THE EFFECTS OF ANTERIOR INFARCTION COMPLICATED BY BUNDLE BRANCH BLOCK UPON THE FORM OF THE QRS COMPLEX OF THE CANINE ELECTROCARDIOGRAM

FRANCIS F. ROSENBAUM, M.D., HERMAN ERLANGER, M.D., NELSON COTRIM, M.D., FRANKLIN D. JOHNSTON, M.D., AND FRANK N. WILSON, M.D.
ANN ARBOR, MICH.

IN MAN the characteristic changes in the form of the ventricular complex usually produced by myocardial infarction are often greatly modified or absent when one of the two main subdivisions of the His bundle is blocked. When the conduction defect is on the left side it is rarely possible to diagnose infarction with certainty by means of either limb leads or precordial leads. Block on the right side usually prevents the occurrence of characteristic signs of anterior infarction in the standard limb leads, but not in the precordial leads. Such data as are available suggest that, as a rule, it does not seriously obscure the diganostic signs of posterior infarction, which ordinarily appear in Leads II and III.

Because the number of cases in which both bundle branch block and infarction were known to be present is relatively small, it seemed expedient to undertake an experimental study of the electrocardiographic changes produced by this combination of lesions. The methods employed in our experiments were those used in the electrocardiographic studies of infarction carried out by Wilson, Hill, and Johnston.[1]

Dogs of large or medium size were used. The heart was exposed, the right or left branch of the His bundle was cut in the usual way, and in most of the experiments the anterior descending coronary artery was ligated in its mid-portion. The chest was then carefully restored. These surgical procedures were carried out under aseptic conditions. After a period of seven to forty days, when the animal had recovered completely from the operation, the electrocardiographic observations were made. The standard limb leads and unipolar precordial leads were taken with the chest intact. Then the thorax was opened by splitting the sternum, and the anterior surface of the heart was explored by means of unipolar direct leads.

The standard limb leads of nine dogs are reproduced in Fig. 1. In the first three animals (57, 58, and 61) the right branch of the His bundle

From the Department of Internal Medicine, University of Michigan Medical School.

The observations reported in this article were made with the aid of a grant from the Horace H. Rackham School of Graduate Studies. They were summarized in a preliminary report published in the Proceedings of the Thirty-Third Annual Meeting of the American Society for Clinical Investigation, May 5, 1941 (J. Clin. Investigation 20: 460, 1941).

Received for publication May 15, 1943.

was cut, but there was no gross infarction, either because the anterior descending coronary artery was not ligated or because the attempt to occlude it was unsuccessful. The electrocardiograms of these animals are of the kind ordinarily seen in canine right branch block. Below these curves are three sets from animals (59, 62, and 66) with both right branch block and anterior infarction. It will be noted that no changes characteristic of infarction appeared in the ventricular complexes of the limb leads. In dogs with normal intraventricular conduction, infarcts similar in size and location to those induced in these experiments usually give rise to large Q deflections in Lead I.

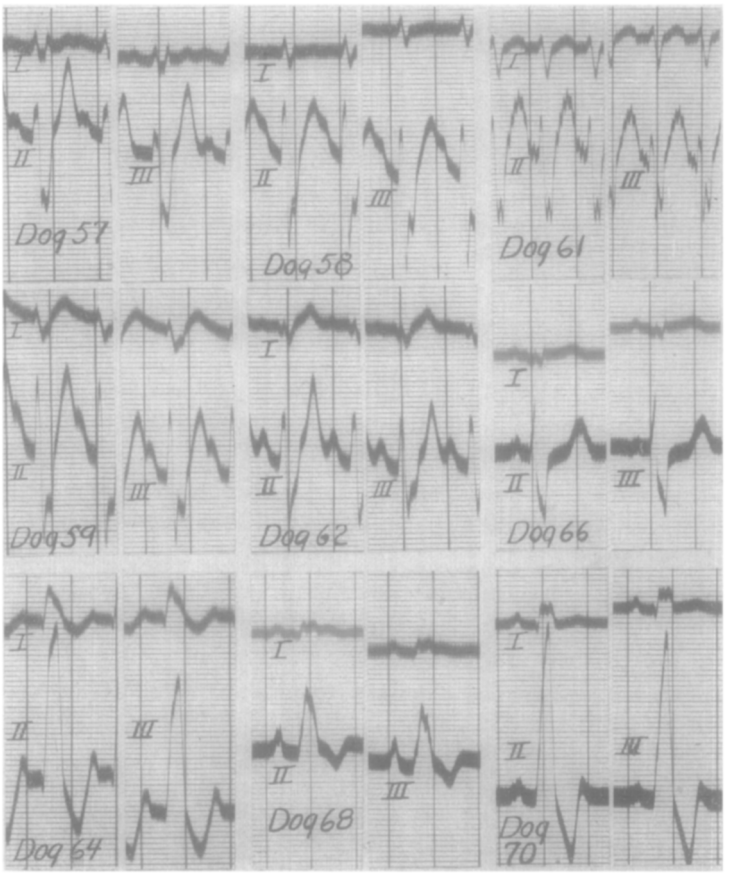

Fig. 1.—Standard limb leads with simultaneous Lead I. Upper row—three dogs with right bundle branch block. Middle row—three dogs with both right branch block and anterior infarction. Lower row—Dog 64 had left bundle branch block; Dogs 68 and 70 had left branch block and anterior infarction.

The first set of curves of the last row (Fig. 1) are from an animal (64) with left branch block only, and the second and third sets are from animals (68 and 70) with both left branch block and anterior infarction. Here also the limb leads display no changes in the ventricular complex that suggest the presence of infarcted cardiac muscle.

The precordial curves were obtained by moving the exploring electrode across the precordium in the same way as when taking comparable human curves. The exploration was begun at a point to the right of the right margin of the sternum, and was extended to a point well to the left of the apex beat. The electrode consisted of a stiff copper wire, insulated by enamel except at the ends. One end was sharpened and was brought into contact with the subcutaneous tissues by making a small slit in the skin. The enamel was removed from the opposite end for a

