Electrocardiographic Changes in a Case of Left Ventricular and Septal Hypertrophy Resembling Anterior Myocardial Infarction

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In certain instances of left ventricular hypertrophy, deep Q waves observed in the precordial electrocardiogram may resemble those of anterior myocardial infarction. Such a case is reported in a patient with syphilitic aortic insufficiency. Pathologic examination ruled out the diagnosis of infarction and revealed marked hypertrophy of left ventricular wall and the interventricular septum. The latter was of unusual degree and was probably responsible for the presence of large Q waves in precordial leads representing, the normal activation of the upper part of the interventricular septum which in cases of septal hypertrophy presumably originates a vector of greater magnitude.

The presence of essentially negative QRS complexes displaying a QS configuration in right precordial unipolar leads has been observed in patients with uncomplicated left ventricular hypertrophy; the absence of an initial R wave probably indicates a neutralization of the normal positivity in these leads by contralateral potential variations of greater magnitude owing to activation of the free wall of the enlarged left ventricle.

These changes are frequently indistinguishable from those due to a healed anteroseptal infarction, particularly in the absence of a positive clinical history or of serial tracings showing the characteristic RS-T and T-wave changes during the acute and subacute stages of coronary occlusion. In leads from the left side of the precordium, Q waves are usually recorded and are due to early activation of the interventricular septum from left to right; the presence of these deflections may occasionally give rise to a problem of differential diagnosis, anterolateral infarctions being suspected particularly when the Q waves are abnormally large. Wilson and associates have pointed out that the R-wave amplitude decrease, or disappearance of this deflection as the exploring electrode is moved from the right to the left side of the precordium, may be considered as more reliable criteria for the electrocardiographic diagnosis of anteroseptal infarction than is the complete absence of R waves in all right precordial leads.

In a study of the electrocardiographic and pathologic findings in 20 patients with anteroseptal infarction, Myers, Klein, and Stofer observed 8 subjects whose electrocardiograms showed an initial R wave of normal amplitude in Lead V1 and abnormal QRS complexes of the QR or QS type in one or more of the following three precordial leads; in 5 patients all leads from the right side of the precordium displayed essentially negative QRS deflections. Although definitely abnormal Q waves were not found in Leads V5 and V6, there were two instances in which the amplitude of the initial negative deflection in these leads was at the upper limit of normal. This configuration was regarded as the transmission of the potential variations of the anteroseptal infarct to the left axilla due to marked clockwise rotation of the heart.

In addition to the absence of R waves in Leads V1 to V4 which occurs quite frequently in patients with marked left ventricular hypertrophy, we have observed several instances in which an initial upstroke was present in Lead V1 and decreased in amplitude as the exploring electrode was moved from this lead to V4; in occasional tracings, deep Q waves were recorded in Leads V5 and V6. In a recent publication, Quintiliano de Mesquita reports
a case of marked left ventricular hypertrophy with large Q waves in Lead CF4 in which the abnormal configuration was interpreted not as an evidence of myocardial infarction but as a transition complex associated with an intraventricular conduction defect although pathologic examination was not obtained.

A definite diagnosis is often difficult to establish in these cases particularly in the absence of a positive clinical history; although the presence of infarction was considered highly improbable in our cases, its possibility could not be excluded merely on a clinical basis, particularly since all electrocardiographic criteria for the diagnosis of old anterior infarction in precordial leads were observed.

In a patient with aortic insufficiency and marked left ventricular hypertrophy who has recently come under our observation, the electrocardiogram was extremely suggestive of anterior myocardial infarction in view of the decrease in amplitude of the R wave from V2 to V4 in addition to the presence of an abnormally large Q wave in Lead V6. Since no evidence of myocardial infarction could be demonstrated at autopsy despite a careful pathologic examination, a report of this case was considered to be of interest, from the electrocardiographic standpoint, investigating the possibility of interpreting the QRS changes in precordial leads as due to left ventricular and septal hypertrophy. It is our object in this article to suggest caution in the electrocardiographic diagnosis of anterior myocardial infarction in certain patients with left ventricular hypertrophy. Although such electrocardiographic patterns do not seem to be of frequent occurrence in these patients, Myers and collaborators have recently emphasized the difficulty in the interpretation of tracings showing respiratory variations in the configuration of the QRS complexes in precordial leads simulating those due to anteroseptal infarction; furthermore, a patient is mentioned, who as yet has not been reported, whose electrocardiogram showed an initial R and a deep S wave in Leads V1 and V2 and a Q wave varying from 2 to 5 mm. in Lead V6, followed by an R deflection which also varied in amplitude. Autopsy revealed both left and right ventricular hypertrophy but no evidence of infarction.

