The Q Wave in Precordial Electrocardiograms Overlying the Hypertrophied Right Ventricle: Intracavity Leads

By Noble O. Fowler, Jr., M.D., Richard N. Westcott, M.D., and Ralph C. Scott, M.D.

Right ventricular cavity electrocardiograms were obtained in six subjects having qR patterns in V leads made from precordial sites overlying the hypertrophied right ventricle. Four of the six, unlike control subjects, had no initial R wave in the right ventricular cavity; one had only a questionable initial R wave. Simultaneous intracavity and precordial electrocardiograms suggested that the initial Q wave over the hypertrophied right ventricle is due to abnormal depolarization of the interventricular septum, and that the large R wave over the hypertrophied right ventricle is the result of delay in activation of the free wall of that chamber.

SEMIDIRECT unipolar electrocardiographic leads (V leads) from sites on the precordial overlying the normal human right ventricle usually reveal a QRS pattern characterized by a small initial R wave and a large final S wave (rS pattern*). When the right ventricle is hypertrophied, V leads obtained from the right precordium may show one of four types of QRS complex: (1) the normal rS pattern; (2) right bundle branch block; (3) evidence suggesting dilatation but not hypertrophy of the right ventricle; (4) the QRS pattern which is considered diagnostic of right ventricular hypertrophy. This latter pattern, with which we are primarily concerned, is a QR pattern associated with a QRS of normal duration. Occasionally, there may be seen an rSR or qRs pattern. The Q wave in precordial leads overlying the hypertrophied right ventricle may be quite deep in proportion to the succeeding R wave, resulting in Q:R ratios ordinarily associated with myocardial infarction.2

Since the origin of the Q wave in electrocardiographic leads from the chest overlying the hypertrophied right ventricle is in consider-
able dispute it was believed that electrocardiographic leads taken from the cavity of the right ventricle in subjects showing such a Q wave might shed light on the mechanism of its production.

Material

Six patients having a QR or qRs pattern in electrocardiographic leads overlying the right ventricle were obtained from the medical wards of the Cincinnati General Hospital and from the medical wards of the Veterans Hospital, Dayton, Ohio. All patients had roentgenographic evidence of right ventricular hypertrophy.

As controls, right ventricular cavity leads were obtained in three subjects who had rS patterns in precordial leads overlying the right ventricle. Two of these subjects had emphysema of the lungs.

Method

Cardiac catheterization was performed by methods described previously, using catheters containing a wire electrode buried in their walls. In all instances the position of catheter tip was verified both by fluoroscopy and by viewing the pressure curve on an oscilloscopic viewing screen before taking the intracavity electrocardiograms. Simultaneous precordial and intracavity electrocardiograms were obtained with the Technicon Triagram electrocardiograph. Electrocardiograms were recorded at two speeds: slow speed at 25 mm. per second and fast speed at 50 mm. per second. Nonsimultaneous electrocardiograms were obtained with the Sanborn Visocardiette. In some instances the precordial and intracavity electrocardiograms were timed for superimposition by a simultaneous recording of the right ventricular pressure curve. Intracardiac leads were recorded at one-tenth to one-half normal sensitivity. Intracardiac pressures were recorded by means of

From the Cardiac Laboratory, Cincinnati General Hospital, Department of Internal Medicine, University of Cincinnati.

This study was supported in part by Research Contract V1001 M-432 Veterans Administration.

* In discussing the QRS complex of the electrocardiogram, small letters will be used to designate relatively small deflections; capital letters will be used to designate large deflections.

Circulation, Volume V, March, 1950
the five-channel Hathaway pressure recording apparatus. Blood samples, collected under oil and stored in ice over mercury, were analyzed for oxygen and carbon dioxide in the Van Slyke manometric apparatus. Duplicate samples were required to check within 0.2 volume per 100 cc.

Intracavity leads (fig. 2) were taken from the pulmonary conus, midzone of the right ventricle, low in the right ventricle, right ventricular side of the tricuspid valve and low in the right atrium. In the intrinsicoid deflection is delayed, the R is prominent, and the S is small.

RESULTS

Case Reports

D. H., a 61 year old white man, had pulmonary emphysema confirmed by spirometry, chronic cor pulmonale, and secondary polycythemia. An angiocardiogram revealed right ventricular hypertrophy. Cardiac catheterization performed May 26, 1950 revealed a pulmonary artery pressure of 75/25 mm. Hg. Arterial oxygen saturation was 68.27 per cent of capacity. Hemoglobin was 17.45 Gm. per 100 cc. of blood (Van Slyke oxygen capacity method). Arterial carbon dioxide content was 66.84 volumes per 100 cc. of blood. The arterial hypoxemia and hypercapnia were considered characteristic of severe pulmonary emphysema. The pulmonary arterial hypertension was in keeping with the impression of cor pulmonale.

