THE LOCALIZATION OF VENTRICULAR EXTRASYSTOLES*

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The interest in localization of the site of origin of ventricular extrasystoles by study of their electrocardiographic configuration does not arise from a sense of the importance of such a localization in the case of the majority of ventricular extrasystoles observed in man. It is, rather, founded on the recognition in the earliest days of electrocardiography[15, 26, 27, 28] that the electrocardiographic configuration of a ventricular extrasystole is to be interpreted in terms of an altered sequence of excitation and recovery in the heart, and that study of the configurations obtained when the sequence of excitation is altered in a predetermined manner by stimulation of chosen localities in the heart should permit inferences to be drawn regarding the location and nature of pathological processes when these are responsible for alterations in the distribution of the cardiac impulse.

There is, however, an apparent disagreement over the localizing significance of the various electrocardiographic patterns, which is particularly marked in the case of the so-called "discordant" patterns, in which the major deflections of the QRS portion are oppositely directed in leads I and III.[29] The classical interpretation of Lewis, in which an extrasystole with a QS_1 and an R_3 was considered to have a right-sided origin, has been largely supplanted by a diametrically opposite interpretation. Others have held that changes in the position of the heart in the chest and other factors may so alter the configuration of extrasystoles that localization is, for all practical purposes, impossible.[12] The apparent difficulties attending the application of experimental results in the dog to man have brought repeated warnings of the hazards of such attempts.[28, 29]

At least three factors appear to have contributed to the lack of agreement that at present exists. These are as follows:

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(*a*) *The failure to provide for normal conduction from the heart to the distant leads.* The usual technic in animal experiments has been to open the chest widely and suspend the heart in a sling formed by the opened pericardium.[2, 15, 26, 27, 28] The lungs were inflated and the chest wall was closed only in experiments reported by Lewis in a study of endocardial extrasystoles.[17] Most experimental studies on human ventricular extrasystoles were performed on the heart whose surface had been exposed over a relatively wide area,[3, 18, 19, 20, 25] although there are a few references to mechanical stimulation of the heart in individuals with a defect in the bony structure of the anterior wall of the thorax.[4, 11, 24, 25]

The importance of this factor was recognized as early as 1912 by Hering,[6] and has received full experimental support from the work of Katz and his collaborators.[13] The measurements of conductivity through the lungs recently reported by Kaufman and Johnston emphasize the necessity for inflation of the lungs around the heart if a normal electrocardiogram is to be recorded.[14] Finally, in the present report will be found examples of the very great alteration produced by deflation of the lungs and the elevation of the heart away from conducting surfaces. While these experiments, as well as previous reports, deal solely with experimental animals, the inference is clear that the configuration of an extrasystole observed in man when any significant portion of the heart is exposed, may not necessarily resemble the configuration of an extrasystole from the same point on the heart when the thorax is intact.

(*b*) *Preoccupation with the site of origin to the neglect of the sequence of excitation of adjacent and distant regions of heart.* Almost without exception, the attempt has been made to interpret all patterns in terms of an exclusive right or left ventricular localization. While it has been recognized that excitation originating in one ventricle will ultimately involve the opposite ventricle, no attention has been given to an equally obvious fact, namely, that extrasystoles arising at or near the septum spread simultaneously to adjacent portions of both right and left ventricle before they involve the more distant regions of the ventricles in which they arise. Such extrasystoles might more properly be termed anterior or posterior extrasystoles, without reference to the ventricle in which they arise.

It is well to point out that in the original report of Barker, Macleod, and Alexander[3] it was noted that the configuration now interpreted as a left ventricular extrasystole was obtained by stimu-

lation only at a point near the anterior septum on the left ventricle, and that when points near the apex, or more laterally, were stimulated, concordant records were obtained, in which the initial deflections were downward in both lead I and lead III. Why the "concordant" records should be so largely neglected is perhaps best explained in the next paragraph.

(*c*) *Inferences from experiments on bundle-branch block.* "It is apparent also," state Barker, Macleod, and Alexander, "that the current view regarding the site of origin of extrasystoles represented in clinical electrocardiograms are [*sic*] not based upon satisfactory direct evidence in man, nor upon direct observations in animals but upon inference." This inference, as the above authors point out, has been largely derived from a study of bundle-branch block. The experimental work of Lewis, upon which the classical view was largely based, deals in its essential details entirely with bundle-branch block; in the experiments on ventricular extrasystoles lead II alone was recorded.[16] The attempt to interpret all ventricular extrasystoles in terms of the results of bundle-branch block fails in two regards, practical and theoretical. Barker, Macleod, and Alexander called attention to the fact that, "on the other hand it is obvious that stimulation of all points on the right or left ventricle does not necessarily give complexes bearing a close resemblance in all leads to those produced by cutting the opposite branch of the His-bundle." Nor is there any reason why they should be. An extrasystole spreads from a single focus, which may be anywhere in the heart, to adjacent, and then to more distant regions. In bundle-branch block the whole of a single ventricle is excited normally, while the opposite ventricle is stimulated by spread from the contiguous borders of the normally activated ventricle. This does not imply that the whole of the normally stimulated ventricle is necessarily activated in advance of the other. It is, for example, universally recognized that the earliest region on the surface of the dog heart to be activated lies at the anterior septum. In the event of right bundle-branch block this region would remain presumably the earliest to be excited, and it is entirely reasonable to suppose that the adjacent regions of the right ventricle might be excited well in advance of other regions, even in the left ventricle. As far as the dog's heart is concerned, this factor can adequately account for the so-called "discordant" electrocardiograms seen in the bundle-branch block.

These considerations make it clear that attempts to assay the differences between observations in man and experimental animals are premature since strictly comparable observations have not been made in adequate number and under conditions that approach the normal as closely as possible. This is particularly true in the case of animal experiments, where technical defects and unwarranted inferences from other types of experiments have obscured the issue. In the following experiments, therefore, the entire epicardial surface of the heart and much of the endocardial surface have been explored with stimulating electrodes under conditions that approach the normal as closely as possible, namely, a virtually intact pericardium, fully expanded lungs, with, in most instances, spontaneous respiration, and maximum reconstruction of the chest-opening by approximation of underlying muscles and skin edges.

Method

Fifty dogs were employed in the experiments reported here. They were anesthetized with Dial or Nembutal. The heart was exposed through a skin incision parallel to the lower border of the pectoral muscles, and by removal of the sternal portions of the 4th or 5th rib on the right or left side. After opening the pericardium bipolar electrodes (polar distance 1 to 2 mm.) were stitched in place through the epicardium, or a variety of self-retaining electrodes were employed. In all cases the lungs were fully inflated and the chest opening was closed by skin-clips before records were taken. In some instances artificial respiration was suspended during recording, and the animal breathed spontaneously. In others, an excessive period of apnea ensued before spontaneous respiration was resumed and artificial respiration was maintained to prevent anoxia. Intermittent break shocks were delivered approximately every second by means of a thyratron stimulator; an occasional extrasystole was thereby elicited against a background of normal complexes. The strength of the stimulus was adjusted to be slightly above the threshold at the end of the relative refractory period. Records were taken with the animal on its back, although significant variations did not result from changes to other positions. Records were made by means of a Sanborn Cardiette or Sanborn Tribeam, and the present report is concerned only with the standard leads I, II, and III.

