QRS-T Patterns in Multiple Precordial Leads that May Be Mistaken for Myocardial Infarction

II. Right Ventricular Hypertrophy and Dilatation

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The electrocardiograms of patients with pathologic evidence of right ventricular hypertrophy and/or dilatation and exclusion of myocardial infarction are presented to bring out certain features likely to be mistaken for myocardial infarction: (1) abnormal qrS patterns or cove plane inversion of the T waves in leads from the right precordium; (2) reduction in the amplitude of the initial R wave or replacement by a QS deflection and/or change from an upright to an inverted T wave as the electrode is moved from the V1 position to the transitional zone; (3) abnormal qrS deflections in leads from the left axilla.

Detailed descriptions have been published of the Wilson precordial electrocardiogram in right ventricular hypertrophy and dilatation,1-4 and in anteroseptal infarction,1, 6-11 but little attention has been devoted to the differentiation of these two conditions. The precordial leads in uncomplicated right ventricular hypertrophy and/or dilatation often show abnormalities that resemble the pattern associated with infarction of the septum and/or anterior wall of the left ventricle. To bring out the differential diagnosis, 15 previously unreported cases have been selected because of (1) findings in the precordial leads likely to be mistaken for myocardial infarction, (2) postmortem demonstration of right ventricular hypertrophy and/or dilatation and pathologic exclusion of myocardial infarction. The method of electrocardiographic and pathologic study is similar to that employed in the preceding study.12

I. RIGHT VENTRICULAR HYPERTROPHY

In right ventricular hypertrophy, leads from the right precordium may display one of the following signs1-3: (A) right bundle branch block; (B) a diagnostic pattern appearing in Lead V3R, usually in V1, and occasionally in V2, characterized by (1) a QRS of normal duration, (2) a prominent late upstroke, usually preceded by either a relatively small q wave or by a minute r and narrow s, resulting in a qR or rsR′ configuration, * (3) a delayed intrinsicoid deflection that terminates at the isoelectric line or is followed by an abnormally small s (or s′) wave, (4) depression of the RS-T junction, and (5) inversion of the T wave; (C) evidence suggesting dilatation of the right ventricle, but not hypertrophy; (D) an electrocardiogram showing no abnormality. In this section, it is our purpose to present cases of right ventricular hypertrophy with more or less diagnostic patterns in V3R, V1, and/or aVR to bring out certain features that may be mistaken for those of myocardial infarction. Cases without diagnostic signs of right ventricular hypertrophy in precordial leads, but with changes referable to right ventricular dilatation, will be presented in Section II. Right bundle branch block will be deferred until the next communication.

Electrocardiographic patterns referable to right ventricular hypertrophy may present certain features that might be mistaken for those of myocardial infarction, namely: (A) the qR deflection in right precordial leads may be abnormally broad, suggesting right bundle branch block, due to septal infarction; (B)

* The lower case letter is used to indicate a relatively small deflection; the upper case letter, to indicate a relatively large deflection.
the Q wave in right precordial leads may be unusually deep in proportion to the succeeding R, resulting in abnormal ratios in the range customarily associated with myocardial infarction; leads at the transitional zone, either finding suggesting anteroseptal infarction; (F) the normal Q wave may persist in left ventricular leads, accompanied by marked reduction in the

![Figure 1](image1.png)

**Fig. 1.-**Serial electrocardiograms in Case 13

![Figure 2](image2.png)

**Fig. 2.-**Electrocardiographic findings in right ventricular hypertrophy. A, Case 14; B, Case 15; C, Case 16; D, Case 17.

### Notes

- (C) sharp inversion of the T wave without the usual Rs-T depression may occur in leads from the right precordium, and may resemble the "coronary" T wave; (D) localized reduction in the amplitude of the R wave, or (E) replacement by a QS deflection, may occur in R and exaggeration in the S wave secondary to right ventricular hypertrophy, thus producing a pattern suggestive of anterolateral infarction. These features are collectively illustrated by the electrocardiograms of Cases 13 to 17, inclusive, reproduced in figures 1 and 2.
A. Abnormally Broad qR Deflection in Right Precordial Leads. As exemplified by the tracing of July 31, 1947, in figure 1, abnormally broad qR deflections in right precordial leads brings up the question of right bundle branch block, due to infarction of the septum. Both electrocardiograms in figure 1 were obtained from Patient 13, a boy aged 13 years, who was admitted to the hospital with congestive failure associated with rheumatic mitral stenosis and insufficiency complicated by a recent right subclavian thrombophlebitis and pulmonary infarction. Despite maintenance of prothrombin concentration between 20 per cent and 30 per cent, the thrombophlebitis extended into the right jugular, the innominate, and left subclavian veins, and repeated pulmonary emboli occurred, resulting in increasing right ventricular dilatation and failure.

The tracing of July 11, obtained upon admission, was considered diagnostic of right ventricular hypertrophy, because of (1) an abnormally large R wave in Leads V₃R and V₁ with small antecedent Q wave in the former lead, but without succeeding S wave in either lead; (2) a time interval from beginning of the QRS complex to the onset of the intrinsicoid deflection that was abnormally long in V₃R and V₁ and greater than in V₅ and V₆; (3) a prominent S wave in Leads V₅ and V₆.*

*Since this conclusion was reached, Kossmann and associates have advanced a different interpretation based on new evidence, which must be reviewed and re-evaluated. These workers took leads around the circumference of the chest and from within the right ventricular cavity in patients with clinical or pathologic evidence of right ventricular hypertrophy and found patterns in the customary precordial leads similar to that in the tracing of July 11 in Case 13. The qR pattern of Leads V₁ and V₃R was demonstrated over the entire right side of the chest as far posteriorly as the V₃R position, whereas the rS pattern of V₆ was distributed over the entire left side of the chest from the V₃ to the V₆ position. They contended that (1) the rS pattern was transmitted from the right ventricle because of its presence in Leads V₂ through V₄, which were anatomically over the right ventricle; (2) the qR pattern in all leads from the right side of the chest was transmitted from the left ventricle because of their observation that the hypertrophied right ventricle does not become as thick as the normal left ventricle and thus would not be expected to give rise to an R wave that is greater in amplitude and longer in duration than the normal R from the left ventricle.

