The Effect of a Threshold Electrical Stimulus upon the Repolarization Process of the Left Ventricle of the Intact Dog Heart

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A threshold stimulus applied either at apex or base of the intact dog ventricle at any depth of the myocardium produces a delay in repolarization during the following three to four normal beats, in an area equivalent to the size of the proximal zone of the precordial lead. This change in repolarization is not caused by postextrasystolic distension of the ventricle. The question is discussed whether a threshold stimulus initiates a propagated disturbance from the point of the electrodes or the larger area whose repolarization is altered.

In the course of a study on the excitability cycle of the apex and base of the normal dog heart performed with an intact chest wall and with the heart beating spontaneously in situ, it was observed that after a forced extrasystole the oncoming beats showed T-wave alterations with a definite pattern.

This phenomenon has already received attention from the clinical point of view. Levine and co-workers have pointed out that the T-wave alterations in postextrasystolic beats are most likely to occur in hearts that are already the seat of damage, but that they can be found in a small proportion of normal hearts though not in normal individuals. In speculating on the possible explanations for this phenomenon various authors have suggested several causes:

(1) Changes in repolarization as a result of alterations in cycle length.

(2) Mechanical effects of overdistension of the ventricular wall, particularly in those cases that already suffer from coronary artery insufficiency of either the inner or the outer layers of the myocardium.

The production of forced beats has become an important technic in the study of localization of activity in the heart as well as the spread of excitation through it. In fact, the theory of limited potential differences in regard to the explanation of the ventricular complex was originally propounded on the basis of a study of the configuration of the initial phases of the ectopic beat in endocardial and epicardial stimulation.

The T waves of postextrasystolic beats have received little attention experimentally, since the repolarization process in exposed hearts is subject to alterations from temperature, water, and carbon dioxide changes at the cardiac surface. If the stimulation can be effected in a known region of a normal heart in situ, the repolarization of the ventricle following a forced beat can be studied, and light thrown on the problem of the area involved by a threshold stimulus. It is already known that changes in the local excitatory state can be produced by a subthreshold stimulus, but no evidence on the area involved is at hand. One can make deductions about the size of this area from a study of the T waves following a forced beat, since if changes are present they would be due to involvement of a measurable portion of the myocardium.

Method

The apical and basal regions of the hearts of anesthetized dogs were stimulated by means of a bipolar needle electrode inserted into the myo-
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cardium through the intact chest wall. Threshold stimuli were introduced at two-minute intervals from a rectangular wave generator through a mercury key. The hearts were allowed to beat spontaneously, and by means of a delayer system the stimuli could be introduced at any desired instant.

The type seen in figure 1B were observed. In this example the T wave in the complex preceding the extrasystole was diphasic, while the succeeding beat exhibited an upright T wave.

FIG. 1. Extrasystoles and postextrasystolic T-wave changes produced by rectangular wave stimulation in the dog. Unipolar precordial lead from over left apex. In A, stimulating electrode at apex of left ventricle, paper speed 25 mm. per second. In B, stimulating electrode at base of left ventricle, paper speed 75 mm. per second.

RESULTS

In 27 animals forced beats from stimulation of the left apex were uniformly followed by a compensatory pause, after which the succeeding beats showed inversion of the T wave which progressively decreased in amplitude until the fourth or fifth postextrasystolic beat, at which time the T wave had reverted to its prestimulation pattern (fig. 1A).

In experiments in which the ventricular base was the site of origin of the extrasystole, the apical precordial lead did not constantly reveal changes in the configuration of the postextrasystolic T wave. At times alterations of

The Q-T intervals in the complexes following the extrasystoles were always prolonged, as indicated in the following tabulation:

Apical Stimulation (fig. 1A)
Beat preceding extrasystole: .24 second
First beat following extrasystole: .29 second
Second beat following extrasystole: .27 second
Third beat following extrasystole: .25 second
Fourth beat following extrasystole: .25 second
Fifth beat following extrasystole: .24 second

Basal Stimulation (fig. 1B)
Beat preceding extrasystole: .26 second
First beat following extrasystole: .29 second
Second beat following extrasystole: .26 second
DISCUSSION

The evidence reported indicates that an electrical stimulus of threshold intensity applied to any region of the ventricle produces a short period of delayed repolarization around the point of application of the shock, as evidenced by an alteration in the T wave of the precordial lead. When the shock is applied underneath the exploring electrode, prolongation of the Q-T interval and conversion of an upright to a sharply inverted T wave takes place in the four or five beats following the extrasystole. This change is transient, and both the Q-T interval and the T wave return to the previous pattern after five or six beats.

