

Correlation of Type of ST Segment Elevation in Acute Anterior Wall Myocardial Infarction on Electrocardiogram with Left Ventricular Ejection Function

Ahmad Hasan*, Muhammad Muzamil, Umer Aftab, Aqib Javed, Imran Ullah, Zain Mehmood, Muhammad Usama

FCPS Cardiology, Allama Iqbal Medical College, Jinnah Hospital, Lahore, Pakistan

*Corresponding author: Ahmad Hasan, FCPS Cardiology, Allama Iqbal Medical College, Jinnah Hospital, Lahore, Pakistan, Tel: 923214937381; E-mail: ahnmalik@hotmail.com

Received date: November 03, 2018; Accepted date: November 23, 2018; Published date: November 29, 2018

Copyright: © 2018 Hasan A, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Background: The implication of the shape of ST segment in acute phase of myocardial infarction (MI) remains unclear.

Objectives: Myocardial ischemia is clinically expressed by chest pain and various ST segment patterns on ECG. It was categorized into three grades. Type 1 shows only hyper acute T waves, type 2 shows hyper acute T waves +ST segment elevation and in type 3 tombstone appearance having distortion of terminal portion of ST. We had assumed that there was maximum reduction of Left ventricular (LV) function in Type 3. Our objective was to see this correlation between type of acute ST segment in anterior wall myocardial infarction on ECG and LV function on echocardiography.

Study design: Non probability purpose sampling. Duration: Jan 2017 to Mar 2017 in the CCU of Jinnah Hospital Lahore.

Results: We examined 50 patients with anterior wall myocardial infarction (MI) presenting within 12 hours symptoms, thrombolized by streptokinase. We determined the correlation between types of ST elevations of acute anterior wall MI assessed on ECG with the LV function assessed on echocardiography. 50% were found between the 46 to 60 years, while 34% were below 45 years and 16% were above 65 years. Mean age was 52.66+ SD10.87. Male and female distribution was 88% and 22% respectively with 20% having DM and 45% having hypertension. There were 20 (40%) patients who were falling in the category 1 with the mean EF 48.25+ 8.926, while 11 (22%) patients in type 2 and mean EF was 35.45+6.502 while 19 (38%) patients fell in type 3 group having maximum decrease in EF with mean 31.05+7.375.

Conclusion: LV function was preserved in type 1, intermediate damage in type 2 and maximum damage in type 3. This simple classification is useful for the prediction of left ventricular function at discharge.

Keywords: ST segment elevation anterior wall MI; Echocardiography; Electrocardiography; Cardiovascular

Introduction

ST-segment elevation myocardial infarction (STEMI) is the most severe form of acute coronary syndrome (ACS) after sudden cardiac death. In USA at present, approximately 25% to 40% of all MI presentations [1] are of STEMI. Acute myocardial infarction (MI) resulting from an occlusive thrombus is recognized on an electrocardiogram by ST-segment elevation [2]. The Universal Definition of Myocardial Infarction is: a new ST elevation at the J point in at least two contiguous leads of ≥ 2 mm (0.2 mV) in men or ≥ 1.5 mm (0.15 mV) in women in leads V2-V3 and/or of ≥ 1 mm (0.1 mV) in other contiguous chest leads or the limb leads [3]. Anterior wall contributes maximum to the ejection fraction and similarly when it is damaged it may cause maximum damage to the heart.

Ischemia produced by obstruction in coronary artery, if is prolonged can produce infarction. An increase in demand is

manifested by changes not only in the ST segments but a decrease in perfusion can produce a wide range of changes in the ST segments, T waves, and QRS complexes [4,5].

Sciarovsky and Birnbaum have developed a method for classifying the gradation of changes (Figure 1) observed in decreased supply [6]. According to them, in grade/type 1 there are changes only in T wave and the ST segment is of concave type, in type 2 there are changes not only in T wave but also in ST segment which becomes straight type and the last type 3 which includes the changes in T wave, ST segment and QRS complex that makes the shape as convex type or tombstone appearance.

In earlier studies, epicardial and precordial mapping showed that the amount of ST segment elevation reflects the extent of myocardial injury [7-10]. So, ST segment elevation was used to define subsets of ischemic patients benefitting most from early reperfusion therapy [11-13]. The amount of ST elevation is influenced not only by the extent and severity of the ischemia, but also by variation in the shape and size of the chest and by localization of infarction [14]. Birnbaum et

al. [4,15] observed that on admission ECG, distortion of the terminal portion of the QRS complex was associated with larger infarct size and increased mortality.