Results

I. The influence of conduction through tissues in contact with the heart

Figure 1 shows examples of the alterations in configuration of the electrocardiogram of ventricular extrasystoles produced by deflat-

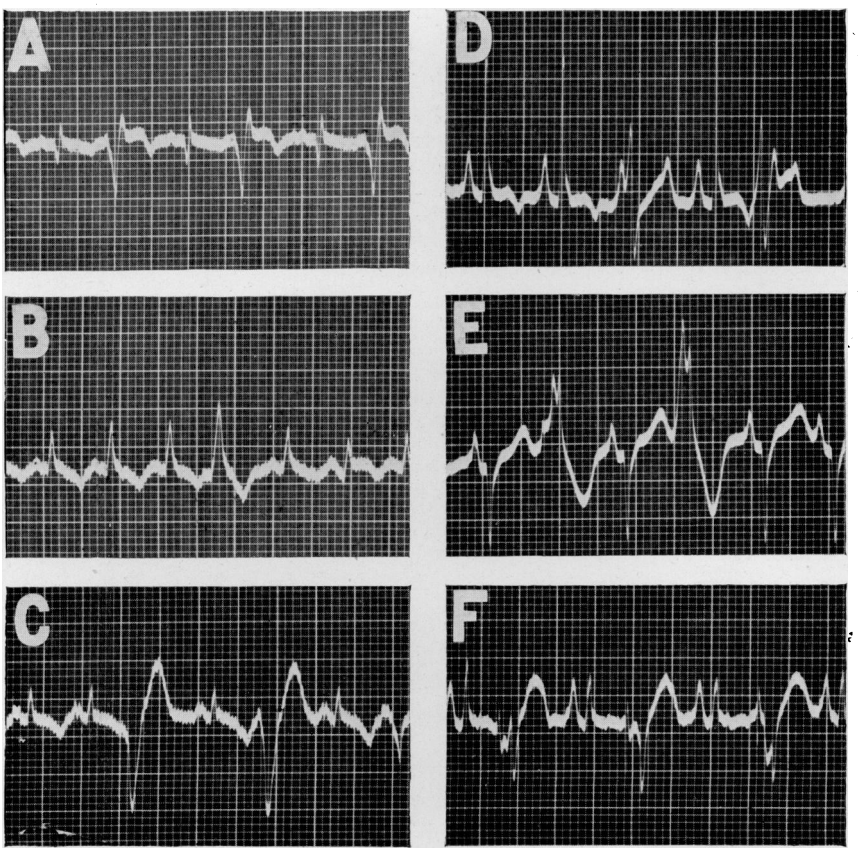

FIG. 1. Oct. 23, 1942. Dog, 10.0 kg. **A.** Control, lead I. Lungs fully expanded, chest closed, spontaneous respiration. The ventricular extrasystole, from the anterior surface of the right ventricle, shows Q and R components. **B.** The same, except that artificial respiration was started and the anterior surface of the left ventricle was exposed. The Q wave has disappeared. **C.** The same, except that the lungs were allowed to deflate and the posterior right ventricle was lifted up away from the lungs by traction on the pericardium. The R wave is not present. **D.** Control, lead III. Ventricular extrasystole from the anterior surface of the left ventricle. Lungs fully expanded, chest closed, spontaneous respiration. R and S components are present. **E.** The same, except that the posterior surface of the left ventricle was lifted away from the lungs by traction on the pericardium. Artificial respiration with lungs partially deflated. The S component has disappeared. **F.** The same except that the anterior surface of the right ventricle is exposed. The R component is absent.

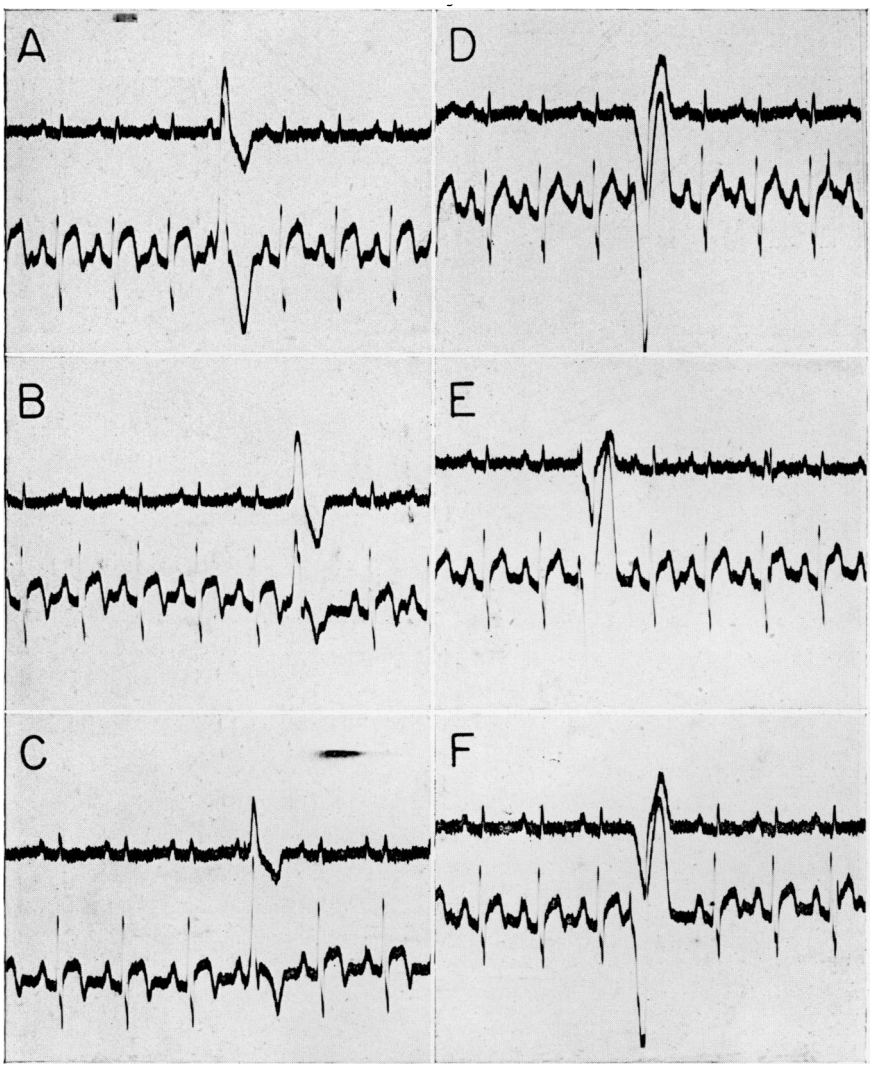

FIG. 2. April 26, 1943. Dog, 9.0 kg. Dial anesthesia. Lungs fully inflated, chest closed, and spontaneous respirations during recording. Leads I and III. **A, B, C.** Ventricular extrasystoles elicited along the lateral margin of the right ventricle at the base (**A**), midway between base and apex (**B**), and within 1 cm. of the apex (**C**). **D, E F.** Ventricular extrasystoles elicited from the lateral margin of the left ventricle at base (**D**), midway between base and apex (**E**), and the apex of the left ventricle (**F**).

ing the lungs and lifting the heart and pericardium away from surrounding structures. They demonstrate clearly that the electrocardiogram of a ventricular extrasystole taken under such abnormal conditions cannot be relied on as a guide to the configuration of the same extrasystole arising in a heart in its normal position, nor can evidence obtained in such a fashion be utilized in localizing extrasystoles arising spontaneously in an intact subject.

II. Configuration

A. Basic localizing patterns.

The general nature of all ventricular extrasystoles derived by stimulation of the surface of the heart consists in variations imposed upon four fundamental patterns, which are displayed by extrasystoles elicitable from four areas as follows:

(*1*) *Right lateral.* This pattern is obtained by stimulation within a region approximately 1 cm. wide, extending from the base of the right ventricle to within a few millimeters of the septum at the apex. This zone, descending along the right lateral margin of the heart, comes to lie somewhat anteriorly near the apex. From any region in this area extrasystoles show in both lead I and lead III, and consequently in lead II, an uncomplicated R complex followed by an inverted T (Fig. 2, **A**, **B**, **C**; Fig. 3, 1).[21]

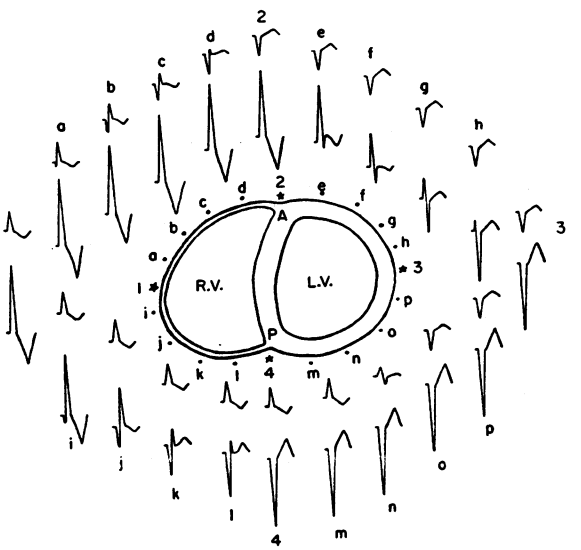

FIG. 3. A semi-schematic summary of the configuration in leads I and III of the electrocardiogram of ventricular extrasystoles elicited from various points around the heart midway between apex and base. The configurations 1, 2, 3, and 4 represent the four basic patterns, namely, right, anterior, left, and posterior, respectively, while the configurations lettered a to m indicate the transitional patterns described in the text.

