Cardiac Resuscitation Following Experimental Arrest by Procaine and Ether

By Martin L. Ryan, A.M., Richard A. Salvador, A.M., and Chester W. White, Jr., M.D.

A method of producing experimental cardiac arrest is described, and the characteristics of arrest are reported. The efficacy of epinephrine, norepinephrine, phenylephrine and acetyl strophantidin is compared in the resuscitation of hearts stopped by this method.

Many instances of cardiac arrest during surgery have been reported.1-3 In this emergency cardiac function may frequently be restored by appropriate early intervention. Methods of resuscitation of the arrested or fibrillating human heart have been the subject of numerous reports and of several reviews.1,4-6 Although it has been generally recognized that manual compression of the heart and artificial respiration are important in maintaining oxygenation and circulation of the blood during arrest in addition to promoting the return of cardiac function,1,6 conclusions as to the clinical use of drugs to stimulate the heart have been variable.5-9 Experimental cardiac arrest has been produced by a variety of methods.10-13 In many of these studies, however, as in the reported clinical cases, the cardiac arrest has not been conclusively established. Resuscitative efforts have frequently been successful, but the value of restorative drugs has remained uncertain either because of inexact diagnosis of arrest or because of the lack of proper controls.

Eggleston and Hatcher14 reported in 1919 on the actions of intravenous procaine in unanesthetized cats, in which phenol was used as a local anesthetic for surgery. They noted the uniform occurrence of respiratory arrest and a profound blood pressure fall. These events sometimes took place simultaneously, but more frequently the respiratory arrest preceded failure of the circulation. They established cardiac arrest in some of their animals by direct visualization, and stated that an effective heart beat returned spontaneously in some cases. In resuscitation attempts they found artificial respiration and cardiac massage (applied through the chest wall) of little value, unless epinephrine or ouabain was also used to stimulate the heart. It should be noted that their local anesthetic, phenol, may produce depression of the circulation and respiration by itself.15

These findings differ from those of Isenberg16 who used amobarbital sodium before procaine in his animals. He reported that respiratory arrest consistently preceded an asphyxial depression of the circulation which could be reversed by artificial respiration alone. Hulpieu and Cole17 also reported that intravenous procaine produced respiratory failure before cardiac failure and that artificial respiration with oxygen or air greatly increased the amount of procaine required to produce the circulatory depression. They stated that the “heart stopped” in their experiments, but no criteria for the arrest were given. Schumacker18 found similarly that respiratory arrest always developed before circulatory stasis when procaine was given intravenously during ether anesthesia; he noted, when the chests of his animals were opened, however, that their hearts were beating weakly after apparent cardiovascular collapse. He found in addition that ether increased the toxicity of procaine in guinea pigs and stated that death was due to medullary paralysis. Other workers have reported that ether increases the toxicity of procaine19 and cocaine.20

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Intravenous procain, then, has severe respiratory and cardiovascular depressant effects, and its toxicity is apparently increased by ether anesthesia. It seemed reasonable that these agents might be used together to achieve a reproducible cardiac arrest, and it was hoped that the circumstances would be benign enough to allow resuscitation.

The purposes of this study were (1) to effect and characterize a reproducible cardiac arrest, and (2) to evaluate several cardiovascular stimulants in resuscitation of the arrested hearts.

Methods

Anesthesia was induced in unselected adult mongrel dogs using ether by a semiopen mask technique. It was maintained by auffed endotracheal tube attached to a Wolff bottle. Mean arterial pressure was recorded by a mercury manometer attached to a femoral artery. Standard lead II electrocardiograms were taken. When upper plane 3 surgical anesthesia with constant blood pressure had been maintained for a period of 5 to 10 minutes, attempts to produce arrest were begun.

Procaine hydrochloride was injected intravenously in successively increasing doses of 2, 4, 8, 16 and 32 mg per kilogram in 0.4 ml per kilogram. If the initial dose (2 or 4 mg per kilogram) failed to produce cardiac arrest, a 10- to 15-minute period was allowed for blood pressure and respiration to return to normal before giving the next larger dose. In subsequent injections at 10- to 15-minute intervals, the amount of procaine was doubled each time until cardiac arrest ensued.

