Editor I. HOFFMAN
Co-editor R. I. HAMBY

VECTORCARDIOGRAPHY 3

Proceedings of the Symposium,
held in New York City, 1975

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Proceedings of the Symposium on Clinical Vectorcardiography 1975, held in New York City, May 10-11, 1975

IRWIN HOFFMAN, *Editor*
ROBERT I. HAMBY, *Co-editor*

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PREFACE

This volume presents the papers presented at "Clinical Vectorcardiography 1975", a meeting held in New York City, May 10-11, 1975, and sponsored jointly by the Long Island Jewish Hillside Medical Center, and the American College of Cardiology.

The editors wish to acknowledge the patience and devotion to this project of the Communities Relations Department of the Long Island Jewish Hillside Medical Center, particularly Mrs. June Lewis, Mrs. Lola Sher and Mrs. Ruth Cook. The authors especially wish to thank Dr. Robert K. Match, President of the Long Island Jewish Hillside Medical Center, Dr. James E. Mulvihill, Dean of the Clinical Campus of the State University of New York at Stony Brook, and Dr. Edward Meilman, Chairman of the Department of Medicine, LIJH, and Professor of Medicine at Health Sciences Center, State University of New York at Stony Brook, for their encouragement and invaluable help in preparing and organizing the symposium and its publication.

Irwin Hoffman, M.D., Editor

Robert I. Hamby, M.D., Co-Editor

LIST OF CONTRIBUTORS

The papers comprising this volume were presented at "Clinical Vectorcardiography 1975" on May 10-11, 1975 in New York City. The scheduled speakers for that program, and their academic titles and affiliations, are listed below. Their cooperation in organizing the meeting, and in submission of manuscripts, is gratefully acknowledged by the editors.

J.A. ABILDSKOV, M.D., F.A.C.C., Professor of Medicine and Director of Cardiovascular Research and Training Institute, University of Utah College of Medicine, Salt Lake City, Utah

ALBERTO BENCHIMOL, M.D., F.A.C.C., Director, Institute for Cardiovascular Diseases, Good Samaritan Hospital, Phoenix, Ariz.

MARY JO BURGESS, M.D., Associate Professor of Medicine, Cardiovascular Division, University of Utah College of Medicine, Salt Lake City, Utah

AGUSTIN CASTELLANOS, JR., M.D., F.A.C.C., Professor of Medicine and Director, Clinical Electrophysiology, University of Maimi School of Medicine, Maimi, Fla.

TE-CHUAN CHOU, M.D., F.A.C.C., Professor of Medicine, University of Cincinnati College of Medicine, Cincinnati, Ohio

ROLF M. GUNNAR, M.D., F.A.C.C., Professor of Medicine and Chief, Section of Cardiology, Loyola University of Chicago, Stritch School of Medicine, Maywood, Ill.

ROBERT I. Hamby, M.D., F.A.C.C., Attending in Charge, Cardiology Division, Long Island Jewish-Hillside Medical Center, New Hyde Park; Associate Professor of Medicine, School of Medicine, Health Sciences Center, State University of New York at Stony Brook, Stony Brook, N.Y.

JOSEPH HILSENRATH, M.D., Internist and Cardiologist, Department of Medicine, Long Island Jewish-Hillside Medical Center, New Hyde Park; Assistant Professor of Clinical Medicine, School of Medicine, Health Sciences Center, State University of New York at Stony Brook, Stony Brook, N.Y.

IRWIN HOFFMAN, M.D., F.A.C.C., Attending in Charge, Electrocardiography-Vectorcardiography, Long Island Jewish-Hillside Medical Center, New Hyde Park; Associate Professor of Clinical Medicine, School of Medicine, Health Sciences Center, State University of New York at Stony Brook, Stony Brook, N.Y.

RICHARD J. KENNEDY, M.D., F.A.C.C., Director, Electrocardiographic Department, St. Vincent's Hospital and Medical Center of New York; Clinical Professor of Medicine, New York University-Bellevue Medical Center, New York, N.Y.