(*2*) *Left lateral.* This pattern characterizes extrasystoles elicited from a zone on the left lateral margin of the heart about

1 cm. wide, which maintains its lateral position to include the apex of the left ventricle and extends to within approximately 5 mm. of the septum. From this area extrasystoles show only a QS complex followed by an upright T (Fig. 2, **D, E, F**; Fig. 3, **3**).[21]

(*3*) *Anterior septal.* This pattern is exhibited by extrasystoles elicitable from a zone no more than 0.5 cm. wide, which extends from the base to within 1.5 to 2.0 cm. of the apex. This area actually lies on the left ventricle, just to the left of the anterior descending artery, which runs down over the septum. Initial complexes are oppositely directed in leads I and III; lead I shows a QS, while lead III shows an R (Fig. 3, **2**; Fig. 4, **A, B**).[22] The T wave is oppositely directed to the primary deflection.

(*4*) *Posterior septal.* The zone from which the posterior septal pattern is derived consists of an area varying from 0.5 to 1.0 cm. in width, which extends from the apex, or even a few millimeters anterior to it, to the base. From this posterior region simple complexes are also derived, and leads I and III are again oppositely directed, only they are the reverse of the anterior pattern. The initial complex in lead I is R, and in lead III it is QS (Fig. 3, **4**; Fig. 4, **C, D, E, F**).[22] The T wave is oppositely directed to the primary deflection.

B. Transitions between the four basic patterns.

(*1*) *Anterior septum to right lateral.* No transition occurs in lead III. In lead I, however, there is a gradual change from a QS to an R. This occurs through the development of a small upright complex closely following the initial downward complex, giving the appearance of a small R wave following a Q wave.[9] As the point of stimulation is moved toward the right, R increases and Q diminishes until in the right lateral zone only an R remains (Fig. 3, **a, b, c, d**).

(*2*) *Right lateral to posterior septum.* Here the change is in lead III, while lead I remains the same. A small Q appears and grows as R diminishes, until at the posterior septum R has disappeared and only a simple QS remains (Fig. 3, **i, j, k, l**).[9]

(*3*) *Anterior septum to left lateral.* Lead I is unchanged. Lead III first shows, to the left of the anterior septal zone, a small S wave, which grows progressively in amplitude as the point of stimulation is moved toward the left, while R diminishes. Finally,

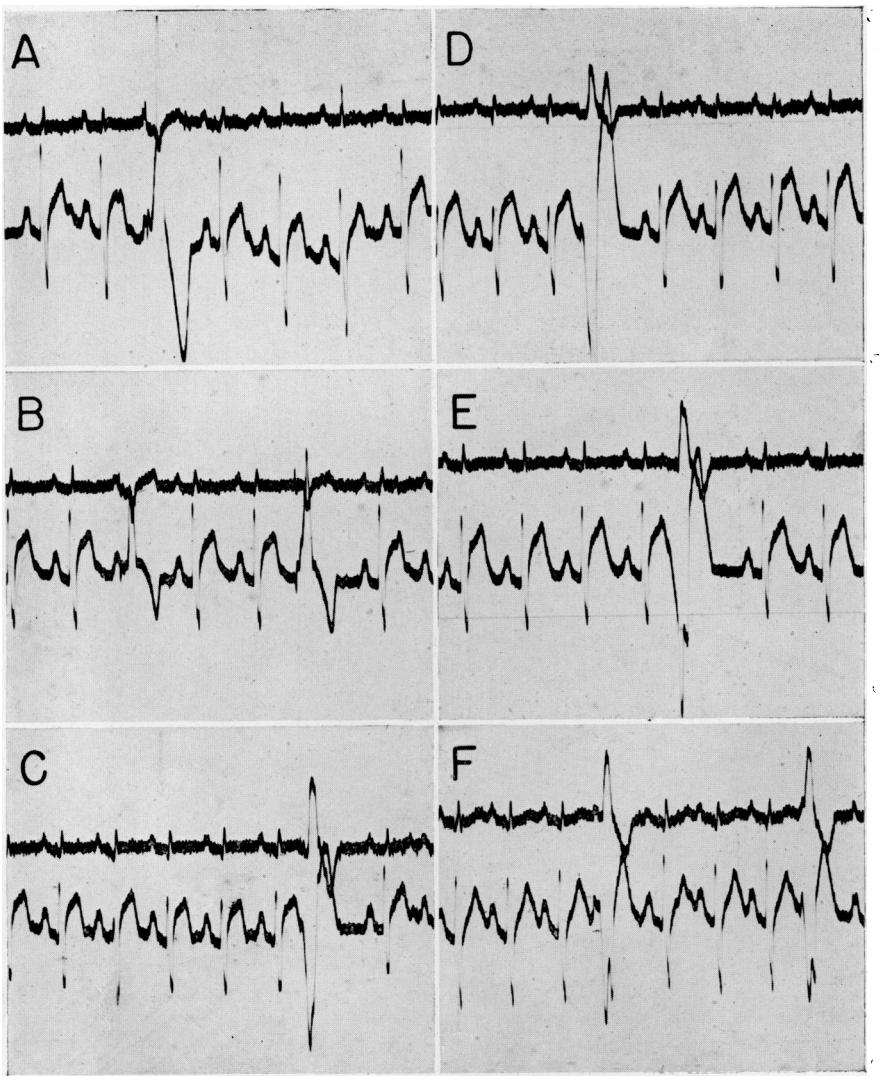

FIG. 4. April 26, 1943. Dog, 9.0 kg. Dial anesthesia. Lungs fully expanded, spontaneous respiration. A series of records in leads I and III of ventricular extrasystoles elicited over the interventricular septum. The point of stimulation was moved progressively from the anterior base to the posterior base. **A.** Anterior septum at base. **B.** Anterior septum 2.0 cm. from the apex. **C.** Septum at the apex. **D.** Posterior septum 1.0 cm. from the apex. **E.** Posterior septum midway between apex and base **F.** Posterior septum at base.