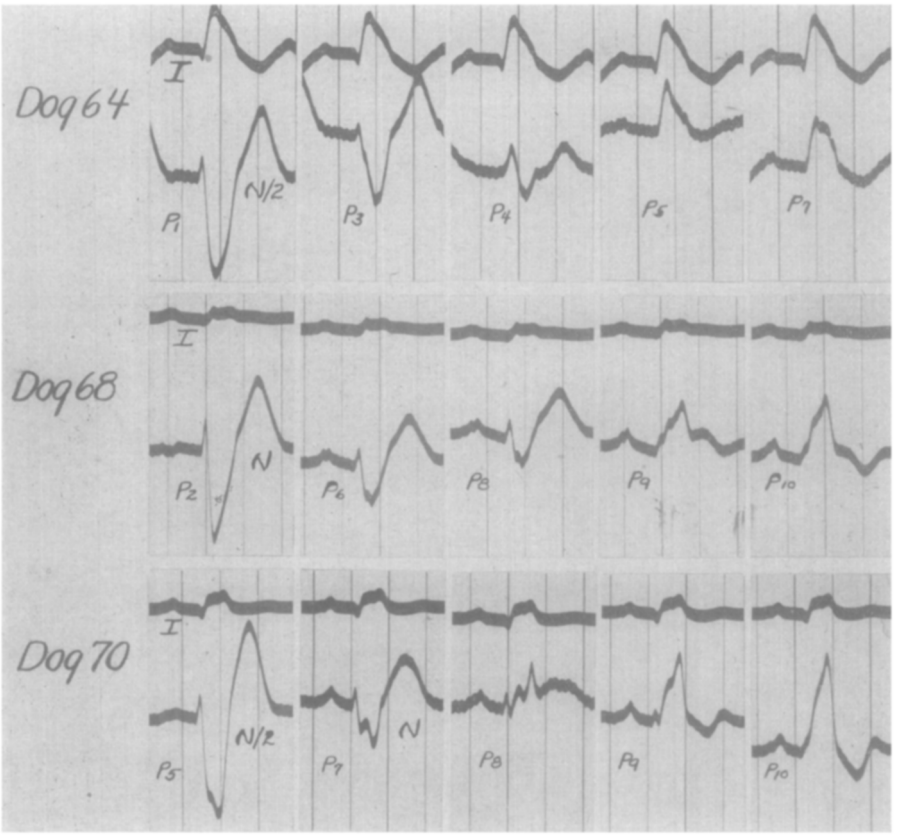

Fig. 2.—Unipolar precordial leads. Dog 64 had left bundle branch block. Dogs 68 and 70 had left branch block and anterior infarction.

short distance so that the electrode could be connected to the appropriate lead wire. The precordial points explored were arranged along a broken line running across the precordium from right to left, and were approximately 2 cm. apart. The exploring electrode used in taking both the precordial and the direct leads was paired with a central terminal connected through resistances of 5,000 ohms to electrodes on the two forelegs and on the right hind leg. Leads taken in this way are referred to as "unipolar leads."

The unipolar precordial curves obtained in uncomplicated canine bundle branch block are strikingly similar to those that represent human bundle branch block, apart from the length of the QRS interval, which is considerably shorter in the dog than in man. In left branch block there are no other easily detectable differences. In leads from the right side of the precordium the QRS complex consists of a small initial R component, followed by a deep, broad S deflection. In leads from the left side of the precordium it is monophasic, and is represented by a broad-topped, slurred, notched, or bifid R wave (Fig. 2, Dog 64).

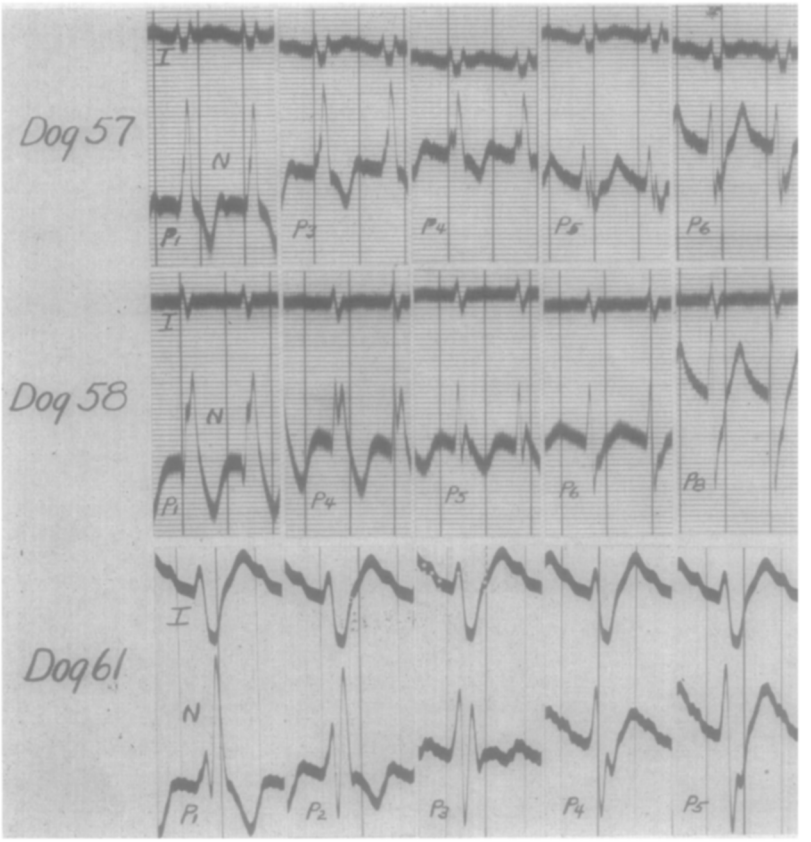

Fig. 3.—Unipolar precordial leads from three dogs with right bundle branch block.

In right bundle branch block the leads from the right side of the precordium display a large R or R′ deflection which reaches its apex late in the QRS interval. In some instances this deflection is preceded by an initial downward movement, or Q wave (Fig. 3, Dogs 57 and 58), in which case its ascending limb is usually slurred or notched. In other instances (Fig. 3, Dog 61) it is preceded by a small initial R component, followed by a conspicuous dip, and notching of its upstroke is absent or less conspicuous. As the exploring electrode is shifted to

the left this initial R rapidly increases in height and is transformed into a slender, tall, upward deflection which reaches its apex early in the QRS interval. At the same time, the downward movement which follows it increases in depth, and the late secondary R′ component rapidly diminishes in size until it is represented by a notch on a deep, broad S wave. Precordial electrocardiograms of this second type are very much more common in human right branch block than those of the first type. In leads from the left side of the precordium the QRS complex always has essentially the same form, and consists of a narrow R deflection, sometimes preceded by a small Q, and a broad, slurred, or notched S wave. The voltage of the slender R is usually smaller in comparison with that of the broad S in the curves of the dog than in those of man.

When right branch block is complicated by anterior infarction (Fig. 4), leads from the right side of the precordium display a tall, late R wave, preceded by a deep Q deflection. As the exploring electrode is shifted to the left the R wave rapidly diminishes in size, and disappears or is submerged below the isoelectric level. In leads from the left side of the precordium the early, slender R wave which is present when there is no infarction is absent, submerged, or greatly reduced in height. In other words, the characteristic changes in the QRS complex consist in the development of large Q or QS deflections in leads from points overlying the infarct, and large Q waves, followed by late R waves, in leads from the right side of the precordium. In one of our experiments (Dog 66) the QRS complex of the lead taken farthest to the right (P_{10}, Fig. 4) showed no changes suggesting infarction. When left bundle branch block is present, anterior infarction gives rise to no characteristic modification of the QRS complex in precordial leads (Fig. 2, Dogs 68 and 70).