FRED KORNREICH, M.D., Director, Unit for Cardiovascular Research, University of Brussels, Brussels, Belgium

HENRI E. KULBERTUS, M.D., F.A.C.C., Associate Professor of Cardiology, Institute of Medicine, University of Liege School of Medicine, Liege, Belgium

JEROME LIEBMAN, M.D., F.A.C.C., Professor of Pediatrics, Case Western Reserve University School of Medicine; Director of Pediatric Cardiology, University Hospitals of Cleveland, Cleveland, Ohio

B. LYNN MILLER, M.D., F.A.C.C., Assistant Professor of Pediatrics, University of Florida College of Medicine J. Hills Miller Health Center, Gainesville, Fla.

HUBERT V. PIPBERGER, M.D., F.A.C.C., Professor of Clincial Engineering and Medicine, The George Washington University Medical Center ; Chief, Veterans Administration Research Center for Cardiovascular Data Processing, Veterans Administration Hospital, Washington, D.C.

PENTTI RAUTAHARJU, M.D., Ph.D., Professor-Director, Department of Physiology and Biophysics, Dalhousie University Faculty of Medicine, Halifax, Nova Scotia

HAROLD G. RICHMAN, M.D., F.A.C.C., Associate Professor of Medicine, University of Minnesota, Minneapolis, Medical School; Staff Physician, Cardiovascular Division and Director, Electrocardiography Laboratory, Veterans Administration Hospital, Minneapolis, Minn.

BORYS SURAWICZ, M.D., F.A.C.C., Professor of Medicine and Director, Cardiology Division, University of Kentucky College of Medicine, Lexington, Ky.

OLGA ZONERAICH, M.D., Associate Attending in Medicine and Cardiology, Long Island Jewish-Hillside Medical Center, Queens Hospital Affiliation, Jamaica; Associate Professor of Medicine, State University of New York at Stony Brook, Health Sciences Center, School of Medicine, Stony Brook, N.Y.

SAMUEL ZONERAICH, M.D., F.A.C.C., Head, Division of Cardiology, Long Island Jewish-Hillside Medical Center, Queens Hospital Center Affiliation, Jamaica; Associate Professor of Medicine, State University of New York at Stony Brook, Health Sciences Center School of Medicine, Stony Brook, N.Y.

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INTRODUCTION

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DEFENSIVE VECTORCARDIOGRAPHY

Irwin Hoffman

From the ECG-VCG Department, Long Island Jewish Hillside Medical Center,
New Hyde Park, N.Y. and The Health Sciences Center,
State University of New York at Stony Brook

Vectorcardiography is a powerful diagnostic tool, facilitating the recognition of myocardial infarction, ventricular conduction disturbances, and ventricular hypertrophies. However, the QRS and T-loop alterations in various disease states overlap considerably, leading to inevitable diagnostic error. Such mistakes are most easily detected when the vectorcardiogram is followed up by a complete hemodynamic investigation in the invasive cardiac laboratory.

In general, infarction shifts portions of the QRS loop away from the damaged area, while conduction disorders or hypertrophies shift the QRS forces towards the affected zones. These shifts may occur in any of the six directions of the XYZ orthogonal lead system employed. Unfortunately, the timing, shape and magnitude of loop displacements may be very similar in widely different disease states, making precise anatomic diagnosis of the VCG extremely difficult.

Nevertheless, the information content in the vectorcardiogram can and should be used by the cardiologist. Based on current and always changing knowledge in this field, certain precautions are herein proposed which may serve to protect the vectorcardiographer and his patient from the hazards implicit in diagnostic error.

(1) The patient has a normal vectorcardiogram, but has been submitted for study because of clinical suspicion of heart disease. The normal VCG does not rule out cardiac pathology. Indeed, coronary disease of single, double or triple extent may be present, as well as ventricular asynergy, mitral stenosis or hypertrophic subaortic stenosis. The principle to remember is that a normal vectorcardiogram does not rule out heart disease.