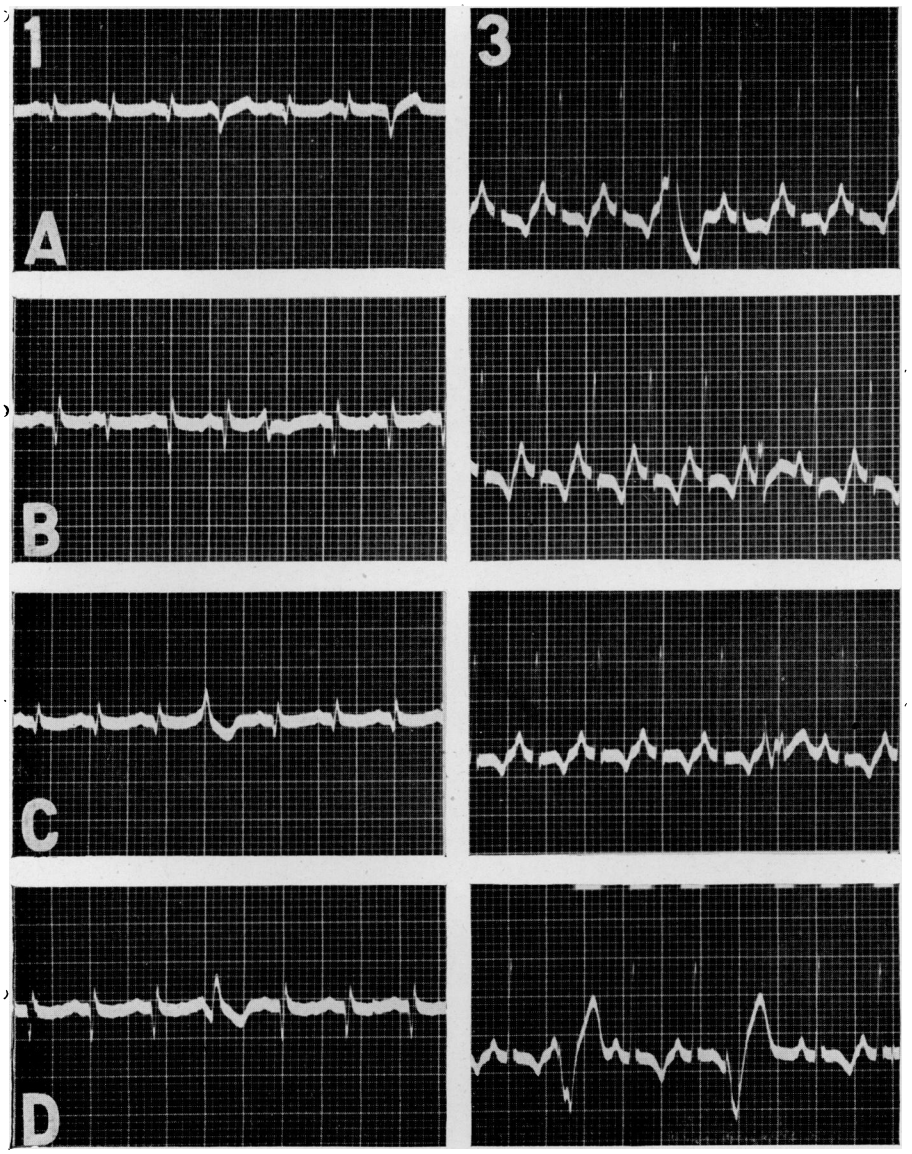

FIG. 5. Sept. 8, 1943. Dog, 11.0 kg. **A.** Leads I and III. Ventricular extrasystoles elicited at the anterior septum, 2.0 cm. from the apex showing the typical anterior pattern. **D.** In these records is shown the posterior pattern exhibited by extrasystoles elicited at the apex. **B** and **C** show transitional types of extrasystoles elicited 1.5 cm. from apex (**B**) and 0.5 cm. from apex (**C**).

at the left lateral margin, R has disappeared and only QS remains (Fig. 3, **e, f, g, h**).[23]

(*4*) *Left lateral to posterior septum.* In lead III there is no change. In lead I the transition is as in (*3*), namely, via the development of an R wave. In most instances, possibly associated with the small amplitude of lead I, the transition was by means of low voltage bizarre, often W- or M-shaped, complexes (Fig. 3, **m, n, o, p**).[23]

(*5*) *The apex.* The apex is the region where the four major areas as well as the zones of transition meet. It is here that the maximum changes take place with the minimum of movement of the stimulating electrodes. With constant position of electrodes, there may be considerable variation in the nature of the complex, depending upon the position of the extrasystole in the cycle, a phenomenon sometimes noted, but less markedly, in other zones of transition. The "apical zone" is a region, approximately 1.5 to 2.0 cm. long and 0.5 to 1.0 cm. wide, lying over the anterior septum, a little more to the left than to the right, starting at the very apex. Typically, transition occurs from the posterior to the anterior septal pattern as follows: The first change is in QS_3, which loses amplitude and assumes an M or W configuration of low amplitude. An R wave remains in lead I. In extrasystoles arising somewhat nearer the base an R appears in lead III, while in lead I R loses amplitude and is replaced by QS. (Fig. 5.) An S_3 is occasionally observed in the complex resulting from stimulation of points near the apex on the posterolateral surface of the right ventricle.

C. *Extrasystoles of endocardial origin.*

Extrasystoles arising from stimulation of the endocardium exhibited the same four basic patterns shown by their epicardial counterparts (Fig. 6). The same transitional patterns were also encountered. When endocardial extrasystoles were matched with their epicardial counterparts it was noticed that the right and left lateral zones were somewhat more restricted in area in the endocardium than on the epicardium (Figs. 7 and 8). When an extrasystole arising anywhere in the right ventricle was viewed by itself, it was impossible to decide whether its site of origin was endocardial or epicardial. Extrasystoles arising in the endocardium of the left ventricle often were characterized by a shorter QRS interval than

were epicardial extrasystoles (Fig. 6, **C, D**). This was the only detail of configuration that might permit differentiation between endocardial and epicardial extrasystoles of left ventricular origin.[7]

D. Extrasystoles whose configuration closely resembles the normal complex.

Inasmuch as the electrocardiogram of both the extrasystole and the supraventricular beat reflects the particular sequence of excitation and recovery of the various regions of the heart represented in a given lead, it should be possible to select points of stimulation which reproduce the normal sequence of activation and recovery, thereby yielding a complex closely resembling the supraventricular complex. Fig. 9 shows examples of this. Bearing in mind the general patterns summarized in Fig. 3, one can reproduce almost any given complex, though each lead must be considered separately. It was impossible to produce ventricular extrasystoles resembling the normal complex in both lead I and lead III by stimulating a single point. When such extrasystoles occur spontaneously they must be considered to have originated in the auriculoventricular node.

Discussion

In these experiments the endocardial and epicardial surfaces of the heart have been mapped under conditions which approach the normal more completely than any previously reported. The configuration of extrasystoles so obtained conforms to four basic patterns and the progressive transitions between them. This makes it possible to locate with considerable accuracy the site of origin of every variety of ventricular extrasystole. No practical distinction can, however, be made between extrasystoles arising on the epicardial surface of the heart and those which originate on the endocardium. The apparent disagreement between these conclusions and those of Lewis[17] have been discussed in detail elsewhere.[7]

In the subsequent section theoretical considerations are presented which permit an explanation of the patterns found in these experiments. It must be emphasized that theoretical explanations in no way affect the validity of the key to localization of the ventricular extrasystole described above.

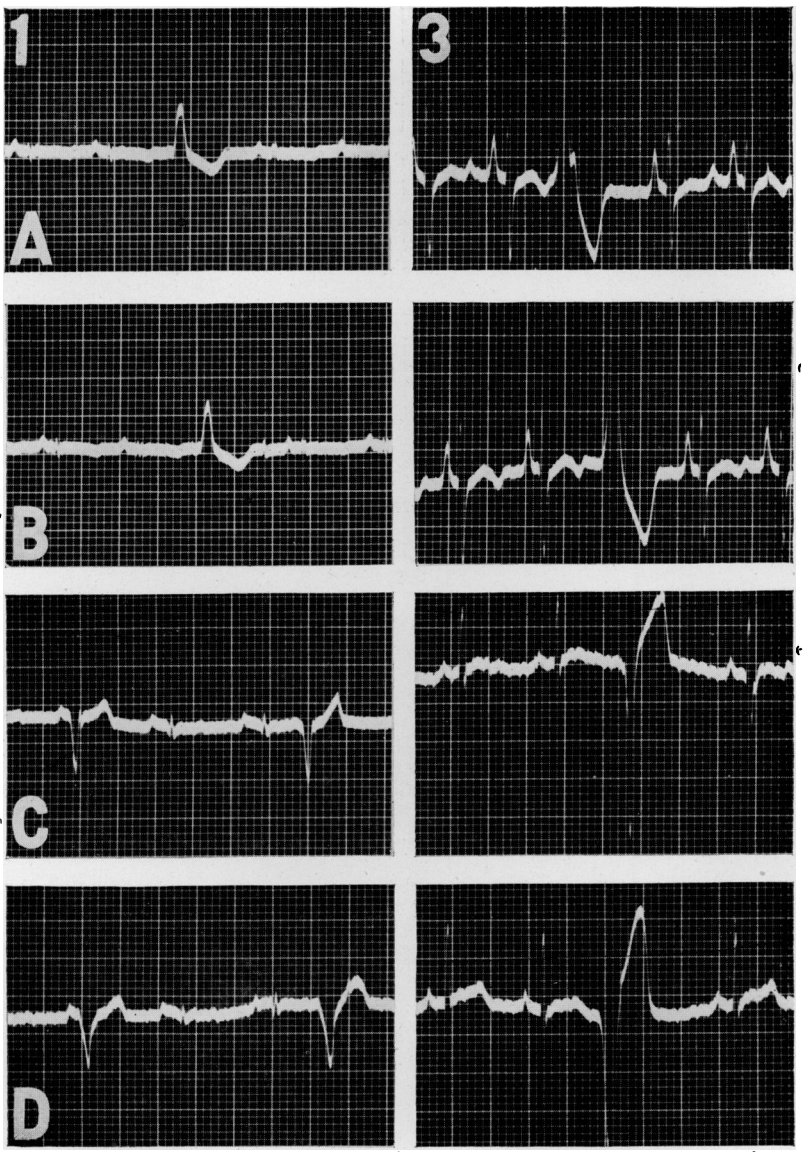

FIG. 6. Nov. 19, 1942. Dog, 6.0 kg. **A.** Leads I and III, showing extrasystoles elicited by stimulating the right lateral ventricular endocardium approximately at the center. **B.** Leads I and III, showing extrasystoles elicited by stimulating a point on the center of the right ventricular epicardium directly external to the point stimulated at **A.** **C.** Leads I and III, showing extrasystoles elicited by stimulating a point in the left lateral ventricular endocardium approximately at the center. **D.** Leads I and III showing extrasystoles elicited by stimulating a point on the left lateral ventricular epicardium directly external to the point stimulated in **C.**

