High Anterior Myocardial Infarction

XX. Studies on the Mechanism of Ventricular Activity

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Out of a large series of patients with coronary artery disease 6 were selected with high anterior myocardial infarction. These cases are presented in detail and their special electrocardiographic characteristics are described. The difficulty in making the diagnosis of infarction by the usual leads and the need for special leads are pointed out.

The most widely accepted electrocardiographic classification of myocardial infarction is that proposed by Wilson and his associates. They classified myocardial infarction “on the basis of the leads in which characteristic modifications of both the QRS deflections and the T complexes appear and have been given names indicative of the parts of the ventricular wall known, or thought, to be involved.” Eight types of infarction were recognized: anteroseptal, anterolateral, extensive anterior, high anterolateral, plain posterior, posterolateral, posteroinferior or posteroseptal and high posterolateral.

Another classification, based on the spatial situation of the area of body surface in which infarction Q waves are found (“Q area”), has been recently proposed by Grant and Murray. After analysis of 115 cases of myocardial infarction, the following 5 general locations of the Q area were described: strictly anterior, anterolateral, inferior or diaphragmatic, strictly posterior, and high lateral.

The conventional 12-lead electrocardiogram explores a given number of positions on the body surface, namely, the roots of the right arm, left arm, left leg, and the 6 precordial positions. If the electrocardiographic manifestations of infarction should arise outside these positions, characteristic changes of infarction will not be reflected in the electrocardiogram.

In an effort to explore the extent of diagnostic assistance that can be derived from the electrocardiogram, exploration of the body surface by multiple leads was systematically carried out in all patients with proved or suspected myocardial infarction during the past 1½ years.

In the majority of cases the area of infarcted changes invaded 1 or more of the standard lead positions and hence caused diagnostic changes in the standard electrocardiogram. In the remaining few this area lay outside the conventional lead positions. This presentation concerns those cases in which changes from the area of infarction were strictly high anterior, usually at the level of the first 3 intercostal spaces and extending from the right to left parasternal areas. Had the additional leads not been taken from the high anterior area, the correct diagnosis would have been missed. An attempt will be made to draw some practical conclusions from this experimental method of electrocardiographic study, and electrode positions that constitute the minimum number of leads sufficient for detection of the high anterior infarction will be suggested.

The characteristics of the normal electrocardiogram of the high anterior area based on a study of 106 normal subjects are discussed in the Appendix.

Methods

One hundred forty-nine patients with various manifestations of coronary artery disease have been studied by means of multiple-lead electrocardiograms. Of these, 66 had clinical and electrocardiographic evidence of myocardial infarction of various types and ages. There were 6 cases of infarction considered to be high anterior that will be presented below.
Electrocardiograms

In addition to the conventional 12 leads each, 84 to 105 additional leads were taken. The lead positions were at the intersection of 7 horizontal lines and 12 to 15 vertical lines. The horizontal lines consisted of the intersections of 7 horizontal planes (passing by the sternal ends of the first 5 intercostal spaces, the epigastrium midway between the fifth intercostal space and the umbilicus, and finally the umbilicus) with the body surface. The vertical lines were drawn at the positions midsternal, V2-R, spinal, V1, V6R, V5R, and V4R. For the sake of expediency in some cases 2 to 3 of the vertical lines, usually V5 and V6R, were omitted. Thus, the torso was explored from the first intercostal space to the umbilicus both anteriorly and posteriorly. The patients were supine except that the back leads were taken in the right lateral decubitus position. The apparatus used was a direct-writing Sanborn Visocardiette at a paper speed of 25 mm. per second.

The electrocardiograms were pasted on diagrams representing an unfolded torso. One complex of each lead was affixed on the area of the diagram corresponding to the location from which the lead was taken. The various electrocardiographic inscriptions could then be studied with ease and their distributions in various regions of the body determined. From serial electrocardiograms studied in this manner valuable information was derived concerning the changing nature of QRS, ST segment, and T waves in cases of infarction.

Case Reports

Case 1. A 56-year-old diabetic man was admitted to the hospital on January 11, 1955, because of severe pain in the anterior chest with radiation to both shoulders for approximately 24 hours. He reported a mild, transient episode of precordial pain that had occurred 2 weeks prior to admission. The blood pressure was 150/90, pulse rate 84, leukocyte count 13,000, and sedimentation rate 14 mm. per hour (Wintrobe); temperature was normal. His blood pressure subsequent to admission dropped to 115/70. Except for another mild attack of chest pain associated with ectopic beats on the tenth day after admission the hospital course was uneventful.

Figure 1 represents the conventional and a portion of the multiple-lead electrocardiogram taken on the second hospital day. Although the conventional electrocardiogram is abnormal with upwardly convex coving of the ST segments in leads V3, V4, V5, and aVL, associated with significant T-wave inversion in leads V2 to V5 and suspicious Q wave in aVL, no definite diagnosis of myocardial infarction can be made. However, the leads taken from the high sternal area revealed unmistakable changes of acute myocardial infarction consisting of QS waves (complete loss of R) in leads from the first 3 intercostal

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**Fig. 1. Case 1.** Tracing taken on second day of admission. Top 3 rows were taken from first 3 intercostal spaces. Note that QRS, ST, and T changes in V1, midline, V2, and V3 positions are characteristic of acute infarction. In the standard limb and chest leads, however, QRS abnormalities are absent.
spaces and occupying the positions V₁, midline, V₃, and V₅. In these same leads there is considerable degree of upwardly convex S-T elevation and T wave inversion rendering the QRS changes more significant.

Comment

An electrocardiogram taken on November 27, 1955, 11½ months after the attack showed complete reversal of the changes to normal (fig. 2). The S-T segment and T waves were normal and the R waves in high sternal leads, which had disappeared after the acute attack, have reappeared. This return of R indicates that the initial QRS negativity observed during the acute stage was the result of myocardial infarction. The Q area has shrunken remarkably.

The loss of the R waves in a relatively large area associated with elevation and coving of the S-T segments and symmetrical inversion of the T waves in some leads during the acute phase and complete reversal of all these alterations after recovery confirmed the clinical diagnosis of myocardial infarction. Noteworthy is the fact that the Q area did not invade any of the conventional lead positions; the definitive diagnosis, therefore, could not have been made without additional leads. The absence of the R waves in the high sternal area cannot be explained on the basis of positional changes inasmuch as the electric axis of the heart showed no change from one record to the other. Theoretically, pronounced clockwise rotation of the heart on its longitudinal axis can cause the normal Q area to extend from its normal position in the right upper back and right shoulder to the anterior midline; such marked rotation occurs from right ventricular dilatation. In the case under discussion, however, there is no evidence of clockwise rotation. The infarction Q area is not an extension of the normal Q area because it is separated from the latter by a zone of small right chest R waves seen in leads V₃ of the third and fourth intercostal spaces and V₄R of the second, third, and fourth intercostal spaces in figure 1. Moreover, while the normal Q area is smooth in contour and merges subtly with the adjacent
R areas, the infarction Q area tends to be irregularly shaped and to end abruptly as in this case. Above all, the normal Q area remains constant in size; the Q area in this case decreased in size as S-T and T changes of infarction regressed.

