracoronary thrombolytic therapy. If this treatment fails to open the LAD coronary artery, the thrombus is perforated and emergency PTCA performed. During the same catheterization, a Swan-Ganz catheter and a temporary pacing lead are inserted. The Swan-Ganz catheter will provide information about pump function. If the patient survives the first few days, continuous rhythm monitoring is still required because of the chance of developing sustained ventricular tachycardia or fibrillation 1 to 2 weeks after the onset of anterior wall myocardial infarction. Table 2 summarizes some of the features of AV conduction disturbances complicating myocardial infarction.

Right Bundle Branch Block

In acute myocardial infarction the appearance of right bundle branch block is usually associated with distal conduction system block secondary to anterior rather than inferior wall infarction. The risk of progression to complete heart block is twice that of left bundle branch block especially when associated with fascicular block.

ECG Recognition

In right bundle branch block, the QRS pattern in lead V_1 is broad with a terminal R wave (classically the pattern is a triphasic rSR'); in leads 1, a VL, and V_6 there is a terminal S wave (qRS). In right bundle branch block secondary to acute anterior wall myocardial infarction, the ventricular complex in lead V_1 looks different than it would had there not been such a complication. That is, because of septal involvement, the initial R wave is missing and the triphasic pattern (rSR') changes to a biphasic pattern (QR). The little q wave in lead V_6 that reflects normal septal activation is also absent.

Mechanism

In right bundle branch block the two ventricles are activated one after the other instead of simultaneously. Septal and left ventricular activation proceed normally; the sole abnormality is late activation of the right ventricle, which explains the late R in lead V_1 and the S wave in leads 1, aVL, and V_6. However, in
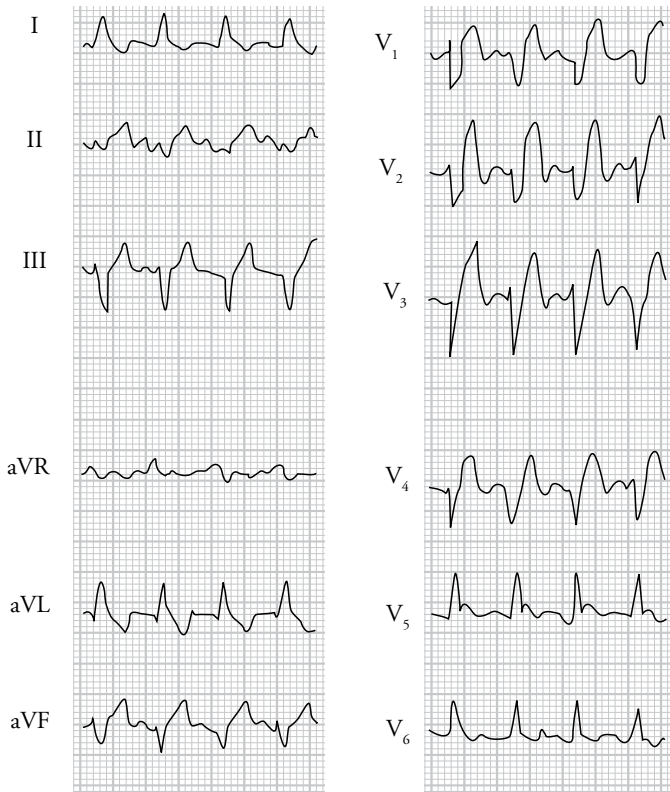
Fig. 11 : Left bundle branch block and anteroseptal myocardial infarction. Note that small Q waves are present in leads I, aVL and V6.

acute anteroseptal infarction the septal forces are lost and lead V_1 shows a QR pattern.

Left Bundle Branch Block

In acute myocardial infarction, left bundle branch block (LBBB) may occur secondary to inferior and anterior wall myocardial infarction because the main stem of the left bundle branch may receive blood supply from either the AV nodal or the LAD coronary arteries. When ischaemic heart disease is excluded, left bundle branch block is usually associated with hypertensive, primary, or degenerative myocardial disease.

ECG Recognition

ln left bundle branch block, the QRS complex in lead V_1 is broad and negative (QS or rS). In leads I, aVL, and V_6 the complex is totally positive (no q and no s). The intrinsicoid deflection over the left ventricle (V6) is delayed. If the left bundle branch block is associated with anteroseptal infarction; a Q wave may be present in leads I, aVL, V_5 and V_6 (Fig. 11).