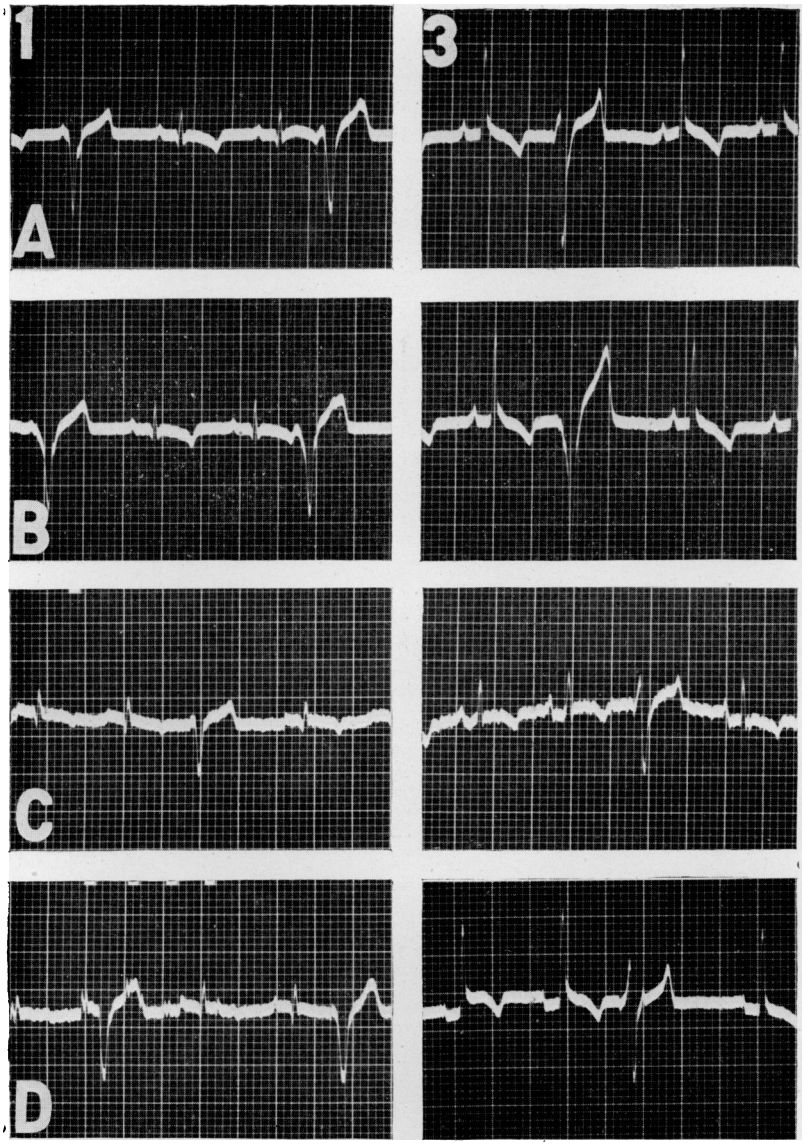

FIG. 7. Nov. 18, 1942. Dog, 7.0 kg. **A.** Leads I and III, showing extrasystoles elicited by stimulating a point about 2 cm. anterior to the endocardial point stimulated in Fig. 6, **C**. **B.** Leads I and III, showing extrasystoles elicited by stimulating the epicardium of the left ventricle directly above **A.** Extrasystole in **A 3** shows an R but in **B 3** does not. **C.** Leads I and III, showing extrasystoles elicited by stimulating a point in the left ventricular endocardium midway between point **A** and the anterior septum. **D.** Leads I and III, showing extrasystoles elicited by stimulating a point on the left ventricular epicardium directly external to **C.** Extrasystole of **C 3** shows an **R** of about the same amplitude as that of **D 3.**

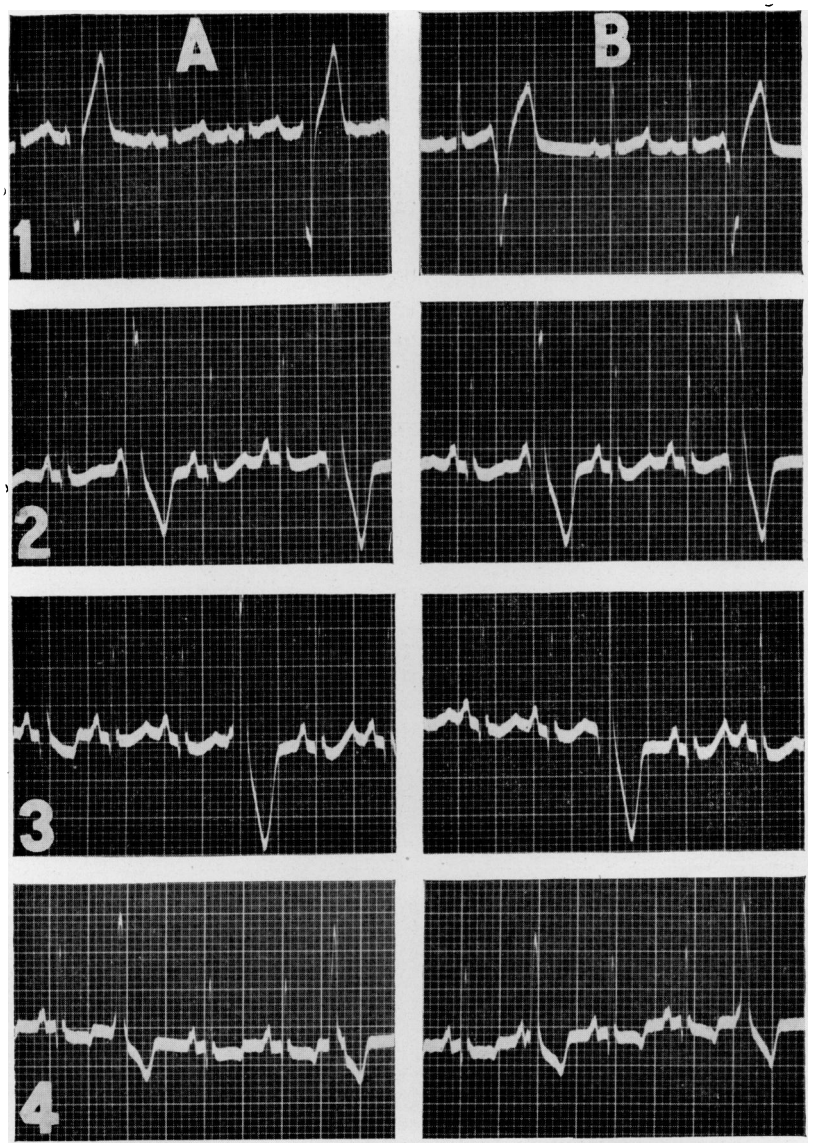

FIG. 8. Nov. 24, 1942. Dog, 9.5 kg. Lead III. **1 A.** Extrasystoles elicited by stimulating a point on the epicardium at the posterior septum midway between apex and base. **1 B.** Extrasystoles elicited by stimulating the right ventricular endocardium just beneath point **1 A.** **2 A.** Extrasystoles elicited by stimulating a point on the right ventricular epicardium in the posterolateral region about 2 cm. from the septum. **2 B.** Extrasystoles elicited by stimulating a point in the right ventricular endocardium just beneath **2 A.** The extrasystole shows a Q in **2 A** and **2 B.** **3 A.** Extrasystoles elicited by stimulating a point on the epicardium midway between the posterior septum and the right lateral position. **3 B.** Extrasystole elicited by stimulating a point in the endocardium directly underneath point **3 A.** Endocardial extrasystole alone shows a Q wave. **4 A.** Extrasystole elicited by stimulating the epicardium at the center of the right ventricle. **4 B.** Extrasystole elicited by stimulating the center of the right ventricular endocardium directly beneath point **4 A.** Neither complex possesses a Q wave.