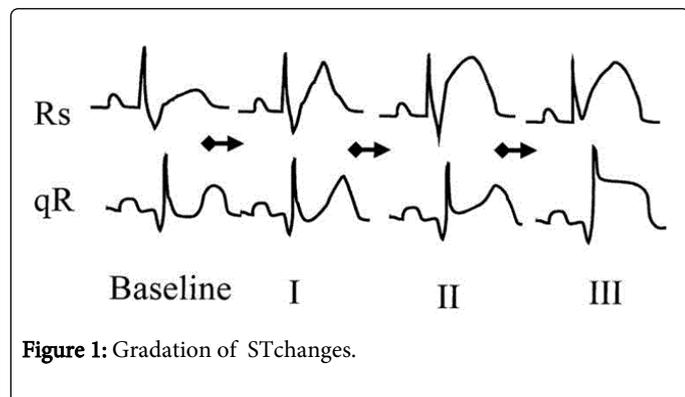


Figure 1: Gradation of ST changes.

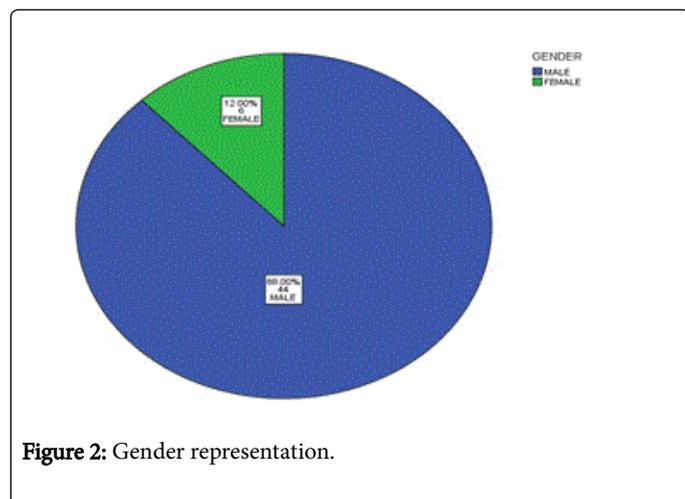


Figure 2: Gender representation.

In a study by Kosuge et al. [16], the correlation between infarct size and shape of ST elevation in total 77 patients who had anterior wall myocardial infarction and reperfusion was done within 6 hours of onset of symptoms. The calculated ejection fraction on left ventriculography after 14 days was 58% for concave type, 48% for straight type and 41% for convex type ($P < 0.05$; concave type versus the other 2 types).

The current study is carried to find out the same relation of type of ST segment in acute infarction with the changes in LV function after thrombolysis.

Material Methods

Inclusion criteria

Both males and females, of age above 15 yrs having typical ischemic chest pain fulfilling the ECG criteria for acute anterior wall MI (as new ST elevation at the J point in at least 2 contiguous leads of ≥ 2 mm (0.2 mV) in men or ≥ 1.5 mm (0.15 mV) in women in leads V2–V3 and/or of ≥ 1 mm (0.1 mV) in other contiguous chest leads or the limb leads 3 as per operational definition).

Exclusion criteria

Following were excluded from the study.

Patient with previous documented infarction, ascites by history, physical examination and laboratory investigations, prior cardiac surgery, history of stroke (ischemic or hemorrhagic), any malignancy bleeding diathesis, pregnancy, active peptic ulcer, valvular heart disease, AV malformation diagnosed on computed tomography (CT) scan, renal failure, Left ventricular aneurysm on the basis of history, previous echo reports and electrocardiogram.

