Vulnerability of the Dog Ventricle and Effects of Defibrillation

By BRIAN F. HOFFMAN, E. E. SUCKLING AND CHANDLER McC. BROOKS

Vulnerability to ventricular fibrillation has been studied in 20 dogs by means of test stimuli of known strength and duration in order to ascertain the relationship between the vulnerable period and the recovery of excitability and to determine the effects of countershock defibrillation. The relative refractory period is found to be the only portion of the cycle during which fibrillation could be produced by this technique. Defibrillation was accomplished without producing significant changes in vulnerability or excitability.

The existence of a "vulnerable period" in which the dog ventricle is susceptible to fibrillation by means of a single test stimulus has been known for many years. Recent studies of the time course of excitability changes throughout the entire cardiac cycle have demonstrated that in the auricle of the dog and in the ventricle of the dog, cat and turtle this vulnerable period is associated with dips or irregularities in the strength-interval curve. Detailed studies of ventricular vulnerability have been made for cat and turtle hearts; here, as in the dog auricle, discrete, constant thresholds for multiple extrasystoles and for true ventricular fibrillation are obtained during the relative refractory period. Throughout the remainder of the cardiac cycle test shocks of any strength elicit only single extrasystoles.

The present report is an extension of this work to the dog ventricle. It is concerned both with a description of vulnerability as well as with the effects of countershock defibrillation on the ventricular excitability cycle.

METHODS

Mongrel dogs were anesthetized with pentobarbital sodium, 30 mg./Kg., and maintained on artificial respiration. The chest was opened by a sternum-splitting incision and silver electrodes embedded in small lucite plaques were attached to the right auricle and right ventricle. After the S-A node had been crushed, the chest was closed and the heart driven by means of auricular electrodes at a constant rate. Ventricular excitability was determined by means of rectangular test stimuli of known duration and strength applied at any desired interval of the cardiac cycle. Stimulus strength in milliamperes was measured by means of a calibrated oscilloscope. The ventricular response was recorded with a 12" oscilloscope from contiguous punctate electrodes attached to the right ventricle. The details of the testing technique have been described previously.

Thresholds for multiple extrasystoles or for fibrillation were determined by progressively increasing the strength of the test stimulus above the value adequate for eliciting a single premature response until multiple extrasystoles or true fibrillation resulted. Since it has been demonstrated that at any interval during the vulnerable period the threshold for true fibrillation is only slightly above that for multiple extrasystoles and since it was desirable to avoid too frequent fibrillation and defibrillation because concurrent studies imposed a time limitation, an attempt was made to produce only the latter. The term "fibrillation threshold" thus refers to the stimulus strength which resulted in either multiple extrasystoles or ventricular fibrillation.

Vulnerability of the Dog Ventricle

In twenty satisfactory experiments on as many dogs it was possible to delineate the boundaries of the vulnerable period within the cardiac cycle and to plot the strength of stimulus required to elicit either multiple extrasystoles or fibrillation at any interval between these boundaries. As in the case of the cat and turtle ventricle, vulnerability to single supra-threshold test shocks was found only during the relative refractory period. Following the restoration of full excitability, a single extrasystole...
tole was the sole response which resulted from stimuli even of maximal strength and duration (fig. 1a). This was true, too, in those instances in which a phase of supernormality followed the completion of the total refractory period.

In most ventricles, a plot of the fibrillation thresholds within the vulnerable period inscribed a V-shaped pattern overlying the major dip of the strength-interval curve (fig. 1a). A progressive increase in stimulus strength at any interval in this vulnerable period produced first a single extrasystole, then multiple extrasystoles, and finally ventricular fibrillation. As seen in Table 1, the relationship to the major dip varied only slightly from dog to dog. In some instances, greatest susceptibility to fibrillation was found at the same interval as the lowest point of the dip; in other animals the interval of maximum vulnerability preceded or followed this low point in the dip by 5 to 15 msec. In the occasional experiment in which an obvious dip in the strength interval curve was not demonstrated, the vulnerable period maintained a configuration and relative position comparable to that found in the presence of a typical course of recovery (fig. 1b).

Fibrillation thresholds, including the minimum values obtained at the apex of the vulnerable period, varied little over prolonged periods of time.