Mechanism

The sequence of ventricular activation during left bundle branch block is totally different from that during normal conduction. The normal narrow QRS reflects synchronous activation of the ventricles by simultaneous conduction over the right and left bundle branches. When the left bundle is blocked, the impulse reaches the right ventricle first by way of the right bundle branch. Then the septum is activated from right to left, followed by the remainder of the left ventricle. Fig. 15 compares ventricular activation in left bundle branch block

with and without myocardial infarction. Without infarction the septum and right ventricle are activated together; in anteroseptal infarction the right ventricle is activated without opposing septal forces, causing a tall narrow R wave in lead V_1 and a Q wave in lead V_6. In lead V_1 during left bundle branch block without infarction, an initial small, narrow R wave may be present, probably due to activation of the anterior papillary branch of the right bundle. Because the two ventricles are activated one after the other, first the right ventricle and then the left, the main current goes leftward, away from lead V_1. This accounts for the broad negative deflection in that lead and the broad positive one in lead V_6. If left bundle branch block develops secondary to a large anteroseptal infarction, loss of septal tissue and late left ventricular activation cause unopposed right ventricular activation, causing the QRS to have an initial narrow R wave in lead V_1 and a Q wave in lead V_6.

Left Anterior Hemiblock

Left anterior hemiblock may occur secondary to anterior wall myocardial infarction.

ECG Recognition

In left anterior hemiblock there is left axis deviation of -30 degrees and a small q wave in leads I and aVL. The QRS duration is not prolonged beyond normal.

Mechanism

The anterior fascicle of the conduction system is relatively thin and therefore vulnerable to injury. A block in this division of the left bundle branch results in delayed activation (40 msec.) of the anterolateral area of the left ventricle, causing marked left axis deviation because the impulse activates the left ventricle through the posterior fascicle, spreading upward and to the left.

Left Posterior Hemiblock

Left posterior hemiblock rarely occurs in the setting of acute myocardial infarction. If it develops associated with right bundle branch block, it indicates a poor prognosis because, as shown in Fig. 9, this requires occlusion in both the LAD and the right coronary arteries.

ECG Recognition

With left posterior hemiblock there is right axis deviation of greater than +120 degrees and a small r wave in leads I and aVL.

Mechanism

A block in the posterior fascicle causes marked right axis deviation because the impulse activates the left ventricle through only the anterior fascicle, spreading downward and to the right and causing lead I to be mainly negative and leads II, III, and aVF to be mainly positive. The initial forces (first 0.02 sec) travel from the anterior papillary muscle, upward and leftward. This is why there is a small initial r wave in leads I and aVL and a q wave in leads II, III, and a VF.

SUMMARY

The treatment of acute myocardial infarction has changed dramatically with the introduction of thrombolytic therapy and the availability of other methods of reperfusion, such as PTCA and emergency coronary artery bypass surgery. Essential in the

management of acute myocardial infarction is stratification according to infarction location and size and time interval between onset of pain to admission. The 12-lead ECG is of great value in (1) making decisions as to the necessity of thrombolytic therapy (using ST-T segment score and presence or absence of Q waves); (2) recognizing reperfusion (by the appearance of accelerated idioventricular rhythm); (3) diagnosing the site of occlusion in the coronary artery responsible for inferoposterior infarction; (4) identifying right ventricular involvement; (5) recognizing patients with inferoposterior infarction at high risk for developing AV nodal block; and (6) risk stratification because of the emergence of AV conduction disturbances. The bundle branches receive their blood supply from the same artery that supplies the anterior wall of the heart. Thus, bundle branch block and hemiblock are serious complications of acute anterior wall myocardial infarction. Prognosis in these patients is mainly determined by their extensive anterior wall infarction, which may lead to pump failure and early death due to ventricular tachycardia and fibrillation. There is a high incidence of complete AV block in patients with anterior wall infarction who develop right bundle branch block and hemiblock. Complete block, however, is usually transient, and if the patient survives the acute phase and the ventricular tachycardia and ventricular fibrillation that often develop 1 to 2 weeks later, implantation of a permanent pacemaker is rarely needed.

Value of the electrocardiogram in localizing the occlusion site in the left anterior descending coronary artery in acute anterior myocardial infarction

OBJECTIVES

The study assessed the value of the electrocardiogram (ECG) as predictor of the left anterior descending coronary artery (LAD) occlusion site in relation to the first septal perforator (S1) and/ or the first diagonal branch (D1) in patients with acute anterior myocardial infarction (AMI).