Intelligent interpretation of the ventricular complexes of unipolar precordial leads must be based upon the relations between the components of these complexes and the corresponding deflections of unipolar leads from the anterior surface of the exposed heart. In the animals with bundle branch block but no infarction, there was a very close resemblance between the ventricular complexes of the leads from the right side of the precordium and the ventricular complexes of the leads from the exposed surface of the right ventricle, and likewise between the complexes of the leads from the left side of the precordium and those of the leads from the exposed surface of the left ventricle. Curves depicting these relations in right branch block without infarction (Dog 58) and in left branch block without infarction (Dog 64) have recently been published in a general article on the precordial electrocardiogram.[2] We shall, therefore, confine the present discussion to experiments in which bundle branch block was complicated by infarction. In order to conserve space and to avoid needless repetition, comments and discussion necessary to the understanding and interpreta-

tion of the observations made in the experiments described here in detail have not been relegated to a separate section, but are interspersed with the presentation of the relevant data.

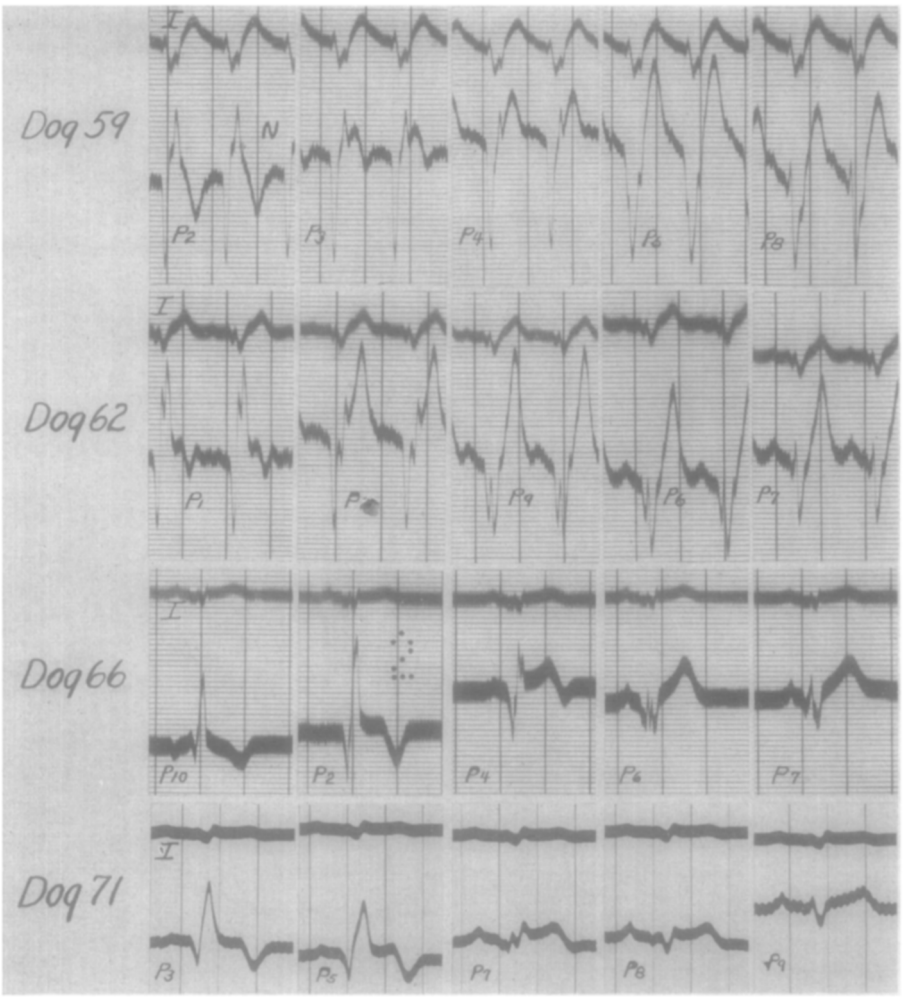

Fig. 4.—Unipolar precordial leads from four dogs with right bundle branch block and anterior myocardial infarction.

Dog 66.—The locations of the points on the epicardial surface explored in this experiment are shown on the outline drawing of the heart reproduced in Fig. 5. The points indicated by numbers not followed by a letter were studied with a soft-tipped electrode consisting of a small glass tube connected to a short length of soft rubber tubing cut on a bias at its lower end. The glass tube was stoppered with salted kaolin, and filled with 10 per cent copper chloride solution in which a long coil of copper wire was immersed. The short rubber extension was packed with cotton wool saturated with isotonic saline. When this electrode

was pressed lightly against the epicardium, no injury or only minimal injury to the underlying muscle resulted. The points marked by numbers followed by the letter *s* were explored with the sharp electrode used in taking precordial leads. This electrode was employed to distinguish dead from living muscle. When it was brought into contact with the former there were no injury effects in the curve obtained, but the latter yielded prominent RS-T displacement or completely monophasic ventricular complexes. The points designated by numbers followed by the letter *c* are those at which the sharp electrode was thrust through the ventricular wall in order to record the potential variations of the ventricular cavity. The first of the precordial curves reproduced (P_{10} of Fig. 6) was obtained from a point approximately 4.5 cm. to the right of the midsternal line. The ventricular complexes which it displays are

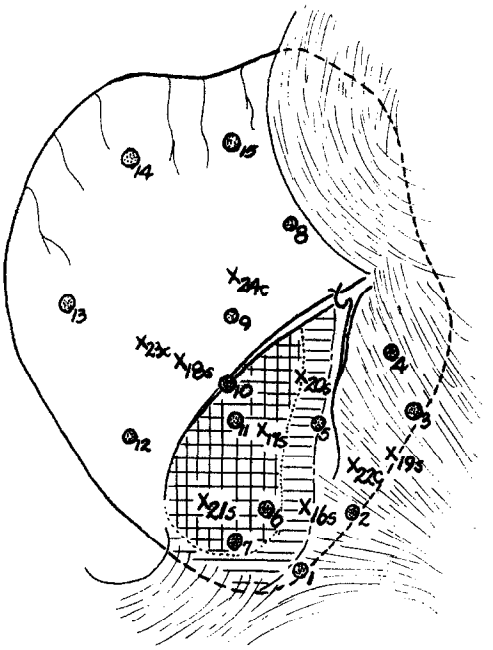

Fig. 5.—Dog 66. Outline drawing of anterior surface of exposed heart. Points explored with soft-tipped electrode indicated by stippled circles. Points explored with sharp electrode indicated by *X*; records taken from the surface are lettered *s*, and from the ventricular cavity are lettered *c*.

strikingly similar in outline to those of the direct leads from points *8, 12, 13, 14,* and *15,* which were all on the right ventricle. Since the deflections of all these leads are similar, only one, that from point *14,* is reproduced (Fig. 6). In these tracings the first QRS component is a small R wave, followed by a dip which reaches or barely crosses the isoelectric level. The second component is a tall R′ deflection which reaches its apex late in the QRS interval and displays a shoulder on its ascending limb. In right bundle branch block the muscle of the free wall of the right ventricle is not activated until late in the QRS

interval. During the earlier phases of this interval it contains no boundary between active and resting muscle and produces no electromotive forces. So long as this is the case, the potential of its outer surface is practically identical with that of the neighboring part of the ventricular cavity. It is not surprising, therefore, that in the tracings obtained from the ventricular cavity by thrusting a sharp electrode through the wall of the right ventricle at points *23c* and *24c* (Fig. 6), the earliest phases of the QRS complex are similar to the corresponding phases of the QRS complexes of the epicardial leads. The small, early R wave of these leads is faithfully reproduced in the internal leads.