Case Report

S. S. V., a 47 year old Negro mechanic with syphilitic aortic insufficiency, was admitted to the hospital on August 16, 1948, with advanced congestive heart failure. He had been in good health until three years prior to admission when he first noticed dyspnea on exertion, orthopnea, palpitation, and ankle edema which later became generalized. Since that time he complained of constant pain in the left lower portion of his back and in the lumbar region. He was treated by a local physician and obtained relief from these symptoms which had recently recurred. At the age of 26 years, he contracted gonorrhea, but denied ever having had a chancre, although reactions to serologic tests for syphilis had been repeatedly positive in spite of anti-syphilitic therapy.

Admission examination revealed a severely dyspneic patient with Cheyne-Stokes respiration and long periods of apnea during which the patient became markedly agitated. There were typical signs of aortic regurgitation, including an average blood pressure reading of 240/40 with marked fluctuations to the extremes of 300 systolic and 0 diastolic levels.
LEFT VENTRICULAR HYPERTROPHY

FIG. 2.—Electrocardiogram made shortly after the patient’s admission showing auricular fibrillation with a slightly irregular ventricular rate of 107 per minute. Precordial leads taken at half normal sensitivity revealed left ventricular hypertrophy in addition to a decrease in amplitude of the R wave in Leads V2 to V4 and a deep Q wave in Lead V6. The RS-T segment is elevated in Leads V4 and V5 and depressed in Lead V7. The tracing is diagnostic of left ventricular hypertrophy and suggestive of anterior myocardial infarction and digitalis effect.

The heart was enlarged and a gallop rhythm was audible. The rate was 104 per minute, and the rhythm was slightly irregular. The liver was enlarged and tender to palpation. Rales were present at both lung bases. There was generalized edema.

Laboratory studies showed the following: Positive serologic reactions (to Kahn and Kline tests), a moderate amount of albumin in the urine, the specific gravity of which was 1.012; blood urea value of 70 mg. per 100 cc.; creatinine value of 1.3 mg. per
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100 cc.; 16,750 white blood cells per cu. mm. of blood, with 92 per cent neutrophils; total serum protein of 6.1 grams per 100 cc., with 3.54 grams albumin, 2.56 grams globulin, and an index of 1.3; x-ray film of the chest obtained on April 28, 1948, in the outpatient department (fig. 1) showing marked cardiac enlargement particularly of the left ventricle in addition to a dilatation of the aorta. Diffuse atheromatosis of the abdominal aorta was demonstrated radiologically with no evidence of erosion of the lower thoracic or lumbar vertebral bodies. An electrocardiogram made shortly after admission (fig. 2) showed auricular fibrillation with a slightly irregu-

Fig. 3.—Pathologic specimen showing a transverse section of the heart which reveals a marked degree of left ventricular hypertrophy in addition to a greatly thickened interventricular septum which bulges into and reduces the capacity of the right ventricle. VE = left ventricle; VD = right ventricle.

lar ventricular rate of 107 per minute, slurred QRS complexes, and signs of marked left ventricular hypertrophy in precordial leads. There was a decrease in the size of the R wave from V2 to V5 and a deep Q deflection in Lead V6. The RS-T segment was elevated in Leads V1 and V2 and depressed in Lead V5. The tracing was strongly suggestive of anterior myocardial infarction mainly in view of the characteristic QRS changes, since the alterations in RS-T segment and T waves could be attributed, at least partially, to the effects of digitalis. High precordial leads were essentially similar to those obtained at the usual levels. It is likewise conceivable that incomplete left bundle branch block could be indicated in this tracing notwithstanding the presence of a deep Q wave in Lead V4 and a smaller initial negative deflection in Lead V1, which, according to Sodi-Pallares, could be explained by the location of the block below the origin of the initial vector of septal activation. In one electrocardio-gram this type of block was suggested by the configuration of Lead I showing a slurred upstroke of the R wave which is strikingly similar to the tracings obtained experimentally by Sodi-Pallares and co-workers.

