LIDOCaine in Cardiac Resuscitation from Ventricular Fibrillation

By Norman L. Carden, B.S. and John E. Steinhaus, M.D.

The interruption of ventricular fibrillation following coronary occlusion in dogs was effected with the use of lidocaine. This technic caused no obvious sequelae and the recurrence of fibrillation was rare. It was used successfully in both normothermic and hypothermic animals although the proportion of successful resuscitations was less in the latter group.

INTERRUPTION of ventricular fibrillation with the use of drugs has generally been found to be unsuccessful. Previous work on local anesthetic agents suggested that such compounds could terminate ventricular fibrillation; however, Stearns, Maisin and Stutzman reported little success with the use of procaine. Wegria and co-workers conclude in their studies that procaine may stop fibrillation; however, the rhythmicity of the heart is depressed to such a degree that the subsequent massage results in a recurrence of fibrillation. It was a purpose of this study to develop a method of cardiac resuscitation from ventricular fibrillation with the use of drugs.

Preliminary studies in our laboratory with procaine, procaine amide, and dibucaine were not consistently successful, whereas, studies with lidocaine (α-Diethylamino-2,6-acetoxylidide) were more promising and led to further investigation. Since Covino, Wright, and Charleson found that some drugs, such as procaine amide, actually induce or lower the threshold for ventricular fibrillation during hypothermia, a second series of dogs was subjected to hypothermia before the fibrillation was produced and resuscitation instituted.

METHODS

Dogs were anesthetized with pentobarbital, 30 mg./Kg. administered intravenously, and the chests were opened with the resection of the fourth rib. Artificial respiration was instituted, using a pneumatic balance respirator, and the circumflex branch of the left coronary artery was isolated. Ventricular fibrillation was induced by a temporary occlusion of the circumflex artery and electrocardiogram and blood pressure were recorded with the use of a strain gage and a twin-channel electrocardiograph. Within 1 to 2 min. after the onset of fibrillation cardiac massage was instituted. When adequate circulation was achieved, 15 mg./Kg. of lidocaine was injected directly into the left ventricle and the heart massaged for 30 to 60 sec. with the aorta occluded to insure maximal distribution of the drug to the coronary arteries. The aorta was then released and massage was continued until an organized ventricular beat was initiated. At that time, levarterenol 0.004 mg./Kg. was injected into the left ventricle to stimulate cardiac contraction and elevate blood pressure. In addition, a solution of this drug containing 0.004 mg./ml. was administered by intravenous infusion as required to maintain normal levels of blood pressure. Dextran or 5 per cent dextrose was also given to compensate for blood loss incurred during the surgical procedure.

In the first series, resuscitation was considered successful when fibrillation was stopped and an effective, regular cardiac contraction with an adequate blood pressure was maintained for a minimum of 1 hour. The recovery of dogs resuscitated in this manner was determined in a second series of animals. After resuscitation by the above-described technic the dog's chest was closed and thoracic suction and artificial respiration were maintained until the animal regained consciousness. Recovery in this series was considered successful when the dog lived a minimum of 24 hours.

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rewarmed by immersion in a 45 C. water bath and recovery was considered successful when they lived a minimum of 24 hours.

RESULTS

In a group of 23 dogs, 21 or 91 per cent were successfully resuscitated using the lidocaine technic. Figure 1 reveals the electrocardiographic (lead II) and blood pressure tracings from a typical resuscitation experiment. The first tracing, recorded 2 min. after lidocaine was injected, shows the development of a rapid ventricular rhythm although there was no effective myocardial contraction. In the second tracing the first evidence of effective myocardial contraction is noted, 14 min. after the onset of ventricular fibrillation. The time required for the onset of organized contraction in this series varied from 2 to 27 min. after the onset of the arrhythmia, with a mean of 7 min. The third tracing of this figure demonstrates the effect of levarterenol. A systolic blood pressure of 80 mm. Hg was recorded 1 min. following the injection of levarterenol. Further improvements in myocardial function occurred rapidly and at the end of 1 hour an adequate blood pressure and a more normal electrocardiogram were present as illustrated in the final tracing. Occasionally the electrocardiogram, which was recorded following the injection of lidocaine, was more irregular than the one illustrated (fig. 1). This rhythm usually reverted abruptly to a ventricular rhythm resembling that shown in the second or third tracing. Another variation, noted in one instance, was a short period of asystole followed by an abrupt return to effective myocardial contraction which did not require the use of levarterenol for the maintenance of effective blood pressure.

In order to determine whether complete recovery could be obtained with lidocaine resuscitation, the chests were surgically closed in a group of 21 dogs. Recovery was based on a minimum survival time of 24 hours, although all except 4 of these dogs were observed for a longer period. The results in table 1 reveal a recovery rate of 76 per cent or 16 of the 21 dogs in this group of experiments. It should be noted that the hearts of all dogs were successfully defibrillated and the apparent causes of death observed or determined at postmortem were respiratory failure, cardiac hemorrhage, cardiac arrest and ventricular fibrillation as listed in table 1. Upon postmortem examination no conspicuous myocardial damage was observed.

