The formation of a model is an important phase of all scientific thinking in that it serves to demonstrate or explain the workings of an inherently complex natural system in terms that can be more readily understood.

In electrocardiography, the Einthoven triangle has been used for years as a relatively simple geometric model of an essentially complex volume conductor, the human torso.

This model, in spite of many oversimplifications, has served for over 50 years as a useful reference frame for a vast amount of data and theory. More recently, the definitive mapping study by Scher of the sequence of myocardial depolarization in dogs, again a simple model of a very complex phenomenon, has served to illuminate and clarify a vast body of empirical data. This model has added greatly to our understanding of previously confusing findings in electrocardiography and vectorcardiography.

The introduction of computer technics

Figure 1

In the simulation the heart was pictured as consisting of 20 segments; this diagram gives an approximation of the direction assigned to the vector representing each segment.
(analog or digital) now makes it possible easily and rapidly to study more sophisticated models, especially those that are non-linear. These technics have had wide application in engineering and physical sciences and are currently being utilized to an increasing extent in biology and medicine. It seems conceivable, therefore, that such a model applied to the electric field of the heart might enhance the contributions of Scher to our understanding of the relationship between myocardial pathology and observed electrocardiographic and vectorcardiographic changes.

Furthermore, the value of a good mathematical model of the heart and the surrounding volume conductor is clear-cut. If the simulation is adequate, then a large number of experiments could be run mathematically and the key experiments validated by observations either in animals or in human beings. Such an approach may well eliminate vast amounts of time in unproductive experimentation and pinpoint the areas of animal experimentation and human documentation that are most likely to yield significant results.

The work reported here is a study of the feasibility of simulating the vectorcardiogram by means of a relatively simple analog computer model.

**Methods and Materials**

The approach used in this simulation is based on concepts derived from Scher’s work in the sequence of ventricular myocardial depolarization in dogs. Two obvious modifications were necessary to simulate the human vectorcardiogram. The duration of depolarization was increased to correspond with that observed in man and the directional forces were chosen to allow for the anatomic differences observed in the orientation of the heart in the dog chest as compared to the human. An additional liberty was taken by

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**Figure 2**

Shown here is the time history of the field strength generated along the axis of a typical segment of the model. Segment 2 from the apex of the interventricular septum is shown with its assigned direction in space. The contribution of this time varying vector to one of the orthogonal axes, here the Y axis, will be the strength of that vector (R) at a given instant, times the cosine of the angle that the vector makes with the Y axis.
Figure 3
The time history of the current field strength from each of the 20 segments is shown in the central portion of this figure. To the right are the direction cosines (I, M, and N) for each of...
increasing the disparity between the leftward and rightward forces in the septum. Instead of the ratio of 2/3 left to right and 1/3 right to left, as would be suggested by Scher, the left-to-right forces were assumed to be 80 per cent of the total energy from the septum. These changes were suggested by vectorcardiographic findings in our laboratory, particularly in varying degrees of right ventricular hypertrophy.

In this simulation the heart was pictured as consisting of 20 segments of myocardium; seven for the septum, nine for the left ventricle, and four for the right ventricle (fig. 1). The heart was assumed to be in a purely resistive, homogeneous, conducting medium of infinite size. The pickup electrodes were assumed to be on orthogonal axes at such distances that the effect of the dimensions of the heart was negligible. These are the same assumptions made in clinical vectorcardiography.

The current fields from each of the 20 segments were assigned as fixed direction vectors approximately perpendicular to the epicardial surface of the center of each segment. These directions were chosen to correspond to standard textbook anatomy of the heart. These current fields were also assigned a magnitude that varied with time during the passage of the depolarization wave through the segment. Because of the rapid endocardial conduction of impulses as compared with the slower propagation of the wave front through the myocardium, the wave front will enter any given geometric segment somewhat obliquely and, therefore, the current buildup will always be somewhat gradual. The wave front will leave the segment in the same manner so that decay of the current field strength will also be gradual. The value of the field strength from moment to moment along the chosen vector is assumed to be proportional to the effective area of the wave front within that segment. The duration of the current field from any given segment will be a function of the thickness of that segment. Figure 2 represents a typical segment showing the field strength as a function of time that will be generated by this segment.