Data collection procedure

Total 50 patients, with non-probability purposive sampling technique, presenting in the cardiology emergency department and fulfilling the clinical and ECG criteria were included in the study. All of these patients were thrombolysed with streptokinase (SK) and shifted to cardiology ward. To rule out other co-morbid conditions, a thorough clinical examination (to rule out the secondary causes of pseudo ST elevations or previous history of infarction) and all other necessary investigations (Chest X-ray, CT Brain and abdomen where required, laboratory investigations including renal profile, liver profile, bleeding profile etc.) were also done. Standard settings and protocol (paper speed of 25 mm/sec and voltage of 10mm/mv) was opted to do the ECG by a single technician. Three groups depending on the morphology of ST segment were assigned. Group 1-Hyperacute T waves, Group 2-ST elevation >0.1 mV without distortion of the terminal portion of the QRS, Group 3-ST elevation with distortion of the terminal portion of the QRS (emergence of the J point $>50\%$ of the R wave in leads with qR configuration, or disappearance of the S wave in leads with an Rs configuration). They were anonymized, read by 2 experienced observers, blinded to all other study characteristics and outcomes. Similarly when patients were shifted to cardiology ward, their ECHO was done on 4th to 5th post admission day, by a single expert consultant and on same echocardiography machine i.e., Gee Machine model Vivid 7, to minimize inter observer variability. Ejection fraction was calculated by using M- mode on parasternal long axis, parasternal short axis and then Simpson method (by measuring LVIDD, LVIDS Septal and posterior wall thickness etc.). The average of the three values was taken the final and recorded.

Results

Variables like age was presented by calculating their mean and standard deviation and frequency. Qualitative variables like gender and true positive cases were presented as frequency and percentage. Anova test was applied that showed significant P value ($P = .000$). In this study, a total of 50 patients were recruited that were fulfilling the inclusion/exclusion criteria to determine the correlation between type of ST elevation in acute anterior wall myocardial infarction and ejection fraction on echocardiography.

Age	Frequency	Percentage
30-45 years	17	34.0
46-60 years	25	50.0
61-75 years	8	16.0
Total	50	100.0

Table 1: Age distribution.

This research work shows majority of the patients between 45-65 years of age 50% (n=25), while 34% (n=17) patients were found between 30 to 45 years and 16% (n=08) were found between 61 to 75 year (Table 1).

Mean and standard deviation was found on 52.66+10.873.

When age distribution of patients was calculated it was found that 88% were males and only 22% were females who presented with anterior wall myocardial infarction (Figure 2).

Regarding risk factor assessment, 20% of the participants were diabetic, 45% were hypertensive while 10% were having positive family

history (1st degree relative having coronary artery disease with female having <50 years of age and male <40 years of age).

When analysis of correlation between type of ST segment elevation and EF was made, it showed that 20 (40%) patients presented with concave type 1 ST segment elevation and their mean EF was 48.25% +8.926, a minimum decrease in EF, while mean EF of the 22% of patients who presented with straight type (type 2) ST elevation (n=11) was 35.45%+ 6.502 and large decrease in the EF was noticed in type 3 (38%) which showed mean EF of 31.05%+7.375(n=19) showing a P value of .000 (Table 2).

	N	Frequency	Mean EF	P Value
Concave Type 1	20	40%	48.25%	.000
Straight Type 2	11	22%	35.45%	
Convex Type 3	19	38%	31.05%	
Total	50	100%		

Table 2: Correlation between ST Segment type and EF.

Discussion

Clinical outcome after AMI largely depends on the final size of infarct [17-19], rendering a need for early and timely need for reperfusion therapy. An ability to estimate the ischemic area at risk and the severity of ischemia immediately on admission may enable physicians to identify patients in whom reperfusion could not be expected to salvage myocardium and conversely, those who might benefit from reperfusion even if much time has elapsed from onset of symptoms. Imaging methods like ECHO, thallium spect. scan can quantify the extent of ischemia and infarct size but on the same time delaying the treatment. So various ECG patterns/criterias have been proposed in the early recognition and sorting out of high and low risk patients for prioritizing the reperfusion therapy.

Some investigators [20-22] have found a correlation between the number of leads with ST segment elevation and infarct size or mortality. Some have studied the magnitude of ST elevation with mortality [23,24] Our study is done in correlation with a study done by Birnbaum et al. [25] showing a relation of initial ST segment pattern with in-hospital mortality in a first anterior wall myocardial infarction. Studies of the ability to estimate the area at risk or final infarct size by the admission ECG have conflicted results [26-28].