(2) The patient under study for cardiac disorder has small or absent anterior QRS forces on the Z-axis, resembling the picture of anterior wall infarction. Non-cardiac causes must also be considered. Left ventricular hypertrophy of any etiology, but especially aortic stenosis, should come first to mind. Chronic obstructive lung disease may result in a very similar VCG picture. Cardiomyopathies of any etiology, including amyloidosis, may result in decreased or absent anterior forces. Less commonly, mitral valve stenosis or prolapse of the mitral valve may cause a similar picture. Indeed, in the presence of left ventricular hypertrophy, any initial anterior forces should call the diagnosis of anterior wall infarction into question.

(3) Large anterior forces on the Z-axis are observed in a patient undergoing cardiovascular examination. These large forces may suggest the diagnosis of dorsal wall infarction or right ventricular hypertrophy. In this 1975 VCG symposium, the concept of anterior conduction delay as a cause for prominent anterior QRS forces was proposed [1]. If this concept is confirmed in the future, it is likely that many degrees of anterior conduction delay will be observed, similar to various degrees of right ventricular conduction delay which are so commonly encountered. When the diagnosis of true dorsal infarction is suspected, the diagnosis is more difficult in the absence of QRS or T-wave evidence of lateral or inferior wall infarction.

(4) Left bundle branch block is diagnosed from the vectorcardiogram and initial anterior forces are not seen, suggesting anterior wall infarction. Occasionally, in left bundle branch block in patients completely free of heart disease, no anterior forces are visible even with amplification. Occasionally, such anterior forces are present but are so small that even amplified loops do not reveal the initial QRS forces which are superimposed upon the E-point or on the P or T-loop. A high amplitude Z-axis lead, swept in a scalar manner, will occasionally reveal such small initial anterior forces.

(5) The patient under investigation for heart trouble has right bundle branch block. Under these circumstances, especially if the anterior conduction delay theory holds up, many right bundle branch blocks with anterior displacement on the Z-axis will represent combinations of right bundle branch block with mid-septal (anterior) branch block of the left division. Therefore, it seems likely that the diagnosis of true dorsal wall infarction will simply not be possible in patients with right bundle branch block. It is presently known that the combination of right bundle branch block and left anterior hemiblock, readily diagnosable from the frontal plane vectorcardiogram, presents as two families of loops in the horizontal plane--one anterior and one posterior. It seems likely that the anterior family of loops represents additional involvement of the left mid-septal branch.

Also, in right bundle branch block, Q-waves of small magnitude are commonly seen in leads V-1 and V-2 which, in the vectorcardiogram, are written as initial direct posterior forces. Goldman et al. [2] have clearly shown that such posterior initial QRS forces are frequently not correlated with the presence of anterior wall infarction at postmortem examination.

(6) The patient under investigation for heart trouble has a right anterior T-loop in the horizontal plane which rotates clockwise. This abnormality is not specific for coronary artery disease, although it may occur in that condition. Such clockwise right anterior horizontal T-loops have been seen in left ventricular hypertrophy, left bundle branch block, and right ventricular pacing. They frequently reflect severe underlying disease and may at times represent a combination of ventricular hypertrophy and ischemia, secondary to coronary obstruction. However, the clockwise rotation itself is not specific for coronary artery disease.

(7) The patient under investigation for heart pathology has a VCG interpreted as inferior wall myocardial infarction. The cardiologist must consider non-coronary conditions which may mimic inferior wall infarction. The commonest of these are hypertrophic subaortic stenosis, and aortic insufficiency. Occasionally right ventricular hypertrophy will present with initial superior forces meeting criteria for inferior wall M.I. Rarely, a similar picture is seen in the syndrome of mitral valve prolapse.