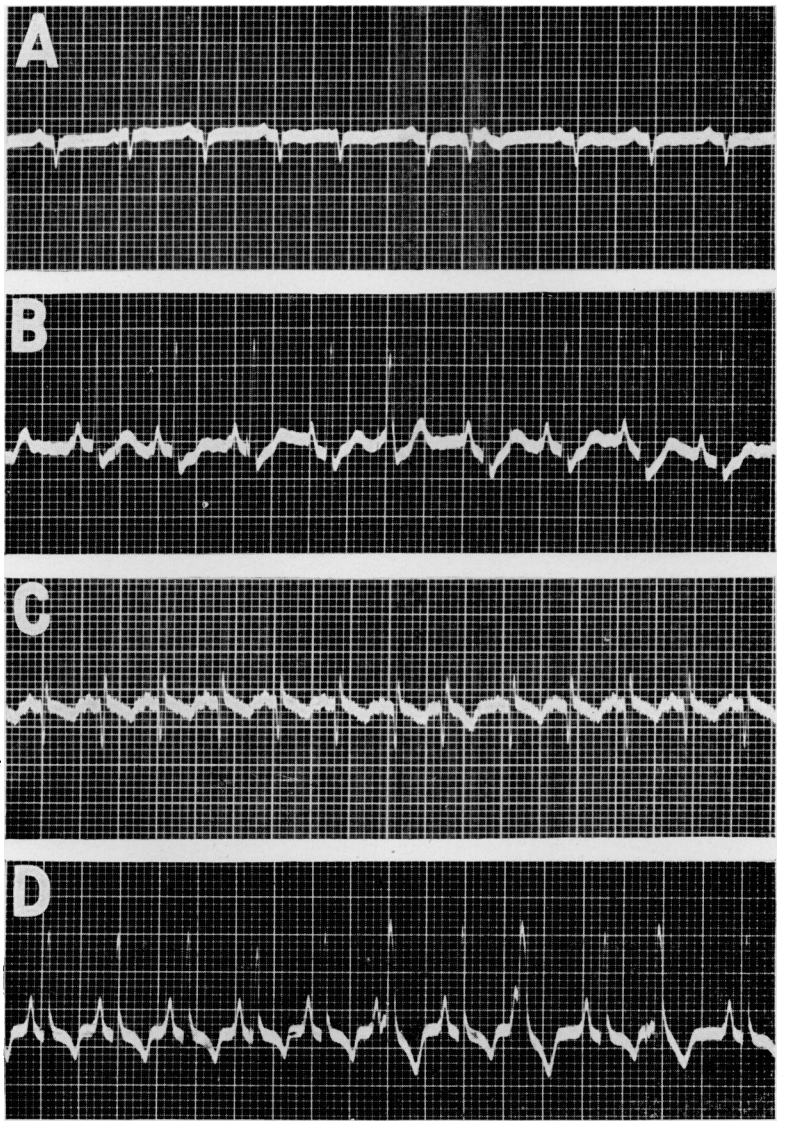

FIG. 9. Extrasystoles resembling the normal complex. **A** and **B**. Oct. 22, 1943. Dog, 15.0 kg. **A**. Lead I. Two ventricular extrasystoles closely resembling the normal complex elicited from the anterior septum midway between apex and base. A P wave distorts the second extrasystole. **B**. Lead III. A single complex resembling the normal complex, elicited from a point to the left of the septum and somewhat nearer the apex than the base. **C** and **D**. Oct. 23 1943. Dog, 10.0 kg. **C**. Lead I. A single ventricular extrasystole arising from the anterior right ventricle very close to the septum. **D**. Lead III. Four ventricular extrasystoles arising from the anterior right ventricle near the apex.

Interpretation

A. Principles of interpretation.

In order to interpret the electrocardiogram of a ventricular extrasystole in terms of the sequence of excitation and recovery in the heart, it is desirable to consider the following three principles:

(*1*) *Contribution of the surface to the electrocardiogram.* Although it is generally recognized that the cardiac impulse normally arrives at the endocardium before it extends to the epicardium, no evidence is available to indicate that this progress of endocardial excitation and outward passage is reflected in the electrocardiogram as recorded from the standard leads. On the contrary there is strong indication that excitation and recovery on the surface determine the electrocardiogram. Thus, an endocardial extrasystole is virtually indistinguishable from an epicardial extrasystole and shows no wave which might represent excitation in the endocardium.[7] Additional evidence is afforded by the readiness with which the various components of the ventricular complex of supraventricular as well as ectopic ventricular beats may be modified by altering conditions at the surface.

(*2*) *Recognition of surface involved by the direction of deflection of the electrocardiogram.* Excitation of the surface of the right ventricle has been shown to produce an upward deflection in the electrocardiogram as recorded from leads I, II, and III. On the contrary, excitation of the left ventricle causes a downward deflection.[21] Studies involving application of known potentials across the surfaces of the right and left ventricles indicate that such deflections must be associated with negativity of the active surface with respect to the inactive surface of the opposite ventricle.[10] It is immaterial for the purposes of the present exposition whether the so-called "membrane" or "dipole" theory is invoked to explain the observed difference in potential.[8]

In viewing any ventricular complex, therefore, an upward movement during QRS should be ascribed to excitation at the surface of the right ventricle, while a downward movement of the string should be attributed to excitation of the surface of the left ventricle. During phases of recovery, a downward movement indicates right ventricular recovery, while an upward movement indicates left ventricular recovery.

(*3*) *The significance of leads I and III.* Attention has been

called to the fact that lead I records preponderantly along an axis passing through the centers of the anterior left ventricle and the posterior right ventricle, while lead III records preponderantly along an axis passing through the center of the anterior right ventricle and the posterior left ventricle.[10, 21] This information permits division of the right and left ventricles into anterior and posterior subdivisions.

B. Principles of electrocardiographic interpretation apply equally to ectopic and supraventricular complexes.

All experimental procedures which modify the several components of the normal ventricular complex of an electrocardiogram in a special way produce similar modifications in the same component of a ventricular extrasystole. Some of these are listed below:

(1) *The R wave* of ventricular extrasystoles, as well as of normal complexes, has been increased in amplitude by cooling the left ventricle or warming the right ventricle.[21] Cooling the right ventricle or warming the left ventricle diminished its amplitude. These procedures, carried out on the anterior surface of the right ventricle and on the posterior surface of the left ventricle, modified R_3. When they were applied to the anterior surface of the left ventricle and the posterior surface of the right ventricle R_1 was modified.

(2) *The Q wave,* whether of ventricular extrasystole or of normal complex, has been increased in amplitude by warming the left ventricle and cooling the right ventricle.[9] Its amplitude was diminished by the opposite treatment of these regions. The lead which showed changes was determined by the regions of the heart subjected to heating or cooling (as in paragraph 1 above). In addition, application of KCl solution to the appropriate surface of the left ventricle resulted in the diminution or disappearance of Q, while KCl applied to the surface of the right ventricle increased the amplitude of the downstroke of Q but reduced or abolished its upstroke.[9]

(3) *The S wave* responded in every way like the Q wave to the various treatments outlined above.[23]

(4) *The S-T segment* was not a prominent feature in the electrocardiogram of most ventricular extrasystoles, due to the usual prolongation of the QRS interval. When one was present, however, it could be caused to appear elevated by damage to the left ventricle and depressed by right ventricular damage (Fig. 10). Damage to