It is apparent from Figure 2a that the dimensions of the vulnerable period depended in part on the duration of the test stimulus. Long duration shocks elicited multiple responses from a wider area of the relative refractory period at lower strengths than did short pulses. In addition, when 10 or 12 msec. duration stimuli were employed, it was sometimes possible to demonstrate two peaks of vulnerability—one during the major dip area and another,

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**Fig. 1.** a. Strength-interval curve depicting the time course of the recovery of excitability of the dog ventricle (---) and the boundaries of the vulnerable period (O—O). Note relationship of vulnerability to the dip of the strength-interval curve. Ordinate—stimulus strength in ma.; abscissa—interval of the cardiac cycle in m sec. b. Strength-interval curve as in a. Note absence of dip and position of the vulnerable period.
TABLE I.—The Table Depicts the Temporal Relationship Between the Vulnerable Period and Several Aspects of the Strength-Interval Curve in Twelve Experiments

<table>
<thead>
<tr>
<th>Exp.</th>
<th>Cycle Length msec</th>
<th>Interval of Cycle</th>
<th>Position in Cycle of Lowest Threshold</th>
<th>Total Refractory Period—Position in Cycle</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Dip</td>
<td>Vulnerability*</td>
<td>Dip</td>
</tr>
<tr>
<td>6/24/53</td>
<td>250</td>
<td>115-140</td>
<td>115-125</td>
<td>125</td>
</tr>
<tr>
<td>12/22/53</td>
<td>250</td>
<td>110-130</td>
<td>120-125</td>
<td>135</td>
</tr>
<tr>
<td>7/8/53</td>
<td>300</td>
<td>100-115</td>
<td>110-115</td>
<td>125</td>
</tr>
<tr>
<td>7/8/52</td>
<td>250</td>
<td>90-105</td>
<td>85-95</td>
<td>100</td>
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<td>125-150</td>
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<td>10/13/53</td>
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<td>300</td>
<td>110-150</td>
<td>115-115</td>
<td>120</td>
</tr>
</tbody>
</table>

* In all cases the range of vulnerability is that determined with a short-duration stimulus (1-3 msec).

Table depicts the temporal relationship between the vulnerable strength-interval curve and several aspects of the cycle length and position in the cycle of lowest threshold. The table includes data for twelve experiments, each with a different cycle length and interval of vulnerability. The position in the cycle of lowest threshold and the total refractory period—position in cycle are also listed.

As observed occasionally in the auricle of the dog, and in the ventricle of the cat and turtle, suprathreshold stimuli which failed to elicit any response became effective in producing single or multiple extrasystoles when stimulus strength was increased or decreased by a fraction of a milliampere. This "no response" phenomenon has been discussed previously.

DEFRIBRILLATION TECHNIQUE

The technique employed in defibrillation was as follows: Immediately after fibrillation had been

FIG. 2 a. Strength-interval curve (— — -) and boundaries of the vulnerable period as determined by 3 msec. (O—O) and 10 msec. (X—X) stimuli. b. Strength-interval curve and vulnerable period prior to (solid lines) and immediately following (dotted lines) fibrillation and defibrillation.
recognized on the monitoring oscilloscope, all electrode connections to the dog were open-circuited, approximated margins of the sternum were parted and two large, moist, padded electrodes were firmly applied to opposite sides of the ventricles. A burst of 60 cycle current was given from a defibrillator with an "on" cycle of 0.4 to 0.6 second and an "off" cycle of 0.5 or 1.0 seconds. During the "off" cycle the electrogram obtained on the oscilloscope and this permitted recognition of defibrillation if accomplished by the first pulse. If fibrillation persisted a second countershock was given. Two to three amps at 160 to 180 volts were usually used although less current would probably have been effective in most instances. In each instance defibrillation was accomplished within 30 to 45 seconds by one, or very rarely, two countershocks. Because the period of interrupted circulation was brief, no cardiac massage was required. Since the experiments performed did not permit a prolonged rest period following defibrillation to allow excitability to return to normal it was necessary to study the effects of countershock immediately after defibrillation and at intervals ranging up to 10 minutes.

**Effects of Defibrillation**

In over 50 per cent of tests, the procedure employed to accomplish defibrillation produced no significant change in either the strength-
interval curve or the position or magnitude of vulnerable period (fig. 2b). Furthermore, as seen in Table 3, in many cases even repeated fibrillation and defibrillation failed to alter either minimum diastole thresholds, minimum fibrillation thresholds, the duration of the absolute, total and relative refractory periods, or the mean blood pressure by more than the normal variation between tests. When an immediate change in excitability did result from defibrillation, it was usually apparent only in the minimum diastolic and minimum fibrillation threshold (table 3). Both increases and decreases in thresholds were observed. Such effects were prolonged, persisting after 30 to 60 minutes or more. The observation that these thresholds were changed when refractory periods were unaltered suggests that displacement of the test electrodes rather than a true alteration in tissue excitability accounts for the effects of countershock.

SUMMARY
A study of ventricular fibrillation in the dog showed that the relative refractory period was the only portion of the cardiac cycle during which fibrillation could be produced by single test stimuli. The period of vulnerability usually coincided with the major dip of the strength-interval curve. Defibrillation by means of an effective countershock technique produced no significant changes in either excitability or vulnerability.

REFERENCES
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