Comparison of the electrocardiogram obtained on July 31, one day before death, with that taken on admission revealed a length-

In an effort at an anatomic rationalization of their premise of predominant transmission of the potential variations of the left ventricle to the entire right side of the chest, Kossmann and associates postulated an extreme degree of clockwise rotation. Sufficient cardiac rotation to permit transmission of the potential variations of the posterobasal wall of the left ventricle to the right arm and thereby produce a prominent late R wave in Lead aV₆ is a common observation, both in normal individuals and in patients with isolated left ventricular hypertrophy. On the other hand, sufficient cardiac rotation to permit transmission of the potential variations of the posterobasal wall of the left ventricle to the right precordium and thereby produce a prominent late R wave in V₁R and V₅, like that in figure 1, has not been encountered in any of our patients proved to have a normal heart or preponderant left ventricular hypertrophy at autopsy. All of our patients with such findings in V₅ and V₁, who have come to autopsy, have had definite evidence of right ventricular hypertrophy. If the positive potentials responsible for the late upstroke in V₅R and V₁ of Patient 13 had been transmitted from the posterobasal wall of the left rather than from the right ventricle, a relatively larger R wave would have been expected in Lead aV₆. The fact that the R waves in V₁ and V₅R were much greater than the R wave in aV₆, both in absolute voltage and in relative amplitude in respect to the antecedent Q wave, was in keeping with a right ventricular origin, but was strongly against a left ventricular source.

It would appear that the studies of Kossmann and associates, made with simultaneous leads from the right ventricular cavity and precordium in another case of right ventricular hypertrophy, provide a more satisfactory explanation for the registration of a prominent late R wave in leads to the right of the midline and an rS complex in leads to the left. They found an rS' complex in V₅ and V₆ and an rS'R' complex in Leads V₃R to V₅R and demonstrated conclusively that the R' deflection, which was comparable in amplitude to the R wave in Lead V₅R of our Patient 13, was produced by activation of the free wall of the right ventricle. They postulated that the late R wave was derived from the base of the hypertrophied right ventricle, which at autopsy is often thick and solid, in contrast to the apex, which is thin and trabecular. This may well account for the registration of a prominent late R wave in Leads V₁ and V₃R, facing the thickened base of the hypertrophied right ventricle in Patient 13, and for the registration of an R' deflection in Leads V₅, V₆, and V₄ over the relatively thin apex of the hypertrophied right ventricle. They also demonstrated that the minute initial r wave in leads from the right precordium was produced by septal activation and the
enning of the QRS interval from 0.09 to 0.12 second without significant change in the relative amplitudes of the component phases of the QRS in the limb leads or in Leads V3R and V1. In the remaining precordial leads, the R wave increased at the expense of the S. If the tracing of July 31, 1947, were examined without benefit of history or without access to previous electrocardiographic studies, the possibility of right bundle branch block due to septal infarction would demand serious consideration. The broad, slurred Q wave and late peak of the R wave in V3R and V1 were comparable to the findings in patients with pathologically proved septal infarction.\textsuperscript{11} The depression of the RS-T junction and straightening of the RS-T segment were atypical of infarction, but could be attributed to digitalization. However, in the clinical appraisal of this patient, no consideration was given to the possibility of septal infarction because of the history and previous electrocardiographic findings.

The question arose as to whether the terminal lengthening in time of the QRS interval was due to right bundle branch block or to a conduction defect in the free wall of the right ventricle. The possibility of uncomplicated right bundle branch block was practically excluded by the Q waves in V3R and V1, since activation of the entire septum from left to right should have produced sufficient electro-

motive force to have led to the registration of an initial R wave in these leads. Delay in arrival of the impulse at the base of the dilated right ventricle presumably permitted registration of a Q wave in V3R and V1, whereas slowing in the passage of the impulse through the hypertrophied ring of muscle near the tricuspid orifice probably accounted for the prolonged slurred R wave in the same leads. The increased amplitude and duration of the R wave in leads from the left precordium may have been due to a lesser degree of prolongation in activation of the thinner apical portion of the right ventricle.

The heart weighed 357 grams and showed marked right ventricular hypertrophy, as evidenced by a ratio of 1.1:1.0.\textsuperscript{13} The basal portion of the right ventricular wall measured 1.1 cm. in thickness, whereas the anteroapical one-third of the left ventricular wall measured 1.2 cm. in thickness. There was a marked rheumatic mitral stenosis, admitting only the tip of one finger. The coronary tree was widely dilated and there was no evidence of myocardial infarction. There were multiple pulmonary infarcts of various ages, involving the greater portion of both lungs.

Upon reanalysis of the electrocardiograms in the light of the pathologic findings and the observations of Kossmann, it was concluded that the prominent late R waves in Leads V3R and V1 of the first tracing were a manifestation of right ventricular hypertrophy and that the increased amplitude and duration of the Q and R waves in the same leads in the final tracing were referable to a conduction defect in the free wall of the dilated and hypertrophied right ventricle. A right bundle branch block with loss of the customary initial R wave was considered unlikely in view of the hypertrophy of the septum and dilatation of the right ventricle.

B. QR Complex of Normal Duration with Relatively Deep Q Wave in Right Precordial Leads. This finding, resulting in abnormal ratios in the range customarily associated with myocardial infarction, may occur in association with right ventricular hypertrophy, as exemplified by Patients 14 and 15, whose electrocardiograms are reproduced in figure 2, A and
B. The QRS interval was normal in both cases, measuring 0.09 second in the former and 0.08 second in the latter. Lead V₁ of figure 2, A displayed an initial downstroke, 3 mm. in depth and 0.03 second in duration, followed by an upstroke extending 3.5 mm. above the isoelectric line and attaining a peak at the end of 0.06 second. Lead V₂ also showed an equiphasic QR complex, lower in voltage, but comparable in time relationships to the QR deflection of V₁. In Lead V₁ of figure 2, B there was a Q wave 4 mm. in depth and 0.02 second in duration, followed by an R wave extending 8 mm. above the isoelectric line and attaining a peak 0.05 second after the onset of the QRS complex. The QR deflection in Lead V₂ consisted of a downstroke 5 mm. deep and an upstroke of 3 millimeters.

If QR patterns, like those in V₁ and V₂ of figure 2, A and B were recorded in leads facing the epicardial surface of the left ventricle, a diagnosis of infarction of the subendocardial layer of the subjacent left ventricular wall would be justified. However, the V₁ and V₂ positions were over the right atrium in Patient 14, as evidenced by the diphasic P waves with steep intrinsicoid downstroke, and were apparently in the vicinity of the right atrium in Patient 15, as indicated by the fact that distinct f waves of auricular fibrillation were made out in V₁, V₃, and V₅, but not in precordial leads further to the left or in the limb leads.

In the antemortem interpretation of both tracings, the prominent late R waves in V₁ and V₂ were believed to have been derived from the basal portion of the right ventricle and were taken as evidence of right ventricular hypertrophy. Since then, Kossmann and associates have advanced the opinion that the qR deflections registered in leads from the right side of the chest in cases of right ventricular hypertrophy are transmitted from the posterobasal wall of the left ventricle to the right atrium, whereas the Rs deflections recorded in leads from the entire left side of the chest are transmitted from the right ventricle. This necessitated a reanalysis of our cases in the light of their interpretations.