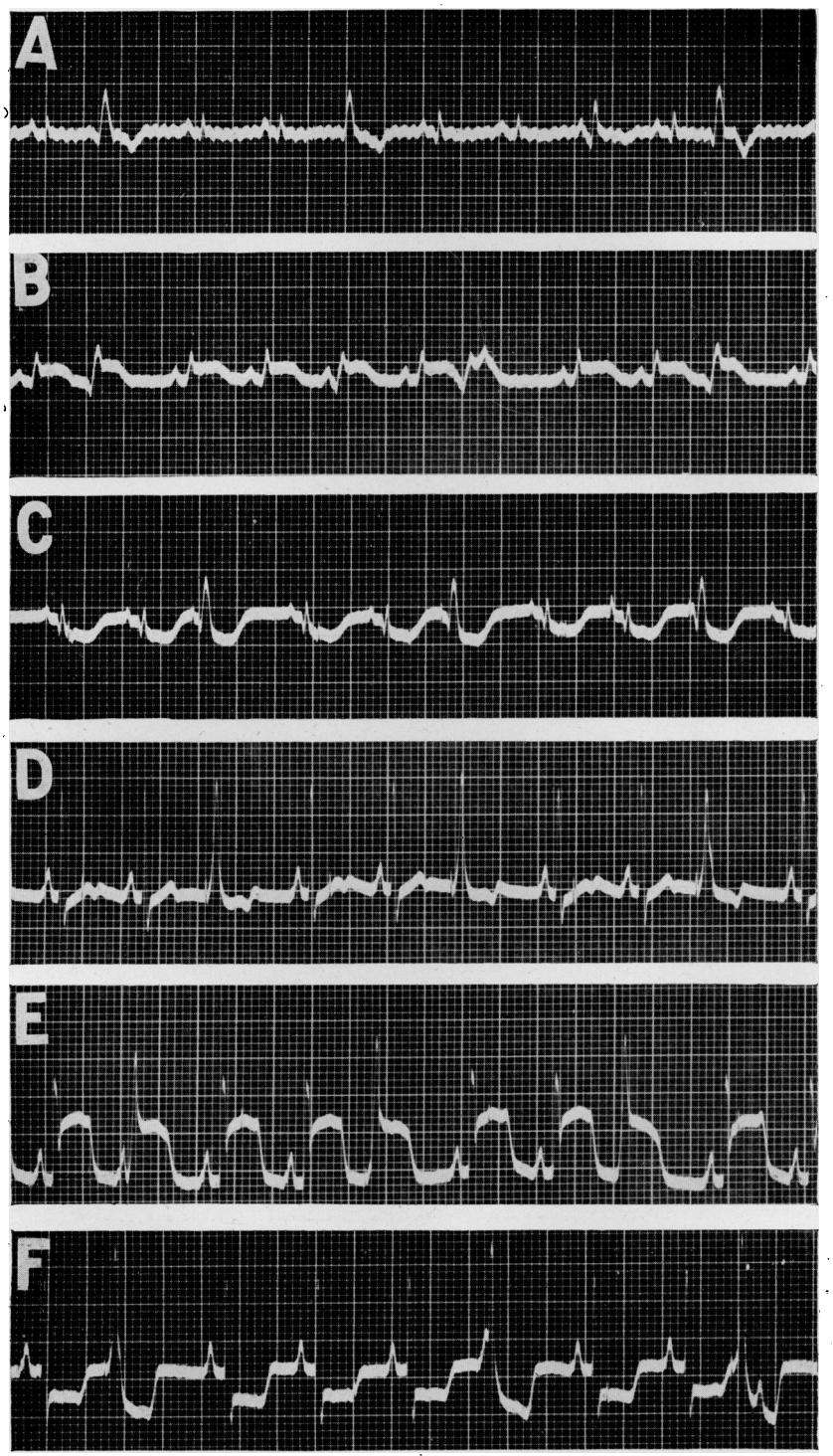

FIG. 10. Nov. 5, 1943. Dog, 8.0 kg. Lungs fully inflated and chest wound closed with clips during recording. **A.** Control, lead I. Ventricular extrasystoles elicited from a point on the anterior surface of the right ventricle some distance from the anterior septum. **B.** The same after application of *M*/5 KCl to the anterior surface of the left ventricle. Depression of diastolic base-line in both supraventricular beats and extrasystoles. **C.** The same after applications of KCl to the posterior surface of the right ventricle. Elevation of the diastolic base-line in the extrasystoles as well as in the normal complexes. **D.** Control, lead III. Extrasystoles elicited from the anterior surface of the left ventricle near the septum. **E.** The same after application of KCl to the posterior surface of the left ventricle. Diastolic base-line depressed in all complexes. **F.** The same after application of KCl to the anterior surface of the right ventricle. Diastolic base-line elevated in extrasystoles as well as in the normal beats.

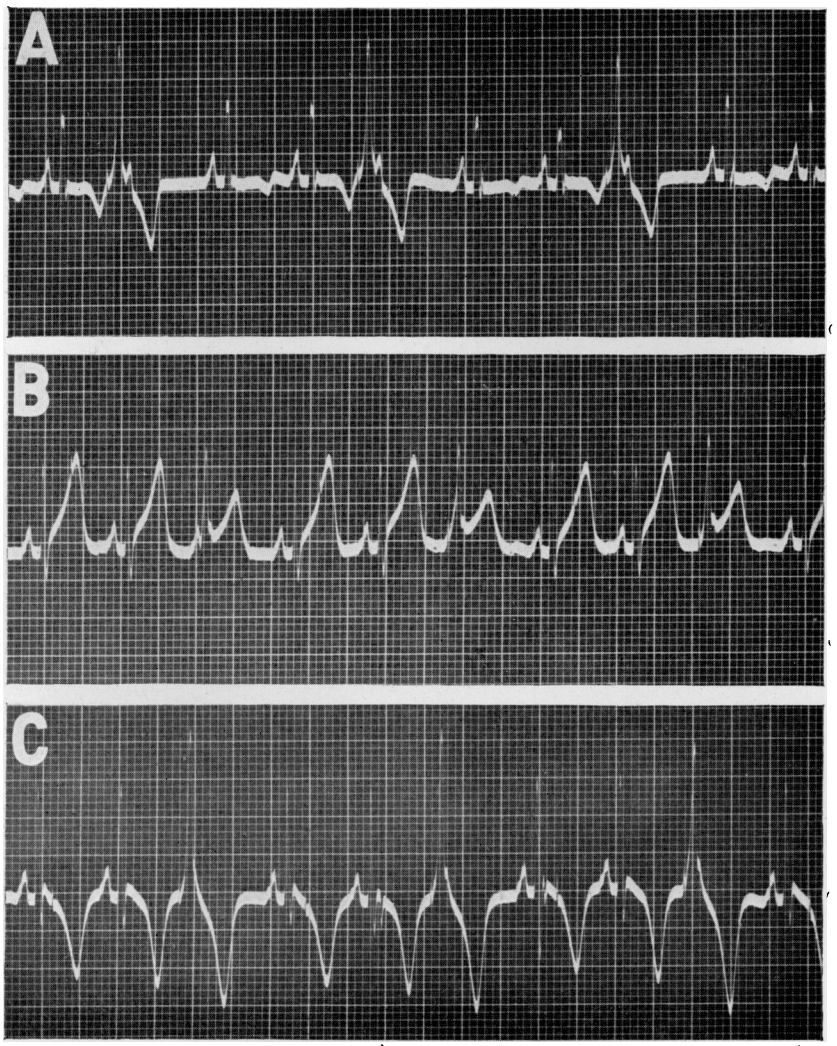

FIG. 11. Nov. 5, 1943. Dog, 8.0 kg. **A.** Control, lead III. Ventricular extrasystoles elicited from a point on the anterior surface of the left ventricle. **B.** Left apex warmed by thermal chamber placed over the left apex, circulated with water at 50° C. T waves of normal complexes as well as ventricular extrasystoles are now upright. Note also the reduction in amplitude of R of the extrasystoles. **C.** Left apex cooled by circulating chamber with water at 15° C. T waves of extrasystoles and normal complexes are now markedly inverted, and the R waves are increased in amplitude.

the anterior right or posterior left ventricle was manifest in lead III, while damage to the anterior left ventricle or posterior right ventricle was reflected in lead I.

(5) *The T wave.* Usually, though not always, the T wave of the extrasystole was directed oppositely to the main deviation of the initial part of the complex. This was not invariable, however, and extrasystoles could be evoked which showed T waves no different than in normal complexes of supraventricular origin. A normally upright T wave could be converted to a negative T, or it could be made more negative if already so, by cooling the surface of the left ventricle or warming the surface of the right ventricle (Fig. 11). It could be made upright, or its amplitude increased, by cooling the right ventricle or warming the left ventricle. Changes appeared in T_1 when the anterior surface of the left ventricle or the posterior surface of the right ventricle was affected, and in T_3 when the anterior surface of the right ventricle or the posterior surface of the left ventricle was treated.