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these segments. They represent the contribution to the X, Y, and Z orthogonal axes respectively of each segment.
The diagrams published by Scher were used to assign the time of onset of each segment and the duration of each segment. The published time intervals were scaled upward to produce a total QRS duration of 80 milliseconds. The 20 segments and their time of onset, duration, magnitude, wave form, and the direction cosines assigned are shown in figure 3. The size of the segments in this simulation varied, and hence the magnitude of the function representing that segment varied proportional to the effective surface area that is perpendicular to its vector.

Since this model was to be viewed in an orthogonal system, it was necessary to determine what the value of each segment would be as seen in each of three axes (X, Y, Z). These axis contributions were determined by the product of the scalar magnitude from that segment and the cosine of the angle between each segment’s force and the axis in question (fig. 2).

The simulation was performed on an EA1 231 R analog computer. In the computer (fig. 4), each segment was embodied in the form of a variable diode function generator. When driven by a ramp voltage, it delivered the wave form which had been previously stored in it. The rate at which it scanned this wave form was determined by the slope of the ramp. All function generators were driven by the ramp voltage from a single ramp generator, whose role might be considered roughly analogous to the AV node. This ramp voltage was fed through each one of 20 operational amplifiers to their corresponding variable diode function generators. These amplifiers acted as a conduction system in that a bias voltage on the amplifier determined the time of initiation of each segment. Following each function generator, an attenuator determined the scalar magnitude from each segment. This allowed for changes in the magnitude from any

![Figure 5](image)

Three planar projections of the computer output. The horizontal plane was produced by plotting the summed output from the computer for the X and Z axes, the frontal plane the summed X and Y output, and the sagittal plane the summed Y and Z output.
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HORIZONTAL PLANE

SIMULATED RVH
GAIN INCREASED ON ALL RIGHT VENTRICULAR SEGMENTS

Figure 6

The simulation of right ventricular hypertrophy was accomplished by increasing the output from the four right ventricular segments by increments as shown. The computer's normal horizontal plane vectorcardiogram is shown in the upper left-hand corner for reference. The middle figure at the bottom is an actual spatial vectorcardiogram recorded from a patient with congenital pulmonary stenosis whose right ventricular systolic pressure was 75 per cent of systemic pressure. The computer simulation resembles the reality in all significant aspects.

Results

When the summed outputs from the computer, representing each of the orthogonal axes (X,Y,Z), were plotted in three planar projections, as seen in figure 5, the summed outputs did indeed resemble a normal vectorcardiogram.

Right ventricular hypertrophy was simulated by increasing the voltage from right ventricular segments in a stepwise fashion. Figure 6 demonstrates that a reasonable simulation of right ventricular hypertrophy was accomplished in this manner and that the changes produced did indeed resemble varying degrees of right ventricular hypertrophy. A clinical vectorcardiogram is shown.
for comparison from a patient with valvular pulmonary stenosis in whom the systolic right ventricular pressure is 75 per cent of systolic systemic pressures. Although varying in minor detail, it will be noted that the general configuration as well as the amplitude and direction of changes in the model are all quite similar to that observed in pathologic right ventricular overload.

Left ventricular hypertrophy was simulated by increasing the scalar magnitude of all left ventricular segments in a stepwise fashion; this is recorded in the horizontal plane in figure 7. A spatial vectorcardiogram from a patient with moderately severe systemic hypertension for the past 10 years is included for comparison.

A number of simulations of myocardial infarction were done by eliminating one or more segments at a time. An example of such a simulated infarct is given in figure 8, in which three apical segments are removed, one from the septum and two from the left ventricular wall. For comparison, a clinical vectorcardiogram is presented which was taken from a patient who had an infarction involving approximately the same mass and location of myocardium. It should be noted that the clinical material for comparison was found after the computer work had been done and the computer work was not based on an attempt at curve fitting.