If the potential variations of the posterobasal wall of the left ventricle had carried through to the right precordium to produce the late R wave in V₁, one would have expected better transmission to the right arm and a relatively larger R wave in Lead aV_R. The fact that the late R wave in V₁ of figure 2, B was much greater than that in aV_R, both in actual amplitude and in relative amplitude, was in keeping with a right ventricular origin, but was strongly against a left ventricular source. On the other hand, the similarity of the QR patterns in Leads V₁ and aV_R of figure 2, A did not permit conclusions as to their source and necessitated utilization of other evidence. This was obtained through a comparison of the QRS patterns in the first two with those in the last four precordial leads. The minute initial R and prominent S waves in V₃ were evidently transitional and the findings in V₁, V₃, and those in V₄ through V₆ were thus transmitted from opposite ventricles. The progressive increase in the R at the expense of the S deflection as the electrode was moved from the V₄ to the V₆ position was compatible with transmission from the anterolateral wall of the left ventricle, but not from the apex of the right ventricle. The comparative QRS patterns in the last four precordial leads constituted strong indirect evidence, signifying that the QR complexes in V₁ and V₂ were derived chiefly from the right ventricle. Hence, reanalysis of these electrocardiograms in the light of the evidence brought forward by Kossmann and associates confirmed the original conclusion, namely, that the findings in V₁ and V₂ of both cases were referable to right ventricular hypertrophy.

Patient 14 was a 29 year old housewife, who gave a history of rheumatic heart disease since the age of 15 years and congestive heart failure of three months’ duration, precipitated by pregnancy. The electrocardiogram in figure 2, A was taken on admission, while the patient was in advanced congestive failure, but after partial digitalization. Death occurred two weeks later, as a result of pulmonary infarction, and autopsy revealed fish-mouth mitral stenosis with marked right ventricular hypertrophy, the right ventricle equaling the left in weight. There was no evidence of myocardial infarction.

Patient 15 was a 52 year old man, who had had rheumatic heart disease since the age of
21 years and was admitted to the hospital with acute congestive failure secondary to lobar pneumonia. The electrocardiogram reproduced in figure 2, B was obtained after the administration of 0.8 mg. of Cedilanid. Death occurred four days later and autopsy revealed a 478-gram heart with fish-mouth mitral stenosis and marked right ventricular hypertrophy. The basal portion of the right ventricular wall measured 1.3 cm. in thickness, whereas the apical portion of the left ventricular wall measured 1.4 cm. in thickness. There was no evidence of myocardial infarction. It was, therefore, concluded that the abnormal QR patterns in Leads V1 and V2 of this and the preceding patient were due to right ventricular hypertrophy.

C. Sharp Inversion of the T Wave without the Customary RS-T Depression in Leads From the Right Precordium. This finding may occur in association with right ventricular dilatation and hypertrophy and the pattern may resemble the "coronary" T wave. This is exemplified by Lead V1 of Patient 16, figure 2, C. This patient was a 25 year old man, who gave a history of rheumatic heart disease since the age of 19 years and was admitted to the hospital because of sudden severe orthopnea and pleural pain referable to pulmonary infarction.

The electrocardiogram was obtained on admission before the administration of cardiac glycosides. The terminal portion of the inverted T wave of V1 was interrupted by a large diphasic P wave. The broad slurred postintrinsicoid negative phase of this P wave was ascribed to left auricular hypertrophy. The QRS interval was 0.08 second. Careful scrutiny of Lead V1 revealed an rsR' complex of the type previously attributed to incomplete right bundle branch block. However, as Kossmann and associates4 have pointed out, the brief duration of the initial R wave was compatible with normal septal activation and militated against a conduction defect in the septum. The prominent late R' deflection was attributed to activation of the hypertrophied free wall of the right ventricle and was indicative of right ventricular hypertrophy. It is noteworthy that the minute initial R wave of V1 could not be made out in Lead V3R. This lead displayed merely a qR complex comparable to the sR' deflection of V1. The slight elevation of the RS-T junction in V1 and the sharp inversion of the T wave resulted in a pattern suggestive of the "coronary" T wave. However, sharp inversion of the T wave of this degree may be associated with right ventricular hypertrophy, particularly when accompanied by acute right ventricular dilatation, as in acute pulmonary embolism. The elevation, rather than the customary slight depression of the junction following the rsR' complex in V1, was unusual in right ventricular hypertrophy, but could be explained by superimposition of a prominent upright auricular T wave. The latter would have been expected after the deep, broad negative phase of the P wave.

Death occurred on the third hospital day from infarction of both lower lobes and autopsy revealed a fish-mouth mitral valve. The right ventricle was markedly hypertrophied and almost equaled the left ventricle in weight and thickness. The base of the right ventricle was 1.2 cm. in thickness, whereas the anteroapical wall of the left ventricle was 1.4 cm. in thickness. There was no evidence of myocardial infarction. Thus, the rsR' complex and the sharply inverted T wave of Lead V1 were referable to right ventricular hypertrophy.

D. Localized Reduction in the Amplitude of the R Wave at the Transitional Zone. This is a common finding in uncomplicated right ventricular hypertrophy and must be differentiated from localized reduction in the amplitude of the R wave in a left ventricular lead occurring as a residue of healed anteroseptal infarction.1-7

The initial R wave in Lead V3 in Case 14, figure 2, A, was reduced below 1 mm., but was readily recognized as a manifestation of the transitional zone by comparison with patterns in leads to the right and left. In the presence of definite signs of right ventricular hypertrophy in V1 or in both V1, and V2, no significance should be attached to localized reduction in the amplitude of the R wave in a lead further to the left unless one can be certain that the electrode is well beyond the transitional zone and over the anterolateral wall of the left ventricle.

E. QS Deflection in Leads at the Transitional Zone. This may occur in association with uncomplicated right ventricular hypertrophy and
may be mistakenly interpreted as due to anteroseptal infarction. The diagnostic difficulties are exemplified by Leads V₂ and V₄ in Case 15, figure 2, B. These leads displayed a QS deflection containing an R equivalent in the form of slurring located near the beginning of the downstroke. These findings would have been interpreted as evidence of old anteroseptal infarction, if a normal rS deflection had been present in right ventricular Leads V₁ and V₂ or if an abnormal Q wave had been recorded in left ventricular Lead V₆. The QS deflections in V₃ and V₄ and the minute R wave and early notch on the downstroke of the S wave in V₆ were considered a transitional zonal effect because of the prominent late R wave referable to right ventricular hypertrophy in Leads V₁ and V₂ and because of the distinct initial upstroke in V₆, the first lead distinctly to the left of the transitional zone. In patients with right ventricular enlargement, the pathway of the electrode is often parallel to the long axis of the septum, and the electrode may lie directly over the anterior terminus of the septum in positions V₂, V₃, and/or V₄. The QS complex occasionally recorded in such leads in the absence of infarction probably represents cavity potentials, transmitted because of cancellation of opposing vectors, derived from septal activation by impulses proceeding from its two endocardial surfaces.