(8) Technical problems occur in vectorcardiography as well as in standard ECG studies. Reversal of electrodes, especially the A and I electrodes, may occur. This interchanges the voltages from the left and right axillae and results in a deep negative deflection in lead X, and therefore a rightward horizontal and frontal loop resembling right ventricular hypertrophy. Since there is no internal check in the vectorcardiogram, the XYZ leads observed will be consistent with the vectorcardiographic loops. However, comparison with the 12-lead tracing in the particular case should give the clue that a technical error has occurred. Similarly, if the VCG leads are placed too high or too low around the chest wall, significant changes in magnitude and direction of the XYZ leads resulting are very likely.

(9) Do not deprive a patient of cardiac catheterization and possible surgical help on VCG evidence alone of complicating disease. Specifically, if a patient is studied for valve disorder with left ventricular hypertrophy, but the VCG indicates anterior or inferior M.I., this latter diagnosis should be confirmed by coronary angiography. Pseudoinfarction patterns in LVH are very common and should not deprive a patient of his needed valve replacement.

(10) If a patient is perfectly normal clinically and has no chest pain, heart murmur or other cardiac symptomatology, but an ECG and VCG are somewhat bizarre, do not perform cardiac catheterization. Rarely, if ever, is significant pathology uncovered under these circumstances, and the patient is submitted to a procedure which is not without hazard.

(11) Any cardiologist reading vectorcardiograms should have some continuing contact with an invasive cardiac lab. In this way his VCG diagnoses will be subject to hemodynamic and anatomical confirmation. In no other way will the vectorcardiographer realize all the surprises, combinations and errors that are possible using the technique of vectorcardiography.

(12) Vectorcardiograms should not be read "cold." Even very simple preliminary information as the patient's underlying symptoms such as chest pain or dyspnea, or perhaps heart murmur, is sufficient to start the interpretation in the correct direction.

References

[1] I. Hoffman et al., Anterior conduction delay--A possible cause for pseudodorsal infarction, in present volume.

[2] M. Goldman and H. Pipberger, Analysis of the orthogonal electrocardiogram and vectorcardiogram in ventricular conduction defects with and without myocardial infarction, Circulation 39 (1969) 243.

SECTION 1.
MYOCARDIAL INFARCTION AND HYPERTROPHY

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VECTORCARDIOGRAPHIC DIAGNOSIS OF MYOCARDIAL INFARCTION AND LEFT VENTRICULAR HYPERTROPHY

Rolf M. Gunnar, John F. Moran, M. Ziad Sinno, and Dilip Shah

From the Sections of Cardiology, Departments of Medicine, Loyola University Stritch School of Medicine, Maywood, Illinois, and the Veterans Administration Hospital, Hines, Illinois

In order for the vectorcardiogram to be a useful diagnostic tool in patients with myocardial infarction, we must understand the genesis of the QRS loop and how infarction, by removing electrically active tissue, will deform that loop. Although the lower left septal endocardium and the apical anterior endocardium are the earliest areas of the heart to be activated, much of the cavity of the left ventricle has been activated by 25 msec. By 30 msec, the entire cavity, even the posterobasal segment, is activated. Thus, infarction of any area of the left ventricle will tend to deform the early forces of the QRS loop. Since the loop defines the resultant instantaneous vectors, infarction of any particular area will tend to enlarge the resultant vector, and, therefore, the QRS loop, in a direction away from the infarcted area.

There is additional information relating to myocardial infarction in the later portions of the QRS loop. Attempts have been made to analyze these terminal forces, but the value of criteria derived from these analyses remains to be proven. Since the velocity of movement of the wave of depolarization through the myocardium is much more variable than the direction of the initial forces, it is more difficult to codify the effects of loss of myocardium on terminal forces than it is to derive criteria for infarction from the initial forces.

Hugenholtz, working in Dr. Harold Levine's laboratory, proposed and tested criteria for myocardial infarction based on the deformity of the initial portion of the QRS loop [2]. These criteria have been re-tested and expanded. This has led to a better understanding of the accuracy of the various criteria and conditions other than myocardial infarction which may lead to similar deformities of the QRS loop [3, 4].