Case 2. A 50-year-old woman was admitted to the hospital on July 18, 1954, because of anterior chest pain of 2 days' duration. The blood pressure was 135/85 and pulse 84 per minute and regular. The sedimentation rate, leukocyte count, and temperature were normal. A conventional electrocardiogram taken at this time showed symmetrical T wave inversion in leads I and aV R, without any changes in QRS or ST segment. The electrocardiogram was unremarkable otherwise.

A second conventional electrocardiogram taken on the third hospital day was identical with that of admission. On the fifth hospital day a multiple-lead record showed an area of T wave inversion beginning at the left midaxillary line and merging into the normal negative T area to the right of the sternum. The latitude of the negative T area was highest at the V 3 position, descended to the fourth intercostal space, and thence tapered off, giving the negative T area a triangular shape with the apex at the V 3 position in the fourth intercostal space and the base in the first intercostal space. Within this area the S-T segments showed up to 1 mm. elevation. Noteworthy, however, was the size of the R waves which, in an area extending from the midternal line to V 4 position and from the first intercostal space to the third, did not increase in magnitude. This finding was considered particularly significant because these low amplitude R waves were situated in the center of the negative T area. There was no Q area because the initial R waves did not disappear completely. Lead aV R exhibited a normal QRS complex, isoelectric ST segment and inverted T wave.

Another multiple-lead electrocardiogram taken on the following day showed marked changes toward normal. The R waves now displayed gradual progression in magnitude toward the left, the ST segments were isoelectric and the T waves were upright (fig. 3). A third tracing taken on the eighth hospital day, following a recurrence of pain, showed only T wave inversion in this area. The tracing of the tenth hospital day was again within normal limits. Tracings taken 39 days and 15½ months after admission showed normally increasing R waves in the high anterior area, isoelectric ST segments, and upright T waves.

Comment

The serial electrocardiograms in this case showed fleeting changes involving the QRS complexes, the S-T segments, and the T waves. Although the area of T-wave inversion reached the root of the left arm and, therefore, brought about T-wave abnormality in leads I and aV R, the area of QRS changes and S-T elevation were smaller and confined to the high anterior region of the chest. Only myocardial ischemia could have been suspected from a study of the conventional leads. However, additional leads taken from the high anterior region showed significant QRS and ST changes that warranted the diagnosis of acute myocardial infarction. The clinical picture was indicative of a relatively mild attack of myocardial infarction. The fleeting nature of the electrocardiographic changes was consistent with such a diagnosis. An interesting feature of this case was that the diminution in the size of the R wave was just as transient as the ST and T changes. This phenomenon has been observed in a few other patients with classical types of myocardial infarction in whom multiple-lead records were taken at frequent intervals.

Case 3. A 58-year-old man was admitted to the hospital on February 26, 1955, because of pain in the left chest radiating down the left arm. For 2 days prior to admission, he had had episodes of pain lasting as long as 1 hour. His blood pressure had begun to rise in 1948 and reached 190/100 early in 1955. On admission he was found to be in acute distress with chest pain. The blood pressure was 170/100. The leukocyte count was 5,300 and the sedimentation rate 1 mm. per hour. On the sixth

![Fig. 3. Case 3. The large dotted area shows the area of T-wave inversion during the acute stage; the small dotted area depicts the area of reduced R waves. No true Q waves developed. All the changes regressed after recovery.](http://circ.ahajournals.org/content/58/5/783)
hospital day, the leukocyte count had risen to 9,200 and the sedimentation rate to 50 mm. per hour (Wintrobe). The blood pressure had dropped to 150/70. The leukocyte count showed further rise to 10,200 on the eighth hospital day. The serum transaminase level obtained on this day for the first time was reported as 90 U. (normal 10–40 U.).

The standard electrocardiogram taken on admission showed equivocal Q waves in aVL and failure of the R wave to increase in amplitude from V₁ to V₄. There was 1-mm. ST elevation in aVL and 3- to 4-mm. elevation in leads V₁ to V₄. The T waves were symmetrically inverted in I, aVL, and V₂ to V₆. There was reciprocal ST depression in leads II, III, and aVF. This tracing showed characteristic ST-segment and T-wave changes of acute anteroseptal infarction. However, the R waves did not disappear completely. The standard tracing of March 1, 1955, showed some decrease in the magnitude of ST elevation and increase in the size of the R wave in leads V₁ to V₄.

A multiple-lead record made on March 4 showed the true extent of the electrocardiographic changes. There was a high Q area that started at the left anterior axillary line and merged into the normal Q area of right shoulder. The latitude of this Q area included the first 3 intercostal spaces at V₂ and V₃ positions and only the first 2 intercostal spaces at midline, V₁, V₄, and V₅ positions. Lead aVL contained a 0.5-mm. Q wave of 0.02-second duration. There was S-T elevation ranging from 1 to 3.5 mm. in these chest leads; the T waves, however, were tall and upright.

An electrocardiogram on December 1, 1955, 9 months after the attack, showed return of the R waves in leads that had exhibited QS waves. The Q area now began on the left shoulder and extended posteriorly. The S-T segments maintained a 1- to 2-mm. elevation. The T waves remained unchanged. This electrocardiogram was consistent with left ventricular hypertrophy and strain.

Comment

The clinical picture as well as the ST elevation in the standard leads were those of acute myocardial infarction. The reduction in the size of the R waves in leads V₂ to V₄ suggested the possible proximity of these leads to infarcted myocardium; however, this was not proof of myocardial infarction for such QRS variations in the transitional zone are compatible with normalcy. No further information as to location and size of the Q area could be derived from the standard electrocardiogram.

A multiple-lead record taken on the sixth hospital day delineated the Q area and showed it to be high anterior in location. The disappearance of the Q waves and the return of the R waves that took place after recovery from the acute attack are evidence that the Q waves were due to infarction. There was no change of the electric axis capable of explaining the QRS changes. The fact that the T waves within the Q area remained upright is explicable on the basis of the hypertensive changes that caused the T vector to rotate anteriorly, superiorly, and to the right.

Case 4. A 58-year-old woman with a history of hypertension and diabetes was admitted, on August 15, 1955, because of severe anterior chest pain of 12 hours’ duration. She had had previous episodes of severe precordial pain diagnosed as “coronary insufficiency.” The present attack, however, was more persistent and more intense than the previous ones. Blood pressure was at its usual level of 220/96. There was evidence of cardiomegaly and slight ankle edema. The cardiac rhythm was regular. The blood pressure dropped to 180/80 on the second day and to 150/80 on the third hospital day. The leukocyte count on admission was 10,950. Temperature ranged from 100 to 101 F. for 5 days.

The conventional electrocardiogram taken on the first day was consistent with left ventricular hypertrophy. On the second day there was ST elevation in all the precordial leads without significant T-wave changes. The QRS complexes were normal. The multiple-lead record taken on the fourth hospital day showed some ST elevation in the high precordial leads and a small Q area extending from V₁ to V₄ and present only in the first 2 intercostal spaces. The T waves in this area were upright or flat, hence not significantly different from those seen in hypertension. The conventional electrocardiogram taken at this time did not differ materially from those

![Fig. 4. Case 4. Convention. Note shrinkage of the Q area after recovery.](image-url)
taken on the first 2 days. As yet there were no QRS changes. Another multiple-lead electrocardiogram taken 3 months after discharge showed complete disappearance to marked reduction in size of the Q waves in most of the leads from the high sternal area, return of the R waves, and also regression of the ST elevation (fig. 4).

