Electrocardiogram and Spatial Vectorcardiogram of Localized Myocardial Hypertrophy

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MYOCARDIAL HYPERTROPHY is usually considered to be either concentric or eccentric.\(^1\) Although generalized hypertrophy of the various chambers of the heart has been studied, relatively little attention has been given to hypertrophy localized to only a portion of the ventricle. Localized myocardial hypertrophy may develop as a result of compensatory hypertrophy following death of a large area of myocardium due to coronary artery disease,\(^2\)\(^3\) or it may develop in the absence of coronary artery disease when there is a greater demand for work from one portion of the myocardium than from another, as in the presence of a left-to-right intracardiac shunt.\(^4\)\(^7\)

The purpose of this paper is to describe the electrocardiographic and spatial vectorcardiographic findings in certain types of localized hypertrophy.

Hypertrophy Localized to the Posterobasal Aspect of the Left Ventricle

When there is extensive arteriosclerotic disease and occlusion of the left coronary artery, particularly of its anterior descending branch, there may be widespread loss of muscle in the anteroseptal and apical portions of the left ventricle. If the right coronary artery remains patent, compensatory hypertrophy will develop in the remaining or posterobasal portion of the left ventricle that is called upon to maintain cardiac output and to perform all the work previously done by the entire left ventricle.\(^2\) This phenomenon is illustrated by the two patients whose spatial vectorcardiograms are shown in figures 1 and 2.

The spatial vectorcardiogram shown in figure 1 is from a 64-year-old patient with occlusion of the left anterior descending coronary artery 1.5 cm. below its origin from the left coronary artery. The myocardium of the anteroseptal and apical aspects of the left ventricle was thin and atrophic. An aneurysm was present at the apex of the left ventricle. The right coronary artery showed minimal narrowing and was patent throughout its entire course. The circumflex branch of the left coronary artery was moderately narrowed but was not occluded. The left ventricle measured 1.2 mm. in thickness at the apex and 3 mm. in thickness at a point just below the anterior papillary muscle. The high posterobasal portion of the left ventricle measured 20 mm. at its thickest portion.

The spatial vectorcardiogram and the electrocardiogram shown in figure 2 are from a 62-year-old patient with severe coronary arteriosclerosis. The left coronary artery was occluded 0.5 cm. below its origin from the aorta. Both the left anterior descending and circumflex arteries were markedly narrowed throughout. The right coronary artery was dilated and tortuous and contained several subintimal plaques; however, the artery was widely patent throughout its course. The apical portion of the left ventricle measured 2 mm. in thickness. In the region of the anterior papillary muscle the left ventricle measured 5 mm. in thickness. The myocardium was 21 mm. thick in the high basal portion of the left ventricle.

The electrocardiogram and spatial vectorcardiogram of patients with localized posterobasal hypertrophy displayed a fairly characteristic and uniform pattern. Because of the loss of muscle mass in the anterior portions of the left ventricle and because of the hypertrophy at the base of the ventricle, the early
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Vectors of ventricular activation are shortened, whereas the late vectors are increased in both magnitude and duration (figs. 1, 2). The electrocardiogram from patients with localized posterobasal hypertrophy had deep, slurred S waves in leads II, III, and aVF, as well as deep S waves in all precordial leads except leads V₅ and V₆ (fig. 2).³

The spatial vectorcardiogram recorded by means of the equilateral tetrahedral reference system⁸ in patients with localized posterobasal hypertrophy was very similar to that previously described for apical myocardial infarction.² The early portion of the QRS sE-loop was directed for a short time inferiorly and to the left in the frontal plane projection. However, the loop soon turned upward to become directed superiorly, to the left, and posteriorly. Thus, the early vectors of the QRS sE-loop were foreshortened, but the late mean instantaneous vectors were of relatively great magnitude (figs. 1, 2).