Anteroseptal Infarction (figure 1)

The loss of the lower portion of the septum and of the adjacent anterior wall of the left ventricle destroys the early forces whose vector is right and anterior. Thus, in anteroseptal infarction, the initial QRS forces move quickly posterior and to the left so that the .02 sec vector is behind the null point. This deformity of the QRS loop can occur with marked fibrosis of the septum and anterior wall as well as with infarction, or with replacement of this area with amyloid [4]. The presence of marked posterobasal hypertrophy in patients with severe aortic stenosis or hypertrophic cardiomyopathy can also overwhelm the normal anterior forces generated by the septum and cause the .02 sec vector to move posteriorly. Loss of early anterior forces has been described in patients with severe emphysema [5]. The effect of emphysema may be particularly evident in the Frank lead system, since the correcting resistances do not allow for the imposition of a great deal of lung tissue between the anterior chest wall and the left ventricle (table 1).

Anterolateral Infarction (figure 2)

An infarct involving the anterior and lateral free wall of the left ventricle, but leaving the septum mostly intact, will allow the early septal vectors directed anteriorly and to the right to persist until the forces from the posterior free wall turn the loop posteriorly and to the left. This will allow the initial portion of the QRS loop to rotate in a clockwise direction in the horizontal plane. If the area of involvement of the lateral wall is extensive, the entire loop may rotate in a clockwise direction. Thus, anterolateral wall infarction is diagnosed by clockwise rotation of the initial QRS loop around the E point in the horizontal plane. This rotation is a rather specific deformity having a small number of false-positives, when severe fibrosis of the lateral wall occurs in patients with marked left ventricular hypertrophy [4]. Right bundle branch block and severe right ventricular hypertrophy will rotate the loop clockwise around the null point in the horizontal plane. In this deformity, the entire loop lies anterior while in anterolateral infarct the loop is directed posteriorly and to the left. Two additional criteria have been proposed. Though not well tested, they may give strength to the diagnosis of anterolateral infarction. The initial rightward forces may exceed the upper limits of normal of 0.16 mv [6]. The loop in the frontal plane may rotate in a counterclockwise direction when the maximal QRS vector is greater than 40°. This abnormal rotation in the frontal plane would be a reflection of the clockwise

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rotation in the horizontal plane if the loop did, indeed, remain vertical.

Extensive Anterior Infarction (figure 3)

The loop begins posteriorly and to the right, and the loop has a counterclockwise rotation. This is reflected in the frontal plane loop by prolongation of the initial rightward forces. In the standard electrocardiogram, it is seen as QS deformity over much of the precordium with large Q waves in leads V_5 and V_6 and standard leads I and AVL.

Table 1

Comparison of vectorcardiographic measurements in patients with emphysema and patients with anteroseptal infarction. Adapted from the report of Watanabe et al. [5]

	Emphysema VS ASI (m ± S.D.)					
	N	$R_X + R_Z$	0.025 sec Z	$S_X/R_X + S_X$	QRS Duration	Comb.
Emphysema	52	0.85 ± 0.36	.087 ± 0.25	0.42 ± 0.29	93.6 ± 20.5	88%
Anteroseptal Infarction	35	1.64 ± 0.68	0.35 ± 0.35	0.25 ± 0.26	105.5 ± 14.0	91%
Discrimin.		1.12mv	0.10mv	0.33mv	100.7	