**Discussion**

This analog computer model of the electrical events of the heart has been based on experimental knowledge of ventricular depolarization. Not only did these results resemble

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*Figure 7*

Left ventricular hypertrophy is simulated by increasing the output from all left ventricular segments by increments. The lower right figure is a horizontal plane spatial vectorcardiogram for comparison from a 72-year-old woman who had had systemic hypertension for at least 10 years.
Myocardial infarction was simulated on the computer by reducing the output to zero from certain segments. Here a lateral infarct was simulated. The lower right-hand figure is a spatial vectorcardiogram from a patient with a large lateral myocardial infarct at autopsy.

normal vectorcardiographic loops, but also changes based on concepts of pathologic physiology produced configurations resembling abnormal clinical vectorcardiographic findings.

When this model has been perfected, it would seem feasible to introduce these 20 functions as dipoles into the electrolyte medium in either cadaver or artificial torso studies. Although 20 dipoles is a rather small number, it appears that a reasonable simulation can be accomplished with this number. Such applications should greatly enhance our understanding of the validity of various lead systems, since we would have a physical and an electrical model of the heart where the input is entirely specified.

Furthermore, it appears quite likely that most vectorcardiographic diagnostic "patterns" can be simulated with this simple model. Experimentation may allow us to detect previously unrecognized alterations in wave form, permitting diagnosis of infarction or other processes not possible by present knowledge and criteria.

The work to date leaves little doubt that a more sophisticated model can be developed with computer technics that simulates more closely the electrical events of the cardiac generator. It seems likely that a digital program can be written which will simulate the direct propagation of a wave front as seen in extrasystoles in addition to normal conduction. It would then be a simple matter to simulate a combination of normal conduction and direct propagation as seen in bundle-branch block.

The simulation reported here was based on
the assumption that the heart was small (that is, all dipoles could be considered as having a common origin and proximity effects to any given electrode could be ignored). In reality this obviously is not true. With digital computer technics it appears possible to measure the contribution of each one of these dipoles located at differing distances from each of the pickup electrodes and to test the actual proximity effects of each. Wilson and Bayley\(^3\) have proposed analytical methods of measuring eccentric dipoles within a spherical conducting medium and Okada\(^4\) has developed a computational solution for a finite cylinder with an eccentric dipole. A rather long iterative, digital, computational method has recently been developed by Gelernter and Swihart\(^5\) to simulate both the irregular boundary of the thorax and irregular areas of tissue inhomogeneity. It must be confessed immediately that this is a far cry from the simple analog simulation reported here. With such a sophisticated model, many of the problems that plague electrocardiography and vectorcardiography can certainly be evaluated and placed on a rational foundation. It is our hope that the further exploration of a combination of our simulation of the cardiac generator and Gelernter-Swihart simulation of the volume conductor, will remove much of the empiricism and dogmatism from the field of clinical electrocardiography and vectorcardiography and place these studies on a more rational basis.

**Summary**

This study demonstrates the feasibility of developing an analog computer model of the electrical events of the heart, based on a rational application of experimental knowledge of ventricular depolarization.

Not only did the results resemble normal vectorcardiographic loops, but also changes were analogous from concepts of pathologic physiology that produced configurations resembling known clinical vectorcardiographic abnormalities.

It is expected that all known vectorcardiographic deviations can eventually be simulated on a rational basis, and, further, that previously unrecognized alterations may be found permitting diagnosis of infarction or other processes not possible by present knowledge and criteria.

The present study strongly supports the view that most, if not all, of the factors known to influence the electrical field of the heart can be simulated by computer technics. When this is accomplished, it may be possible to remove much of the dogma and empiricism from traditional electrocardiographic theory and place it on a more rational foundation. The need for further exploration in this area seems imperative.

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**References**
