Circus Movement in Canine Right Ventricle

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SUMMARY The present study reports on the epicardial spread of excitation during premature beats and during the initial stages of ventricular fibrillation, both of which were induced by single-test stimuli during regional ischemia or local hypothermia. Simultaneous recording of the activity at 48 epicardial sites on the right ventricle of dog hearts enabled us in some instances to demonstrate a circus movement. Circ Res 45: 374-378, 1979

VENTRICULAR arrhythmias in principle can be caused by automatic foci or by reentry. To prove reentry, the spread of activation during the arrhythmia must be known in detail. Thus, Allessie et al. (1973, 1976, 1977) were able to demonstrate a circus movement in rabbit atrial muscle by sequential mapping during a stable tachycardia. To analyze the activation pattern during nonperiodic arrhythmias, simultaneous registration of the electrical activity at many sites is required.

Methods

Experiments were performed on mongrel dogs, weighing 20-30 kg, which were anesthetized by intravenous injection of sodium pentobarbital (30 mg/kg). Under artificial ventilation, the chest was opened at the midline and the heart was supported by the opened pericardial cradle. The aortic pressure was recorded continuously.

Forty-eight bipolar electrodes (copper wire; diameter, 80 \mu m; interelectrode distance, 0.2-0.4 mm) were embedded in a quadrate (3 x 3 cm) high flexible network of silicone rubber (electrode distance, 4 mm). To record epicardial potentials, the network was placed on the right ventricle as shown in Figure 1. In those experiments with coronary occlusion the center of the network was placed above the ischemic border zone at the visible edge of the cyanotic area. For stimulation we used a central bipolar tungsten electrode (interelectrode distance, 1 mm) which was inserted 2 mm into the wall. Stimulation was performed by applying rectangular pulses 2-4 times threshold and 1 msec in duration. A pericardial ECG served as time reference. After amplification (bandwidth of the amplifiers, 10 Hz to 10 kHz), the signals were multiplexed and then stored on a 14-channel analog tape recorder. Depending on the configuration of the electrogram complex, the maximum of the slope or the peak value of the signal was taken as the moment of activation. Thus in almost all cases it was possible to determine this moment with an accuracy better than 5 msec. A single premature stimulus (intensity of 2-4 times diastolic threshold) was applied after every 10th basic stimulus to induce extrasystoles or ventricular fibrillation. To facilitate the initiation of these arrhythmias, regional ischemia was produced by occlusion of the left anterior descending coronary artery, or local hypothermia was induced by applying 0.9% saline at 0°C by drops (30 drops/min) onto the epicardial surface close to the center, and removing the liquid by suction.

Results

Spread of Excitation during Induced Premature Beats (Five Experiments)

Regional Ischemia

Figure 2 shows the patterns of activation of basic beats and of induced and nonstimulated premature beats 5 minutes after coronary artery occlusion. The grid of electrodes was on the right ventricle, at the border between cyanotic and normal myocardium. Basic stimuli (interval, 240 msec) were applied at the center of the grid; after every 10th basic stimulus (SA), a test stimulus (SB) [given at the end of the refractory period (162 msec) and with an intensity of 0.44 mA = 4 times diastolic threshold] induced a premature beat (B) and a nonstimulated premature beat (C). The spread of activity during the basic beat (A) is centrifugal, with slightly varying conduction velocities in different directions; maximal conduction velocity is 0.47, minimal is 0.36 m/sec. Conduction velocity was determined as the quotient of the distance and the mean conduction time between site of stimulation and the corners of the grid. The spread of excitation during the induced premature beat (B) is similar to that of the basic beat, except that maximal and minimal conduction velocities are reduced to 0.27 and 0.18 m/sec, respectively. During the nonstimulated premature response (C), the pattern of excitation is totally different: the area under the electrode grid is excited almost simultaneously (between 263 and 268 msec after the test stimulus). Although there are two separate epicardial sites that are activated...
earlier, it is clear that these sites do not serve as sources from which activity spreads radially over the epicardial surface.

Local Hypothermia (Five Experiments)

Figure 3 shows similar excitation patterns for basic beats (interval, 160 msec) and stimulated and nonstimulated premature beats during local hypothermia, 10 minutes after the application of cold drops. The test stimulus in this case had an intensity 2 times diastolic threshold, and its preceding interval was 115 msec (= refractory period).

In one heart (basic stimulus interval, 170 msec; extrastimulus interval, 130 msec; intensity, 4 times threshold) a nonstimulated premature beat could be elicited in control conditions. As shown in Figure 4, the activation pattern of the nonstimulated beat was similar to those shown in Figures 2 and 3.

Spread of Activation during the Initial Phase of Ventricular Fibrillation (Eight Experiments)

Ventricular fibrillation could be induced only by a single test pulse of 2-4 times diastolic threshold strength during regional ischemia or local hypothermia. As shown in Figure 5, the pattern of excitation during the first five nonstimulated responses is similar to that shown for single nonstimulated premature beats in Figures 2-4. However, during the sixth premature impulse (panel H), local conduction block was observed in the upper left quadrant, and the excitation wave turns around that area of block and reexcites the zone proximal to it (see panel I). During beat I, the zone of block shifts toward the middle, and again a clockwise circus movement is observed. Throughout beats H, I, and J, continuous activity is recorded by the electrodes, and two complete circus movements did occur within the area under the electrode grid.

In the experiment of Figure 6, the premature beat elicited by the test pulse already shows unidirectional block (panel B). Activity spreads around the blocked area in a counterclockwise manner and excites the blocked area 95 msec after the stimulus (panel C). This counterclockwise circus movement continues in the next beats (D and E), the site of block gradually shifting to the right.