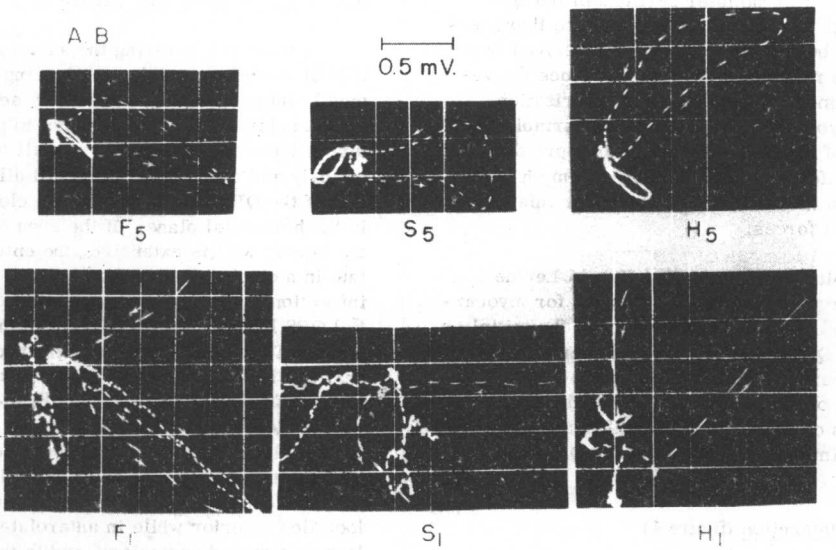

Fig. 1. Anteroseptal infarction with the 0.02 sec vector posterior. Interruptions are at 400 per sec (0.0025 sec per dot).

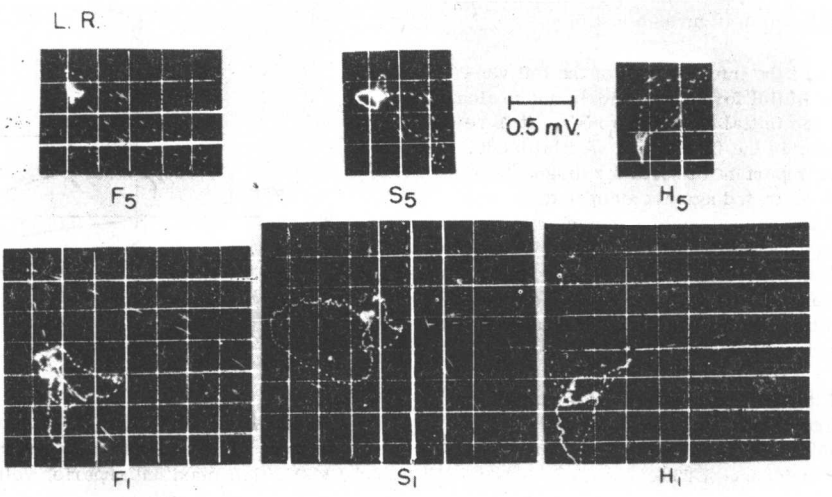

Fig. 2. Anterolateral infarction with clockwise initial forces in the horizontal plane. Also meets criteria for inferior infarction: 0.025 sec vector superior and left maximal superior vector > 0.4 mv with a clockwise QRS_F.

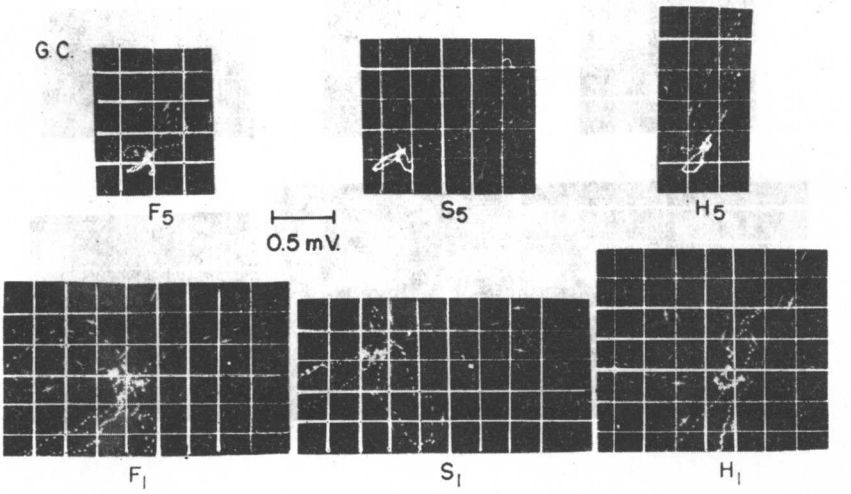

Fig. 3. Extensive anterior infarction with clockwise rightward and posterior rotation of the initial QRS_H and the entire QRS_H rotates in a clockwise manner.