In a study by Kosuge et al. in 2001 [29] they compared the type of ST-T changes in lead aVR with the LV function on echocardiography in 105 patients who had anterior wall MI and had gone successful reperfusion within 6 hrs. In group A they included those patients who had ST segment elevation > or = 0.5 mm, in group B they included those patients who did not have ST deviations and in group C they included those patients who had ST segment depression > or = 0.5 mm in aVR. They concluded that EF was maximally decreased in those patients who had type C changes on ECG and ST-T changes in lead aVR is a good predictor of infarct size and LV function before discharge. In another study by Kosuge et al. [16] in 1999 also showed maximum loss of EF in type C. Birnbaum, et al. [30] found that patients with terminal QRS distortion on admission ECG in STEMI

have worse prognosis, larger infarct size and less benefit from thrombolysis.

Another report by Birnbaum, et al. [4,5,31,32] also predicted the same results of final infarct size by the admission ECG based on the type of ST Elevation. In the TIMI-4 trial, patients having Type C ST Elevation on admission had a larger infarct as assessed by creatine kinase (CK) release over 24 hours, and a larger cardiac defect size shown by predischarge 99mTc sestamibi [4]. It was also found that final QRS Selvester score was less by thrombolytic therapy only among patients with Type B (Type 2 in our study) ST, but not Type C (Type 3) ST segment, having first anterior myocardial infarction [5]. Overall, final QRS Selvester score was higher for patients with grade C than grade B ST segment, both in those who either received or not receive thrombolytic therapy. In the GUSTO I angiographic substudy, patients with grade B Pattern ECG had higher left ventricular ejection fraction at 90 minutes than patients with grade C ECG pattern [31]. So, it's the amount and severity of ischemia in grade C which is causing this difference, rather due to the amount of the area at risk or duration of ischemia [31]. They also showed that patients with grade C (type 3) pattern on enrollment have larger final infarct size, but not larger initial ischemic area at risk [32]. In addition, same patients are less likely to get benefit from thrombolytic therapy than patients with grade B ST [5].

Birnbaum et al analyzed 2603 patients with acute myocardial infarction who received thrombolytic therapy as reperfusion strategy within six hours of the onset of symptoms showing comparable results regarding the In-hospital mortality when treated within 02 hours from onset of symptoms between patients with grade C and grade B. However, If the time interval of treatment exceeded more than two hours after onset of symptoms, mortality increased to 7.4% in patients with grade C as compared to 3.6% in patients with grade B type, showing that the beneficial effect of salvaging myocardium with reperfusion therapy is lost after two hours due to a more rapid rate of progression of necrosis in Grade C patients [15].

Findings from the AMISTAD study also confirmed the same hypothesis of more necrosis progression in grade C patients as

compared to grade B confirmed by 99mTc sestamibi SPECT myocardial perfusion scan on admission as well as pre-discharge. It also showed that in patients with grade C necrosis progresses rapidly and less myocardial preservation is expected, and patients with grade C ST Segments usually have larger infarct size [32].

Patients with grade 3 (Type 3) on the admission ECG have a higher mortality [33,34] and re-infarction rate [4,25], reduce risk of re-infarction but no effect on reduction of mortality, regardless of the treatment strategy, either primary angioplasty or the thrombolysis, shown by GUSTO IIb [33].

The Purkinje system is less sensitive than the myocytes in resisting ischemia [35,36]. Hence, only a very severe and long term ischemia that affects the Purkinje fibers could alter the terminal portion of the QRS complex [37,38] also showing that type C (Type 3) pattern is well correlated with the severe form of ischemia.

The limitation of our study is a small sample size but keeping in mind the results of previously conducted studies and duplication of the same result in our population, it appears that we can safely endorse to our emergency physicians, to promptly recognize and stratify the high risk patients depending upon the shape of the acute ST Segment. Another difference between other studies and our study was that for the calculation of EF they used ventriculography, that was done after 14 days of infarction but in our study EF was calculated by echocardiography which is more sensitive method for calculation of EF strengthening the current study protocol.

Conclusion

We conclude that those patients that showed type 3 or convex type ST elevation with terminal distortion showed the maximum damage of EF which may lead to future grave cardiovascular sequels, so require prompt diagnosis and early activation of Cath lab as the first line reperfusion strategy.

