Electrical Effects of Stimulating the Endocardial Surface of the Right Ventricle of the Dog

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The endocardial surface of the right ventricle in four dogs was stimulated by a unipolar electrode introduced into the cavity of the right ventricle by means of a catheter. Electrical changes at a suprajacent point on the right wall were recorded by the V lead method of Wilson. In every experiment the induced QRS complexes were initiated by a positive electrical change.

There is considerable difference of opinion regarding the polarity of the initial electrical change recorded from a point on the thoracic wall when the endocardial surface of the subjacent ventricular wall is stimulated. Lewis and Rothschild reported that for the free wall of the right ventricle the initial change in potential, relative to a point transversely across the thorax is positive, whereas an initially negative deflection appears when the epicardial surface is stimulated. On the other hand, Nahum and Hoff report that both endocardial and epicardial stimulation produce the same effect, namely, an initially negative deflection at a suprajacent point on the thoracic wall. Of course the thorax, previously opened, had been closed, and the lungs inflated, when the stimuli were applied. Hoff's view is that the endocardial response may contribute to electrical changes, but that the sign of the electrical effect is the same, whether endocardium or epicardium is first stimulated.

In a previous paper, reasons were given for doubting the adequacy of the method employed. The results reported might have been due to an air pocket between the external pair of stimulating electrodes and the inflated lungs, to the use of too strong a stimulus, or to refractoriness of the endocardial surface at the moment the stimulus was applied in the rapidly beating heart. The usual form of the T wave, at least in the human electrocardiogram, suggests slower electrical recovery of the endocardial than of the epicardial muscle surface, and this indicates that the inner surface has a longer refractory period.

For these reasons, we decided to stimulate the endocardial surface of the right ventricle by means of a wire passed through a catheter, the end of which, after the catheter was passed through the jugular vein, was in contact with the inner surface of the free wall of the right ventricle. The tip of the wire was rounded to prevent injury by attaching a small globule of soldering metal to it. The stimulus was unipolar, being delivered from the secondary of an inductorium. The indifferent electrode was a metal plate placed at a distance on the dog's body. The stimulus strength was adjusted so that responses were obtained on the break, but not on the make. This precluded too strong stimulation. Before stimulating, the right vagus was faradized, this being continued for several seconds while the stimuli were being applied to the ventricle. Arresting the heart by vagus stimulation accomplished two things: it assured complete recovery of the muscle from refractoriness before the shock was applied, and it permitted the tip of the catheter and wire to come to rest in contact with ventricular wall. Fluoroscopic examination was used to place the catheter, and a roentgenogram was taken to show the positions of stimulating and recording electrodes. Furthermore, the location of the tip of the catheter was confirmed at autopsy. No effort was made to stimulate the epicardial surface, since all observers agree that with such stimulation, the initial electrical
change is negative. For recording, a string galvanometer was connected to the precordial electrode and Wilson’s central terminal. The precordial electrode was placed on the right precordium suprajacent to the part of the right wall which was stimulated. Positivity at the electrode yielded an upright deflection. Four dogs were used.

**Fig. 1.** V lead from right precordium. The first three complexes are the regular sinus rhythm. The vagus is stimulated with cardiac arrest. A break shock is applied, at the time of the white dot to the left of the S wave of the fourth QRS complex. This is followed by the upward portion of the diphasic artefact due to the shock. The R wave, with a white dot at its apex, follows. At the position of the next dot, the diphasic artefact due to the subthreshold make shock is seen, and this is followed by two other break shocks, with response, and an intervening make, without response. Time, 0.04 and 0.20 second.

**Fig. 2.** V lead from right precordium. More rapid film, taken during vagus stimulation. Time intervals, 0.10 sec. At left, the diphasic artefact due to an ineffective break shock. Just left of center, make shock artefact. This is followed by a break and the RS form of QRS complex. Two premature beats, probably mechanically induced, follow the QRS complex being of RS form in each. The R wave of the second premature beat is reduced in apparent amplitude because it occurs on the rapidly descending limb of a T wave.

**Results**

The results were the same in all four of the animals. Among hundreds of responses, none began with a negative deflection. A relatively narrow R wave (positive) appears immediately after the artefact signalling stimulation by the break shock, and this is followed by a much wider S wave (negative). This is the same as the result reported with endocardial stimulation by Lewis and Rothschild, except the arrangement of the electrodes in their experiments caused initial positivity to produce a downward deflection. One of their records is shown in Lewis’ book. Samples of results from two dogs are shown in figures 1 and 2, and are described in the legends. In two of the four dogs, ectopic beats appeared, probably as a result of mechanical stimulation. Although the locus of stimulation cannot be determined with certainty, it was presumably the same as the locus of electrical stimulation. Associated with these responses there is, of course, no electrical signal of stimulation. Their form is identical.

**Discussion**

These results are in agreement with those to be expected on the basis both of electrical theory and of findings with ventricular strips or whole turtle ventricles immersed in a volume conductor. When the inner surface of the right ventricle is activated, the wave of excitation begins to spread outward through the wall. A positive field is in advance of the wave, whereas the ventricular cavity would be rendered negative. This wave, approaching the precordial electrode, makes it positive, relative to the
central terminal. As soon as the entire thickness of the right wall is activated over an area of critical size, the wave of excitation, now retreating from the electrode through the septum and left ventricle, renders the electrode negative and an S wave is produced. Since the right wall is thin, the R wave is narrow in comparison with the S wave.

Conclusion

Contrary to another report, but in agreement with Lewis and Rothschild, it was found that electrical stimulation of the endocardial muscle surface of the free wall of the right ventricle produced initial positivity of a supraventricular galvanometer electrode in all trials in the hearts of four dogs.

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