Inferior Wall Infarction (figures 4 and 5)

Infarction of the inferior wall of the left ventricle shifts the initial forces superiorly and prolongs the duration of the initial superior forces. It is reflected in changes in the frontal and sagittal leads. The three most important criteria for diagnosis of this deformity, as tested against autopsy data, are: (1) the .02 and .03 second vectors superior to the X axis; (2) the point at which the initial superior forces traverse the X axis (left maximal superior voltage) > 0.4 mv to the left; and (3) persistence of clockwise rotation of the initial QRS forces when the maximal QRS vector is shifted to the left of 0^{o} [4].

If two of these three criteria are met, the incidence of inferior infarction, as proven at autopsy, approaches 100%. These criteria, however, have a false-negative incidence of 23%.

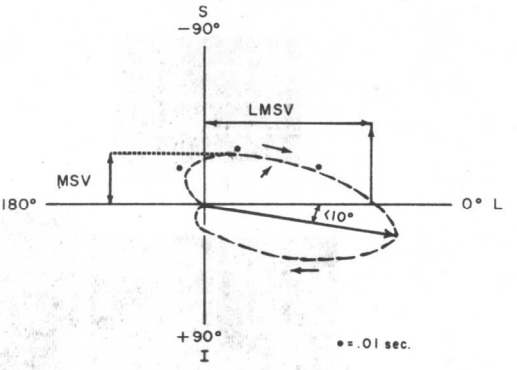

Fig. 4. Diagram of the QRS_F loop in inferior infarction. The left maximal vector is at less than 10^{o}. LMSV = left maximal superior voltage. MSV = maximum superior voltage.

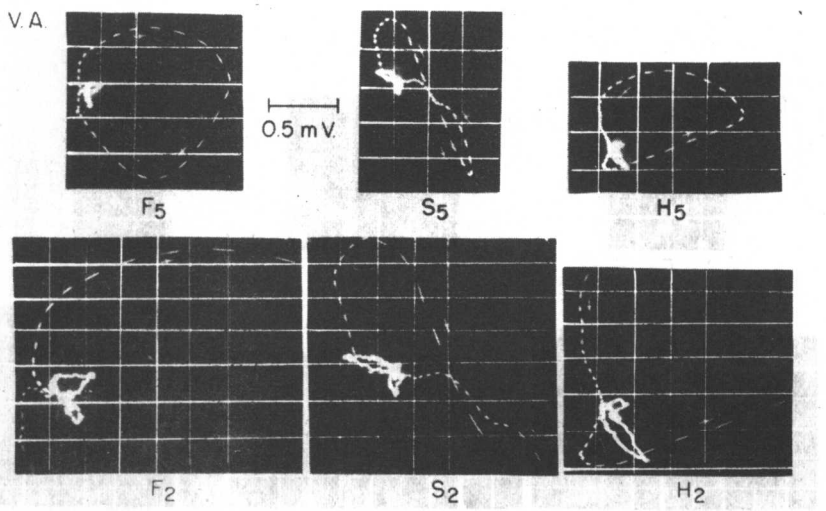

Fig. 5. Inferior infarction meeting the following criteria: 0.02 and 0.03 sec vectors superior. LMSV > 0.4 mv, clockwise QRS_F with left maximal vector above 0^{o}, and maximum superior voltage > 0.1 mv, and a Q_y:R_y ratio > 1:5.

The criterion of LMSV = or > 0.4 mv is an extension of the findings of Hoffman et al. [7] who found the LMSV = or > 0.3 mv in 63 of 76 (83%) patients with documented previous inferior myocardial infarction, and proposed this as the criterion to be used for this measurement. However, they only decreased their yield slightly to identify 59 (78%) patients when the criterion was changed to LMSV = or > 0.4 mv. There have been instances of normal patients with frontal QRS loops meeting the less stringent criterion. These authors also tested the 0.025 second vector superior, and this identified 66 of the 76 patients (87%). The combination of these two criteria identified 72 (95%) of the patients. However, there was no control group to test how many patients without infarction would be identified by using these combined criteria.