References

1. Mehta RH, Parsons L, Rao SV (2012) Association of bleeding and in-hospital mortality in black and white patients with ST-segment-elevation myocardial infarction receiving reperfusion. *Circulation* 125:1727-1734.
2. DeWood MA, Spores J, Notske R, Mouser LT, Burroughs R, et al. (1980) Prevalence of total coronary occlusion during the early hours of transmural myocardial infarction. *N Engl J Med* 303: 897-902.
3. Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, et al. (2012) Third universal definition of myocardial infarction. *Circulation* 126: 2020-2035.
4. Birnbaum Y, Kloner R, Sclarovsky S, Cannon CP, McCabe CH, et al. (1996) Distortion of the terminal portion of the QRS on the admission electrocardiogram in acute myocardial infarction and correlation with infarct size and longterm prognosis (Thrombolysis in Myocardial Infarction 4 Trial). *Am J Cardiol* 78: 396-403.
5. Brinbaum Y, Maynard C, Wolfe S, Mager A, Strasberg B, et al. (1999) Terminal QRS distortion on admission is better than ST segment measurements in predicting final infarct size and assessing potential effects of thrombolytic therapy in anterior wall acute myocardial infarction. *Am J Cardiol* 84: 530-539.
6. Billgren T, Birnbaum Y, Sgarbossa E, Sejersten M, Hill NE, et al. (2004) Refinement and interobserver agreement for the electrocardiographic Sclarovsky Birnbaum ischemia grading system. *J Electrocardiol* 37: 149-156.
7. Maroko PR, Libby P, Covell JW, Sobel BE, Ross J, et al. (1972) Precordial S-T segment elevation mapping: an atraumatic method for assessing alterations in the extent of myocardial ischemic injury. The effects of pharmacologic and hemodynamic interventions. *Am J Cardiol* 29: 223-230.
8. Muller JE, Maroko PR, Braunwald E (1975) Evaluation of precordial electrocardiographic mapping as a means of assessing changes in myocardial ischemic injury. *Circulation* 52: 16-27.
9. Madias JE, Venkataraman K, Hodd WB (1975) Precordial ST-segment mapping 1. Clinical studies in the coronary care unit. *Circulation* 52: 799-809.
10. Muller JE, Maroko PR, Braunwald E (1978) Precordial electrocardiographic mapping. A technique to assess the efficacy of interventions designed to limit infarct size. *Circulation* 57: 1-18.
11. Bren GB, Wasserman AG, Ross AM (1987) The electrocardiogram in patients undergoing thrombolysis for myocardial infarction. *Circulation* 76: II18-II24.
12. Vermeer F, Simoons ML, Bär FW (1986) Which patients benefit most from early thrombolytic therapy with intracoronary streptokinase? *Circulation* 74: 1379-1389.
13. Bar FW, Vermeer F, de Zwaan C, Ramentol M, Braat S, et al. (1987) Value of admission electrocardiogram in predicting outcome of thrombolytic therapy in acute myocardial infarction. A randomized trial conducted by The Netherlands Interuniversity Cardiology Institute. *Am J Cardiol* 59: 6-13.
14. Yusuf S, Lopez R, Maddison A, Maw P, Ray N, et al. (2017) Value of electrocardiogram in predicting and estimating infarct size in man. *Br Heart J* 42: 286-293.
15. Birnbaum Y, Herz I, Sclarovsky S, Zlotikamien B, Chetrit A, et al. (1996) Prognostic significance of the admission electrocardiogram in acute myocardial infarction. *J Am Coll Cardiol* 27: 1128-1132.
16. Kosuge M, Kimura K, Ishikawa T, Kuji N, Tochikubo O, et al. (1999) Value of ST-segment elevation pattern in predicting infarct size and left ventricular function at discharge in patients with reperfused acute anterior myocardial infarction. *Am Heart J* 137: 522-257.
17. White HD, Norris RM, Brown MA, Brandt PW, Whitlock RM, et al. (1987) Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction. *Circulation* 76: 44-51.
18. Sheehan FH, Mathey DG, Schofer J, Krebber HJ, Dodge HT (1983) Effect of interventions in salvaging left ventricular function in acute myocardial infarction: a study of intracoronary streptokinase. *Am J Cardiol* 52: 431-438.
19. Hamer AW, Takayama M, Abraham KA, Roche AH, Kerr AR, et al. (1994) End-systolic volume and long-term survival after coronary artery bypass graft surgery in patients with impaired left ventricular function. *Circulation* 90: 2899-2904.
20. Aldrich HR, Wagner NB, Boswick J, Corsa AT, Jones MG, et al. (1988) Use of initial ST-segment deviation for prediction of final electrocardiographic size of acute myocardial infarcts. *Am J Cardiol* 61: 749-753.
21. Christian TF, Gibbons RJ, Clements IP, Berger PB, Selvester RH, et al. (1995) Estimates of myocardium at risk and collateral flow in acute myocardial infarction using electrocardiographic indexes with comparison to radionuclide and angiographic measures. *J Am Coll Cardiol* 26: 388-393.
22. Mauri F, Gasparini M, Barbonaglia L, Santoro E, Franzosi MG, et al. (1989) Prognostic significance of the extent of myocardial injury in acute myocardial infarction treated by streptokinase (the GISSI trial). *Am J Cardiol* 63: 1291-1295.
23. Nielsen BL (1973) ST-segment elevation in acute myocardial infarction: prognostic importance. *Circulation* 48: 338-345.
24. Marik PE, Lipman J, Eidelman IJ, Erksine PJ (1990) Clinical prediction of early death in acute myocardial infarction: a prospective study of 233 patients. *S Afr Med J* 77: 179-182.
25. Birnbaum Y, Sclarovsky S, Blum A, Mager A, Gabbay U (1993) Prognostic significance of the initial electrocardiographic pattern in a first acute anterior wall myocardial infarction. *Chest* 103: 1681-1687.