Although overdistension of the ventricle with impairment of nutrition of endocardial lamellae has been proposed as the explanation for T-wave changes following extrasystoles, this is probably not the basis for the changes reported here. In these experiments basal extrasystoles caused a delay in repolarization in the area around the point of stimulation at the base, while apical extrasystoles produced a similar delay in the corresponding area at the apex. If ventricular distension were the cause, one would not expect such selective effects to appear, and the configurational changes would be the same regardless of the site of stimulation. Furthermore, since the hearts of these animals were presumably normal, the effects of overdistension should disappear after the first oncoming beat following the extrasystole rather than after the fourth or fifth beat. The presumption is justified that the change has been caused by an electrical effect at the point of application of the shock and in a sizeable area of myocardium around it.

The size of the area involved by the shock is the major portion, if not the entire area, of the proximal zone of the precordial lead. If only the point of stimulation were the seat of the alteration in repolarization, no significant changes would appear in the T waves of the oncoming beats following the extrasystole. Pinpoint currents of injury and pinpoint changes in repolarization caused by heating and cooling are not of sufficient magnitude to affect the precordial T wave. Furthermore, a pinpoint change in the distal zone would most certainly not be detectable in the precordial lead, since it has been shown that alteration in the distal zone must involve a considerable portion of it to produce a configurational change in the T wave. In these experiments basal stimulation did produce transient changes in the apical lead in the postextrasystolic beats. The presumption is strong, therefore, that a shock applied to either the base or the apex of the left ventricle alters the repolarization of a zone of myocardium almost equivalent in size to the area of the proximal zone. It has already been established that this area is at least 2 to 3 cm. in diameter.

The striking difference in the magnitude of effect upon the T wave of the postextrasystolic beats produced by apical as compared with basal stimulation may be explained by the relation of the size of the areas involved to the size of their respective zones. If the major portion or the entire area of the proximal zone is involved by a shock, the changes in the T wave will be more pronounced than if an area of the same size is affected in the distal zone, since the latter is very large in comparison to the proximal zone. Thus an effect in a distal area comparable in size to the proximal zone would produce relatively minor alterations. This was encountered in these experiments and is readily explained on the basis of the view that a precordial V lead results from the interference of electrical potentials generated in the proximal and distal zones of that lead.

Since the cylindrical electrode was inserted at random depth from one experiment to the next, the resulting extrasystoles must have arisen at various depths in the left ventricular wall. This variation made no difference in the character of the T-wave changes in the oncoming beats since apical stimulation, at whatever depth the electrode happened to be, produced inversion of the T wave and prolongation of the Q-T interval in the several beats following the extrasystole. This demonstrates in another way the finding previously described from this laboratory that endocardial and epicardial stimulation produced extrasystoles of the same configuration in a precordial lead. There is, therefore, no support from these obser-
observations for the view that the T wave is the result of the interference between potentials generated at the endocardial and epicardial surfaces beneath the exploring electrode.

The final problem raised by these observations is whether the applied shock causing the extrasystole depolarizes a small point between the stimulating electrodes but affects the repolarization of a large area around it, or whether a threshold shock in the heart brings into activity not the muscle at the point of the electrodes alone but a much larger area, perhaps comparable with the area whose repolarization is affected in the succeeding beats. This question is important, since much electrocardiographic experimentation has depended on the assumption that a threshold shock depolarizes the area immediately around the stimulating electrode and that the propagated disturbance begins from this point. These experiments do not actually disprove this assumption, but they do cast some doubt upon it, since the shock affects a large area in the myocardium around the point of stimulation insofar as the repolarization process is concerned. The concept that the depolarization caused by a threshold shock in the ventricle begins at a point cannot be assumed and requires further experimentation. In this connection it is worth while to point out that Lewis did make this assumption when he proposed the theory of limited potential differences on the basis of a difference in appearance of endocardial and epicardial extrasystoles.  

Summary

A threshold rectangular wave shock applied to either the base or the apex of the left ventricle in situ was shown to produce an extrasystole and a delay in repolarization of an area around the stimulating electrode of about 2 to 3 cm. in diameter, which endured for four or five succeeding beats.  

**Sumario Español**

Un estímulo de umbral aplicado al ápice o a la base del ventrículo canino intacto a cualquiera profundidad del miocardio produce una demora en la repolarización durante las próximas tres o cuatro pulsaciones normales, en un área equivalente al tamaño de la zona próxima al terminal de la derivación precordial. Este cambio en repolarización no se debe a distensión postextrasistólica del ventrículo. El problema de que un estímulo de umbral inicia un disturbio propagado desde el punto del electrodo, o desde el área mayor cuya repolarización es alterada se discute.

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