C. The interpretation of the four basic patterns and transitional forms.

(1) *The right lateral pattern.* The exclusive presence of an R wave in both lead I and lead III indicates that both the anterior and the posterior surface of the right ventricle have been excited in advance of their counterparts in the left ventricle. This is consistent with the view that the extrasystole, which began at the center of the right ventricle, spread to adjacent regions before it arrived in more distant areas.

The termination of the complex by a T wave inverted in both lead I and lead III indicates that recovery was complete in the right ventricle before it was in the left.

(2) *The left lateral pattern.* The QS wave in both lead I and lead III is indicative of primary activation of the anterior and posterior surfaces of the left ventricle in advance of their right ventricular counterparts, and the configuration is understandable in view of the site of stimulation at the center of the left ventricle, from which an impulse would spread to the anterior and posterior surfaces of the left ventricle before reaching the opposite ventricle.

The upright T_1 and T_3 indicate that the left ventricle has recovered before the right.

(3) *The anterior septal pattern.* QS_1 indicates that the ante-

rior left ventricle was excited in advance of the posterior right ventricle. R_3 indicates that the anterior right ventricle was excited before the posterior left. This sequence of excitation is to be expected from spread of an impulse arising at the anterior septum.

(*4*) *The posterior septal pattern.* The presence of R_1 and QS_3 is explained by the extension of the impulse to the posterior surfaces of the right and left ventricles before it has travelled to the anterior surfaces.

(*5*) *Transitional stages.* These are readily explained on the basis of gradual transition between the four most simple configurations. In the case of complexes showing S waves, it must be assumed that some regions of the right ventricle are the last to be excited. This assumption fits the observed facts that the S wave may be altered by procedures influencing the time of discharge of the right ventricle, and by the observation that in normal beats the surface of the right ventricle at the conus may be the last to be excited.[13, 21, 22]

The septum at the apex is understandably the region in which maximum changes in configuration result from minimum movement of the stimulating electrodes. From this region it might be expected that all parts of the heart would be excited with almost equal rapidity, and thus give rise to the almost formless complexes of small amplitude actually observed.

As far as experimental studies in the dog are concerned, the major investigation with which the present experiments may be compared is that of Rothberger and Winterberg.[28] They differ in experimental method since the chest remained open in the studies of Rothberger and Winterberg, the state of the lungs was not mentioned, and an ano-esophageal lead was employed in place of lead III. Despite this, there is agreement in many points. In the first place, stimulation of a point just to the left of the septum at the base of the left ventricle gave a downward initial complex in lead I and an upward initial complex in the A-O lead which may be taken to correspond roughly with lead III. Moving the stimulus to the right from this point produced no change in A-O, but eventually produced an upright initial complex in lead I. Movement of the point of stimulus to the left produced complexes which were again concordant, but in which the initial complex was inverted in both leads. Finally, when the posterior septum was attained, discordant initial complexes appeared, being upright in lead I and downward in A-O. The apex of the left ventricle yielded concordant patterns, the

initial deflections being downward in lead I as well as in A-O. In short, there is complete accord in the actual results of the present experiments and those of Rothberger and Winterberg as far as the essential details of configuration are concerned. In both, the four basic patterns, anterior, posterior, right, and left, are agreed upon.

The question of the applicability of these results to man demands a review of the points of agreement and disagreement between human and animal experiments. The most significant of the former are those of Barker, Macleod, and Alexander,[3] not only in regard to the number of points explored and the accuracy with which they were localized, but also because of the clear recognition by these authors of the distinction between observed facts and theoretical considerations. The facts are that stimulation of the left ventricle, which was largely not exposed, at the base and near the anterior septum gave a complex resembling that obtained in the dog by stimulation of the same region; namely, an "anterior" extrasystole showing a QS_1 and an R_3. At the apex of the left ventricle, as in the dog, complexes followed the "concordant" true left pattern; i.e., both leads I and III showed downward initial complexes. The same pattern was found after stimulation of two points (nos. 11 and 12) on the posterolateral surface of the left ventricle near the base and apex, respectively. There is, finally, agreement that stimulation at the conus near the right base (point no. 10) yields a "concordant" pattern with initially upright complexes in both lead I and lead III. It is possible that point no. 4 in the Barker, Macleod, and Alexander study is in agreement with the present animal experiments, inasmuch as the change from the "anterior" to the "posterior" septal pattern takes place somewhat anterior to the apex.

This leaves disagreement concerning points 5, 6, 7, 8, and 9 on the anterior and lateral surfaces of the right ventricle. In the present animal experiments extrasystoles arising in these regions give rise to right lateral or anterior patterns, or transitional stages between them, while in the experiments of Barker, Macleod, and Alexander "posterior" configurations were observed. It appears more logical to suppose that this discrepancy is due to the fact that it was this very region that was exposed, rather than that a fundamental distinction between dog and human electrocardiograms exists. There is a certain amount of evidence to support this view. Prinzmetal, Oppenheimer, and Dach report studies on a patient in whom, because of an operation for constrictive pericarditis, an attempt was made to

stimulate the left ventricle which was completely exposed over a wide area.[25] Stimulation of a point on the upper part of the left ventricle 3 cm. from the anterior septum yielded complexes with upright initial deflections in both lead I and lead III. Later, after recovery, stimulation of the heart by mechanical stimulation by tapping gave typical "anterior" extrasystoles. Here a discrepancy was clearly noticeable, not only between these results and those on animals, but also between them and those of Barker, Macleod, and Alexander. When, however, the heart was again covered by a continuous layer of tissue, the discrepancy disappeared.

This experiment and others of a similar nature, in which the anterior surface of the heart has been stimulated by tapping the overlying skin, have been criticized because of the inaccuracy with which the exact point stimulated can be determined. This is an interesting question that has an immediate bearing upon the present problem. The criticism is probably true that no exact localization can be made, and that some stimuli fell on the right ventricle while others fell at the septum or on the left ventricle itself. Nevertheless, there is complete agreement that the pattern of extrasystoles so obtained is of the "anterior" type; namely, a downward initial complex, or QS in lead I, and an upward initial complex, or R in lead III. This is particularly noticeable in the experiments of Oppenheimer and Stewart where two points were tapped at each level, one toward the right and one toward the left.[24]

Toward the apex transitional complexes were found, i.e., low-voltage complexes in which lead I showed a small R and lead III a small QS,[4, 24, 25] which is entirely in agreement with the present experiments on dogs. Taps as far as possible to the left and toward the apex of the left ventricle showed the appearance and development of S_3 (Oppenheimer and Stewart, points 3, 4, 5, 6) and the final appearance of purely downward initial complexes in both lead I and lead III (Fossier, point 6). In summary it can, therefore, be stated that there is complete agreement between experiments on man and those on dogs when the heart is fully covered by at least some conducting medium. When, as has been the case in acute experiments on man, a portion of the heart has been exposed, results are in essential agreement when the points stimulated have been covered, and not in agreement when the region stimulated has been exposed. Clearly, there is need for investigation in man in which the question of exposure of the heart is adequately dealt with. The evidence

already at hand, however, indicates the probable validity of the localization of ventricular extrasystoles in man according to the patterns described here for the dog.

Summary

1. The endocardial and epicardial surfaces of the dog's heart have been systematically explored to correlate the configuration of ventricular extrasystoles with their site of origin.

2. No practical distinction can be made between an epicardial extrasystole and its endocardial counterpart.

3. Four basic localizing patterns have been described. These consist of right and left, and anterior and posterior septal configurations.

4. Transitional patterns intermediate between the four basic configurations were also described. Among these was a pattern characteristic of extrasystoles arising at the apex.

5. The ventricular extrasystole reflects, as does the normal complex, the sequence of excitation and recovery in the ventricles, and should be interpreted in accordance with principles found to apply in the interpretation of the normal complex. These principles are restated.

6. There is close agreement between these results and those obtained in man when the heart is not exposed, or when the point of stimulation is in a non-exposed region of the heart.

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