Comment

The presence of myocardial injury could have been suspected from the conventional leads; however, a diagnosis of infarction could not have been made owing to failure of these leads to show QRS changes. The changes in the high sternal leads supported the diagnosis of myocardial infarction that was already suggested by the clinical picture.

Case 5. A 72-year-old man was admitted to the hospital on April 3, 1956, following a prolonged attack of precordial pain requiring opiates for relief. The blood pressure was 140/90 and pulse rate 65 and regular. The temperature was normal and remained so after admission. The laboratory tests were within normal limits. The chest x-ray revealed moderate left ventricular hypertrophy. The multiple-lead record taken on the second hospital day showed left axis deviation and T-wave inversion over the left anterolateral chest wall consistent with left ventricular hypertrophy. In the high sternal area, however, there was up to 2 mm. ST elevation followed by terminal T-wave inversion. In this same area extending from the midline to V5 in transverse direction and from the first to third intercostal space in vertical direction the R waves appeared reduced in size. This finding, coupled with ST elevation, was considered to be evidence of myocardial infarction and the patient was treated accordingly.

Subsequent multiple-lead tracings taken on April 6, 10, 13, 16, and 20 showed gradual return of the R waves to normal size. Q waves did not appear at any time. The ST elevation receded only partially and the T waves remained unchanged. The standard limb and precordial leads failed to disclose these evolutionary changes.

Comment

In this case of clinically mild acute myocardial infarction, the standard limb and chest leads failed to show acute changes. Leads taken from the high sternal area, however, displayed evolutionary changes of the QRS and ST that, in the light of the clinical picture, were thought to be diagnostic of acute myocardial infarction. Noteworthy is the fact that QRS alterations of infarction in the high sternal leads were confined to a reduction in amplitude of the R wave without Q waves appearing at any time. Reduction of R amplitude, occurring during the acute stage of a coronary attack, therefore may have a similar diagnostic significance to that of infarction Q wave.

Case 6. A 43-year-old man was admitted to the hospital on May 17, 1955, for cardiopericardioscopy for treatment of persistent angina pectoris. He had a classical acute myocardial infarction on February 2, 1955. After discharge from the hospital he was totally incapacitated with recurrent severe anginal pain both on exertion and at rest. Physical examination revealed a blood pressure of 110/80; pulse rate of 76 per minute. Examination of the heart was not remarkable, as was the remainder of the physical examination. The serologic test was 4+ positive and the patient gave a history of syphilis.

The tracings obtained after the acute attack of February, 1955, were identical with the previous one and showed normal limb leads except for an equivocal Q wave with T-wave inversion in aV6. There were QS waves in V1, QR waves in V6, and normal QRS complexes in V3 to V6; the ST segments in V1 to V4 were persistently elevated and the T waves were within normal limits. No definitive diagnosis of myocardial infarction could be made on the basis of these conventional leads. Q waves in V6 and V5 were of uncertain significance. A multiple-lead electrocardiogram taken on June 24, 1955, revealed a very large Q area starting around the right shoulder but extending for a considerable distance beyond its normal anterior limits and reaching the left shoulder in the transverse direction and the fourth intercostal space in the vertical direction. Small r waves, normally encountered in right chest leads, were absent.

This Q area was decidedly too large to be considered normal (fig. 5, Left). It could not be explained on the basis of rotation of the heart, for there was no indication of any positional changes. The S-T segments within this Q area were sharply inverted. These changes were most pronounced in high sternal leads. A diagnosis of large high anterior infarct extending over the right ventricle was made.

We had the good fortune of studying this case under direct vision after the chest was opened for the cardiac operation. After the pericardium was opened, the epicardial surface of the heart was inspected. Patchy wrinkled scars were found over the basal third of the anterior septal area and the anterior wall of the right ventricle and the basal one third to one half of the anterior surface of the left ventricle.

Direct electrocardiograms taken from the epicardial surface of the heart revealed dis-
tinctly abnormal QRS complexes in the scarred areas consisting of pure QS, QRS, and rSr' type complexes (fig. 5 Right). These changes were most marked over the right ventricle and the septal area. Normally, the anterior surface of the right ventricle, the septal area, and the juxta-ventricular area of the left ventricle exhibit rS or RS type of depolarization complexes. The presence of scars in these areas left little doubt concerning the significance of the QRS changes.

Comment

The Q area in this case is perhaps too large to permit its inclusion in the high anterior infarct group. Actually leads V1 and V2 did show QRS changes of infarction. However, the main area of infarction Q waves was situated above the level of V1 and V2. This case is interesting in that it demonstrates the good correlation between the Q area on the body surface and the location of infarction in the heart.* Assuming that a similar correlation existed in other patients of this group, it would be permissible to place the high anterior infarct in the basal anterior portion of the septal area and the juxta-ventricular area of the ventricles.

* We wish to express our thanks to Dr. Alfred Goldman of the City of Hope Hospital, Duarte, California, for permission to take these direct leads.

DISCUSSION

With the exception of case 6, the cases described are examples of myocardial infarction of mild to moderate severity. The clinical picture in all these patients was definitely suggestive of myocardial infarction. However, a definitive electrocardiographic diagnosis could not be made from the conventional limb and chest leads because the interpretation in these cases might have been “myocardial ischemia,” “coronary insufficiency,” or “non-specific changes.” The leads from the high sternal area substantiated the diagnosis of infarction on the basis of the following: (a) a Q area situated on and to the sides of the upper sternal region; (b) the dynamic serial changes of the QRS complexes within the Q area, including the gradual decrease in the size of the Q area associated with recovery; (c) the coexistence of the characteristic ST elevation and T-wave changes within and beyond the Q area and their return to normal after recovery; and finally (d), absence of factors such as marked positional changes or acute right ventricular dilatation to account for the QRS alterations. In cases 3 and 5 a reduction in the size of the R waves in the high sternal area made diagnosis of myocardial infarction possible. In our experience a significant loss of R potential in association with ST elevation and
T-wave inversion has proved to be of diagnostic implication. This is particularly true if reduction in R amplitude is transient, appearing during the acute phase and returning to normal in the recovery period. The significance of the reduction of the R wave in myocardial infarction has been demonstrated in experimental animals in this laboratory.  

What separates the high anterior from other varieties of anterior wall infarction, i.e., massive anterior, antero-apical, anterolateral, high anterolateral and, finally, anteroseptal (often referred to as supra-apical in the European literature), is the location and extent of the Q area, which to a great extent depend on the topography and size of infarction of the anterior wall. For example, the Q area of the massive anterior infarction is exceedingly large, extending from the right sternal border to left anterior axillary line and covering all the first 5 to 6 intercostal spaces. The Q area of anterolateral infarcts is large and extends laterally to the mid- and even posterior axillary line and creates typical changes in left arm leads I and aV_L. The Q area of anteroseptal infarcts forms a vertical band on and to the left of the sternum and extends above and below the level of the standard V_1 and V_2 leads. In all these varieties of anterior infarction 1 or more of the conventional 12 leads fall within the Q area, hence classical changes of infarction appear in the standard electrocardiogram. In the so-called “supra-apical” infarction described by Holzman and Mussafia, the conventional leads V_1, V_3, and V_5 show classical changes while the complexes of the limb leads remain normal. Electrocardiographically then, this type of infarction is very similar, if not analogous, to the anteroseptal infarction. However, it is entirely possible that the “supra-apical” variety would include a proportion of the presently described high anterior infarction.