The clockwise rotation commonly associated with right ventricular dilatation and hypertrophy facilitates transmission of the potential variations of the transitional zone or right ventricle to the left arm. The QS deflection in Lead aV₁ was attributed to transmission of the potential variations of the transitional zone rather than to lateral infarction in the ante-mortem interpretation, but high precordial and axillary leads should have been taken for more definite exclusion of the latter.

The foregoing electrocardiographic analysis was confirmed at autopsy, which, as already described, revealed fish-mouth mitral stenosis, marked right ventricular hypertrophy, and no evidence of infarction. A number of microscopic blocks were made in order positively to exclude the presence of infarction.

F. Persistence of the Normal Q Wave in Left Ventricular Leads Accompanied by Marked Reduction in the R and Exaggeration of the S Wave. This condition, when secondary to right ventricular hypertrophy, may result in a precordial electrocardiogram that may be readily mistaken as representative of anterolateral infarction. This is illustrated by figure 2, D, reproducing the electrocardiogram in Case 17. The patient was a woman, 74 years of age, with a marked pigeon-breast deformity. She was hospitalized because of strangulated hernia and was studied electrocardiographically during convalescence from an exteriorization of the gangrenous bowel together with ileostomy.

In Lead V₁, a normal rS complex was recorded; in Lead V₂, a QS deflection, containing an R equivalent in the form of a small early notch; and in V₃, V₄, V₅, and V₆, a triphasic QRS was registered. The last four leads displayed a Q wave of constant depth and duration (2 mm. in amplitude and 0.02 second from onset to nadir), an R wave which decreased in height as the electrode was moved to the left, and a prominent S wave throughout. This latter finding, together with the large R wave in Lead aV₃R, raised the question of right ventricular hypertrophy and led to the taking of Lead V₃R. This lead showed an rS complex like that in V₁, and thus failed to support the suspicion of right ventricular hypertrophy. An antemortem diagnosis of old healed anterolateral infarction was accordingly made because of the QS deflection in V₂ and the abnormal QR ratio in V₃ through V₆ in the presence of a normal rS complex in V₁R and V₁. The notched QS deflection in Lead aV₇ was interpreted as evidence of continuation of the infarct into the posteroapical wall of the left ventricle.

Death occurred four weeks after admission following a second operation, and autopsy revealed chronic cor pulmonale with moderate right ventricular hypertrophy, as evidenced by a ratio of 1.2 and a right ventricular thickness of 1.0 centimeter. No evidence of infarction was found on careful gross examination or in multiple microscopic blocks.

Upon reconsideration of the electrocardiogram in the light of the pathologic findings, it would appear that more significance should have been placed on the prominent double-
peaked R wave in Lead aV₉. The fact that the ventricular complex in aV₉ began with an upstroke rather than a Q wave indicated transmission from the right rather than the posterobasal wall of the left ventricle. The double-peaked R of right ventricular origin in aV₉ thus contrasted sharply with the qR deflection of left ventricular origin in aV₉. The initial upstroke in aV₉ was probably due to passage of the impulse through the septum, the second upstroke to activation of the hypertrophied free wall of the base of the right ventricle. An attempt to trace the source of the QRS pattern in aV₉ would probably have led to the correct ante-mortem diagnosis and should have been made in this case because of the chest deformity and the discrepancy between the findings in aV₉ and V₉. The fact that the Q wave in left ventricular Leads V₂ through V₆ was normal in duration, constant both in size and duration, and essentially the reciprocal of the initial R wave in V₁ and aV₉ should have led to the suspicion that it was a normal manifestation of septal activation rather than a result of infarction. The notched QS in V₂ was evidently a manifestation of the transitional zone between the anterior walls of the right and left ventricles, whereas the notched QS in Lead aV₉ was apparently due to predominant transmission of the potential variations of the posteroseptal region to the left leg.

II. RIGHT VENTRICULAR DILATATION

The electrocardiographic diagnosis of right ventricular dilatation is difficult in the absence of classic signs of associated hypertrophy. Findings referable to right ventricular dilatation are likely to be mistaken for those of anteroseptal myocardial infarction. One reason for the confusion is that Leads V₁, V₂, and V₃ may reflect the potential variations of the free wall of the right ventricle, the right side of the septum, and, to a variable extent, the anterior wall of the left ventricle. Acute right ventricular dilatation may be manifested by striking, though not pathognomonic, changes in the RS-T segments and T waves in Leads V₁, V₂, and V₃, which resemble those found in the same leads in the presence of acute infarction of the anteroseptal wall of the left ventricle.

Another reason for the confusion is that enlargement of the right ventricle, together with the usually associated dilatation of the right atrium and clockwise rotation of the heart, facilitates the predominant transmission of the potential variations of the apical portion of the right ventricle as far to the left as the midclavicular line, sometimes to the anterior axillary line, and occasionally even to the midaxillary line. Furthermore, the transitional zone is not only displaced to the left into Leads V₄, V₅, and/or V₆, but is likely to be broad, probably owing to a shift in the long axis of the septum to a plane more nearly parallel with the pathway of the electrode. The anatomic relationship of the heart to fixed points on the chest wall is further altered when the right ventricular dilatation is associated with pulmonary emphysema and lowering of the diaphragms. Under the foregoing circumstances, errors may be made if one fails to recognize that the patterns in leads from the left precordium are transmitted predominantly from the right ventricle and interprets the findings as if the patterns had been derived from the left ventricle.

The electrocardiographic features of right ventricular dilatation that might be mistaken for those of myocardial infarction include: (A) sharp inversion of the T waves with elevated or isoelectric RS-T junctions in the first three or four precordial leads; (B) rapid changes in the direction and amplitude of the T waves of right precordial leads in serial tracings; (C) QS patterns in the first three or more precordial leads; (D) localized reduction in the amplitude of the initial R wave as the electrode is moved leftward from the V₁, and V₂ positions, particularly when accompanied by a change from an upright to an inverted T wave; (E) progressive diminution of the initial R wave as the electrode is moved leftward from the V₁, and V₂ positions into the transitional zone, particularly when accompanied by change from upright to inverted T waves; (F) replacement of the initial R wave by a QS deflection or W-shaped complex in leads near the transitional zone, particularly when accompanied by inversion of the T wave. These features are collectively illustrated by the electrocardiograms in Cases 18 to 27, inclusive, reproduced in figures 3, 4, and 5.
A. Sharp Inversion of the T Wave with or without Elevation of the RS-T Segment in the First Three or Four Precordial Leads. This may occur as a manifestation of acute right ventricular dilatation\textsuperscript{14-18} and may resemble the RS-T changes associated with recent anteroseptal infarction. It is illustrated by the electrocardiograms reproduced in figure 3, obtained in Cases 18 to 21, inclusive.