Anomalous origin of the left coronary artery from the pulmonary artery is another disease in which there is likely to be marked fibrosis of the area of the myocardium supplied by the left coronary artery and hypertrophy of the portion supplied by the right coronary artery.⁹-¹² In this disease the left coronary artery usually behaves as a vein draining the right coronary artery. The only blood supply to the anterior aspect of the left ventricle is from the cavity of the left ventricle.¹³, ¹⁴ Autopsy studies in this anomaly have shown the apical region of the left ventricle frequently to be aneurysmally dilated, whereas the high posterobasal portion of the left ventricle is hypertrophied.⁹-¹² The basal hypertrophy is greatest in adults, presumably because the myocardium has had sufficient time to hypertrophy.⁹ However, marked basal hypertrophy has been reported even in infants a few months of age.¹⁰

The electrocardiogram from patients with anomalous left coronary artery is similar to those described above for patients with coronary arteriosclerosis and diffuse myocardial disease in the area supplied by the left coronary artery. Deep S waves are present in leads II and III (fig. 3) and in the precordial leads with the exception of leads V₅ and V₆ (fig. 4).¹⁵ Thus, nature has provided an ideal experiment to reveal the influence of localized myocardial hypertrophy on the electrocardiogram and vectorcardiogram of man.

Hypertrophy Localized to the Crista Supraventricularis

Hypertrophy of a localized area of the myocardium may develop when the demand for mechanical work is greater for one area of the myocardium than for another. The crista supraventricularis is a muscular ridge of tissue that is located in the posterior aspect of

![Spatial vectorcardiogram of a patient with localized posterobasal myocardial hypertrophy. Heart weight 395 Gm. At postmortem examination the left anterior descending coronary artery was occluded. There was extensive scarring in the area of the myocardium supplied by this artery. The left circumflex and right coronary arteries were patent. The posterobasal and lateral aspects of the left ventricle were hypertrophied, whereas the anteroseptal and apical aspects were thin and fibrotic. The initial portion of the QRS sE-loop is oriented inferiorly, to the left, and anteriorly. However, the QRS sE-loop is foreshortened, so that the late and mid vectors and terminal vectors are oriented superiorly, to the left, and posteriorly. Note that the degree of foreshortening of this QRS sE-loop is not so great as that of the loop shown in figure 2, and the mid temporal vectors are of greater magnitude in this QRS sE-loop. This is probably explained by the fact that the left circumflex artery was patent and the compensatory hypertrophy included a portion of the lateral wall as well as the posterobasal portion of the left ventricle.](http://circ.ahajournals.org/lookup/vol26/issue10/545)
Electrocardiogram and vectorcardiogram of a patient with localized posterobasal hypertrophy. Heart weight 380 Gm. The left coronary artery was occluded 1.5 cm. from its origin. The right coronary artery was dilated and tortuous. The posterobasal aspect of the left ventricle was hypertrophied. The QRS sE-loop is remarkably foreshortened, so that almost all of the mean instantaneous cardiac vectors are directed superiorly, to the left, and posteriorly.

Despite many publications,4-7, 18-18 many cardiologists still do not realize that in the presence of an uncomplicated atrial septal defect hypertrophy of the right ventricle is restricted primarily to the outflow tract (crista supraventricularis). Hypertrophy of the crista supraventricularis is also a prominent feature of ventricular septal defect.18 The evidence for localized hypertrophy of the crista supraventricularis has been obtained primarily from electrocardiographic and vectorcardiographic studies. Kossmann and co-workers19 have indicated that the anatomic position of the crista supraventricularis is such that activation of this structure is likely to result in a mean vector that is directed superiorly and to the right, and that is re-
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flected as a late positive deflection in precordial leads recorded to the right of the sternum (R' wave). This fact explains the occasional occurrence of a small r' wave in precordial leads recorded to the right of the sternum of normal subjects. In the presence of a left-to-right shunt, the volume of blood that must be ejected by the right ventricle is greater than normal (diastolic overload). This apparently places a greater burden on the crista supraventricularis than on the remainder of the right ventricle, so that the crista undergoes work hypertrophy. The remainder of the ventricle dilates primarily to accommodate the greater volume of blood that reaches the right ventricle because of the left-to-right shunt.