BACKGROUND

In anterior AMI, determination of the exact site of LAD occlusion is important because the more proximal the occlusion the less favorable the prognosis.

METHODS

One hundred patients with a first anterior AMI were included. The ECG showing the most pronounced ST- segment deviation before initiation of reperfusion therapy was evaluated and correlated with the exact LAD occlusion site as determined by coronary angiography.

RESULTS

ST-elevation in lead aVR (ST+ aVR), complete right bundle branch block, ST- depression in lead V5 (ST- V5) and STV1 $\geq$ 2.5 mm strongly predicted LAD occlusion proximal to S1, whereas abnormal Q-waves in V4-6 were associated with occlusion distal to S1 (p = 0.000, p = 0.004, p = 0.009, p =0.011 and p = 0.031 to 0.005, respectively). Abnormal Q wave in lead aVL was associated with occlusion proximal to D1, whereas ST- aVL was suggestive of occlusion distal to D1 (p = 0.002 and p = 0.022, respectively). For both the S1 and D1, inferior ST- $\geq$ 1.0 mm strongly predicted proximal LAD occlusion, whereas absence of inferior ST- predicted distal occlusion (p $\leq$ 0.002 and p $\leq$ 0.020, respectively).

CONCLUSIONS

In anterior AMI, the ECG is useful to predict the LAD occlusion site in relation to its major side branches.

Abbreviations

AMI=acute myocardial infarction;

cRBBB=complete right bundle branch block;

D1=first diagonal branch;

ECG=electrocardiogram;

LAD=left anterior descending coronary artery;

Qx=abnormal Q-wave in lead x;

ST+=ST-segment elevation;

ST-=ST-segment depression;

ST+x=ST-segment elevation in lead x;

ST-x=ST-segment depression in lead x;

S1=first septal perforator.

In acute anterior myocardial infarction (AMI), the site of occlusion in the left anterior descending (LAD) coronary artery is related to the extent of the myocardial necrosis and prognosis. Electrocardiographically, anterior myocardial infarction is classified as anteroseptal, anterolateral and apical. With a few exceptions, such as the study of Ideker et al., most studies show poor correlation between the electrocardiogram (ECG) and the exact extent of myocardial involvement as determined by autopsy. In the present descriptive study, we assessed the value of the ECG to predict the occlusion site of the LAD in relation to the first septal perforator (S1) and/or the first diagonal branch (D1).

METHODS

Patient group: One hundred consecutive patients admitted to the coronary care unit of the University Hospital Maastricht with the diagnosis of anterior AMI, defined as chest pain lasting more than 30 min. accompanied by ST- segment elevation (ST+) $\geq$ 2.0 mm in V2 and V3 (ST+ V2-3), were studied In 98 patients AMI was confirmed enzymatically. The remaining two patients were treated for AMI but did not show an enzyme rise because of early reperfusion (one after thrombolysis and the other after primary percutaneous transluminal coronary angioplasty). Patients with complete left bundle branch block, left ventricular hypertrophy (Sokolow index), ECG signs of an old M1 or previous cardiac surgery were excluded.

This study was approved by the institutional ethics committee, and informed consent was obtained in all patients.

Coronary angiography

All patients underwent coronary angiography: 89 patients during the acute phase (70 of 89 after thrombolytic therapy) and eleven patients 3 to 14 days after the acute episode (10 of 11 after thrombolytic therapy). In the latter 11 patients, immediate angiography was planned but logistically not possible. The severity of the stenosis was graded by using the CAAS II (Coronary Angiogram Analyzing System II, Pie Medical, Maastricht, The

Netherlands). The culprit lesion in the LAD was defined as the most severe and/or that lesion with local dissection or thrombus and was related to the take-off of S1 and D1. Flow over the culprit lesion was graded using Thrombolysis in Myocardial Infarction Trial (TIMI) criteria and collateral circulation was classified according to Rentrop et al. The presence of a wrap-around LAD (partially supplying the left ventricular inferior wall) was also assessed.