The shape of the Q varies greatly from one case to another. In 1 case of acute anterior myocardial infarction with typical QRS, ST and T changes in leads V_2, V_5, V_4, and V_6, the multiple-lead records showed the Q area to be in the form of a transverse belt extending from the midline to left anterior axillary line in the third, fourth, and fifth interspaces. The term “intermediate anterior infarction” would be applicable to this type. Other Q areas exhibited triangular forms with the apex in the region of V_3 or V_4 and the base situated in the epigastrium or up in the infraclavicular area. The Q area of infarction does not follow any particular pattern, since the myocardial lesion itself does not. The marked individual variations in the distribution and arborization of the coronary arteries on the one hand, and alterations in the relationship of the arterial branches to various segments of the myocardium as a result of pre-existing narrowings and occlusions (so commonly found in patients with coronary arteriosclerosis) on the other, adequately explain the variability of infarction Q areas. It is important therefore to conceive of the Q area spatially and bear in mind that it can occupy any area of the body surface and be of any size and shape. This fact can be appreciated best by study of the multiple-lead records. The standard leads showing infarction Q waves, however, represent only a few selected points from the Q area. This approach is not unlike viewing an object through a few openings in its casing—an adequate knowledge of the physical characteristics of the object cannot be secured in this way.

The Q area of high anterior infarction is unique in that it is usually small and is so situated that it avoids all the standard electrode locations; hence it inscribes no Q waves in any of the conventional leads. The areas of ST elevation and T-wave inversion are usually larger that the Q area, so that in the acute stage of high anterior infarction, ST elevation and T-wave inversion may be seen in the conventional chest leads and lead I and aV_L. On the other hand, ST elevation and T-wave inversion may be found in some of the precordial and limb leads of patients exhibiting symptoms of acute myocardial infarction in whom no Q area is to be found on the body surface. We have seen 1 such case in a 46-year-old man with clinically indisputable evidence of myocardial infarction. The precordial leads V_2 to V_5 exhibited upwardly convex ST elevation of 2 to 3 mm. in amplitude and terminal inversion of the T-waves. Similar but less pronounced changes were observed in aV_L. No Q waves of infarction were found in the high sternal area or any other part of the body.
Another tracing taken 3 weeks after the acute attack showed complete disappearance of the ST and T changes. Comparison of the 2 records revealed that the R waves in the high sternal area had lost considerable amplitude during the acute stage and returned to normal after recovery. For example, R waves in leads taken from the midline at the level of the first intercostal space and from V2 at the third intercostal space were 1.5 mm. and 5 mm. in amplitude respectively; after recovery, their respective amplitudes had risen to 2.5 and 12 mm. It will be clear, from the consideration of this case and of cases 2 and 5, that reduction in the size of the R waves within the area of ST elevation and T-wave inversion may be of great diagnostic significance. It has been found in dogs with experimentally produced myocardial infarction that the small r wave results from a mixture of live and dead epicardial muscle. As the infarct "heals," the number of cells of the outer myocardial layers recover and add to the height of the R wave.

A few words may be said about the retrospective diagnosis of old high anterior myocardial infarction. In our experience the electrocardiogram rapidly reverted to normal in all cases except case 6 in which a large infarction was found at operation. It may be assumed therefore that small infarcts of high anterior variety usually leave little or no residual electrocardiographic changes and hence are difficult to diagnose in retrospect. An interesting observation, however, is that, not infrequently the Q or QS waves of infarction present in the conventional precordial leads of acute anteroseptal and anterior infarcts disappear completely during the recovery period. The conventional chest leads then may appear normal. High sternal leads in such cases may disclose a Q area obviously residual to the original larger Q area. In fact, Benckimol, Schlesinger, and Cotrim in 1947 and Alzamora and Mispireta in 1948 reported on the usefulness of high chest leads in uncovering residual QRS changes of old anterior myocardial infarcts. We have observed 2 similar cases, 1 of which showed classical acute anterior myocardial infarction with Q waves in leads V2-6 in March 1954. A multiple-lead record taken in 1956 shows practically normal QRS complexes in leads V2-6 and a distinct Q area confined to the high sternal region. In the second case the multiple-lead record showed a relatively large Q area in the high sternal region. The standard leads V1 to V2 show only small r waves probably due to close proximity to the Q area. This Q area is thought to be residual of a larger Q area of a preexisting anterior infarction.

In regard to the anatomic location in the various types of anterior infarction, one may speculate that there is a fair topographic and quantitative correlation between the Q areas on the body surface and on the infarcted wall of the myocardium. All things being equal, larger Q area probably represent larger infarcts; and a Q area high in the chest corresponds to an infarction high (basal) in the heart; a Q area toward the midline would be a reflection of an infarct near or in the septal area; an "intermediate" Q area placed transversely across anterior chest wall represents an infarction of the anterior wall intermediate between base and apex, and so on. Accordingly, therefore, the type of lesion responsible for the electrocardiographic findings in this paper would be a small infarction located anteriorly, near the septum, and adjacent to the A-V groove. Occlusion of a small branch of the anterior descending coronary artery near its origin is most probably the cause. Although the assumption on which these deductions are based has not been confirmed, the available evidence and our own experience (case 6) give validity to such an assumption.

Clinically, the high anterior infarction runs a benign course. Those clinical features that denote a grave prognosis, namely, fever, congestive heart failure, shock, and protracted pain, have been conspicuously absent in our cases. The benignity of high anterior infarction might explain the lack of postmortem information concerning its characteristics.

The incidence of high anterior infarction cannot be inferred from the available electrocardiographic and postmortem correlation in the literature for the selection of cases in such studies would tend to exclude mild types of infarct that do not cause immediate death. Nor would it be fruitful to go over the files of hospital electrocardiograms, as here, too, high anterior infarcts would not be represented for
the same reasons. On the basis of our own experience, however, which is decidedly too limited to allow any definitive conclusions, 6 cases of high anterior infarction out of 66 electrocardiographically proved instances would suggest an incidence as high as 10 per cent of all infarcts. The occurrence of high anterior infarction may be of sufficient frequency to warrant a systematic search for it by taking additional high anterior chest leads in selected cases.

It would not be feasible to subject all patients to the multiple-lead exploration used in this study. As a routine, however, one may attempt to identify a limited number of high sternal leads in which an initial R wave is present in almost 100 per cent of normal subjects (Appendix). Then QR or QS waves or marked reduction of the R wave in such leads may signify high anterior infarction. On the basis of the study of 106 normal subjects, it seems sufficient for diagnostic purposes to record a set of only 2 high sternal leads: V₂ at the level of the second intercostal space (V₂, 2 i.c.s.) and midline lead at the level of the third intercostal space (Mid, 3 i.c.s.). These 2 leads show an R wave in nearly 100 per cent of normal subjects, irrespective of the electric position of the heart. Like any other electrocardiographic item, the diagnostic value of a QR or QS in the above 2 leads must depend on the clinical picture and the presence or absence of other situations causing marked changes in the electric axis of the heart (right ventricular hypertrophy or dilatation, right bundle-branch block, left ventricular hypertrophy, left bundle-branch block). Elevation of the ST segments in these 2 leads were observed in all our cases of acute high anterior and other types of anterior infarct reaching the second and third intercostal spaces. Nevertheless this electrocardiographic item cannot be relied upon, for unless it is marked (Appendix), moderate ST elevation may be observed in these 2 leads in normal individuals. For a similar reason, T-wave inversion in these leads is not of diagnostic significance.