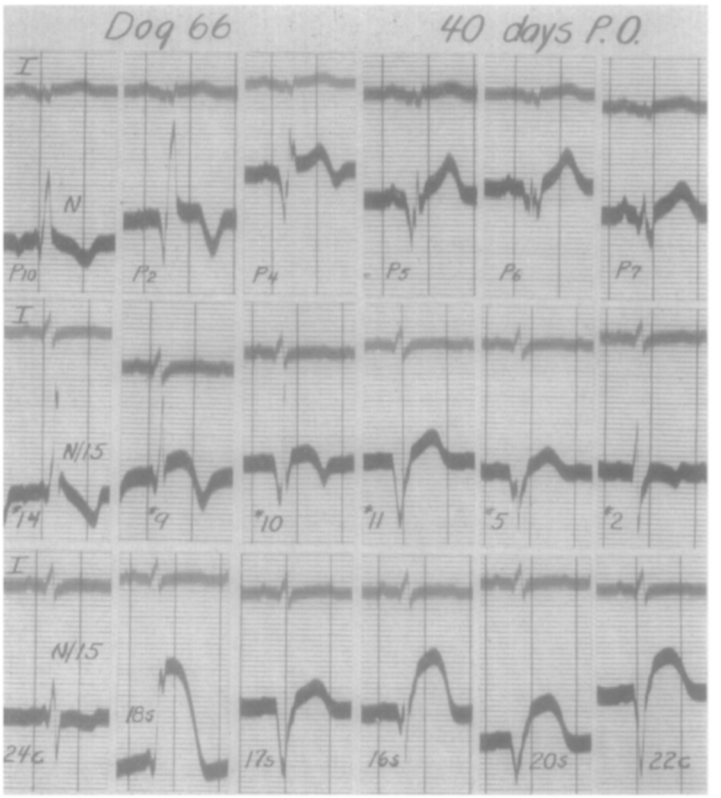

Fig. 6.—Dog 66. Right bundle branch block and anterior infarction. Upper row—unipolar precordial leads. Middle row—epicardial leads obtained with soft-tipped electrode. Lower row—epicardial and cavity leads obtained with sharp electrode.

The second R peak of the latter, which represents the maximum positivity attained by the ventricular cavity, evidently corresponds to the shoulder or thickening of the ascending limb of the late R′ deflection of the external leads. During the latter part of the QRS interval the epicardial surface is positive and the ventricular cavity negative, indicating that there is then a large electromotive force in the right ventricular wall. This force is responsible for that part of the late

R′ wave of the epicardial leads which follows the shoulder on its ascending limb.

It will be noted that the ventricular complexes of the lead from point *18s*, in which there is almost complete fusion of QRS and T due to the injury to the subepicardial muscle induced by the sharp electrode, do not differ, as regards those QRS components which precede the peak of the late R′ deflection, from the complexes obtained when a soft electrode was used. It has been pointed out in a previous publication[3] that injury to the subepicardial muscle can have no effect upon the form of the ventricular complex up to the instant at which the excitatory process arrives at the injured region. Since the subepicardial muscle of the right ventricle is not activated in right bundle branch block until the peak of R′ is inscribed, injury to this muscle has no effect on the earlier phases of the QRS complex.

The ventricular complexes of the direct leads from points *9* and *10* (Fig. 6) display a slender R or R′ deflection which reaches its apex late in the QRS interval and is preceded by a conspicuous downward movement. The RS-T segment is convex upward and the end of the T wave sharply inverted. The QRS complexes of the lead from point *9* begin with a small initial R, which is missing in the lead from point *10*. Both of these points were close to the interventricular sulcus, but their exact relation to the anterior attachment of the septum is not known. It is often difficult to interpret the deflections of leads of this kind because of uncertainty as to whether the recorded potential variations of the epicardial surface were more closely related to those of the right or to those of the left ventricular cavity. In the case of the lead from point *10*, it appears probable that the early phases of the QRS complex were determined by the potential variations of the cavity of the left ventricle; in the case of the lead from point *9*, the corresponding phases of QRS are apparently due in part to the potential variations of the cavity of the right ventricle. The ventricular complexes of these leads display many of the features often seen in leads from the marginal parts of an infarct which involves the left ventricular wall and is more extensive on its endocardial than on its epicardial side.

Unipolar epicardial leads from transmural infarcts that contain no appreciable amount of muscle capable of responding to the excitatory process yield ventricular complexes almost identical with those of leads from the adjacent part of the ventricular cavity. It will be noted that the ventricular complexes of the lead from point *11* are similar to those of lead *22c*, which represent the potential variations of the cavity of the left ventricle, and that the ventricular complexes of lead *17s*, in which a sharp exploring electrode was employed, are of the same kind. Since the last shows no RS-T displacement, it is evident that the muscle in the neighborhood of points *11* and *17s* was dead, or at least not responding to the cardiac impulse. The leads from points *6* and *7* and

the lead from point *21s* gave complexes practically identical in outline with those of the leads from points *11* and *17s*.

Unipolar epicardial leads from parts of left ventricular infarcts that contain appreciable amounts of muscle which responds to the cardiac impulse and lies in the outer layers of the ventricular wall yield QRS complexes characterized by large QS deflections notched by submerged R waves, or by abnormally large Q components followed by R waves of subnormal voltage. Infarction of the outer layers of muscle without involvement of the inner layers should theoretically reduce the size of the R component without producing abnormal Q waves, but this appears to be rare or nonexistent. In the present instance, notched QS deflections occurred in the lead from point *5*. The leads from points *16s* and *20s* show definite RS-T displacement, indicating the presence of living muscle. In the former the small R component is preceded by a broad Q deflection, which suggests that the subendocardial muscle was involved. Points *5*, *16s*, and *20s* evidently lie near the left margin of the infarct. The ventricular complexes of precordial leads P_4, P_5, and P_6 were evidently dominated by the potential variations of the infarcted region; they show many of the features of the ventricular complexes of the direct leads from points *5* and *11*.