The sequence of events following the arrest-producing dose began with respiratory arrest occurring 30 seconds to four minutes later. In the succeeding one-half to three minutes the blood pressure fell to less than 15 mm. Hg, and oscillations in the femoral cannula and mercury manometer disappeared. When this state had been maintained for two to three minutes, the heart was considered in "apparent arrest." Electrocardiograms were taken before and after each procaine injection, at the nadir of blood pressure and at intervals after "apparent arrest." In the first 48 experiments no arrests followed the 2 mg dose of procaine hydrochloride, and it was omitted in the subsequent work.

Immediately following "apparent arrest," the chest was opened in the fourth left intercostal space and the heart was observed directly. If it was quiescent, or if its movements were so feeble that no aortic pulse was palpable for a period of 30 seconds, arrest was considered complete. In spite of the absence of peripheral circulation, electrocardiographic complexes were still present in some instances. Direct visualization of the heart was, therefore, essential to establish the presence or absence of contractions. After arrest was evaluated by direct observation, resuscitative measures were instituted.

In six animals artificial respiration with oxygen for 20 minutes was the only treatment; in eight others an injection of epinephrine (USP) into the left ventricle was also given. In the remaining 91 animals artificial respiration, with oxygen, manual compression of the heart, and the injection of a cardiac stimulating drug or of 0.9 per cent sodium chloride solution, were used. The respirator was a closed circuit, push-pull type with carbon dioxide absorber and adjustment of inspiratory and expiratory pressure by water manometers; it was operated at 17 cycles per minute. In preparation for cardiac massage all obstructing mediastinal connections of the heart were severed, but the pericardium was left intact. The ventricles were then grasped with the fingers on the dorsal surface and the thumb on the ventral surface, and compressed rhythmically at a rate of 25 to 30 per minute.

The stimulants used were epinephrine hydrochloride (USP), 0.05 mg per kilogram; l-norepinephrine bitartrate, 0.02 mg per kilogram; phenylephrine hydrochloride, 0.2 mg per kilogram, and acetylstrophanthidin, 0.1 mg per kilogram. Sodium chloride (2 ml of 0.9 per cent) was the control solution. Intravenous, left atrial and left ventricular injections were tried with each agent, five to eight animals being used for each route and agent combination. The drugs were usually administered at the beginning of massage, except in a few experiments where they were given approximately one minute later. When cardiovascular function was restored, as indicated by maintenance of an adequate blood pressure, the chest was closed. Animals which failed to recover consciousness or which showed obvious deficiencies of behavior on the day following the experiment were called "resuscitations"; those which were apparently normal the next day were called "survivals." The term "revivals" will be used to include both "survivals" and "resuscitations."

Three, six and nine minutes following some of the procaine injections in 20 animals, blood samples were drawn. Analyses of the plasmas for procaine were carried out by the method of Brodie, Lief and Poë.31

In addition to the animals studied under ether, 10 were given procaine hydrochloride in parallel experiments during pentobarbital anesthesia. Total doses of procaine hydrochloride of 240 and 252 mg per kilogram produced arrest in two dogs, one of which was a survivor; in the second, ventricular fibrillation occurred when cardiac massage was begun. In the remaining eight, doses of 96 to 224 mg per kilogram produced ventricular fibrillation. It was apparent that pentobarbital anesthesia was unsatisfactory for this work.
Results

Cardiac standstill was produced by rapid intravenous injection of procaine hydrochloride during ether anesthesia in 106 dogs (table 1). The arrests fell readily into three categories according to the residual activity of the myocardium. In 53 animals no myocardial movements or electrocardiographic activity were detected. Twenty-four animals showed persistent electrocardiographic complexes in the absence of myocardial contractions indicating that the electrocardiogram was not by itself a satisfactory index of cardiac status. In 29 animals both electrocardiographic complexes and myocardial twitches, which were not fibrillatory in character, were seen following the two or three minutes of circulatory collapse. When such action persisted, however, the circulation was ineffective since no pulses were detectable. The probability of successful resuscitation was not influenced favorably by this type of action.