Discussion

Interpretation of Epicardial Patterns of Excitation

Spread of excitation in ventricular muscle is a three-dimensional process and, therefore, mapping of epicardial excitation has serious limitations. To minimize these limitations, we recorded from the thin-walled right ventricle rather than from the...
thick left ventricle. Nevertheless, in many instances we could not rule out that activity did originate in some deeper layers.

In all experiments we found that, following stimulation via the central electrode, activity spread radially over the epicardial surface, except in the case shown in Figure 6, where the premature response showed unidirectional block. The fact that conduction velocity was not the same in all directions most probably is related to differences in fiber direction, conduction velocity being higher along the longitudinal axis of the fibers rather than in a direction perpendicular to it (Clerc, 1976). The slight decrease in conduction velocity after a premature stimulus is due to propagation in the relative refractory period, and our findings confirm those of others (van Dam et al., 1963). The pattern of excitation of the nonstimulated premature responses (Figs. 2-5) shows that the site of origin of these impulses was not in the tissue under the electrodes. The fact that the whole area was excited almost simultaneously suggests that broad wavefronts traveled from endo- to epicardium, possibly in a tangential fashion. A role of the subendocardial Purkinje fibers in the origin of these nonstimulated premature impulses therefore cannot be excluded. They could be involved in a reentrant circuit, but we cannot rule out the possibility of triggered automaticity (Cranefield, 1975). Our experiments do show, however, that reentry does occur in the initial phases of ventricular fibrillation. Activation turning around an area of block and reexciting the tissue proximal to the block was seen repeatedly. The site of block in successive activations shifted, and therefore it is quite possible that reentry did occur also, elsewhere, during those impulses for which no circus movement under the electrode grid could be demonstrated. In one instance (Fig. 6), reentry could be demonstrated for the premature beat elicited by the test stimulus itself, and in this case we may be certain that triggered automaticity is ruled out.

The dimensions of the circus movements were in our experiments in the order of 8–30 mm (Figs. 5I and 6B), which partially agrees with the dimensions found by Allessie et al. (1976) in rabbit atrial muscle. The time needed to complete a circus movement was in the order of 90–100 msec (Fig. 6), sometimes even 50–75 msec (Fig. 5), indicating that refractory periods proximal to the site of unidirectional block may be as short as 50–75 msec. These are short values for ventricular muscle, although they are in the same range as those found in rabbit atrial muscle (Allessie et al., 1976).

The distribution of temperature gradients during local hypothermia in the ventricular wall and its influence on refractory period duration need be investigated. Further measurements with a more extensive spatial resolution are necessary to gain a more complete insight in the spread of activation during the arrhythmias described, but it can be concluded from our experiments that reentry can
account for ventricular fibrillation induced by single premature stimuli during hypothermia.

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Characterization of the Extravascular Component of Coronary Resistance by Instantaneous Pressure-Flow Relationships in the Dog

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SUMMARY To investigate the mechanical effects of the myocardium on the blood perfusion of the canine left ventricle, phasic total left ventricular (LV) coronary blood flow, perfusion pressure, LV pressure, aortic flow rate, and LV segment length were recorded continuously in an open-chested dog heart preparation. These variables were analyzed on a digital computer and time synchronized so that coronary pressure-flow curves could be drawn for various instants in the cardiac cycle. During diastole, the pressure-flow relationship is linear, changing to a nonlinear curve with the onset of systole. To estimate phasic patterns of coronary resistance and intramyocardial pressure (IMP), a model based on the vascular waterfall mechanism was developed and fitted to the experimental data. The results of this operation show inferred coronary resistance patterns that increase during ejection and remain constant during diastole and isovolumic contraction. Assuming LV pressure to represent endocardial IMP, the estimated epicardial IMP signal averages 42.1 ± 13.3% of peak LV pressure at this instant of peak pressure. Furthermore, increases in end-diastolic volume reduced the changes in inferred coronary resistance taking place during ejection, but the epicardial IMP signal remained practically unchanged.

The extravascular component of coronary resistance represents all the mechanical and hemodynamic effects imposed on the coronary circulation by the surrounding myocardial tissue (Snyder et al., 1975). Together with aortic pressure and autoregulation, the extravascular component is an important determinant of coronary blood flow, influencing not only its total value (Sabiston and Gregg, 1957), but also its regional distribution across the left ventricular wall (Moir, 1972). Intramyocardial compression in systole is thought to be greatest in the subendocardium and to decrease in more superficial muscle. Accordingly, during systole, flow is greatest in the subepicardial region and decreases toward the endocardium (Downey and Kirk, 1974; Hess and Bache, 1976), thus suggesting that most of the coronary blood flow to the subendocardial region occurs during diastole (Moir, 1972; Hoffman and Buckberg, 1976).

Under normal physiological conditions, autoregulation always is capable of adjusting coronary blood flow according to the metabolic needs of the myocardium (Mosher et al., 1964). However, in situations of limited supply as in coronary artery disease, this autoregulatory mechanism will become exhausted (Hoffman and Buckberg, 1976), making coronary supply dependent only on aortic pressure and the extravascular component. Therefore, quantification of the extravascular component of coronary resistance and its dependence on hemodynamic conditions has considerable importance for the clinical management of the ischemic heart.

Since different interventions such as changes in preload or contractile state may have distinct effects on diastole and systole, the present work attempts to characterize the extravascular component at each instant of time throughout the cardiac cycle.

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