The electrocardiographic effects of electrical activity in the heart are partially canceled by multidirectional spread of that activity. For example, the effect of excitation directed toward the right may be canceled by a similar process moving toward the left. The extent of such cancellation is of fundamental importance in electrocardiography. It is a measure of the extent to which cardiac electrical activity could be altered without corresponding electrocardiographic changes. It is also the basis for estimates of the degree of cardiac electrical alteration necessary to account for given changes in the electrocardiogram.

This report concerns theoretical and experimental estimates of the spatial cancellation of electrocardiographic effects during ventricular excitation in the dog. A previous estimate of 90 per cent cancellation during ventricular activation in man has been reported on the basis of frontal plane observations. The theoretical estimates in this study were made from data on ventricular excitation sequence, as reported by Scher. Horizontal plane diagrams of the heart published to illustrate the sequence were employed, together with graphic constructions of frontal plane diagrams. To prepare frontal plane diagrams, the horizontal plane outlines were assumed to be superimposed and four levels parallel with the frontal plane were selected. Measurements were made from the edge of the largest horizontal diagram to the edges of each diagram and each activation front as they appeared at the level of the frontal plane section which was being constructed. These values were plotted on lines representing the level of horizontal plane sections viewed from the frontal plane and the frontal diagrams were completed by interpolating lines between the points. Construction of the frontal plane from horizontal plane sections is illustrated in figure 1.

The total length of all excitation fronts on frontal and horizontal diagrams was determined with a map measurer. Wave front length from each diagram was multiplied by a factor proportional to the thickness of the section represented by the outline before being added to the length from other sections. Vectors representing uncancelled portions of the excitation fronts in horizontal and frontal planes were constructed by a method previously employed to derive the electrocardiogram from the excitation sequence. With this method, the ends of excitation fronts interrupted by a surface of the heart were joined with straight lines and vectors were plotted perpendicular to the lines. Each vector was given a magnitude proportional to the length of the line on which it was plotted and also to the thickness of the section represented by the outline before being added to the length from other sections. Vectors representing uncancelled portions of the excitation fronts in horizontal and frontal planes were constructed by a method previously employed to derive the electrocardiogram from the excitation sequence. With this method, the ends of excitation fronts interrupted by a surface of the heart were joined with straight lines and vectors were plotted perpendicular to the lines. Each vector was given a magnitude proportional to the length of the line on which it was plotted and also to the thickness of the section represented by the outline from which it was derived. Vectors representing simultaneous fronts were added vectorially in frontal and horizontal planes. The lengths of all vectors representing separate moments in the excitation process were then added arithmetically and compared with the total length of excitation fronts from which the vectors were derived. This comparison of total length of excitation fronts from which the vectors were derived. This comparison of total length of excitation fronts from which the vectors were derived. This comparison of total length of excitation fronts from which the vectors were derived.

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Figure 1
Diagrammatic representation of the construction of a frontal plane section (B) from horizontal plane cardiac sections (A). The points labeled 1 through 8 on the horizontal sections illustrate the measurements carried out to construct frontal plane sections. These points are shown on the frontal section in their appropriate relation to each other. Lines interpolated between these points define the outer wall of the frontal plane cardiac section. A similar definition of points is employed to define the inner cardiac wall completing the outline of the frontal section. Excitation fronts on horizontal plane sections were used to construct the excitation pattern on frontal sections in the same manner as the cardiac outlines were constructed.

Experimental Estimate

This was obtained by comparing the area of spatial magnitude curves recorded during normal ventricular activation with ectopic excitation originating in the ventricles. Experiments were performed on 10 dogs in the weight range of 5 to 10 Kg., anesthetized with 4 mg./Kg. morphine subcutaneously, followed in one hour by intravenous chloralose, 50 mg./Kg. dissolved in 10 per cent urethane solution. With this anesthesia, high vagal tone resulted in slow sinus rhythm which facilitated control of the cardiac mechanism by ventricular stimulation. An orthogonal lead system designed for the dog was employed to obtain XYZ components of cardiac electrical activity. A bipolar electrode constructed of a 17-gauge hollow needle with a stylet projecting 2 mm. beyond the needle tip was used for ventricular stimulation. Needle and stylet were insulated except at the tips.

Spatial magnitude curves were obtained by means of an analogue computer which continuously performed the calculation \( \sqrt{X^2 + Y^2 + Z^2} \) on the output of the lead system. Curves were recorded as linear functions of time on an oscillograph, using a paper speed of 25 inches per second. Areas of the ventricular excitation portions of the curves were measured with a planimeter and expressed in microvolt seconds.

In each experiment, the stimulating electrode was inserted through the intact chest wall into the ventricular myocardium to preserve the volume conductor characteristics of the chest. Magnitude curves were recorded during sinus rhythm and while the ventricles were being driven with stimulus intensity and frequency regulated in each experiment to give control of the cardiac mechanism. Curves during ventricular stimulation were always compared with those during normal sinus rhythm recorded a few seconds before or after the period of stimulation. Multiple stimulation sites, varying from 4 to 21, were employed in each experiment and a total of 96 stimulation sites were used in all experiments.
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FIGURE 2

Diagrammatic illustration of the method employed to make a theoretical estimate of cancellation. In A, a diagrammatic horizontal plane cardiac section is shown, with activated myocardium shown as the shaded area. Activation wave fronts are indicated by a, b, c, and d. In B, straight lines joining the ends of these wave fronts are shown as a', b', c', and d'. Vectors 1 and 2 are perpendicular to these lines and have a magnitude equal to the length of the lines. In C, vectors 1 and 2 have been added to give the resultant V', which reflects the portions of the original wave fronts in which the electrocardiographic effects are not canceled.