These 2 leads are useful, not only in detecting acute high anterior infarcts, but also in retrospective search for old anteroseptal and old anterior infarcts in which the Q area has treated to the high sternal region. Furthermore, it is suggested that a systematic recording of the leads V₂, 2 i.c.s., and Midline, 3 i.c.s., would allow the separation in symptomatic patients of high anterior infarction from other syndromes caused by various types of myocardial ischemia, such as angina pectoris and "coronary insufficiency," etc.

A question of general interest concerns the possible usefulness of the vectorcardiogram in the diagnosis of high anterior infarction. We have no vectorcardiograms taken during the acute stage, therefore whatever is said on the subject is purely speculative. Vectorcardiograms taken in 2 cases (case 1 and case 4) after the return of the electrocardiogram to normal were within normal limits. It is believed, on theoretical grounds alone, that vectorcardiography is unlikely to be of diagnostic assistance in this type of infarction. That portion of the QRS loop likely to show abnormality in this type of infarction would be the initial one, which is normally directed anteriorly and to the right and, depending on the electric position of the heart, may point superiorly or inferiorly. This portion of the loop is short in duration, 0.01 to 0.02 second; in still vectorcardiograms it is often buried, at least partially, in the zero point. The direction and magnitude of this portion cannot be accurately studied unless the vectors are inscribed on the moving film. In any event, total disappearance of this portion of the loop would not cause the loop to depart significantly from the normal. This may be one of the situations in which standard electrocardiograms may be superior to the vectorcardiograms but this problem needs complete study.

An observation of some interest made during the serial study of multiple-lead electrocardiograms in myocardial infarction was the frequency with which QRS changes of infarction were found to fluctuate within a few days. The Q areas were observed to be dynamic, enlarging in some cases and diminishing in others without necessary correlation with the clinical picture. Reduction in the size of the Q area during the first few days following the acute attack was a particularly common occurrence. Obviously shrinkage and scarring of the infarcted myocardium would not be an acceptable explana-
tion, for such changes cannot occur within a few days. Instead, it would seem more logical to describe the fleeting Q waves to a state of "electrophysiologic necrosis," resulting from severe ischemic injury rather than to histologic necrosis. Since the R wave returns, the QS waves are obviously mural type QS waves as the myocardium contains viable myocardium.13

SUMMARY

Multiple-lead electrocardiographic exploration was carried out in 149 patients with various manifestations of coronary artery disease. Of these, 66 had myocardial infarction.

Six cases of myocardial infarction in which infarction QRS changes were absent in the conventional limb and precordial leads but were present in leads taken from the high sternal region are reported. The name "high anterior infarction" is suggested for this type so that it would be distinguished from other varieties of anterior infarction. Only some type of "myocardial ischemia" would have been recognized in routine electrocardiograms.

The diagnosis of infarction in our cases was based primarily on the characteristic clinical picture and the evolutionary changes in the QRS complexes and in S-T and T of the high sternal leads. The electrocardiograms taken from this region during the acute stage displayed QS waves, S-T elevation, and T-wave inversion. After recovery, the R waves, S-T and T reverted to normal. All these patients recovered, thus no pathologic correlation with the electrocardiographic findings is yet available. Prognosis appears to be very good.

Of interest was the observation that the QRS changes of infarction were not permanent. On the contrary, the fluctuations in the size and location of the Q area pointed to a dynamic rather than static nature of the Q waves. It was suggested that such Q waves represented mural type QS waves.

The conventional precordial leads and aVL taken during the acute stage of high anterior infarction showed only S-T and T-wave changes. It is suggested therefore that the high sternal leads be taken in patients exhibiting symptoms of acute myocardial infarction and isolated S-T and T changes consistent with such a diagnosis in precordial and aVL leads.

This practice may be warranted, for in our small series of 66 infarction cases, 6 instances of high anterior infarct were found.

On the basis of our study of the normal electrocardiograms taken from the high sternal area and the constancy with which an initial r wave occurs, the following 2 leads are suggested as the minimum sufficient number of leads to be taken when high anterior infarction is suspected: V2 at the levels of the second, and midline at the third intercostal spaces. The incidence of an R in these leads is nearly 100 per cent, hence, a Q or QS in these leads in the presence of suggestive clinical symptoms would substantiate the diagnosis of acute myocardial infarction. These leads may show residual QRS changes in healed anterior and anteroseptal infarcts while the standard chest leads have returned to normal.

Vectorcardiography is believed to be inferior to electrocardiography in detecting the high anterior infarction, but more study is necessary.

The systematic use of the 2 high sternal leads may be advisable in selected patients with various manifestations of coronary artery disease. This practice would serve to differentiate between cases with infarction and without infarction in otherwise undiagnosable instances.

Clinical evidence of the "mural" QS wave and the abnormally small R wave in coronary artery disease has been obtained.

SUMMARIO IN INTERLINGUA

Exploraciones electrocardiographic a derivationes multiple eseva execute in 149 patientes con varie manifestationes de morbo de arteria coronari. Inter illes, 66 habeva infarcimento myocardial.

Es reportate sex casos de infarcimento myocardial in que alterationes infarcimental de QRS eseva absente in le derivationes conventional de extremitate e precordio sed presente in derivationes ab le region supero-sternal. Le termino "infarimento ali-anterior" es proponite pro iste typo in distinction ab altere varietates de infarcimento anterior. In le casos mentionate, le electrocardiographia routinari haberea revelate non plus que un typo de "ischemia myocardial."

Le diagnose de infarcimento in nostre casos
esse basate primarmente super le typic tableau clinice e le alteraciones evolutionari del complexos QRS e de S-T e T in le derivationes supero-sternal. Le electrocardiogrammas derivate ab iste region durante le stadio acute exhibiva undas QS, elevation de S-T, e inversion del unda T. Post recuperation del pacientes, le undas R e S-T e T retornava a configurationes normal. Omne iste pacientes recuperava. Per consequente nulle correlacion pathologic con le constatationes electrocardiographic pote esser estabili a iste tempore. Le prognose es apparentemente multo bon.

Un observation de interesse esseva que le alteraciones de QRS in infarcimento non esseva permanente. Al contrario, le fluctuaciones del dimensiones e del location del area Q signalava un natura dynamic plus tosto che static del undas Q. Se suggereva le notion che tal undas Q representava un typo mural de unda QS.