The standard lead electrocardiogram, together with unipolar extremity leads and unipolar precordial leads, is shown in figure 1. The standard leads show right axis deviation. Lead aVR shows a QR complex, consistent with but not diagnostic of right ventricular hypertrophy. Since the QRS pattern of lead V1 was not typical of right ventricular hypertrophy, leads V3R, V5R, V4R and V3R were taken. These V leads all showed the classic QR or qRs pattern of right ventricular hypertrophy. In the leads V7R through V1, the qRs pattern differs from that seen in septal infarction in the fact that here none of these areas was a definite initial R wave seen in the QRS complex.

C. B., a 56 year old white man, had pulmonary emphysema, cor pulmonale, and congestive heart failure. Hematocrit was 59.
On Dec. 22, 1950, cardiac catheterization was performed. Pulmonary artery pressure was 90/45 mm. Hg. Right ventricular pressure was 80/15–7 mm. Hg. Systemic arterial oxygen saturation was 51.31 per cent of capacity. Systemic arterial carbon dioxide content was 65.69 volumes per 100 cc. of blood. Hemoglobin was 16.87 (Van Slyke oxygen capacity method).

The intracardiac electrocardiographic leads and standard leads, unipolar extremity leads and unipolar precordial leads obtained at the time of catheterization are reproduced in figures 3 and 4. The standard leads show right axis deviation and deep Q waves in leads II and III. There is a definite Q wave in leads V₄₅, V₅₆, and V₁; all show the QRS complex characteristic of right ventricular hypertrophy. It was felt that the patient possibly had healed posterior myocardial infarction in addition because of the large Q wave lead in V₆; however, the probability that this Q wave was referred from the posterior transitional zone between right and left ventricles could not be excluded. Simultaneous precordial and intracavity electrocardiograms (fig. 4) revealed that the Q wave in lead V₄₅ was simultaneous in onset with the initial downward deflection in tracings made from the right ventricular cavity. In no part of the right ventricular cavity was the normal R wave pattern obtained. Since there was no electrocardiographic evidence of massive septal infarction, it was felt that the possible old posterior myocardial infarct played no role in the absence of an R wave in the right ventricular cavity leads.

R. P., a 62 year old Negro man, had bronchiectasis, pulmonary emphysema, cor pulmonale, and congestive heart failure.

Cardiac catheterization was performed on Jan. 5, 1951. Brachial artery blood showed oxygen saturation 76.9 per cent of capacity and a carbon dioxide content of 64.22 volumes per 100 cc. of blood. Hemoglobin (Van Slyke) was 14.38 Gm. per

100 cc. Right ventricular pressure was 54/7 mm. Hg.

The standard electrocardiographic leads, unipolar extremity leads, and unipolar precordial leads obtained at the time of cardiac catheterization are reproduced in figures 5 and 6.

The standard leads show right axis deviation. Lead V₄₅ shows a qR pattern with the QRS complex of normal duration. There is a small initial R wave in leads V₃₄ and V₅. Lead V₆ (fig. 6) contains a rather wide S wave in the QRS complex. These findings are considered indicative of right ventricular hypertrophy. Intracavity leads from the apex of the right ventricle revealed a QS pattern.

J. S. was a 54 year old white man suffering from pulmonary emphysema and cor pulmonale without congestive heart failure.

Cardiac catheterization was performed Oct. 27, 1950. Pulmonary artery pressure was 55/32 mm. Hg. Right ventricular pressure was 56/2.5 mm. Hg. The systemic arterial blood was 81.3 per cent saturated with oxygen and contained 52.09 volumes of carbon dioxide per 100 cc. of blood. Hemoglobin (Van Slyke oxygen capacity method) was 14.74 Gm. per 100 cc. of blood.

Standard and unipolar limb leads and unipolar precordial lead electrocardiograms were made. The standard leads revealed right axis deviation. There was a large Q wave in lead III and in lead V₆; this was thought to be referred from the Q wave over the right precardium. Leads V₃₄ and V₅₆ revealed the QRS pattern characteristic of right ventricular hypertrophy. Intracavity leads within the right ventricle taken at one-half and one-third normal sensitivity revealed only a questionable R wave and a large S wave.

Patient E. S. was a 54 year old Negro man who had cor pulmonale in congestive failure. Venous
blood carbon dioxide combining power was 64 volumes per 100 cc.

Cardiac catheterization was performed Aug. 18, 1950. The pulmonary artery pressure was found to be 72/30 mm. Hg. Right ventricular pressure was 60/9 mm. Hg.