Patient 18 was a woman, aged 68 years, who was admitted because of a senile psychosis. Bilateral femoral phlebothrombosis developed in the hospital and was complicated by repeated pulmonary embolism. The electrocardiogram reproduced in figure 3, A was obtained after the development of pulmonary infarction, but before the administration of digitalis. The first three precordial leads showed elevation of the RS-T junction and inversion of the terminal portion of the T wave. Anteroseptal infarction was excluded as a cause of the T-wave inversion in these leads because of (1) the presence of upright T waves in Leads V\textsubscript{4} and V\textsubscript{5} nearer the anteroseptal wall of the left ventricle, (2) the presence of an initial upstroke in all precordial leads, and (3) a progressive increase in the amplitude of the R wave as the electrode was moved to the left. These features, together with the displacement of the transitional zone to the left, led to an electrocardiographic diagnosis of acute right ventricular dilatation.

The patient died of pulmonary embolism three and one-half weeks after admission. There was marked dilatation of the right ventricle and atrium and moderate right ventricular hypertrophy, as indicated by a ratio of 1.1 associated with a cardiac weight of 338 grams.

There was mild coronary sclerosis without narrowing or evidence of infarction. The electrocardiographic findings were believed referable to right ventricular dilatation and failed to disclose the hypertrophy also found at autopsy.

The electrocardiogram reproduced in figure 3, B was obtained from Patient 19 prior to the administration of digitalis. This patient, a housewife aged 47 years, gave a history of pollen asthma since childhood and increasing exertional dyspnea for several years and was admitted to the hospital with left ventricular failure complicated by miliary tuberculosis.
FIG. 4.—Serial changes in acute right ventricular dilatation. A, Case 22; B, Case 23.
The striking features of the electrocardiogram consisted in upward bowing of the RST-segments and sharp inversion of the T waves in Leads V1, V2, and V3. Despite the covelike contour of the T waves, the findings could be ascribed to acute cor pulmonale rather than anteroseptal infarction because of (1) the normal upright T wave in V4, the first lead to the left of the transitional zone; (2) the absence of Q waves from V1 to V4 and the progressive increase in the R waves of these leads; (3) the doubling of the initial R wave in Leads V2 and V3 near the transitional zone (indicating right hypertrophy of hypertensive origin. There was slight hypertrophy and marked dilatation of the right ventricle and atrium, due in part to chronic pulmonary emphysema, in part to recent passive congestion. The miliary tuberculosis did not involve the heart and there was no evidence of pericarditis, subepicardial myocarditis, infarction, or coronary narrowing. The T-wave abnormalities were thus referable to right ventricular dilatation.

The electrocardiogram reproduced in figure 3, C was obtained from Patient 20, a man aged 59 years, hospitalized because of carcinoma of the cecum and thrombophlebitis of the right femoral vein, complicated by repeated pulmonary embolism. No cardiac glycosides were given. The tracing is presented because of the deeply inverted T waves in Leads V1 through V4, resembling those associated with organizing myocardial infarction. However, the electrocardiographic findings were attributed to acute cor pulmonale for essentially the same reasons as in the two preceding cases, namely: (1) the limitation of the inverted T waves to right ventricular leads, (2) the normal initial R waves in these leads.
Death occurred three weeks after the tracings were made from carcinomatous perforation of the cecum and autopsy revealed multiple pulmonary infarcts. The heart weighed 375 grams and showed dilatation of the right ventricle and atrium without hypertrophy. The coronary vessels were moderately sclerotic, but patent throughout. Epicardium and myocardium appeared normal on gross and histologic examination.

Patient 21 was a man, aged 73 years, admitted to the hospital because of a perforated peptic ulcer. Convalescence was uneventful until the ninth postoperative day, when he had a sudden attack of dyspnea and weakness. The electrocardiogram reproduced in Fig. 3, D was obtained two hours later, while the patient was in shock. No cardiac glycosides had been given.

Leads V₁ and V₂ displayed an rSr' complex, indicating that the electrode faced the epicardial surface of the right ventricle, whereas Lead V₃ showed an Rs deflection, signifying that the electrode had crossed the transitional zone. Slurring was present near the end of the intrinsicooid deflection in V₁ and was simultaneous with the R' deflection of V₁ and V₂, but later than the peak of the R in left ventricular leads V₄, V₅, and V₆. Incomplete right bundle branch block was excluded as a possible cause of the pattern in V₁ and V₂ because of the brevity of the initial R wave. The r' deflection in these leads was not derived from the posterobasal wall of the left ventricle because of the absence of a larger late upstroke from Lead aV₉. Activation of the conus pulmonalis was believed responsible for the r' deflection in V₁ and V₂ and the simultaneous slurring of the descending limb of the R wave in V₃.

A more striking feature of the tracing was the RS-T pattern, characterized by elevation and upward convexity of the RS-T segment and terminal inversion of the T wave in Leads V₁, V₂, V₃, and aV₉ and by depressed segments and upright T waves in V₄, V₅, and aVF. Three possibilities were considered: (1) acute myocardial infarction reaching the epicardium in the anteroseptal aspect of the left ventricle and confined to the subendocardial layer in the anterolateral and posterior aspects; (2) recent infarction localized to the anteroseptal aspect of the left ventricle with reciprocal RS-T depression in lateral and posterior leads; (3) acute cor pulmonale with ischemia of the anterolateral and posterior walls of the left ventricle, secondary to shock. The preservation of the initial R wave in all precordial leads was against infarction, but did not exclude the possibility of a very recent lesion. On the other hand, the early transition and the RS-T elevation in left ventricular Lead V₃ were against cor pulmonale, but did not exclude it because Lead V₃ also apparently reflected potential variations of the nearby conus pulmonalis. A positive differentiation, therefore, could not be made from this electrocardiogram.

The patient made a complete symptomatic recovery by the next day and had no further complaints until the thirteenth postoperative day, when a second attack occurred, terminating fatally within fifteen minutes. Autopsy revealed a massive terminal and previous smaller pulmonary emboli. The heart weighed 440 grams and showed right ventricular dilatation and hypertrophy. There was minimal coronary sclerosis, but no narrowing and no evidence of myocardial infarction or pericarditis. The RS-T elevation in V₁, V₂, and V₃ and aV₉ was, therefore, a manifestation of acute cor pulmonale. The RS-T depression in V₄, V₅, and aVF may have been reciprocal to the elevation in right precordial leads, but was more likely due to acute left ventricular ischemia secondary to shock.

B. Rapid Changes in the Inverted T Waves of Right Precordial Leads in Serial Tracings. These changes also occur in association with acute cor pulmonale 14-18 and may be mistaken for those due to recent myocardial infarction. The problem in diagnosis is illustrated by figure 4, which represents the electrocardiograms in Cases 22 and 23, respectively.