The heart of one animal stopped following the 4 mg. per kilogram dose of procaine hydrochloride, 27 stopped following the 8 mg. dose, 58 following the 16 mg. dose, and 20 following the 32 mg. dose. In no case was a dose larger than 32 mg. required (table 1). Ventricular fibrillation occurred after the injection of 32 mg. of procaine hydrochloride per kilogram in 3 of 23 animals. The cumulated 50 per cent arresting dose of procaine hydrochloride was 19.0 mg. per kilogram (confidence limits 17.0 to 21.3) as estimated by the method of Litchfield and Wilcoxon.22

Doses of procaine which were insufficient to produce cardiac arrest uniformly caused a significant fall in blood pressure (table 2), as has been reported by others.23 Recovery from this effect was apparently complete within the 10- to 15-minute interval between injections. The depressor response following each injection was significantly greater than that after the preceding one (table 2), except for the 16 mg. per kilogram dose. The maximal effect, then, was produced by a total dose of 12 to 14 mg. per kilogram, suggesting that doses in excess of this range are likely to arrest the heart (50 per cent arresting dose = 19.0 mg. per kilogram).

The analyses for procaine showed cumulation of this substance; it did not disappear from the plasma within nine minutes after the 4 and 8 mg. per kilogram doses (table 3). Since the effects of this residual procaine cannot be interpreted, results have been reported as “following” a given dose of procaine hydrochloride or on the basis of total dose.

Seventy-one of 91 animals in which manual compression of the heart was employed were “survivors” and an additional five were “resuscitations.” The various states of myocardial and electrocardiographic change which were present at the time of arrest did not appear to influence the effectiveness of the various resuscitative procedures (table 4). One animal was discarded because of mechanical failure of the respirator.

Among the six control animals which had artificial respiration only, and the eight which received intraventricular epinephrine also, one developed ventricular fibrillation and the rest showed no response whatever. The results for

### Table 1.—Cardiac Arrest Produced by Intravenous Injection of Procaine Hydrochloride during Ether Anesthesia

<table>
<thead>
<tr>
<th>Dose procaine HCl following which arrest occurred mg./Kg.</th>
<th>No. Dogs</th>
<th>Quality of Cardiac Arrest</th>
<th>Ventricular fibrillation no. dogs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Complete arrest (no. dogs)</td>
<td>Persisting ECG (no. dogs)</td>
</tr>
<tr>
<td>4</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>8</td>
<td>27</td>
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<tr>
<td>16</td>
<td>58</td>
<td>28</td>
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<td>32</td>
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<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Totals</td>
<td>109</td>
<td>53</td>
<td>24</td>
</tr>
</tbody>
</table>

### Table 2.—The Depressor Effect of Doses of Procaine Hydrochloride Not Producing Cardiac Arrest

<table>
<thead>
<tr>
<th>Dose procaine HCl mg./Kg.</th>
<th>No. dogs</th>
<th>Control blood pressure mm. Hg mean and S.D.</th>
<th>Nadir blood pressure mm. Hg mean and S.D.</th>
<th>Percent fall in blood pressure mean and S.D.</th>
<th>p of difference control cf. nadir</th>
<th>p of % fall cf. preceding dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>48</td>
<td>128 ± 10</td>
<td>114 ± 21</td>
<td>10.6 ± 9</td>
<td>&lt;0.01</td>
<td>—</td>
</tr>
<tr>
<td>4</td>
<td>97</td>
<td>132 ± 21</td>
<td>107 ± 24</td>
<td>13.5 ± 11</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>8</td>
<td>79</td>
<td>130 ± 21</td>
<td>92 ± 20</td>
<td>28.4 ± 9</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>16</td>
<td>17</td>
<td>134 ± 22</td>
<td>90 ± 22</td>
<td>23.6 ± 9</td>
<td>&lt;0.01</td>
<td>&gt;0.1</td>
</tr>
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</table>
TABLE 3.—Procaine Hydrochloride Concentrations in Plasma Following Intravenous Injection