A progressively downward course was observed in spite of intensive therapy consisting of cardiotonic drugs, diuretics, and sedatives. The patient died five days after an episode of pulmonary embolism following a shocklike condition associated with an irregular cardiac rhythm. The electrocardio-

Fig. 4.—Histologic section showing the normal aspect of the myocardial fibers. Essentially similar sections were obtained at other points such as the interventricular septum, the cardiac apex, and the anterior wall of the left ventricle.

gram recorded a half hour before death showed a return to sinus rhythm with periods of bradycardia alternating with sinus tachycardia and partial A-V block with sinus depression.

Pathologic examination by Dr. Penna de Azevedo revealed the following: Syphilitic aortitis, syphilitic aortic valvulitis with deformity of the valves, aneurysmal dilatation of the thoracic aorta, edema and passive hyperemia with hemorrhagic infarcts in both lungs, cardiac hypertrophy, bilateral healed renal infarcts with atrophy of the kidneys, moderate ascites, and chronic bilateral fibrous pleuritis. The heart measured 13.5 by 7.5 by 8.0 centimeters. The left chambers showed a greatly increased capacity. The right ventricular chamber was markedly decreased in size and the extremely hypertrophied (25 mm.) interventricular septum bulged into the
cavity of the right ventricle (fig. 3). The inner aspect of the aorta showed marked irregularities due to yellowish plaques, some of which were calcified, alternating with large areas of tissue retraction. The coronary arteries were patent throughout. The myocardium was of firm consistency and of a dark red color with no apparent increase of fibrous tissue.

The wall of the left ventricle measured 26 mm. from endocardium to epicardium. Just above the diaphragm in the thoracic cavity there was an aneurysmal dilatation, 7.5 by 5.5 cm., to the left of the aorta with which it communicated through an orifice 2 cm. in diameter; the renal arteries were seen to emerge immediately below the aneurysm.

A number of histologic sections of the myocardium were made (fig. 4), particularly from those areas where the electrocardiogram suggested the possibility of infarction; no evidence of this type of lesion in its acute or chronic stage could be found.

**Discussion**

Since the electrocardiographic diagnosis of myocardial infarction was not confirmed at autopsy there did not seem to be a correlation between the tracings and the pathologic findings in this case.

The possibility of a very recent infarct in which the electrocardiographic changes may precede the histologic lesions in the myocardium as shown by Blumgart and associates, and confirmed clinically by others, was discarded in this patient, since not only were the coronary arteries found to be patent at autopsy but also because the patient died seven days after the tracings were obtained, a period of sufficient length for pathologic changes to occur.

The finding at autopsy of an extremely thickened interventricular septum bulging into the right ventricular cavity (anatomic stage of Bernheim's syndrome) suggested the possibility that the initial activation of the upper part of the hypertrophied septum could originate a septal vector of greater magnitude than that of the normal Q wave. According to this interpretation, the deep Q waves recorded in the left precordial leads in this case reflect early activation from left to right of the interventricular septum such as occurs in normal individuals although of greater voltage owing to septal hypertrophy.

The decrease in the amplitude of the R wave from Leads V₃ to V₅ is more difficult to explain according to the foregoing interpretation.

Several other explanations may be offered for the presence of abnormal Q waves in left precordial leads in the absence of infarction. Among these is the possibility that the QR complexes represent mixed cavity and epicardial potentials recorded in these leads as a result of cardiac rotation.

**Summary and Conclusions**

A case history is presented of a patient, with syphilitic aortic insufficiency and marked left ventricular hypertrophy, in whom the electrocardiogram was extremely suggestive of anterior myocardial infarction which was not found to be present at autopsy. Pathologic examination revealed marked hypertrophy of the left ventricle in addition to great enlargement of the interventricular septum which bulged into the right ventricular cavity reducing the capacity of this chamber.

It is suggested that septal forces from left to right of great magnitude could presumably explain the electrocardiographic findings in the absence of infarction.

On the basis of this case and previous ones without pathologic basis, the authors suggest caution in the electrocardiographic diagnosis of anterior myocardial infarction in the presence of marked left ventricular hypertrophy, particularly in patients with a negative clinical history.

**References**


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