Electrocardiography

The MAC VU electrocardiograph (Marquette Medical Systems, Milwaukee, Wisconsin) with a frequency range of 0.01 to 150 Hz was used. In the acute phase every 10 min a 12-lead ECG was recorded and also whenever the clinical condition changed. The one showing the most pronounced ST- segment deviation before start of reperfusion therapy was evaluated. The TP-segment was used as the iso-electric line; the PR-segment was used when the T-wave and the P-wave merged The J-point was determined for each lead independently. Using electronic vernier calipers (Mitutoyo, Kawasaki-Shi Japan), both ST+ and ST- segment depression (ST-) were measured at the J-point in all leads (1 mm = 0.1 mV) The accuracy of these calipers is ± 0.02 mm, and in practice we measured with an accuracy of 0.1 mm. Although this system is semi-automatic, the accuracy of our measurements was clearly higher than usual in clinical practice. Besides ST-segment deviation, the incidence of complete right bundle branch block (cRBBB) and abnormal Q waves on the acute ECG was assessed.

Statistics

The data were analyzed using SPSS 7.0 for Windows (SPSS Inc, Chicago, Illinois). Data were expressed as median plus minimum and maximum for continuous variables and as rates (%) for categorical variables. For comparison of continuous variables, the Mann-Whitney U test was used. For comparison of categorical variables, the chi-square test or the Fisher exact test was used. A probability value < 0.05 was considered statistically significant.

RESULTS

The S1 may have its take-off from the LAD proximal to the D1 and in other patients distal to the D1. The culprit lesion was proximal to both S1 and D1 in 31 patients, proximal to S1 and distal to D1 in 11 patients, distal to S1 but proximal to D1 in 10 patients and distal to both S1 and D1 in 48 patients. For correlation with the ECG patients were divided into those with LAD occlusion proximal (31 + 11 = 42) or distal to S1 (10 + 48 = 58), respectively. The analysis was thereafter repeated in the same group of patients, but this time the patients were allocated to subgroups based on the site of occlusion in relation to D1 patients with LAD occlusion proximal (31 + 10 = 41) or distal to D1 (48 + 11 = 59), respectively.

Demography and clinical data

The baseline characteristics of the patients are listed in Table 3. Subgroups were comparable with regard to gender, mean age and the time delay between onset of chest pain and recording of the ECG When the culprit lesion was proximal to either S1 or D1, the peak serum creatine kinase was significantly higher.

Coronary angiography

Between proximal and distal lesions there were no differences regarding the presence of single-, two- and three-vessel disease or a wrap-around LAD (Table 3). One patient had left main stenosis. No significant differences regarding TlMl or Rentrop flow grade were found. Angiographic signs of thrombus were present in 47 patients.

Electrocardiography

ECG predictors of LAD occlusion proximal to S1 ST+aVR was present in 43% of the occlusions proximal to S1 and in only 5% of the occlusions distal to S1 (p = 0.000). The amount of ST+ was small, the median being 0.4 (0.2 to 1.8) mm (Table 4).

ECG predictors of LAD occlusion distal to S1 Absence of inferior ST-, particularly in leads II and aVF, was strongly related to LAD disease distal to S1. Except for one, all patients without inferior ST- had a wrap-around LAD (Table 5).

Q-waves in V4 (width ≥ 20 ms), V5 (width ≥ 30 ms) and V6 (width ≥ 30 ms) were rather specific for LAD occlusion distal to S1 and were found in 55%, 24% and 17% of the occlusions distal to S1 and in only 31%, 7% and 0% of the proximal occlusions (p = 0.015, p = 0.031 and p = 0.005). ECG predictors of LAD occlusion distal to D1 Absence of inferior ST-, particularly in lead III, was also strongly related to LAD disease distal to D1 (See Table 5). Also, ST- aVL was rather specific for occlusion distal to D1 The amount of ST- was 0.3 (0.2 to 1.1) mm.

DISCUSSION

This article describes several new findings regarding the electrocardiographic prediction of the LAD occluslon site in anterior AMI (Table 6). Lead aVR was found to be very useful to identify LAD occlusion proximal to S1. Besides ST+aVR, both cRBBB, ST- V5 and ST+V1, ≥ 2.5 mm were strongly predictive of LAD occlusion proximal to S1, whereas abnormal Q-waves in V4 through V6 were indicative of occlusion distal to S1. Considering the LAD occlusion site in relation to D1, an abnormal Q-wave in lead aVL was suggestive of proximal occlusion, while ST- in the same lead was associated with distal occlusion. For both S1 and D1, marked ST- in the inferior leads predicted proximal occlusion, whereas absence of inferior ST- predicted distal occlusion.