Intracavity electrocardiograms were taken along with the standard leads, unipolar extremity leads, and unipolar precordial leads. The standard leads showed right axis deviation. Leads $V_{3R}$, $V_{4R}$, and from near the apex of the right ventricle. However, it was believed that the initial deflection in the leads from the mid-right ventricle and apex of the right ventricle is a $Q$ wave; that in the pulmonary conus was definitely a $Q$ wave. An apparent minute initial $R$ wave in the right atrial and tricuspid lead may have come from the left ventricular cavity via the mitral orifice and left atrium. An initial $R$ wave in the pulmonary artery lead probably came from the left ventricular cavity via the aorta.

$V_{3R}$ demonstrated the characteristic QRS pattern of right ventricular hypertrophy. Intracavity leads taken at approximately one-half normal sensitivity revealed no initial $R$ wave in the region of the pulmonary conus or tricuspid valve of the right ventricle.* Unfortunately the onset of the QRS complex was partially obscured by an artefact in leads obtained from the middle of the right ventricle and

* We are indebted to Dr. Gordon B. Myers, Professor of Medicine, Wayne University, for the following analysis of the electrocardiograms of this patient.

R. T., a 22 year old white man, had uncomplicated pulmonic stenosis. Cardiac catheterization was performed Dec. 19, 1950. As the catheter was advanced through the superior vena cava, right atrium, right ventricle, and pulmonary artery, in no instance was there a rise of even 0.1 volume per 100 cc. in oxygen content, thus excluding a left to right intracardiac shunt. Systemic arterial oxygen saturation was 96.15 per cent of capacity, thus militating against a right to left intracardiac shunt. Pressure in the pulmonary artery was 18/10 mm. Hg. and in the right ventricle 165/7 mm. Hg.

Standard electrocardiographic leads, unipolar
Fig. 5. Case R. P. Standard and unipolar electrocardiographic limb leads: unipolar precordial leads. The standard leads show right axis deviation. Lead V₃R shows the qR pattern diagnostic of right ventricular hypertrophy.

Fig. 6. Case R. P. Simultaneous precordial and intracavitary electrocardiogram. Shows the absence of an initial R wave in the right ventricular cavity lead.
limb and precordial leads and simultaneous intracavity and precordial leads were made. The standard leads showed marked right axis deviation. Leads V_R, V_{R}, V_{R}, and V_{R} each revealed the characteristic QRS pattern of right ventricular hypertrophy. Intracavity leads obtained from the midportion of the right ventricle at one-fifth normal sensitivity demonstrated a small initial R wave occurring simultaneously with Q wave in leads V_R and V_{R}.

Control Studies

In each of three control subjects intracavity leads from the right ventricle revealed an rS pattern. All controls had rS patterns in semidirect unipolar leads from the precordium over the right ventricle. Two control subjects had emphysema; the other had no evidence of heart disease.

Discussion

Following the investigations of Hecht and others, Levine found an rS pattern in 24 of 27 normal subjects in electrocardiograms taken from the right ventricular cavity. This normal initial positivity, found also in our control subjects, is interpreted as indicating earlier activation of the left side of the interventricular septum in the normal course of ventricular depolarization.

The cause of the initial Q wave in V leads over the hypertrophied right ventricle is in dispute. One group of investigators has stated that the qR complex in right precordial leads in right ventricular enlargement is due to extreme clockwise rotation of the heart so that the electrode in the V_1 position is facing the posterobasal portion of the left ventricle. Kert and Hoobler have suggested that the initial Q wave over the right precordium in the aforementioned condition may be due to activation of some part of the left ventricle prior to septal depolarization. Sodi-Pallares believes that the initial Q wave in right ventricular hypertrophy is the result of transmission of negative potentials in the right auricle to the right side of the precordium. Myers believes that the initial Q wave over the right ventricle in right ventricular hypertrophy may be due to one of two factors: the interventricular septum may be activated from right to left instead of from left to right as is thought to occur normally; or, the septum may be activated normally from left to right, but the resulting initial R wave of the right ventricular cavity may be so small that is is not transmitted to the right precordium, where an initial Q wave may appear as the result.

Schlesinger and co-workers studied five cases of right ventricular hypertrophy by means of right ventricular cavity leads; one of their cases had a qR complex in lead V_1. This case also had an initial Q wave in the right ventricular cavity electrocardiogram. Kert and Hoobler also found an initial Q wave in the right ventricular cavity lead of a case of right ventricular hypertrophy having a qR complex in lead V_1.