Young and Williams proposed the following criteria for inferior infarction [8]:

(1) Clockwise initial superior forces in an upward convexity and > 0.02 sec in duration, and = or > 0.25 mv leftward in magnitude.

(2) As in (1), but superior forces = or > 0.025 sec if leftward magnitude is < 0.25 mv.

(3) Completely clockwise initial forces if the maximal QRS vector is to the left of 10°.

(4) If the loop is clockwise but the initial forces are inferior and to the right, then subsequent clockwise superior forces must be 0.25 sec in duration.

(5) If there is a short inferior leftward initial vector, then this is quickly reversed and followed by a superior clockwise loop = or > 0.025 sec in duration.

These criteria were derived on 100 patients with prior or present ECG evidence of inferior infarction and tested by comparing with VCGs of 315 normal subjects of all ages.

Starr et al. [9] analyzed the criteria of Young and Williams by testing these criteria in a group of normal individuals and in a group of individuals with clinical inferior infarction or angiographic evidence of inferior infarction. They found a false-positive incidence of 7% and a false-negative incidence of 12%. These authors proposed their own criteria and tested these criteria in a new group of patients. Their criteria are predicated on a clockwise initial loop in the frontal plane and include one of the following:

(1) time at which the QRS crosses the X axis = or > 0.025 sec and at a magnitude > 0.3 mv to the left,

(2) a maximal QRS vector to the left at 15°,

(3) maximal superior voltage = or > 0.1 mv and Q:R ratio in Y axis at least 1:5.

The difficulty in testing against a group of normal subjects is that conditions other than infarction may deform the QRS loop, and such patients would be excluded from the control group. On the other hand, any autopsy study eliminates consideration of healthy normal individuals from the control group. Understanding the genesis of the QRS loop and how it may be deformed allows interpretation of the vectorcardiogram as modified by clinical findings. We suggest that if a clockwise initial frontal plane QRS loop meets the following criteria--the 0.03 second vector above the null point; the LMSV > 0.4 mv; maximal QRS vector to the left of 10°--then one can be assured the patient has an inferior infarction. If the QRS loop is clockwise, if there is 0.025 sec of the initial vector above the X axis and LMSV is > 0.3 mv, and if there is no evidence of severe ventricular hypertrophy, the diagnosis of inferior infarct has a very high probability of being correct. If the QRS loop in the frontal plane is initially clockwise, if the 0.02 sec vector is inferior and the LMSV is < 0.16 mv, and if the maximal QRS vector is greater than 20°, then the diagnosis of inferior infarction is highly unlikely. Between these negative and positive criteria will be patients with and without infarction, and analysis of clinical data becomes important in the diagnosis. In this latter group will be a few normal patients, some patients with myocardial fibrosis and micro-infarction, patients with pulmonary embolism, and patients with left ventricular hypertrophy, particularly of the asymmetric type.

Posterior Infarction (figures 6 and 7)

In the absence of inferior wall infarction, it is difficult to diagnose true posterior infarction. Even though many would insist that the deformity of the QRS loop should be seen in the terminal forces, it is true that the posterior wall begins its activation process early and most of the criteria for diagnosis of this lesion are based on deformity of the initial forces of the QRS loop [2, 3, 10]. The loss of posterior forces allows the entire loop to move anteriorly. The criteria for the diagnosis include maximal anterior QRS voltage greater than 0.55 mv, the maximum horizontal QRS vector anterior to 20°, and the duration of the anterior forces greater than .05 seconds. In analysis of the terminal forces, we have found useful the criterion of a rather straight slow terminal segment directly posterior to the null point. The major causes for false-positive diagnoses of posterior infarction using these criteria are right ventricular hypertrophy and anterior displacement of the heart against the chest wall. This latter deformity is seen not only in the straight-back syndrome and