Results

The theoretical estimate of cancellation made on the basis of Scher's ventricular excitation data was 71 per cent. In the experimental studies, three sites of ventricular stimulation resulted in smaller magnitude curve areas than those resulting from supraventricular excitation. The remaining 93 sites resulted in magnitude curve areas 3 per cent to 77 per cent greater than the areas during normal excitation. In each experiment, the largest area during ventricular stimulation with reference to the area during supraventricular activation was considered the most reliable estimate of cancellation. This estimate varied between 44 and 77 per cent, with an average value of 62 per cent. Examples of spatial magnitude curves obtained during sinus rhythm and during ventricular stimulation are shown in figure 3. Data on which the cancellation estimates were based are summarized in table 1.

Since some cancellation was almost certainly occurring even during ectopic excitation yielding the largest magnitude curve area, the actual cancellation of electrocardiographic effects during normal ventricular activation must have been even higher. The theoretical estimate of cancellation during radial excitation from a point on the lateral wall of the left ventricle was 13 per cent. Increasing the value of the largest experimental magnitude curves during ventricular stimulation by this amount and comparing the new values with the area of magnitude curves during normal activation gave cancellation estimates of 51 to 80 per cent, with an average of 66 per cent.

Discussion

The present study can only be considered to give an estimate of the degree of cancellation of electrocardiographic effects of ventricular excitation. The theoretical estimates necessitated several simplifying assumptions, including consideration of excitation at a limited number of moments and at a limited number of anatomical levels. More accurate estimates will become possible when still more detailed descriptions of the sequence of cardiac electrical events have been obtained. The theoretical estimates were also dependent upon assuming uniformity of the excitatory
TABLE 1

Summary of Data on Which Experimental Estimates of Concentration Were Based*

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<th>Experiment number</th>
<th>Magnitude area, µv. seconds</th>
<th>Per cent cancellation</th>
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*The magnitude curve areas listed represent the paired observations during normal sinus rhythm and during ectopic stimulation which indicated the highest degree of cancellation in each experiment. The adjusted area of magnitude curves and adjusted per cent cancellation reflect the results of considering ectopic stimulation itself to be 13 per cent canceled. This value for cancellation during ectopic excitation was based on a theoretical estimate assuming radial spread of activation from the ectopic site.

process along the length of excitation fronts. This assumption has been useful in electrocardiography, but its validity has not been definitely established.

A further limitation of the cancellation estimates obtained in this study is that they apply only to remote electrocardiographic leads. To whatever extent specific leads, such as those from the precordium, do not operate in this manner, the cancellation estimates reported are not applicable.

Despite all of the limitations of the present study, it is felt that the results have value for understanding the electrocardiogram. Both the theoretical and experimental estimates indicate a high degree of cancellation of electrocardiographic effects of ventricular excitation. The single previous attempt to quantitate such cancellation yielded an even higher value in man than that estimated for dogs in the present study. This difference may be a result of the fact that the estimate for man concerned only the frontal plane or may be related to the different size, shape, and excitation sequence of the human and canine hearts.

The large amount of cancellation of electrocardiographic effects of ventricular excitation gives an important insight into the mechanism of electrocardiographic alterations by disease. As pointed out by Schaefer, alteration of the direction of excitation in a small portion of the ventricular muscle mass may result in a marked alteration of the QRS complex. Based on the average cancellation estimate of 66 per cent obtained in this study, the activation of only 34 per cent of the ventricular mass contributes to the form of the body surface electrocardiogram. Reversal of the direction of excitation in one-half of this or 17 per cent of the ventricular mass could theoretically alter the magnitude curve area by 100 per cent, with correspondingly large changes in the electrocardiogram. These findings provide an explanation for the well-known fact that small localized myocardial lesions sometimes result in marked abnormalities of the QRS complex.

A high degree of cancellation also provides an explanation for some limitations of the electrocardiogram in the recognition of disease. If activation of approximately 66 per cent of the normal ventricular mass does not influence the form of the electrocardiogram, the possibility of considerable alteration of cardiac electrical activity without correspond-
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ing electrocardiographic changes is present. While it is unlikely that a myocardial lesion would involve only portions of the ventricle in which excitation is self-canceling, the extent to which such involvement occurs would be a major determinant of the degree of QRS alteration produced. The findings reported make it unlikely that the size of myocardial lesions can be estimated by the degree of the electrocardiographic alteration.

Summary

Theoretical and experimental estimates of the amount of cancellation of electrocardiographic effects of ventricular excitation have been carried out. The theoretical estimate was based on the excitation sequence reported by Scher and consisted of comparing the total length of excitation fronts at several moments in the activation process with a measurement of the uncanceled portions of these fronts.

Experimental estimates in 10 dogs were based on the relative areas of spatial magnitude curves during normal ventricular excitation and during stimulation of the ventricular site yielding the largest magnitude curve area. A theoretical estimate of the cancellation to be expected during ventricular stimulation was also carried out. The theoretical estimate of cancellation of electrocardiographic effects during normal ventricular activation was 71 per cent, while the experimental estimates varied from 51 per cent to 80 per cent, with an average of 66 per cent.

References

Cancellation of Electrocardiographic Effects During Ventricular Excitation
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