<table>
<thead>
<tr>
<th>Dose procaine HCl mg./Kg.</th>
<th>Minutes after injection</th>
<th>No. dogs</th>
<th>Procaine HCl mg./L plasma, mean and S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>3</td>
<td>4</td>
<td>4.2 ± 1.2</td>
</tr>
<tr>
<td>6</td>
<td>4</td>
<td>4</td>
<td>3.2 ± 0.7</td>
</tr>
<tr>
<td>9</td>
<td>10</td>
<td></td>
<td>2.0 ± 0.5</td>
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<tr>
<td>8</td>
<td>3</td>
<td>8</td>
<td>7.1 ± 2.2</td>
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<tr>
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<td>6.8 ± 1.2</td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>10</td>
<td>4.4 ± 0.7</td>
</tr>
<tr>
<td>16</td>
<td>3</td>
<td>9</td>
<td>17.4 ± 5.2</td>
</tr>
<tr>
<td>32</td>
<td>3</td>
<td>3</td>
<td>19.4 ± 1.9</td>
</tr>
</tbody>
</table>

TABLE 4.—Influence of State of the Arrested Heart on Results of Resuscitation Experiments

<table>
<thead>
<tr>
<th>Quality of arrest</th>
<th>No. “Resuscitations”</th>
<th>No. “Survivals”</th>
<th>No. failures</th>
<th>No. fibrillations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete</td>
<td>2</td>
<td>38</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Persistent ECG</td>
<td>2</td>
<td>15</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>ECG and Twitches</td>
<td>1</td>
<td>18</td>
<td>0</td>
<td>3</td>
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<tr>
<td>Totals</td>
<td>5</td>
<td>71</td>
<td>3</td>
<td>12</td>
</tr>
</tbody>
</table>

The 91 dogs in which an injection of one of the resuscitating drugs was combined with the use of artificial respiration and manual compression are summarized in table 5. The average mean arterial pressures and mean massage times for each of the agents are detailed in table 6.

Eighteen of the 24 animals injected with isotonic salt solution were “survivals” while an additional one was a “resuscitation.” Ventricular fibrillation occurred during massage in four animals, and one failed to respond. The mean arterial pressure maintained by manual compression in the “revivals” was 35 (S.E. = 1.60) mm. Hg. The duration of massage before return of adequate cardiac function was 5.1 (S.E. = 1.44) minutes. (Massage time reached 27 minutes in one survival.)

Among the epinephrine-treated animals, 15 “survived,” one was “resuscitated,” and ventricular fibrillation developed in one. Cardiac massage produced a mean arterial pressure of 79 (S.E. = 6.2) mm. in the “revivals”; average massage duration was 0.8 (S.E. = 0.10) minutes.

Of 15 animals treated with norepinephrine, 12 “survived,” two more were “resuscitated,” and ventricular fibrillation supervened in one. The average mean arterial pressure of massage in the “revivals” was 64 (S.E. = 4.3) mm. while the time to resumption of effective heart action was 1.1 (S.E. = 0.21) minutes.

When phenylephrine was used as the stimulant drug, “survivals” were noted in 13 experiments, “resuscitation” in one, and ventricular fibrillation in three. Manual compression gave a mean arterial pressure of 56 (S.E. = 2.8) mm. Hg, and the average duration of manual compression was 1.3 (S.E. = 0.24) minutes.

The acetyl strophanthidin-treated group showed 13 “survivals,” three cases of ventricular fibrillations, and two completely unresponsive animals. The average mean arterial pressure during massage was 51 (S.E. = 3.2) mm. Hg for the “revivals,” and heart action was effective in an average of 2.6 (S.E. = 0.32) minutes after resuscitative measures were initiated.

Chi-square analyses revealed no over-all differences in resuscitation and survival rates regardless of the agent or route. Table 6, however, shows a number of significant differences among the agents in the blood pressure and in the duration of manual compression. In all, epinephrine appeared the most effective of the drugs in promoting a higher arterial pressure during massage and in hastening the early return of heart action, while sodium chloride solution was the poorest. Complete failure of response was noted only after saline and acetyl strophanthidin. This suggests that stimulation of the circulation by rapidly acting sympathomimetic amines may have some value in cardiac resuscitation.