ST-elevation in lead aVR

ST+aVR in unstable angina in three-vessel or left main stem disease has previously been reported. However, we did not find literature on ST+aVR as an indicator to localize ischemia of the left ventricular anterior wall. In the only two articles on ST+aVR in the setting of ischemia in the perfusion area of the LAD, ST+aVR was not used to define the exact LAD occlusion site in relation to the major side branches. Kataoka et al found that in anterior AMI, ST+ in the right precordial leads was associated with LAD occlusion proximal to S1, but the ST- segment in lead aVR was not mentioned. When present, the amount of ST+aVR is usually small (<1 mm), particularly when measured at the J-point and not 40 to 80 ms thereafter. Therefore, in this selected population with anterior AMI, any ST+aVR is associated with LAD occlusion proximal to S1 and is probably the result of transmural ischemia of the basal part of the septum (injury current directed toward the right shoulder). This theory is supported by

Table 3 : Demographic, Clincial and Angiographic Data legend

	Proximal to S1	Distal	p-Value	Proximal to D1	Distal to D1	p-value
No. of pts.	42	58		41	59	
Gender (M:F)	29 :13	45 : 13	NS	30 : 11	44 : 15	NS
Age (yrs.)	55	59	NS	54	59	NS
	22-77	(35-80)		(22-77)	(36-80)	
Time to ECG	2.0	2.3	NS	2.1	2.3	NS
	(0.1-9.5)	(0.2-19.4)		(0.2-19.4)	(0.1-14.0)	
Peak CK	3948	2238	0.014	3333	2239	
	(12,132)	(78-12,132)	(680-12,132)	(78-10,364)	0.045	
(U/Liter)						
Percentage stenosis	99(50-100)	100 (45-100)	NS	100 (50-100)	99 (45-100)	NS
Left main	2	0	NS	2	0	NS
RCA	10	9	NS	8	10	NS
RCX	14	22	NS	17	21	NS
RCA & RCX	12	14	NS	10	15	NS
Wrap-around	52	42	NS	43	49	NS

Data are presented as median plus minimum and maximum for continuous variables and as rates (%) for categorical variables.

CK = creatine kinase;

D1 = first diagonal branch;

ECG = electrocardiogram;

LAD = left anterior descending coronary artery;

No. of Pts. = number of patients;

RCA = right coronary artery;

RCX = circumflex coronary artery;

S1 = first septal perforator.

our finding that none of the 10 patients with LAD occlusion proximal to D1 but distal to S1 showed ST+aVR. ST+aVR was absent in 57% of the occlusions proximal to S1. The reason for the rat- her low sensitivity of this ECG criterion may be due to the rare dominance of the basal septum, being needed for this criterion to become positive, because of the counter balance of the septal ischemia current by ischemia in other large areas of the left ventricle perfused by the LAD such as the lateral and apical inferior wall.

ST-deviation in leads II, III and/or aVF

The amount of inferior ST- was significantly higher in proximal LAD disease, and particularly ST- ≥ 1.0 mm was highly predictive of a proximal LAD lesion. This finding is consistent with previous studies on inferior ST- in anterior AMl ST-deviation in leads V1-V6. In 57% and 23% of the patients showing an ECG pattern traditionally termed "anteroseptal infarction" (ST+V1-V3), the LAD occlusion was located distal to the first and second septal perforator, respectively. This is in agreement with the study by Shalev et al. Analyzing the ST-deviations in leads V1 through V4, ST+V1 > 2.5 mm was the only ECG parameter that could be defined to discriminate between proximal and distal LAD occlusion in the individual patient. Furthermore, ST- V5 appeared to be very specific for LAD occlusion proximal to S1. The occurrence of ST- V5 was interpreted as reciprocal changes associated with transmural ischemia high in the anteroseptal

area. Although ST- V6 was seen more often in proximal LAD disease, this difference was not significant.

Complete right bundle branch block

The development of cRBBB in anterior AMI is an independent marker of poor prognosis. This is primarily considered to be due to extensive myocardial damage rather than the conduction disorder itself. Only if cRBBB comes together with left anterior hemiblock is there an increased risk for progression into complete AV-block. In our study, cRBBB was registered exclusively in LAD occlusions proximal to S1. As S1 is the main blood supply to the distal part of the bundle of His and the proximal bundle branches, the cRBBB is most likely acquired in the setting of anterior AMI.

Abnormal Q waves

In leads V4, V5 and V6, Q waves were rather specific for LAD disease distal to S1. In distal LAD disease, normal activation of the ventricles starting in the interventricular septum and spreading transversely from left to right through the septum usually remains intact. In case of myocardial necrosis beneath the anterolateral leads, the septal vector in (V5)V6 will facilitate Q wave formation in those electrodes In contrast, in proximal to S1 occlusion the septal vector will decrease or even disappear and thereby hinder Q wave formation. The Q waves occurred already early after onsetof AMI and may indicate local conduction delay rather than necrosis.