The electrocardiograms of patients with atrial and ventricular septal defects show prominent late positive deflections in the precordial leads recorded to the right of the sternum. It seems likely that the R' wave recorded in patients with intracardiac left-to-right shunts is inscribed during depolarization of the same structure that produces the r' wave in normal subjects, namely, the crista supraventricularis. In the former groups, however, this structure is hypertrophied.

Necropsy studies have shown that in uncomplicated atrial septal defect the hypertrophy of the right ventricle is restricted mainly to the crista supraventricularis.

The electrocardiograms of patients with hypertrophy of the crista supraventricularis display deep, wide S waves in leads I, II, and III or in leads I and II. In lead V1 there is an RSR' pattern. The R' wave, inscribed during depolarization of the crista supraventricularis, tends to be relatively wide and slurred. Deep, wide S waves are also usually present in leads V4 and V5 or in V4, V5, and V6 (fig. 5).

The portion of the QRS sE-loop that is inscribed during depolarization of the crista supraventricularis is oriented superiorly, to the right, and either anteriorly or posteriorly (fig. 6). The configuration of the earlier portions of the QRS sE-loop depends upon the type of congenital defect present. For example, in figure 6 the spatial vectorcardiogram of a patient with an ostium primum-type atrial septal defect and of a patient with ventricular septal defect are shown. In both spatial vectorcardiograms the terminal projection is oriented superiorly and to the right, whereas the main portion of the loop is oriented superiorly and to the left of the patient with ostium primum defect and inferiorly and to the left in the patient with the ventricular septal defect.

Discussion

When ischemia or increased cardiac work due to elevated systemic or pulmonary vascular resistance is the stimulus for myocardial hypertrophy, the hypertrophy is usually sym-

\[ \text{Figure 3} \]

The standard limb leads and constructed QRS loop in an adult patient with anomalous origin of the left coronary artery. At autopsy the heart was thin and fibrotic in the area supplied by the left anterior descending coronary artery. Note the similarity between standard leads from this patient and those shown in figure 1. (Reprinted courtesy of the C. V. Mosby Company; from Gouley, B. A.: Am. Heart J. 40: 630, 1950.)
Electrocardiogram from a 26-year-old patient with anomalous origin of left coronary artery from pulmonary artery. Both coronary arteries were patent at necropsy. The major QRS vectors are oriented superiority, to the left, and posteriorly. (Modified from George, J. M., and Knowlan, D. M.: New England J. Med. 261: 993, 1959.)

Electrocardiogram of a patient with ventricular septal defect. The deep S waves in leads I, II, III, and the r' wave in lead V₁ are probably inscribed during the depolarization of a hypertrophied crista supraventricularis.
Spatial vectorcardiograms from a patient with an ostium primum-type atrial septal defect and from a patient with a ventricular septal defect (electrocardiogram shown in fig. 5). The late vectors of ventricular depolarization are oriented superiorly and to the right and are prolonged in both spatial vectorcardiograms. In the superior plane projection the horizontal deflection is derived from the difference in potential between the left and right arms and the vertical deflection from the difference in potential between the central terminal and the back. In the plane sagittal to the superior, the horizontal deflection is derived from the difference in potential between the central terminal and the foot and the vertical deflection from the difference in potential between the central terminal and the back.