Although it has been reported that procaine depresses cardiac irritability, administration of extremely large doses intravenously in this study produced ventricular fibrillation in 8 of 10 dogs during pentobarbital anesthesia. A myocardial depressant action of ether has
Table 5.—Results of Resuscitation and Survival Experiments

<table>
<thead>
<tr>
<th>Route</th>
<th>Range of total dose procaine hydrochloride causing arrest mg./Kg.</th>
<th>No. dogs tested</th>
<th>Complete arrests (ECG's)</th>
<th>Persistent ECG's and twitches</th>
<th>&quot;Survivals&quot;</th>
<th>&quot;Resuscitations&quot;</th>
<th>Fibrillations</th>
<th>No response</th>
<th>Revival attempts</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.9% NaCl Solution (2 ml.)</td>
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<td></td>
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<tr>
<td>Vein</td>
<td>4-60</td>
<td>8</td>
<td>3</td>
<td>5</td>
<td>0</td>
<td>5</td>
<td>1</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Auricle</td>
<td>12-60</td>
<td>8</td>
<td>4</td>
<td>2</td>
<td>2</td>
<td>5</td>
<td>0</td>
<td>2</td>
<td>1</td>
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<tr>
<td>Ventricle</td>
<td>12-28</td>
<td>8</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td>8</td>
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<td>Totals</td>
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<td>18</td>
<td>8</td>
<td>3</td>
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<td>1</td>
<td>4</td>
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<tr>
<td>Epinephrine HCl (0.05 mg./Kg.)</td>
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<tr>
<td>Vein</td>
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<td>4</td>
<td>1</td>
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<td>6</td>
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<tr>
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<td>l-Norepinephrine HCl (0.02 mg./Kg.)</td>
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<td>5</td>
<td>12</td>
<td>3</td>
</tr>
</tbody>
</table>

also been demonstrated; but ventricular fibrillation also followed directly on the injection of procaine in three ether-anesthetized animals (table 1). Furthermore, some of the hearts were sufficiently irritable during the resuscitation period so that fibrillation developed in 12 cases (table 5). By the time cardiac arrest was established in these studies, hypoxia of moderate to severe degree was present. The extent to which this may have
conditioned the fibrillatory response is uncertain.

**Discussion**

The cardiac status which was accepted as arrest in these experiments was variable. The 24 cases in which the animals showed persistent electrocardiographic complexes without any myocardial movement may properly be called complete arrest since the condition of the heart muscle was such that it was not responsive to impulses which in some instances produced fairly normal electrocardiograms. In the 29 experiments in which both electrocardiograms and minimal nonfibrillatory movements of small areas of the ventricle were seen, there were no indications of peripheral movement of blood in that there was no pulsation in major arteries, no movement in the glass arterial cannula, and no oscillation in the mercury manometer. It is apparent, also, that "revival" rates were not influenced by these differences in cardiac state (table 4). The occurrence of electrocardiographic complexes was not a reliable index of the presence of effective ventricular contractions. Further support for this thesis is provided by the 14 control studies (artificial respiration alone or combined with intraventricular epinephrine) in which none of the animals was resuscitated, although two showed persistent ventricular complexes in the electrocardiogram and two showed myocardial twitches in addition. Previous reports have described agonal cardiac contractions. Negovsky, who produced cardiovascular failure by hemorrhage, found them of no consequence; while Danielopolu and Marcou,\(^\text{19}\) who used overdoses of chloroform as an arresting agent, stated that such movements in the heart muscle facilitated resuscitation by epinephrine. In agreement with Negovsky, we found this myocardial activity of no moment.

Since these cardiac arrests were produced under uniform conditions, and since the arrested hearts all responded similarly to resuscitative measures, a comparison of the various drugs or of the routes of injection is felt to be valid. Massage of the heart and artificial respiration were essential to revival. Furthermore, no great superiority was demonstrated for any resuscitative drug or route of administration (table 5). Our impression was, nevertheless, that higher massage blood pressures after epinephrine, norepinephrine and phenylephrine (table 6) were reflected by an increase in the palpable firmness of the heart.