Patient 22 was an obese woman, aged 54 years, who gave a history of hypertension of two years’ duration and increasing congestive failure for three weeks. Auricular fibrillation was consistently present. The electrocardiogram of June 15, 1945, figure 4, A, was obtained on the first hospital day after the administration of 0.8 mg. Cedilanid. Leads V₄ and V₅ displayed prominent late R waves,
slightly depressed RS-T junctions, and sharply inverted T waves compatible with the presence of left ventricular hypertrophy. The T wave was upright and of normal contour in right ventricular leads V₁, V₂, and V₃ and the RS-T junction was isoelectric in V₁ and elevated 1.0 mm. in V₂ and V₃. Slight inversion of the T wave was present in transitional lead V₄.

A second tracing made the following day after full digitalization, but not reproduced in figure 4, showed the classic straightening of the RS-T segments, but no change in the direction of the T waves. A constant maintenance dose of 1½ grains digitalis daily was given for the remainder of her hospital stay.

The course was uneventful until June 21, when she was suddenly seized with dyspnea and stabbing pain in the right side of the chest. Repeat electrocardiogram on June 23 showed striking changes in the RS-T complexes, particularly in right precordial leads. The RS-T junction in V₁ had become elevated and the T wave sharply inverted; the RS-T junction in V₂ showed increased upward displacement and the T wave had also become sharply inverted. Although these RS-T changes resembled those associated with acute anteroseptal infarction, they were definitely attributed to acute cor pulmonale because (1) the RS-T elevation and T-wave inversion were more marked in Leads V₁ and V₂ over the right ventricle than in Leads V₂ and V₄ over the transitional zone and anterior wall of the left ventricle, (2) the absence of reduction or obliteration of the normal initial R waves in the first four precordial leads. It is noteworthy that the terminal portion of the T wave had become upright in the last three precordial leads, thereby maintaining its reciprocal relationships with the T waves in right precordial leads.

The patient was symptom free on June 29 and the electrocardiogram had returned to the original configuration. The rapid change from sharply inverted to upright T waves in Leads V₁ and V₂ together with the decrease in upward displacement of the RS-T junctions was typical of the evolution accompanying recovery from acute cor pulmonale.

The patient had a second attack of abrupt dyspnea and pleural pain on July 1 and died suddenly on July 8, 1945. Death was due to pulmonary embolism and autopsy revealed organizing pulmonary infarcts. The heart weighed 729 grams and showed marked left ventricular hypertrophy and right ventricular dilatation, but no evidence of myocardial infarction or pericarditis.

Patient 23 was a man, aged 47 years, who gave a history of exertional dyspnea for eleven months and paroxysmal nocturnal dyspnea for three months. He was admitted to the hospital during an exceptionally severe attack of acute pulmonary edema.

The electrocardiogram of November 16, 1944, figure 4, B, was obtained soon after admission, following the administration of 3 grains of digitalis leaf. Leads V₃ and V₄ displayed large R waves, slightly delayed intrinsicoid deflections, and diphasic to inverted T waves indicative of left ventricular hypertrophy. The deep broad S waves in right ventricular leads V₁, V₂, V₃, and V₄ were compatible with left ventricular hypertrophy, but the slightly elevated RS-T junctions and cove negative T waves in the same leads could not be explained in this manner.

The RS-T pattern in the first four precordial leads raised the question of recent anteroseptal infarction, ischemia of the anteroseptal wall of the left ventricle, acute pericarditis, and acute right ventricular dilatation. The possibility of acute anteroseptal infarction was virtually excluded by the normal initial R waves in the first four precordial leads. Ischemia of the anteroseptal wall of the left ventricle was regarded as an unlikely factor because of (1) the absence of RS-T depression and (2) the greater depth of the T wave in V₂ than in leads nearer the anteroseptal wall of the left ventricle (V₃ and V₄). The possibility of pericarditis as a cause of the inverted T waves in the first four precordial leads was practically excluded by their replacement by upright T waves of normal contour on the following day. On the other hand, the rapid evolution was consistent with acute cor pulmonale and could be correlated with marked clinical improvement and disappearance of pulmonary edema under therapy, which included 4½ grains of additional digitalis. The RS-T changes
in V₆ and V₆ could be explained by digitalis effect.

The patient died of "malignant" hypertension two months after the tracings of November 16 were made. The heart weighed 672 grams and showed marked left ventricular hypertrophy, but no evidence of infarction or pericarditis. The coronary tree was patent throughout and of normal caliber. There was no evidence of pulmonary infarction. In view of the clinical findings during the first two hospital days together with the subsequent autopsy findings, it was concluded that the RS-T patterns in Leads V₁, V₂, V₃, and V₄ on November 16 were due to acute right ventricular dilatation secondary to extensive pulmonary congestion and edema from acute left ventricular failure.

C. QS Pattern in the First Three or More Precordial Leads. This may occur in association with right ventricular dilatation, with or without accompanying hypertrophy. Zuckermann and associates studied 50 cases of chronic cor pulmonale without clinical signs of myocardial infarction and found either a QS complex, a W-shaped QRS, or a deep Q wave in right precordial leads of 36 per cent of the series. Nevertheless, when a QS deflection is recorded in the first three or more precordial leads, the possibility of anterior infarction extending into the septum must be given serious consideration and a study of additional leads must be made for differentiation from right ventricular dilatation, and from the rare normal variant associated with marked clockwise rotation and/or displacement of the heart to the left.

The problem in differential diagnosis afforded by QS deflections in the first five precordial leads is illustrated by figure 5, A. This electrocardiogram was obtained from a 45 year old man (Patient 24) soon after admission to the hospital with advanced congestive failure and following partial digitalization. Auricular fibrillation was present and was manifested by coarse f waves in V₁, varying fine to moderately coarse f waves in V₂ and V₃ and no undulations in the remaining precordial leads or in any of the limb leads. This suggested that the electrode was in the vicinity of the right atrium at position V₁ and probably also at V₂ and V₃.

The precordial leads in this tracing did not cover a sufficiently wide area to provide adequate exploration of both the right and left ventricles, as shown by the lack of contrasting patterns in the leads at either end (V₁ and V₆ positions). In fact, the change from a QS in V₅ to an rS deflection in V₆ suggested that the electrode was approaching the transitional zone and that Leads V₄ and V₅, as well as V₁, V₂, and V₃, reflected the potential variations of the right side of the septum and epicardial surface of the right ventricle. The absence of a Q wave preceding the upstroke in transitional lead V₅ was against infarction continuing from the septum into the anterior wall of the left ventricle.