Myocardial hypertrophy may be localized rather than symmetrical. Although the concept of localized hypertrophy has received little attention, recognition of this entity permits a better understanding of certain types of electrocardiographic and spatial vectorcardiographic patterns. The localized muscular hypertrophy is associated with "hypertrophy" of the electric forces as they migrate through the area of increased muscle mass. Thus, the vectors resulting at that time are increased in magnitude and prolonged in duration. The configuration of the electrocardiogram and of the spatial vectorcardiogram is greatly influenced by these vectors. The nature of the alterations of the electrocardiogram and the spatial vectorcardiogram depends upon the area of the localized hypertrophy. In certain instances, for example, hypertrophy of the crista supraventricularis and hypertrophy of the posterobasal portion of the left ventricle, diagnostic patterns evolve (fig. 7). These diagnostic patterns can be recognized readily by use of the principles of anatomy, physiology, electrocardiography, and vectorcardiography. Once the diagnosis is made, one's attention is then directed to the lesions that may produce localized types of hypertrophy, such as atrial or ventricular
septal defect, apical myocardial infarction, and anomalous origin of the left coronary artery. Correlation of the autopsy findings with the electrocardiogram and vectorcardiogram for a large series of patients in this laboratory over the past 10 years has indicated that these concepts are useful and reliable in clinical cardiology.

Other types of localized myocardial hypertrophy have been described in addition to those mentioned in this report. However, the associated electrocardiogram and spatial vectorcardiogram have not yet been studied in sufficient detail to permit recognition of reliable diagnostic features of the localized hypertrophy. For example, many families have been studied with a peculiar form of localized hypertrophy of the left ventricle which has been called “asymmetrical hypertrophy of the heart in young adults.” This form of localized hypertrophy was considered at first to be due to a benign tumor or a hamartoma. The lesion was usually confined to the left ventricle and particularly to the interventricular septum. Nine of 23 members of one family had unequivocal evidence of this type of cardiac disease. The disease seems to be genetically dominant but not sex-linked. Histologic examination of the affected area of myocardium showed large, coarse muscle bundles and scattered areas of fibrosis. The electrocardiogram seems to offer almost no diagnostic clues, but the tracings published are limited in number and poorly illustrated. The one exception is the frequent appearance of prominent right atrial P waves in the electrocardiogram. The presence of this finding suggests that the area of localized hypertrophy has produced an obstruction to the inflow tract of the right ventricle. It is of interest that although the lesion involves the interventricular septum, conduction abnor-

![Diagram to show orientation of terminal mean instantaneous QRS vectors in two types of localized myocardial hypertrophy.](image-url)
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malities are infrequent. There have been no spatial vectorcardiograms recorded in this disease.

Isolated subaortic stenosis and infundibular stenosis represent another type of localized hypertrophy. In these diseases the site of the localized hypertrophy is such that obstruction to the outflow tract of the left or right ventricle develops. These defects are probably due to failure of resorption of the bulbus cordis and represent a congenital form of localized hypertrophy. Because of obstruction to ventricular emptying, the entire musculature of the affected ventricle hypertrophies, so that the electrocardiogram and the spatial vectorcardiogram resemble those of diffuse right or left ventricular hypertrophy. However, Braunwald et al.\(^{23}\) have recently described three siblings with idiopathic hypertrophic subaortic stenosis in whom the electrocardiogram showed anomalous atrioventricular excitation (Wolff-Parkinson-White syndrome).

Summary

Diagnostic electrocardiographic and vectorcardiographic manifestations of localized hypertrophy of the posterobasal portion of the left ventricle and of localized hypertrophy of the crista supraventricularis have been described. Recognition of the fact that only a portion of the myocardium may undergo hypertrophy is important in understanding certain types of electrocardiographic and spatial vectorcardiographic patterns.

Several types of localized myocardial hypertrophy for which no characteristic electrocardiographic or spatial vectorcardiographic patterns have been described were also discussed. There is a need for further study of this problem, and, in particular, for detailed correlation of the necropsy data with the electrocardiogram and the vectorcardiogram.

References

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As a clinician, meaning thereby a man who understands the course, the prognosis and the treatment of disease, Sydenham had no rival in his day and I doubt whether later ages have produced one. When he describes the gout or the smallpox, his wordpictures are unsurpassable.—DAVID RIESMAN, M.D. Thomas Sydenham, Clinician. New York, Paul B. Hoeber, Inc., 1926, p. 23.
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