In our series we have studied six cases of right ventricular hypertrophy having a qR complex in V leads over the right precordium. Only one of these had a definite initial R wave in the right ventricular cavity. One other had a questionable initial R wave in the right ventricular cavity, certainly indicating weakness of septal forces. In four cases there was a definite QS pattern in the right ventricular cavity; in two of these, simultaneous records were obtained demonstrating the onset of the QS pattern in the right ventricular cavity to be simultaneous with the beginning of the Q wave in semidirect leads over the right ventricle.

The evidence in the cases we have studied is in accordance with the theories of Myers. In the cases having a QS pattern in the right ventricular cavity leads, one would assume that the interventricular septum is being activated either from right to left, or simultaneously on the two sides. Unfortunately, we do not have intracavity leads from the left ventricle, which would serve to show conclusively whether the septum were activated from right to left initially. Wilson has stated that the initial Q waves in ventricular hypertrophy may be due to a decreased density of the junctions between Purkinje fibers and ordinary heart muscle in certain areas as a result of dilatation of the chamber chiefly affected. In the one and possibly two cases in our group showing a small initial R wave in the right ventricular cavity leads, we believe that left to right septal depolarization has been interfered with, but to a
lesser degree, resulting in a small initial R wave in the right ventricular cavity which is lost in transmission to the V leads over the right precordium.

The mechanism producing the large R wave of the qR complex in right ventricular hypertrophy is also of interest. Kossmann has concluded that this R wave is produced by activation of the free wall of the left ventricle from his finding that the nadir of Q and the peak of R occurred simultaneously in V leads over the posterior left chest and in lead V1 in subjects with right ventricular hypertrophy. McGregor supported Kossmann's concept by demonstrating RS patterns in direct right ventricular epicardial leads taken at operation in 10 cases of tetralogy of Fallot.

In this connection, it is of interest to examine the electrocardiogram of subject R. R. (fig. 6), showing leads V1R, lead V6, and the right ventricular cavity lead recorded simultaneously. In comparing the right ventricular cavity lead with lead V6, one notes that the nadir of the S wave in the former occurs simultaneously with the peak of R in the latter, indicating that both are produced by the arrival of the force of excitation at the epicardial surface of the left ventricle. The nadir of S in V6 occurs simultaneously with the peak of R in V1R, suggesting that both are produced by activation of the free wall of the right ventricle. In comparing the time of onset of the peak of R in V1R with the time of occurrence of the nadir of S in the right ventricular cavity lead in figure 6, one notes that the peak of R in V1R occurs 0.03 second later than the nadir of S in the cavity lead. In figure 4, the record of subject C. B. shows the peak of R in lead V2R to occur 0.04 second later than the nadir of S in the lead taken from the right ventricular cavity near the apex. The occurrence of the peak of R in leads over the right ventricle later than the arrival of the force of excitation at the free wall of the left ventricle would seem to indicate that the R wave in leads over the right ventricle is produced by the activation of the right ventricular wall, since there is no R wave of comparable magnitude in the leads obtained from the right ventricular cavity.

**Summary and Conclusions**

Intracavity electrocardiograms were obtained from the right ventricle in six cases of right ventricular hypertrophy having qR complexes in the V leads made from sites over the right precordium, and in three subjects without right ventricular hypertrophy having RS complexes in the V leads obtained from sites over the right ventricle. In four of the subjects with right ventricular hypertrophy, the right ventricular cavity electrocardiogram showed no initial R wave; in one the R wave was questionable; in one there was a definite initial R wave in the right ventricular cavity leads. The control subjects each showed a definite initial R wave in the right ventricular cavity leads.

It is concluded that the normal early left to right depolarization of the interventricular septum is often interfered with in right ventricular hypertrophy to the extent that the normal initial R wave of the intraventricular QRS electrocardiogram is not produced or is diminished in amplitude. The absence or diminution of this R wave may be responsible for the initial Q wave in V leads obtained from precordial sites over the right ventricle in hypertrophy of that chamber.

It is also concluded that the delayed R wave of the qR electrocardiographic pattern in the V leads obtained from precordial sites over the hypertrophied right ventricle is probably produced by activation of the free wall of the right ventricle.

**Acknowledgments**

The writers wish to thank Dr. C. H. Sears of Wayne University, Detroit, Michigan, for his helpful comments on the electrocardiograms of subjects R. T., J. S., and C. B.

The writers also wish to thank Dr. J. Taguchi and Dr. C. McCord of the Veterans Hospital, Dayton, Ohio for their help in finding the subjects used in this study.

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NOBLE O. FOWLER, JR., RICHARD N. WESTCOTT and RALPH C. SCOTT

Circulation. 1952;5:441-448
doi: 10.1161/01.CIR.5.3.441

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