The question remained as to whether the QS deflection in the first five precordial leads was due to healed infarction confined to the septum or merely to right ventricular dilatation. To settle this question, a repeat electrocardiogram, including leads to the right of the V₁ position and beyond the V₆ position, was necessary. This was obtained after restoration of compensation, and still showed auricular fibrillation with prominent f waves confined to Leads V₃R and V₁. These two leads displayed a QS deflection; V₂, V₃, and V₄ showed an rS complex, the initial upstroke increasing from 1 mm. in V₂ to 3 mm. in V₄; Lead V₅ exhibited an equiphasic RS deflection of transitional origin and Lead V₆ showed a prominent late R wave suggesting left ventricular hypertrophy.

The second electrocardiogram thus revealed no evidence of infarction and consequently ruled out septal infarction as a cause of the QS deflections in the first tracing. Although it was impossible positively to exclude an erroneously high position of the electrode or an abundant application of electrode jelly over the entire pathway of the electrode as an explanation for these QS deflections, the findings could be adequately accounted for on the assumption of a technically accurate tracing. Thus, the registration of prominent f waves as far as the V₁ position, QS deflections as far as the V₅ position, and the transitional zone beyond midaxilla in the first tracing, was compatible with marked dilatation of the right atrium and ventricle, and marked clockwise rotation associated with advanced congestive failure. The subsequent limitation of f and QS waves to the V₃R
and V1 positions and shift of transitional zone to the anterior axillary line in the second tracing, made after restoration of compensation, was compatible with reduction in distention of the right atrium and ventricle and decrease in the degree of clockwise rotation.

The patient died, on the twenty-second hospital day, of intercurrent pneumonia. Autopsy revealed a 566-gram heart, with left ventricular hypertrophy associated with rheumatic mitral insufficiency and hypertension and accompanied by marked right ventricular dilatation. There was no evidence of myocardial infarction. The necropsy findings thus supported the above antemortem interpretation of the electrocardiograms.

D. Localized Reduction in the Amplitude of the Initial R Wave in a Lead to the Left of V1 or V6. This may occur in association with right ventricular dilatation, but may raise the question of anteroseptal infarction, particularly when accompanied by change from an upright to an inverted T wave. This is exemplified by figure 5, B, obtained from Patient 25, a hypertensive man, aged 55 years, who was admitted to the hospital with advanced congestive failure complicated by bronchopneumonia. No cardiac glycosides were given.

The inversion of the T wave in left ventricular leads V5 and aV3, and the slight slurring and prolongation of the ascending limb of the R wave in aV3, suggested left ventricular hypertrophy. Small r and relatively deep S waves were recorded in the first five precordial leads. The initial R wave measured 1.0 mm. in V1, 1.5 mm. in V5, 2.5 mm. in V3, then decreased to 1.5 mm. in V4 and became slurred and 2.0 mm. in amplitude in V6. The localized decrease in the amplitude of the R wave in V4 and V6 might raise the question of anteroseptal infarction, but the accompanying diminution in voltage of the S wave and slurring of the QRS complex suggested that it constituted a manifestation of the transitional zone. The change from an upright T wave with concave RS-T segment in V1, V5, V3, and V4 to an inverted T wave with convex RS-T segment in V5 might also raise the question of anteroseptal infarction. However, the resemblance of the T wave in V6 to that in V5 suggested that it, too, was a manifestation of left ventricular hypertrophy. The findings in V6 could be explained by the assumption that (1) the electrode faced the right ventricular side of the septum during the registration of the QRS, (2) the heart rotated sufficiently during systole so that the electrode faced the left ventricular side of the septum during registration of the T wave. A diagnosis was therefore made of left ventricular hypertrophy and right ventricular dilatation with displacement of the transitional zone to the anterior axillary line.

Death, due to congestive failure and bronchopneumonia, occurred nine hours after admission to the hospital. The heart weighed 490 grams and showed left ventricular hypertrophy, due to hypertension, and acute right ventricular dilatation. The coronary vessels were patent and there was no evidence of infarction. The postmortem findings thus confirmed the electrocardiographic interpretation.

E. Progressive Diminution of the Initial R Wave, Accompanied by Change From an Upright to an Inverted T Wave. As the electrode is moved from the V1 or V2 positions towards the left, progressive diminution of the initial R wave, accompanied by change from an upright to an inverted T wave, may occur in association with right ventricular dilatation, but may be easily mistaken as representative of anterior myocardial infarction, as exemplified by figure 5, C. This electrocardiogram was obtained from a 66-year-old man (patient 26) soon after admission to the hospital and before the administration of cardiac glycosides.

An rS complex was recorded in all six precordial leads and was of normal voltage in V1, V3, V5, and V6, was reduced by half in V4, and again cut in half in V6. The initial R wave measured 5 mm. in V1 and decreased by approximately 1 mm. in each successive lead up to V6, where it was barely perceptible. The T wave in the first four precordial leads was upright and was accompanied by a normal, upwardly concave RS-T segment in V1, V2, and V3 and a convex segment in V4. The elevated, convex RS-T segments and the inversion of the terminal portion of the T waves in Leads V3 and V6 were abnormal and, along with the marked reduction of the initial R wave, strongly suggested a patchy acute anterolateral infarction. However, the alternative possibility of marked right ventricular dilata-
tation with transitional zonal effects in V5 and V6 had to be considered because of (1) the presence of rS patterns in all six precordial leads and (2) the reduced voltage and slurring to notching of the S wave in V5 and V6.

To settle the diagnosis, it was necessary to take sufficient leads to the right of position V1 and beyond position V4 until contrasting QRS patterns were recorded at the two extremes. An rS deflection like that in V1 and V2 was recorded in V5R and V6R, and V5R, indicating transmission of the potential variations of the right ventricle to the right anterior chest. Lead V1 displayed an Rs complex, consisting of a brief initial upstroke 5 mm high followed by a barely perceptible S wave, isoelectric RS-T junction, and inverted T wave; Lead V5 exhibited a small monophasic R wave and similar RS-T complex. These findings signified that the potential variations of the left ventricle were referred to the left side of the back. The absence of a Q wave from V1 and V5 was strongly against myocardial infarction as the cause of the findings in Leads V5 and V6. The low voltage and slurring of the QRS in these two leads indicated that the electrode was in the vicinity of the transitional zone. The rough correspondence of the RS ratio in V5 and V6 to that in leads further to the right suggested that the electrode lay to the right of the septum during ventricular activation, whereas the inversion of the terminal portion of the T wave, like that in V5, suggested that the heart had rotated sufficiently during systole so that the left ventricle faced towards the anterior axilla.

After study of the additional leads, a diagnosis of right ventricular dilatation was made and the abnormal QRS-T patterns in V5 and V6 were ascribed to the transitional zone. This diagnosis was in accord with the clinical findings. The patient gave a history of asthma for thirty years and a chronic productive cough for five years. Physical examination showed clubbing, barrel chest, and obstructive pulmonary emphysema.