The electrocardiographic and blood pressure patterns in lidocaine resuscitations of hypothermic animals were very similar to those obtained with normothermic dogs except that there was more variation. The dosage of lidocaine required was the same as previously used, 15 mg./Kg. Of a total of 46 hypothermic dogs, 38 were defibrillated using lidocaine, and an adequate heart beat and blood pressure were maintained for a minimum of 1 hour. Survival was determined for a group of 9 dogs following surgical closure of the chest and re-

FIG. 1. Electrocardiograms, lead II (upper) and blood pressure tracings (lower) following the administration of lidocaine (15 mg./Kg.) 2 min. after onset of ventricular fibrillation. A. 4 min.; B. organized contraction (14 min.); C. after 0.004 mg./Kg. levarterenol (18 min.); D. recovery (1 hr.).
TABLE 1.—Postoperative Recovery Following Resuscitation with Lidocaine

<table>
<thead>
<tr>
<th></th>
<th>No. of dogs</th>
<th>Defibrilated</th>
<th>Recovered</th>
<th>Died</th>
<th>Cause of death</th>
<th>Time (hrs.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normothermic</td>
<td>21</td>
<td>21</td>
<td>16</td>
<td>5</td>
<td>Respiratory failure</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Respiratory failure</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac hemorrhage</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cardiac arrest</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ventricular fibrillation</td>
<td>2</td>
</tr>
<tr>
<td>Hypothermic</td>
<td>9</td>
<td>9</td>
<td>5</td>
<td>4</td>
<td>Respiratory obstruction (excess mucous)</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Atelectasis (?)</td>
<td>5+</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Atelectasis (?)</td>
<td>10+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Aspirated vomitus</td>
<td>12+</td>
</tr>
</tbody>
</table>

Warming. In this group 5 of the dogs survived; the deaths occurred between 8 and 12 hours postoperatively. Postmortem findings revealed atelectasis to be a complicating factor in these animals. Further studies with hypothermic dogs suggested that primary cardiovascular failure may also be implicated in some of these deaths.

**DISCUSSION**

Ventricular fibrillation was produced in this study by acute coronary occlusion except in those cases where mechanical stimulation was sufficient. This technic is more traumatic than the electric shock method of producing ventricular fibrillation which has been commonly employed in defibrillation studies. The presence of surgical trauma and anoxia in the occlusion technic made it more analogous to the situation encountered clinically and increased the difficulty of the resuscitation procedure. The administration of lidocaine caused the ventricular fibrillation to change to a single or a multifocal ventricular ectopic rhythm within 1 to 2 min. although an organized myocardial contraction did not occur until some minutes later. This later event may occur abruptly following a short period of asystole or there may be a gradual slowing of the ventricular rate before the onset of an organized contraction as illustrated in figure 1. Procaine and procaine amide also produced similar changes; however, the results with these agents were less consistent. Distribution of lidocaine to the myocardium was enhanced by temporary occlusion of the aorta above the origin of the coronary arteries. This procedure was adopted to reduce as much as possible the depressant effects of lidocaine on the respiratory and vasomotor systems which occur with its distribution to the rest of the body. Effective cardiac massage was of great importance during the period of ineffective myocardial contraction. After the onset of an organized ventricular contraction and the injection of levarterenol, the heart was massaged synchronously with the spontaneous rhythm for 30-60 sec. until the strength of the myocardial contraction was adequate. The total period of massage ranged from 3½ to 34 min. with the mean being about 7 min. The stimulation of the heart and vasomotor system with levarterenol was usually required although in several animals the cardiovascular function recovered satisfactorily without the use of this agent. Levarterenol was chosen initially because it was anticipated that lidocaine and anoxia might combine to produce overwhelming vasomotor collapse. However, cardiac stimulation has appeared to be an important part of the action of this drug. Preliminary studies by the authors suggest that epinephrine is an equally effective cardiovascular stimulant and produces no greater tendency toward refibrillation than does levarterenol, although epinephrine appears to be a less satisfactory agent for maintenance after the acute period of resuscitation has passed. When levarterenol was given before the onset of organized ventricular contraction it served only to delay the initiation of effective cardiac function.

No conspicuous myocardial damage was noted in 76 dogs upon which the lidocaine procedure was employed. When it was necessary to continue cardiac massage for prolonged periods, up to 34 min., an area of moderate hyperemia, corresponding to the position of the fingers at the time of massage, was noted. No apparent cerebral damage was observed in any of the recovered animals. The only central
nervous system damage demonstrable was that shown in table 1 in which 2 normothermic dogs failed to respire in the immediate postoperative period. This may have been caused by anoxia resulting from a delay in the institution of cardiac massage, rather than being due to the depressant effect of lidocaine. In only one instance in which the lidocaine procedure was employed did ventricular fibrillation recur.

Lidocaine was somewhat less effective in resuscitating dogs subjected to hypothermia, although the interruption of ventricular fibrillation was accomplished in over 80 per cent of the 46 dogs in this series. Recovery from this procedure was also less certain; however, the 15 min. period of anoxia during the hypothermia may have been a factor.

Summary

The successful use of lidocaine in cardiac resuscitation from ventricular fibrillation was demonstrated in a group of 90 dogs. Ventricular fibrillation was produced by the temporary occlusion of the left circumflex coronary artery, following which lidocaine was injected into the left ventricle. Cardiac massage was maintained until adequate cardiovascular function returned. As soon as an organized myocardial contraction was noted, levarterenol was administered to stimulate cardiac contraction and to increase vasomotor tone. The technic was effective in both normothermic and hypothermic animals although the proportion of successful resuscitations was less in the latter.

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