Le conventional derivationes precordial e le aV1 registrate durante le stadio acute del infarcimento alti-anterior exhibiva alteraciones solmente de S-T e de unda T. Per consequente il es a recommendar che le derivationes supero-sternal es usate in patientes qui manifesta symptomas de acute infarcimento myocardial e isolate alteraciones de S-T e T de character compatible con ille diagnose in derivationes precordial e aV1. Iste practica pare justificate, viste le facto che in nostre serie de non plus che 66 casos de infarcimento nos ha trovate 6 occurrentias de infarcimento alti-anterior.

Super le base de nostre studio del normal electrocardiogrammas derivate ab le area supero-sternal e viste le regularitate del occurrentia de un unda r initial, le sequente duo derivationes es recommendate como le minimo sufficiente in casos de suspiccion de infarcimento alti-anterior: V2 al nivello del secunde spatio intercostal e derivation de linea intermediari al tertie spatio intercostal. Le incidentia de R in iste derivationes es quasi 100 pro cento. Per consequente, un Q o QS in illos in le presentia de suspect symptomas clinice pote corroborar le diagnose de acute infarcimento myocardial. Iste derivationes pote manifestar residuos de alteraciones de QRS in curate infarcimentos anterior e anteroseptal post que le derivationes thoracic standard ha retornate a configurationes normal.

Nos opina que vectorcardiographia es inferior a electrocardiographia in le detection de infarcimento alti-anterior, sed studios additional es necessari.

Le uso systematic del 2 derivationes supero-sternal es a recommendar in seligite patientes con varie manifestationes de morbo de arteria coronari. Iste practica servirea a differentiar inter casos con e sin infarcimento in situationes che escappa a altere mesuras diagnostic.

Manifestationes clinic del "mural" unda QS e del anormalmente reduce te unda R in morbo de arteria coronari ha esseite determinate.

APPENDIX

By Rashid A. Massumi, M.D.

In order to establish standards for multiple-lead electrocardiograms, 106 normotensive (blood pressure 140/90 or below) subjects without history or evidence of heart disease were studied. The electric position of the heart was classified as vertical, intermediate, or horizontal; the so-called semivertical and semihorizontal positions having been grouped together with the vertical and horizontal respectively. Table 1 represents the distribution with reference to age, sex, and blood pressure. The absence of subjects of third and fourth decades in the horizontal group is in keeping with the tendency of the electric position of the heart to shift from vertical to horizontal with advance of age. Selection of the subjects was made at random in every respect except age: an attempt was made to use as many individuals in the coronary age group (40 years and over) as possible. In the following section only those electrocardiographic items pertinent to the discussion of the high anterior infarction will be considered.

QRS Complex. The QRS complexes in the high sternal area were predominately of the rS or rSr' type in V1, midline and V2 positions and of the RS, Rs or qRs type in V3 and V4 positions. The transition between the rS complexes (sum negative) in the former and RS or Rs complexes (sum positive) in the latter group of leads occurred at different positions being generally farther to the left in the first than in the third intercostal space. Indeed, the position of this transition zone depended to a great extent on the electric position of the mean QRS axis being lower and more to the left in vertical hearts than in horizontal hearts. Small q waves normally seen in leads taken from the left lateral chest wall occurred in a few of the V2 and V4 leads. They did not exceed 1 to 2 mm. in amplitude and 0.01 to 0.02 second in duration.

The depolarization complexes in the high sternal area exhibited an initial r or R in a large majority of the subjects. Special attention was given to the initial portion of the QRS complex due to its great
importance of myocardial infarction. Q waves were uncommon in the high anterior leads occurring in the entire population of our sample (106 cases) with the following per cent frequencies: in the first intercostal space, V1, 8.8; Midline, 10.3; V2, 1.9; V3, 0.9; V4, 0; in the second intercostal space, V1, 4.3; Midline, 1.9; V3, 0.9; V5, 0; V6, 0; in the third intercostal space, V1 to V4, 0. Thus the only leads that show initial Q with a significant frequency are V1 and midline in the first intercostal space and V1 in the second intercostal space. Lead V2 in the second intercostal space displayed a Q wave in only 1 case out of 106 (0.9 per cent). Leads V3 and V4 of the same space, and all the 5 leads in the third space showed no Q waves; the depolarization complexes began with an initial positivity. A breakdown of these frequencies according to the electric position of the heart may be found in tables 2, 3, and 4.

Statistical analysis of the data revealed no significant differences in the frequencies of the occurrence of Q waves when corresponding leads in the 3 groups of hearts were compared. Therefore, with certain reservations owing to the small size of our samples, the presence or absence of a Q in high sternal leads does not seem to be a function of the electric position of the heart. We are not in a position to speculate on the possible explanations for the variations in the initial portion of the QRS complexes. Attempts to correlate the distribution of the initial Q with age and body habitus failed to provide any clue.

The amplitude and duration of the initial r waves of the high sternal leads were found to vary markedly. Tables 2, 3, and 4 depict the mean, standard deviation, the mode (that value occurring most frequently), and the range for the amplitude and durations. It may be noted, from the relatively larger standard deviations and ranges for the amplitudes than for the durations, that the former display greater scatter than the latter. This is consistent with the greater variations seen in the amplitude of the entire QRS complexes than in their duration. Noteworthy in these tables is that intergroup variations of the amplitudes and durations are negligible. Stated differently, the amplitudes and durations of the initial r waves in the high sternal leads (all 15 leads taken together) do not appear to correlate with the electric position of the hearts.

A study of the direction of the initial component of the QRS complex in the high sternal area and the adjacent right shoulder reveals that the large Q waves seen occasionally in the V1 and midline leads of the first 2 intercostal spaces represent an anterior medial extension of the normal Q area of the right upper back and right shoulder.1,2,14,15 This fact would suggest a clockwise rotation of the heart—anatomic or electric—around its longitudinal axis. While such rotation was present in a few cases showing Q areas in the above-named leads, it was not evident in the majority. A point of some interest is that the zone of transition between the area of initial negativity and that of initial positivity in the anterior right upper chest is often wide open. The initial negative component becomes isoelectric in this transition zone. A QRS complex with an isoelectric initial component would appear, at a cursory glance, to be short in duration, for a portion of it is consumed by the isoelectric initial portion. Such “isolectric r waves” can be recognized by recording a simultaneous lead with the various chest leads and use it as the time constant against which to time the initial portion of the QRS in chest leads.18

The incidence of r' in the high anterior leads and particularly those from V1, midline and V2 in the first and second spaces was unexpectedly great (tables 2, 3, and 4). This electrocardiographic item, too, seemed to be unrelated to the position of the mean electric axis of the heart.

ST Segments. The ST segments in the high anterior leads often showed a suggestion of elevation and on occasion as much as 1.5-mm. elevation. Similar but less pronounced elevation of the ST segments can be seen in the conventional precordial leads V1 to V4 in a small proportion of normal individuals. No instance of depressed ST segment was seen in any of the high sternal leads.

T Waves. The T waves were invariably negative in V1 position of first intercostal space and invariably positive in V4 positions of all 3 intercostal spaces. The intervening leads showed transitions between the 2 extremes (table 5).