Table 4 : Electrocardiographic Predictors of Left Anterior Descending Coronary Artery (LAD Occlusion Proximal to the First Septal Perforator (S1) and/or the First Diagonal Branch (D1) legend.

Predictors of LAD Occlusion Proximal to S1

	Sens	Spec	PPV	NPV	LR	p-Value
ST+aVr	43	95	86	70	8.6	0.000
ST-II≥1.0 mm	36	100	100	68		0.000
ST-III≥1.0 mm	60	71	60	71	2.1	0.002
ST-III≥2.5 mm	33	97	88	67	11.0	0.000
ST-aVF≥1.0 mm	52	84	71	71	3.3	0.000
ST-aVF≥2.0 mm	26	97	85	64	8.7	0.002
cRBBB	14	100	100	62		0.004
ST-V5	17	98	88	62	8.5	0.009
ST+V1≥2.5 mm	12	100	100	61		0.011

Predictors of LAD Occlusion Proximal to D1

	Sens	Spec	PPV	NPV	LR	p-Value
ST-II≥1.0 mm	34	39	93	68	17.0	0.000
ST-III≥1.0 mm	66	75	64	76	2.6	0.000
ST-III≥2.5 mm	32	95	81	67	6.4	0.001
ST-aVF≥1.0 mm	54	85	71	72	3.6	0.000
ST-aVF≥2.0 mm	27	97	85	66	9.0	0.001
QaVL	44	85	67	69	2.9	0.002

cRBBB = complete right bundle branch block;
LR = likelihood-ratio;
NPV = negative predictive value;
PPV = positive predictive value;
sens = sensitivity; spec = specificity;
ST- = ST- depression;
ST+ = ST elevation;
Q = abnormal Q wave.

Clinical significance

The ECG signs described in this study may prove useful in defining the site of LAD occlusion in relation to S1 and D1 in the setting of anterior AMI In particular, ST+aVR and ST-deviation in the inferior leads were found to be very helpful. We consider our findings useful in identifying patients with proximal LAD occlusion who need a more aggressive approach to revascularization to prevent extensive myocardial damage resulting in pump failure, the possible development of sub-AV-nodal conduction disturbances and the occurrence of life-threatening ventricular arrhythmias in the second and third week after AMI.

Table 5: Electrocardiographic Predictors of LAD Occlusion Distal to S1 and/or D1 legend

Predictors of LAD Occlusion Distal to S1

	Sens	Spec	PPV	NPV	LR	p-value
Absence of SDT-II	67	74	78	62	2.6	0.000
Absence of ST-III	34	86	77	49	2.4	0.020
Absence of ST- aVF	45	90	87	54	4.5	0.000
QV6	17	100	10	47		0.005
QV5	24	93	82	47	3.4	0.031
QV4	55	69	71	53	1.8	0.015

Predictors of LAD Occlusion Distal to D1

	Sens	Spec	PPV	NPV	LR	p-value
Absence of ST-II	66	73	78	60	2.4	0.000
Absence of ST-III	41	95	92	53	8.2	0.000
Absence of ST-aVF	44	90	87	53	4.4	0.000
ST- aVL	22	95	87	46	4.4	0.022

Abbreviations as in Table 4.

Table 6 : Electrocardiographic Predictors of LAD Occlusion Site legend

STI+V1 > 2.5 mm	proximal to S1
CRBBBB	proximal to S1
ST+aVR	proximal to S1
ST+ V5	proximal to S1
QaVL	proximal to D1
Inferior ST-≥1.0 mm	proximal to S1/D1
QV4-6	distal to S1
ST- aVL	distal to D1
Absence of inferior ST-	distal to S1/D1

Abbreviations as in Table 4

Study limitations

The number of patients studied is limited. Therefore, we are now evaluating our findings prospectively in a larger study population. Several ECGs were recorded in the acute stage and the one showing the most pronounced abnormalities was selected for our study. Therefore, our findings may not be reproducible in a setting where fewer ECGs are recorded. Most of the study population had onset of chest pain within 4 hours before the recording of the ECG, and coronary angiography allowing identification of the culprit lesion was performed in the majority of our cases shortly after admission. Thus, we cannot be certain whether our findings are applicable when patients come in later.