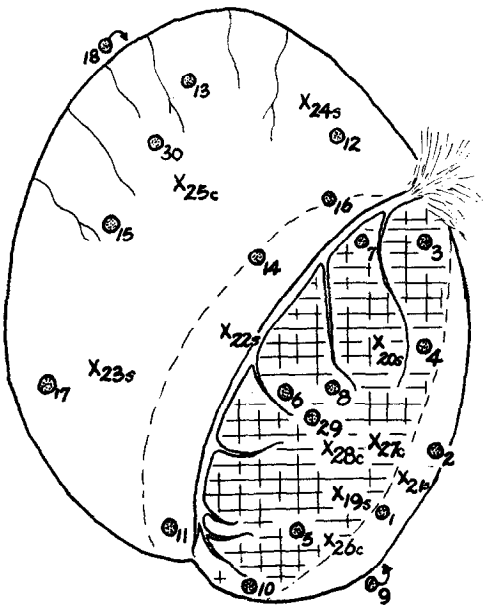

Fig. 7.—Dog. 71. Outline drawing of anterior surface of exposed heart. Symbols as in Fig. 5.

The direct leads from points *1*, *2* (see Fig. 6), *3*, and *4*, which were still farther to the left and in a zone supplied by a branch of the anterior descending artery which came off above the ligature, present ventricular complexes of the normal form. The lead from point *19s* shows conspicuous RS-T displacement, indicating that the muscle in

this zone was responding. All of these points evidently lie to the left of the infarcted region. The ventricular complexes of precordial lead P_7 apparently represent a mixture of the potential variations at the surface of the infarct and those at the surface of the uninvolved wall to the left of it.

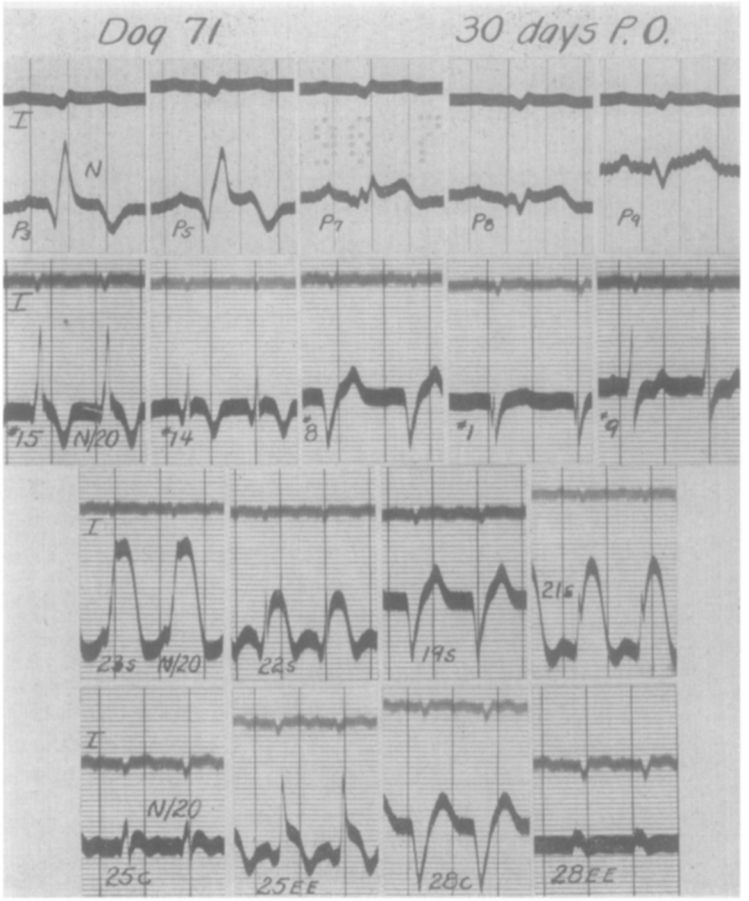

Fig. 8.—Dog 71. Right bundle branch block and anterior infarction. First row—unipolar precordial leads. Second row—epicardial leads obtained with *soft-tipped* electrode. Third row—epicardial leads obtained with sharp electrode. Fourth row—leads from ventricular cavities and transmural leads (marked *EE*).

Dog 71.—The locations of the epicardial points explored in this experiment are indicated in Fig. 7, and characteristic examples of the curves obtained are reproduced in Fig. 8. The ventricular complexes of the leads from points *11, 13, 17,* and *18* are similar in every respect to those of the lead from point *15* (Fig. 8). All of these points were on the free wall of the right ventricle. In the leads from points *12, 14,* and *16,* the QRS complex consists of a prominent Q wave, followed by a late R deflection which is very small in the lead from the last of these points; the T wave is sharply inverted. Points *14* and *16* were close

to the interventricular sulcus, and it is possible that point *12* was much less distant from it than is indicated in Fig. 7. The curves obtained with the sharp electrode at points *22s*, *23s*, and *24s* show pronounced RS-T displacement; in the first the earliest phases of the QRS complex are like those of the lead from point *14*, and in the others they are like those of the lead from point *15* and like those of the lead from the cavity of the right ventricle at point *25c*. It will be observed that the potential variations of the different parts of the epicardial surface and of the cavity of the right ventricle in this experiment were strikingly similar to those recorded in the case of Dog 66.

In the leads from points *3*, *4*, *5*, *6*, *7*, *8*, and *10*, the ventricular complexes consist of large, unnotched QS deflections, followed by positive T waves, and do not differ from those of the leads from the left ventricular cavity at points *27c* and *28c*, or from those of the leads from points *19s* and *20s*. The absence of RS-T displacement in these last two leads indicates that the muscle in contact with the sharp electrode was not responding to the cardiac impulse. The leads from points *1* and *2* present notched QS deflections, and the lead from point *21s* shows conspicuous RS-T displacement. These points were evidently close to the left margin of the infarct. In the lead from point *9*, the ventricular complexes are of the normal form. In this experiment transmural leads were taken in order to measure the variations in the voltage across the ventricular walls during the QRS interval. In these leads a sharp electrode thrust through the wall into the ventricular cavity was paired with a soft electrode on the epicardial surface. The galvanometer connections were made in such a way that relative positivity of the epicardial electrode produced an upward deflection in the finished record. In the lead marked *25 EE*, the soft electrode was on the free wall of the right ventricle at point *30*, and the sharp electrode was in the right ventricular cavity near point *25c*. The QRS complexes of this lead begin with a broad, shallow Q deflection, indicating slight relative negativity of the external surface. Late in the QRS interval there is a tall, sharp R deflection due to activation of the muscle between the two electrodes which made the epicardial surface strongly positive with respect to the ventricular cavity. The RS-T displacement is upward, and probably represents injury due to the pressure exerted by the outer electrode. The lead marked *28EE* was obtained by pairing a soft electrode at point *29* on the outer surface of the infarct with an internal electrode thrust through the ventricular wall at point *28c*. In this case the QRS deflections are very small. Since the infarcted muscle was not responding to the cardiac impulse, this part of the ventricular wall developed no electromotive force between its inner and outer surfaces. The small potential difference recorded is ascribed to electromotive forces generated at a considerable distance from the two electrodes and bearing nearly the same spatial relation to both of them.