It will be evident from this brief discussion and study of tables 2 to 5 that the depolarization complexes display initial positivity in an overwhelming majority of the normal subjects. This r wave is small in leads from the right side of the high sternal area (generally 1 to 3 mm. in amplitude and .01 to .02 second in duration) but it gains both in ampli-

---

**Table 1.—Variation of Cardiac Position with Age, Sex, and Blood Pressure**

<table>
<thead>
<tr>
<th>Decade distribution</th>
<th>Range</th>
<th>Sex</th>
<th>Blood pressure</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>Vertical</td>
<td>41.2</td>
<td>4</td>
<td>13</td>
<td>20</td>
</tr>
<tr>
<td>Intermediate</td>
<td>48.3</td>
<td>3</td>
<td>3</td>
<td>22</td>
</tr>
<tr>
<td>Horizontal</td>
<td>48.3</td>
<td>0</td>
<td>0</td>
<td>11</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

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Table 2.—The r Wave in Vertical Hearts

<table>
<thead>
<tr>
<th>Vertical hearts</th>
<th>1st I.C.S.</th>
<th>2nd I.C.S.</th>
<th>3rd I.C.S.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>V₁</td>
<td>V₂</td>
<td>V₃</td>
</tr>
<tr>
<td>r present</td>
<td>42</td>
<td>41</td>
<td>42</td>
</tr>
<tr>
<td>r' present</td>
<td>21</td>
<td>19</td>
<td>18</td>
</tr>
</tbody>
</table>

Duration of r wave in 0.01 sec.

| Mean 1.6  | 1.6  | 1.8  | 2.2  | 2.4  | 1.7  | 2.0  | 2.3  | 2.6  | 3.1  | 2.0  | 2.2  | 2.7  | 3.2  | 3.8  |
| S.D.± 0.6 | 0.5  | 0.7  | 0.6  | 0.9  | 0.6  | 0.7  | 0.6  | 0.7  | 0.8  | 0.8  | 0.7  | 0.8  | 0.8  | 0.8  |
| Mode 2.0  | 2.0  | 2.0  | 2.0  | 2.0  | 2.0  | 2.0  | 2.0  | 3.0  | 3.0  | 2.0  | 2.0  | 3.0  | 3.0  | 4.0  |
| Range 0.0  | 0.0  | 0.0  | 0.0  | 1.0  | 0.0  | 1.0  | 1.0  | 1.0  | 2.0  | 0.0  | 1.0  | 1.0  | 1.0  | 2.0  |
|           3.0 | 3.0  | 4.0  | 4.0  | 5.0  | 3.0  | 3.0  | 4.0  | 4.0  | 5.0  | 3.0  | 3.0  | 4.0  | 5.0  | 5.0  |

Amplitude of r wave in 1 mm.

| Mean 1.5  | 1.8  | 1.8  | 2.2  | 1.9  | 2.1  | 2.7  | 3.2  | 3.3  | 3.3  | 2.4  | 3.5  | 4.6  | 5.0  | 6.0  |
| S.D.± 0.80 | 0.97 | 0.78 | 0.88 | 0.68 | 1.1 | 1.5 | 1.0  | 1.2  | 1.2  | 1.2  | 1.9 | 2.2  | 1.8  | 2.8  |
| Mode 1.5  | 2    | 2    | 2    | 2    | 1    | 3    | 3    | 3    | 3    | 3    | 6    | 5    | 5    |
| Range 0.0  | 0.0  | 0.0  | 0.0  | 0.5  | 0.0  | 0.5  | 0.5  | 1.0  | 1.0  | 1.0  | 1.0  | 1.0  | 1.0  | 2.0  |
|           4.0 | 4.0  | 4.5  | 4.0  | 5.0  | 7.0  | 7.0  | 6.0  | 6.0  | 8.0  | 10.0 | 11.0 | 9.0  | 13.0 |

Table 3.—The r Wave in Intermediate Hearts

<table>
<thead>
<tr>
<th>Intermediate hearts</th>
<th>1st I.C.S.</th>
<th>2nd I.C.S.</th>
<th>3rd I.C.S.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>V₁</td>
<td>V₂</td>
<td>V₃</td>
</tr>
<tr>
<td>r present</td>
<td>39</td>
<td>40</td>
<td>43</td>
</tr>
<tr>
<td>r' present</td>
<td>16</td>
<td>20</td>
<td>21</td>
</tr>
</tbody>
</table>

Duration of r wave in 0.01 sec.

| Mean 1.4  | 1.5  | 1.8  | 2.1  | 2.8  | 1.8  | 2.2  | 2.4  | 2.9  | 3.6  | 2.1  | 2.5  | 2.9  | 3.7  | 3.9  |
| S.D.± 0.7  | 0.8  | 0.8  | 0.8  | 1.2  | 0.6  | 0.6  | 0.7  | 1.2  | 0.9  | 1.0  | 0.7  | 1.1  | 0.9  | 0.8  |
| Mode 1.0  | 1.0  | 2.0  | 2.0  | 3.0  | 2.0  | 2.0  | 3.0  | 2.0  | 3.0  | 2.0  | 3.0  | 3.0  | 4.0  | 4.0  |
| Range 1.0  | 0.0  | 0.0  | 1.0  | 1.0  | 1.0  | 1.0  | 0.0  | 1.0  | 2.0  | 1.0  | 1.0  | 1.0  | 2.0  | 2.0  |
|           3.0 | 0.3  | 5.0  | 6.0  | 3.0  | 4.0  | 4.0  | 4.0  | 6.0  | 6.0  | 4.0  | 4.0  | 6.0  | 6.0  | 5.0  |

Amplitude of r wave in 1 mm.

| Mean 1.3  | 1.5  | 1.8  | 2.1  | 3.4  | 1.4  | 2.3  | 2.9  | 3.7  | 4.8  | 2.0  | 3.1  | 4.9  | 7.3  | 9.4  |
| S.D.± 0.65 | 0.61 | 1.3  | 1.3  | 2.0  | 1.4  | 2.3  | 2.9  | 3.7  | 4.8  | 2.0  | 3.1  | 4.9  | 7.3  | 9.4  |
| Mode 1.0  | 1.5  | 1.0  | 2.0  | 2.0  | 4.2  | 1.6  | 2.1  | 2.2  | 2.6  | 0.8  | 1.9  | 2.1  | 1.1  | 4.0  |
| Range 0.0  | 0.0  | 0.0  | 0.5  | 0.5  | 0.5  | 0.5  | 0.5  | 1.0  | 2.0  | 1.0  | 1.0  | 2.0  | 2.0  | 3.0  |
|           2.0 | 4.0  | 9.0  | 11.0 | 3.0  | 11.0 | 14.0 | 13.0 | 14.0 | 4.0  | 9.0  | 15.0 | 15.0 | 20.0 |

titude and duration as one proceeds toward the left side of the high sternal area (up to 16 mm. amplitude and up to .06 second duration).

The presence of a normal r in high sternal leads is consistent with vectorcardiographic findings in normal subjects. Fowler and Helm studied the initial portion of the vector loop in 18 normal subjects and found it to be directed rightward and anteriorly in 15, leftward and anteriorly in 12, and straight anteriorly in 1. Indeed, all such orientation of the initial vector would give rise to an initial r in high sternal leads. The observations of Grant and Murray,² Peñaloza and Tranchesi and Wolff, Richmann, and Soffen also indicate that the initial portion of the loop in the majority of normal individuals is expected to point superiorly, anteriorly, and to the right, hence an initial positivity in the QRS complexes recorded from the high sternal area.