26. Birnbaum Y, Sclarovsky S, Herz I, Zlotikamien B, Chetrit A (1997) Admission clinical and electrocardiographic characteristics predicting in hospital development of high-degree atrioventricular block in inferior wall acute myocardial infarction. *Am J Cardiol* 80: 1134-1138.
27. Clements IP, Christian TF, Higano ST, Gibbons EU, Gersh BI (1993) Residual flow to the infarct zone as a determinant of infarct size after direct angioplasty. *Circulation* 88: 1527-1533.
28. Christian TF, Gibbons RJ, Clements IP, Berger PB, Selvester RH, et al. (1995) Estimates of myocardium at risk and collateral flow in acute myocardial infarction using electrocardiographic indexes with comparison to radionuclide and angiographic measures. *J Am Coll Cardiol* 26: 388-393.
29. Kosuge M, Kimura K, Ishikawa T, Endo T, Hongo Y, et al. (2001) ST-segment depression in lead aVR predicts pre-discharge left ventricular dysfunction in patients with reperfused anterior acute myocardial infarction with anterolateral ST-segment elevation. *Am Heart J* 142: 51-57.
30. Birnbaum Y, Herz I, Sclarovsky S, Zlotikamien B, Chetrit A, et al. (1996) Prognostic significance of precordial ST segment depression on admission electrocardiogram in patients with inferior wall myocardial infarction. *J Am Coll Cardiol* 28: 313-318.
31. Birnbaum Y, Criger DA, Wagner GS, Strasberg B, Mager A, et al. (2001) Prediction of the extent and severity of left ventricular dysfunction in anterior acute myocardial infarction by the admission electrocardiogram. *Am Heart J* 141: 915-924.
32. Birnbaum Y, Mahaffey KW, Criger DA, Gates KB, Barbash GI, et al. (2002) Grade III ischemia on presentation with acute myocardial infarction predicts rapid progression of necrosis and less myocardial salvage with thrombolysis. *Cardiology* 97: 166-174.
33. Birnbaum Y, Goodman S, Barr A, Gates KB, Barbash GI, et al. (2001) Comparison of primary coronary angioplasty versus thrombolysis in patients with ST-segment elevation acute myocardial infarction and grade II and grade III myocardial ischemia on the enrollment electrocardiogram. *Am J Cardiol* 88: 842-847.
34. Lee CW, Hong M-K, Yang H-S, Choi S-W, Kim J-J, et al. (2001) Determinants and prognostic implications of terminal QRS complex distortion in patients treated with primary angioplasty for acute myocardial infarction. *Am J Cardiol* 88: 210-213.
35. DeHaan RL (1961) Differentiation of the atrioventricular conducting system of the heart. *Circulation* 24: 458-470.
36. Schieber TH, Stark M, Caesar R (1956) Die Stoffwechselsituation des Reizleitungssystems. *Klin Wochenschr* 34: 181-183.
37. Holland RP, Brooks H (1976) The QRS complex during myocardial ischemia: an experimental analysis in the porcine heart. *J Clin Invest* 57: 541-50.
38. Feldman T, Chua KG, Childres RW (1986) R wave of the surface and intracoronary electrogram during acute coronary artery occlusion. *Am J Cardiol* 58: 885-890.