The precordial leads in this experiment are clearly diagnostic of right branch block complicated by anterior infarction. There are abnormally large Q waves, followed by late R deflections in the leads from the right side of the precordium, and in the leads from the left side of the precordium the early R component ordinarily present in uncomplicated right branch block is absent, submerged, or abnormally small. It is also clear that the potential variations of the precordial points and the potential variations of the nearest parts of the ventricular surface were closely related. The resemblance between the precordial curves and the ventricular complexes of the direct leads is, however, somewhat less striking than in the case of Dog 66. Since large QS deflections occurred in the direct leads from a large part of the anterior surface of the left ventricle, it is surprising that they were not more faithfully reproduced in the leads from the left side of the precordium. If the precordium had been more completely explored, QS deflections larger than those present in precordial lead P_8 might have been obtained.

Dog 59.—The locations of the epicardial points investigated in this experiment are indicated in Fig. 9, and characteristic examples of the curves obtained are reproduced in Fig. 10. The leads from points *3*, *4*, *8*, and *19* on the free wall of the right ventricle display a late R wave, preceded by small preliminary deflections consisting of an initial downward movement followed by a positive deflection of about the same size. This second preliminary deflection notches the ascending limb of R. Similar preliminary deflections are present in the leads from points *13s* and *25s*, which were taken with the sharp electrode, but in the last the downward movement is preceded by a small positive peak. In the lead from the ventricular cavity at point *24c* this positive peak is well developed, but it is not followed by a conspicuous depression. The principal R wave of this internal lead corresponds in time to the notch or shoulder on the upstroke of the R wave of the epicardial leads. The difference between the earliest potential variations of the right ventricular cavity and those of the epicardial surface of the free wall of the right ventricle was not, therefore, as great as the first glance at the tracings suggests.

In the leads from points *2*, *12s*, *17s*, and *21s*, all close to the interventricular sulcus, the ventricular complex consists of a conspicuously notched QS deflection of moderate depth, followed by a positive T wave. Since the sharp electrode produced no upward RS-T displacement in the last three of these leads, it is apparent that most of the muscle in this zone was dead and that the infarct extended across the interventricular sulcus.

In the leads from points *1*, *7*, and *18*, the QRS complex consists of a deep, notched or slurred QS deflection. At points *10s*, *14s*, and *23s*, the sharp electrode yielded deep Q or QS deflections, followed by moderate upward displacement of the RS-T junction, indicating that some

of the subepicardial muscle, but not all of the subendocardial muscle at these points, was responding to the cardiac impulse. The leads from points *5, 6, 9, 11s,* and *20s* indicate that the corresponding parts of the ventricular wall were normal. At point *22s* the sharp electrode yielded a curve which shows no upward RS-T displacement, and in which the QRS complex is unnotched and similar to that of the lead from the cavity of the left ventricle near point *23c,* indicating that in this region the infarct was transmural.

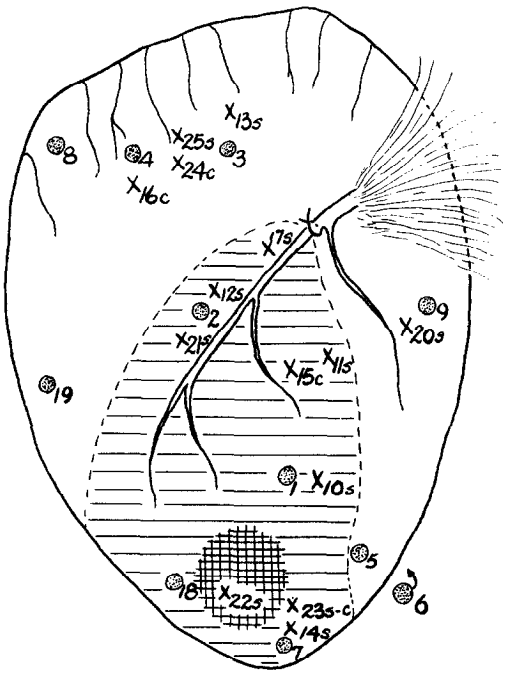

Fig. 9.—Dog 59. Outline drawing of anterior surface of exposed heart. Symbols as in Fig. 5.

There is a close relation between the form of the ventricular complexes of the leads from the left side of the precordium and those of the direct leads from the epicardial surface of the left ventricle. In the leads from the right side of the precordium the initial Q deflection is much larger in comparison with the late R wave than in the two experiments previously described (Dogs 66 and 71). As regards the presence of these large Q components, the ventricular complexes of these leads are unlike those of the direct leads from the epicardial surface of the free wall of the right ventricle. The same phenomenon is seen in the semidirect leads (*SD* in Fig. 10) which were taken from a pad of gauze soaked in isotonic saline solution and laid upon the exposed heart. This pad was approximately 1 cm. thick, and the exploring electrode was moved across it step by step in a base-apex direction, so that the earliest leads (*1* and *2*) were from parts of the pad lying on the right ventricle. The ventricular complexes of these leads resemble those of

the corresponding precordial leads in general contour. It is apparent that in this experiment the potential variations of the surface of the infarct were unusually well transmitted to the right side of the precordium and to the right side of the gauze pad. The reason may lie in the extension of the infarct to the right of the interventricular sulcus. The same phenomenon occurred in the case of Dog 62, but in this instance the animal died prematurely, so that a satisfactory number of direct leads could not be taken. In this experiment, also, there was spotty infarction of the part of the right ventricular wall adjacent to the sep-