Although the general direction of the initial vector in our normal subjects seemed to be superior and anterior, interindividual variations as to the direction in the frontal plane appeared very great. The line of demarcation between the initial Q and initial r varied tremendously from one group to another.
and even in the individuals of the same group. The overlap was so great as to make estimation of the position of this line on the basis of the main vector impossible. This may be due to individual variations in the activation of the septum and/or other structures responsible for the genesis of the initial depolarization forces such as the crista supraventricularis. Such variations have been found in intracavity leads of normal individuals as shown by Levine and co-workers\textsuperscript{18} and Kossman and his associates.\textsuperscript{19}

\textbf{Conclusion.} This study of the normal electrocardiogram of the high anterior area indicates that the presence of an r in these leads taken as a group is very common; a Q wave in the V\textsubscript{1} and midline leads occurs with sufficient frequency to make it useless as a diagnostic item for classical usage; a Q wave in V\textsubscript{2}, V\textsubscript{3}, and V\textsubscript{4} leads is seen very uncommonly, hence of diagnostic significance. The 2 positions pointed out previously, namely V\textsubscript{2}, 2 i.c.s. and midline, 3, i.c.s., show initial positivity in almost 100 per cent of the normal individuals.

Owing to the marked overlapping of the r and Q area of the anterior leads in normal subjects, the finding of a Q wave in the 2 leads mentioned cannot be safely considered pathognomonic of infarction. Like any other electrocardiographic item, this must be interpreted in the light of the clinical condition of the patient. Right ventricular dilatation and hypertrophy, right bundle-branch block, left ventricular hypertrophy, and left bundle-branch block must be ruled out before determining the value of a Q in high anterior leads for such conditions can displace the normal Q area to the left and inferiorly.\textsuperscript{20-22} Likewise it is necessary to differentiate

\begin{table}[h]
\caption{The r Wave in Horizontal Hearts}
\begin{tabular}{|c|c|c|c|c|c|c|c|c|c|c|c|c|c|}
\hline
\textbf{Horizontal hearts} & \multicolumn{4}{|c|}{1st I.C.S.} & \multicolumn{4}{|c|}{2nd I.C.S.} & \multicolumn{4}{|c|}{3rd I.C.S.} \\
\textbf{(18 cases)} & V\textsubscript{1} & Mid & V\textsubscript{2} & V\textsubscript{4} & V\textsubscript{1} & Mid & V\textsubscript{2} & V\textsubscript{4} & V\textsubscript{1} & Mid & V\textsubscript{2} & V\textsubscript{4} \\
\hline
r present & 16 & 15 & 18 & 18 & 16 & 17 & 18 & 18 & 18 & 18 & 18 & 18 \\
r present & 10 & 10 & 8 & 7 & 4 & 2 & 3 & 0 & 1 & 0 & 0 & 0 \\
\hline
\end{tabular}
\end{table}

\begin{table}[h]
\caption{Direction of T Waves, per cent of the Group}
\begin{tabular}{|c|c|c|c|c|c|c|c|c|c|c|c|c|c|c|}
\hline
\textbf{Vertical} & \multicolumn{4}{|c|}{1st I.C.S.} & \multicolumn{4}{|c|}{2nd I.C.S.} & \multicolumn{4}{|c|}{3rd I.C.S.} \\
\hline
\textbf{V\textsubscript{1}} & Mid & V\textsubscript{2} & V\textsubscript{4} & V\textsubscript{1} & Mid & V\textsubscript{2} & V\textsubscript{4} & V\textsubscript{1} & Mid & V\textsubscript{2} & V\textsubscript{4} & V\textsubscript{1} & Mid & V\textsubscript{2} \\
\hline
Positive & 0.0 & 2.3 & 9.0 & 31.8 & 65.8 & 4.5 & 25.0 & 70.4 & 84.2 & 97.7 & 22.7 & 70.4 & 93.3 & 100 & 100 \\
Biphasic & 2.3 & 4.6 & 15.8 & 31.8 & 20.4 & 9.0 & 18.1 & 9.0 & 6.8 & 2.3 & 13.6 & 6.8 & 2.3 & 0.0 & 0.0 \\
Negative & 97.6 & 93.1 & 75.2 & 36.4 & 13.6 & 86.5 & 56.9 & 20.4 & 9.0 & 0.0 & 63.7 & 22.7 & 4.4 & 0.0 & 0.0 \\
\hline
\textbf{Intermediate} & \multicolumn{4}{|c|}{1st I.C.S.} & \multicolumn{4}{|c|}{2nd I.C.S.} & \multicolumn{4}{|c|}{3rd I.C.S.} \\
\hline
\textbf{V\textsubscript{1}} & Mid & V\textsubscript{2} & V\textsubscript{4} & V\textsubscript{1} & Mid & V\textsubscript{2} & V\textsubscript{4} & V\textsubscript{1} & Mid & V\textsubscript{2} & V\textsubscript{4} & V\textsubscript{1} & Mid & V\textsubscript{2} \\
\hline
Positive & 0.0 & 11.3 & 29.5 & 56.9 & 88.5 & 9.0 & 38.7 & 81.9 & 95.4 & 97.7 & 31.8 & 77.3 & 95.4 & 97.7 & 100 \\
Biphasic & 6.8 & 6.8 & 11.3 & 18.1 & 4.5 & 2.3 & 13.6 & 11.3 & 2.3 & 2.3 & 15.9 & 2.3 & 2.3 & 0.0 & 0.0 \\
Negative & 93.2 & 81.9 & 59.2 & 25.0 & 6.8 & 88.5 & 47.7 & 6.8 & 2.3 & 0.0 & 52.2 & 20.4 & 2.3 & 2.3 & 0.0 \\
\hline
\textbf{Horizontal} & \multicolumn{4}{|c|}{1st I.C.S.} & \multicolumn{4}{|c|}{2nd I.C.S.} & \multicolumn{4}{|c|}{3rd I.C.S.} \\
\hline
\textbf{V\textsubscript{1}} & Mid & V\textsubscript{2} & V\textsubscript{4} & V\textsubscript{1} & Mid & V\textsubscript{2} & V\textsubscript{4} & V\textsubscript{1} & Mid & V\textsubscript{2} & V\textsubscript{4} & V\textsubscript{1} & Mid & V\textsubscript{2} \\
\hline
Positive & 5.5 & 16.6 & 44.4 & 94.5 & 100 & 16.6 & 66.6 & 100 & 100 & 100 & 38.8 & 88.8 & 100 & 100 & 100 \\
Biphasic & 0.0 & 11.1 & 22.2 & 0.0 & 0.0 & 11.1 & 22.2 & 0.0 & 0.0 & 0.0 & 16.6 & 0.0 & 0.0 & 0.0 & 0.0 \\
Negative & 94.5 & 72.3 & 33.3 & 5.5 & 0.0 & 72.3 & 11.1 & 0.0 & 0.0 & 0.0 & 44.4 & 11.1 & 0.0 & 0.0 & 0.0 \\
\hline
\end{tabular}
\end{table}
between a true Q and a “pseudo-Q” caused by isoelectricity of the r wave.

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High Anterior Myocardial Infarction: XX. Studies on the Mechanism of Ventricular Activity
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