Death occurred on the tenth hospital day. Autopsy revealed chronic bronchitis, obstructive pulmonary emphysema, but no evidence of myocardial infarction. The heart weighed 327 grams and the right atrium and ventricle were dilated. Relative right ventricular hypertrophy was present, as indicated by a ratio of 1.2 and by comparison of the thickness of the two ventricles. The right ventricle measured 1.2 cm. at the base and 0.8 cm. at the apex; the apical third of the left ventricle measured 1.3 cm. anteriorly and 1.8 cm. laterally. The progressive decrease in the amplitude of the R wave as the electrode was moved from position V1 in the vicinity of the tricuspid ring to position V4, which was presumably near the apex of the right ventricle, could be correlated with the progressive decrease in thickness of the right ventricular wall from base to apex. Although the size and thickness of the right ventricle in this patient closely approached that in Patient 16, classical electrocardiographic signs of right ventricular hypertrophy were not found in this patient, but were present in Patient 16.

F. Replacement of the Initial R by a QS Deflection or W-Shaped Complex Accompanied by Change from an Upright to an Inverted T Wave. This may occur in association with right ventricular dilatation, as the electrode is moved from the V1 or V2 positions toward the left, and may lead to an erroneous diagnosis of myocardial infarction, as in Patient 27, whose electrocardiogram is reproduced in figure 5, D. This patient was a man, aged 78 years, who had a barrel chest secondary to obstructive pulmonary emphysema and had been under treatment for hypertensive heart disease for several years. He was admitted to the hospital with marked congestive failure after neglecting to take digitalis for six weeks.

The electrocardiogram was obtained after the administration of 0.8 mg. Cedilanid. Auricular fibrillation was present and the ventricular rate was approximately 110 per minute. The similarity of the rS complexes in the first three precordial leads indicated that the electrode in all three positions faced the epicardial surface of the right ventricle. The depth and the breadth of the S wave in these leads were suggestive of left ventricular hypertrophy. The reduction in amplitude and slurring of the S wave in V4 suggested that the electrode was approaching the transitional zone, but the general correspondence of the RS ratio to that in V1, V3, and V4 indicated that the electrode was still to the right of the septum. The initial R wave failed to show the usual progressive increase in
amplitude in the first four precordial leads, measuring 1.0 mm. in V₁ and V₂ and only 1.5 mm. in V₃ and V₄. More important, however, was the presence of an initial Q wave in V₃ and the reduction of the R wave to a notch on the downstroke of the QS complex. The T waves in V₁ and V₂ were upright and of normal contour, but those in V₃, V₄, and V₅ were inverted and were associated with slightly elevated convex RS-T segments. The T-wave abnormalities in these leads were believed independent of Cediilanid for the following reasons: (1) the contour of the RS-T segment, (2) the relatively long QT interval, (3) the fact that cardiac glycosides tend to make T waves opposite to the main deflection of the QRS. On the basis of the QRS and T abnormalities in V₃, V₄, and V₅, a diagnosis was made of organizing anteroseptal infarction.

In the electrocardiographic interpretation, too little attention was given to the absence of a Q wave from Lead V₅. If an infarct involves enough of thickness of the wall to result in the registration of a notched QS deflection in a given lead, it should extend sufficiently into the subendocardial layer of the surrounding muscle to result in a marginal qR deflection in the next adjacent lead. This discrepancy should have led to repetition of the electrocardiogram, including leads from additional points on the anterolateral aspect of the left side of the chest, but the patient died of pulmonary embolism before this could be undertaken.

Myocardial infarction was not found on careful gross examination and its possibility was definitely excluded by means of multiple microscopic blocks. The coronary arteries were of normal caliber on injection and showed only Grade 1 sclerosis. The heart weighed 553 grams and showed coexistent left ventricular hypertrophy due to hypertension and right ventricular hypertrophy and dilatation, due chiefly to obstructive pulmonary emphysema. Although the right ventricle measured 1.4 cm. in thickness at the base and 0.8 cm. at the apex and the left ventricle, 1.8 cm. in the anterior and 2.5 cm. in the lateral wall, the electrocardiogram failed to show diagnostic signs of either right or left ventricular hypertrophy, perhaps because of the tendency for the effects of the one to neutralize those of the other. In view of the autopsy findings, the noted QS deflection in V₅ was apparently a transitional zonal phenomenon. During the inscription of the QRS, the electrode at position V₅ probably lay over the anterior terminus of the septum and received negative potentials from the ventricular cavities as a consequence of extinction of positive potentials in the center of the septum. The resemblance of the inverted T waves of V₃, V₄, and V₅ to the terminal portion of the inverted T wave of left ventricular lead V₆ suggested that the transitional zone for the T wave was to the right of that for the QRS because of systolic rotation of the heart between inscription of the QRS and T waves.

**Summary**

In uncomplicated right ventricular hypertrophy and/or dilatation, multiple precordial leads often show abnormalities which resemble those associated with infarction of the septum and/or infarction of the anterior wall of the left ventricle. The differential diagnosis is brought out through the presentation of 15 cases selected because of the presence of (1) electrocardiographic findings originally regarded as indicative or at least suggestive of myocardial infarction, and (2) postmortem demonstration of right ventricular hypertrophy and/or dilatation and pathologic exclusion of myocardial infarction.

In 5 of the cases, the electrocardiogram gave evidence of right ventricular hypertrophy, but exhibited one or more of the following signs likely to be mistaken for those due to myocardial infarction: (A) abnormally broad qR deflection in right precordial leads suggestive of right bundle branch block due to septal infarction; (B) Q wave in right precordial leads that was unusually deep in proportion to the succeeding R wave, resulting in abnormal ratios in the range customarily associated with myocardial infarction; (C) cove plane inversion of the T wave in leads from the right precordium; (D) localized reduction in the amplitude of the R wave or replacement by a QS deflection in leads at the transitional zone, suggesting an-
teroseptal infarction; (E) persistence of the normal Q wave in left ventricular leads accompanied by marked reduction in the R and exaggeration of the S wave, suggesting anterolateral infarction.

In the other 10 cases, the electrocardiogram exhibited one or more of the following signs referable to right ventricular dilatation, but suggestive of myocardial infarction: (A) sharp inversion of the T waves with elevated or isoelectric RS-T junctions in the first three or four precordial leads; (B) rapid changes in the direction and amplitude of the T waves of right precordial leads in serial tracings; (C) QS patterns in the first three or more precordial leads; (D) progressive decrease or localized reduction in the amplitude of the initial R wave as the electrode was moved leftward from the V1 or V2 position, accompanied by a change from an upright to an inverted T wave; (E) replacement of the initial R wave by a QS deflection or W-shaped complex in leads near the transitional zone, accompanied by a change from an upright to an inverted T wave.

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QRS-T Patterns in Multiple Precordial Leads that May Be Mistaken for Myocardial Infarction: II. Right Ventricular Hypertrophy and Dilatation
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