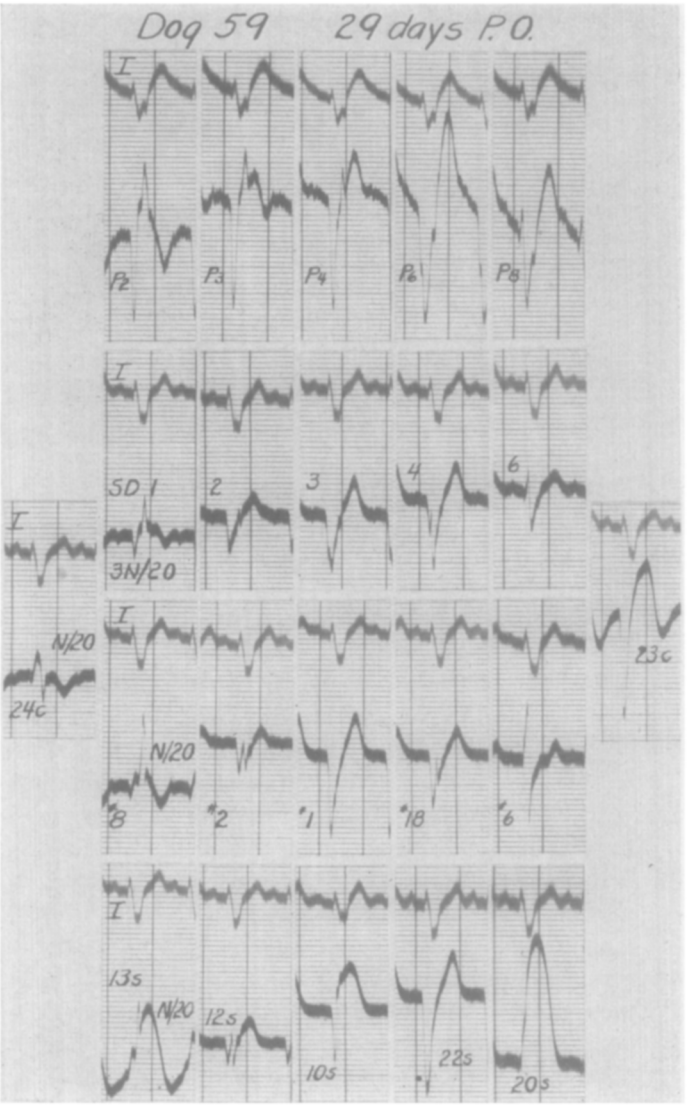

Fig. 10.—Dog 59. Right bundle branch block and anterior infarction. First row—unipolar precordial leads. Second row—semidirect leads from saline soaked gauze pad laid upon exposed heart. Third row—epicardial leads with soft-tipped electrode. Fourth row—epicardial leads with sharp electrode. Tracings labeled *24c* and *23c* are leads from the right and left ventricular cavities, respectively.

tum. It should be noted that, during the earliest parts of the QRS interval, the potential variations of the surface of the infarct were much larger than those of the right ventricular surface, whereas later in this interval this situation no longer existed. This explains why the potential variations of the right side of the precordium were at first like those of the infarcted region, and later like those of the anterior surface of the right ventricle.

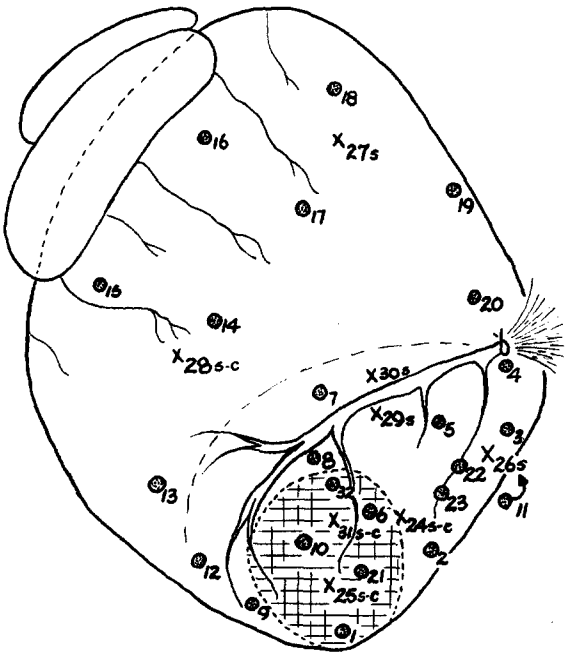

Fig. 11.—Dog 70. Outline drawing of anterior surface of exposed heart. Symbols as in Fig. 5.

Dog 70.—The locations of the epicardial points explored in this experiment are shown in Fig. 11, and some of the tracings obtained are reproduced in Fig. 12. In the leads from the free wall of the right ventricle at points *13, 14, 15, 16, 18,* and *19,* the QRS complex consists of a slender initial R deflection, followed by a deep, broad, and slurred S wave. The T wave is upright. The ventricular complex is of the same form in the leads from points *7, 12,* and *20,* except that in the last there is a mere trace of the initial R wave, and also in the leads from points *4, 5, 8,* and *9,* which were equally close to the anterior descending artery, but on the opposite side of it. In the dog it is the rule that the time of activation of points just below this artery is not much affected by section of either the right or left bundle branch. At points *27s, 28s, 29s,* and *30s,* the sharp electrode induced pronounced upward RS-T displacement.

In the lead from point *2* the QRS complex consists of a tall, late R wave which displays conspicuous slurring of the first part of its ascend-

ing limb. This limb is notched near its onset by a small summit. The T waves are inverted. Ventricular complexes of essentially the same kind are present in the leads from points *11*, *22*, and *23*. In the lead from point *3* the peak of the R wave is earlier, but in this lead, also, this deflection is preceded by a small summit. There are a large S and a

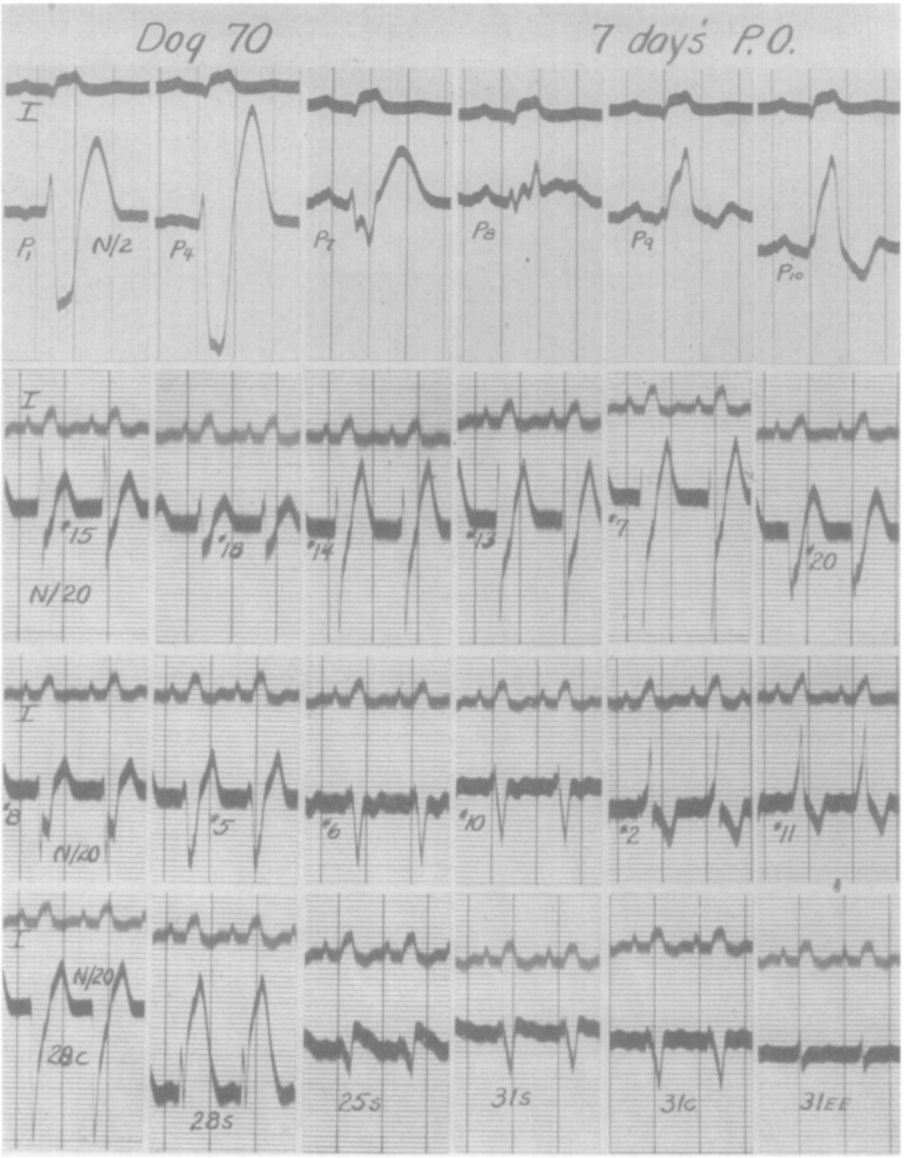

Fig. 12.—Dog 70. Left bundle branch block and anterior infarction. First row—unipolar precordial leads. Second row—epicardial leads from right ventricle with soft-tipped electrode. Third row—epicardial leads from left ventricle with soft-tipped electrode. Fourth row—epicardial and cavity leads with sharp electrode. Transmural lead labeled ***EE***.

positive T wave. The sharp electrode produced a large upward displacement of the RS-T junction in the lead from point *26s*.

In the leads from points *1, 6, 10,* and *21,* the ventricular complexes are of essentially the same form as in the lead from the ventricular cavity near point *24c*. The QRS deflections consist of two sharp, but very low, summits, followed by a deep S deflection, and the T wave is diphasic or inverted. The sharp electrode yielded moderate upward RS-T displacement at point *24s,* slight displacement at point *25s,* and very slight displacement at point *31s*. In all of these leads the initial QRS deflections are similar to those of the lead from the ventricular cavity. It is apparent that a considerable part of the ventricular wall in this region was infarcted.

A transmural lead from the ventricular cavity near point *31c* to the epicardial surface at point *32* (marked *31EE* in Fig. 12) shows very small deflections, indicating that the ventricular wall in this neighborhood was producing no appreciable electromotive force and was not responding to the cardiac impulse. It should be pointed out that in the leads from the infarcted region (points *1, 6, 10,* and *21*) the large downstroke which begins at the peak of R is not nearly as steep as it is in the leads from uninjured parts of the ventricular walls. The absence of a very abrupt downstroke or true intrinsic deflection distinguishes the QRS complex of these leads from those of all the other leads taken with the soft electrode, with the possible exception of the lead from point *5* (see Fig. 12).

The ventricular complexes of the leads from the right side of the precordium (P_1 to P_7, inclusive) are strikingly similar in general outline to those of the direct leads from the free wall of the right ventricle and the zones on either side of the anterior descending coronary artery. The ventricular complexes of the leads from the left side of the precordium (P_9 and P_{10}) resemble those of the direct leads from the uninfarcted anterolateral wall of the left ventricle. None of the precordial leads displays ventricular complexes like those of the direct leads from the surface of the infarct. The reason probably lies in the relatively small size of the region within which a large part of the ventricular muscle was killed. But even if this region had been much larger, and the potential variations over its surface had been more faithfully transmitted to the precordium, it is unlikely that modifications of the precordial curves diagnostic of infarction would have resulted. As has been clearly shown, the potential variations at the epicardial surface of a transmural infarct are always practically identical with those of the adjacent parts of the ventricular cavity. When the left branch of the His bundle conducts normally, the cavity of the left ventricle is negative throughout the QRS interval, and leads from the surface of trans-

mural left ventricular infarcts yield deep QS deflections which clearly indicate the nature of the lesion. If the infarct is anterior and not too small, similar deflections occur in suitable precordial leads. When left branch block is present, however, the cavity of the left ventricle is positive at the beginning of systole because of the electromotive force generated by the spread of the cardiac impulse through the septum from right to left. In this case, leads from the outer surface of a transmural infarct of the left ventricular wall display QRS complexes that consist of an R deflection of variable size, followed by an S wave of like or greater voltage. Such deflections in precordial leads are not sufficiently distinctive to have much diagnostic value. If they occur in leads from the extreme left side of the precordium, which ordinarily yield QRS complexes consisting of a single component, a broad, slurred, notched, or bifid R wave, they may justifiably lead to the suspicion that an infarct is present, but cannot furnish reliable evidence of the existence of such a lesion. The recognition of infarction of the free wall of the left ventricle in the presence of left bundle branch block on the basis of modifications of the QRS complex is, therefore, extremely difficult.

SUMMARY

In dogs, myocardial infarcts induced by ligating the anterior descending coronary artery in its middle third do not usually modify the QRS complexes of the standard limb leads in a characteristic manner when bundle branch block is present.

When such infarcts are complicated by right bundle branch block, the QRS complexes of unipolar leads from the right side of the precordium display a large, initial Q deflection, followed by an R wave which attains its summit late in the long QRS interval. The first component is due to potential variations transmitted from the epicardial surface of the infarcted region, and the second to potential variations transmitted from the epicardial surface of the free wall of the right ventricle. Leads from that part of the precordium overlying the infarct present large, broad QS deflections, often conspicuously slurred or notched.

When left branch block is present, infarction of the kind in question does not give rise to characteristic changes in the QRS complexes of the precordial leads because the potential of the left ventricular cavity and, therefore, of the epicardial surface of the infarcted region is positive during the earliest part of the QRS interval. In direct leads from the epicardial surface of the infarct, the QRS complex consists of an initial R deflection of variable size, followed by an S component of like or greater voltage. In the case of very large lesions, QRS complexes of this kind probably occur in leads from precordial points overlying the part of the left ventricular wall which is affected, but cannot be considered reliable evidence of infarction.

REFERENCES

1. a. Wilson, F. N., Hill, I. G. W., and Johnston, F. D.: The Form of the Electrocardiogram in Experimental Myocardial Infarction. I. Septal Infarcts and the Origin of the Preliminary Deflections of the Canine Levocardiogram, AM. HEART J. **9**: 596, 1934.
   b. Johnston, F. D., Hill, I. G. W., and Wilson, F. N.: The Form of the Electrocardiogram in Experimental Myocardial Infarction. II. The Early Effects Produced by Ligation of the Anterior Descending Branch of the Left Coronary Artery, AM. HEART J. **10**: 889, 1935.
2. Wilson, F. N., Johnston, F. D., Rosenbaum, F. F., Erlanger, H., Kossmann, C. E., Hecht, H., Cotrim, N., Menezes de Oliveira, R., Scarsi, R., and Barker, P. S.: The Precordial Electrocardiogram, AM. HEART J. **27**: 19, 1944.
3. Wilson, F. N., Hill, I. G. W., and Johnston, F. D.: The Interpretation of the Galvanometric Curves Obtained When One Electrode Is Distant From the Heart and the Other Near or in Contact With the Ventricular Surface. I. Observations on the Cold Blooded Heart. II. Observations on the Mammalian Heart, AM. HEART J. **10**: 163, 1934.