The frequent resumption of an effective heart beat with such an increase in cardiac "tone" is in agreement with the findings of Wégria

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**Table 6.—Mean Arterial Pressures and Periods of Massage with the Various Resuscitating Drugs, and Their Differences**

<table>
<thead>
<tr>
<th>Resuscitating Drug</th>
<th>Average Value ± S.E.</th>
<th>No. of dogs*</th>
<th>Differences/ (p) of differences</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Epinephrine</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>79 ± 6.18</td>
<td>15</td>
<td>15/\ (&gt;0.05)</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>64 ± 4.27</td>
<td>14</td>
<td>8/\ (&gt;0.1)</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>56 ± 2.76</td>
<td>13</td>
<td>5/\ (&gt;0.2)</td>
</tr>
<tr>
<td>Acetyl strophanthidin</td>
<td>51 ± 3.17</td>
<td>12</td>
<td>44/\ (&lt;0.01)</td>
</tr>
<tr>
<td>Isotonic saline</td>
<td>35 ± 1.60</td>
<td>19</td>
<td>5.1/\ (&lt;0.01)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Massage Times (Minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isotonic saline</td>
</tr>
<tr>
<td>Acetyl strophanthidin</td>
</tr>
<tr>
<td>Phenylephrine</td>
</tr>
</tbody>
</table>

* Three less animals appear here than in table 5 because of unreadable kymograph recordings.
and his coworkers. The observation that the strength of the returning beat may be suppressed by continuing massage confirms an earlier report by Gunn. When any myocardial activity was detected, it was necessary to stop the manual manipulation of the heart in order to estimate the time of return of spontaneous cardiac action.

Our study did not indicate any superiority of intracardiac as compared with intravenous injections; and the direct stimulation of a flagging heart by needle puncture or by high local concentration of drug may be undesirable. If cardiac stimulating drugs are used, it is perhaps better to choose the more conservative intravenous route.

The mechanism of cardiovascular collapse following ether and procaine is suggested in studies of Brewster and associates, who showed that functional intactness of the sympathoadrenal system is essential to circulatory adequacy during ether anesthesia. In their work blocking of this system or its surgical interruption revealed a direct cardiodepressant effect of ether. Procaine has been reported to interfere with the function of the cardiac nerves and the vagus and to block ganglionic transmission. The cardiac arrest here reported may have resulted from diminution or abolition of reflex control of the heart, or from unmasking the depressant action of ether by procaine blockade of the adrenal medulla, or from a combination of these two effects. Hypoxia and depression of cardiac irritability and/or contractility may also have contributed.

**CONCLUSIONS**

1. A method of producing cardiac arrest by procaine during ether anesthesia has been described, and its mechanism postulated.

2. The nature of arrest was variable; but the presence of agonal contractions of the myocardium or of electrocardiographic change did not improve the probability of resuscitation.

3. Ventricular complexes in the electrocardiogram were found to persist in the absence of observable myocardial contractions in approximately 25 per cent of the animals.

4. When manual compression of the heart was not used, no resuscitations were observed.

5. Although massage blood pressures were higher and massage times were shorter when
epinephrine, norepinephrine or phenylephrine were used in resuscitation than after acetyl-
strophanthidin or isotonic salt solution, no
differences in resuscitation or survival rates
were demonstrated for any agent or route of
administration.
6. An over-all resuscitation rate of 84 per
cent was achieved.

Conclusiones in Interlingua
1. Es describite un metodo pro producer
arresto cardiac per medio de procaina durante
anesthesia a ethere. Le mecanismo del pro-
cesso involvite es describite.
2. In le casos observate le arresto eseva
variable, sed le presentia de contractiones
agonal del myocardio o de cambiantemol
eletrocardiographic non meliorava le probabi-
litate de resuscitation.
3. In circa 25 pro cento del animales com-
plexos ventricular del electrocardiogramma per-
sisteva in le absenta de observabile contrac-
tiones myocardial.
4. Sin uso de compression manual del corde
nulle resuscitation eseva observate.
5. Ben que pressiones sanguinee a massage
eseva plus alte e tempore de massage plus
breve in casos ubi epinephrina, norepinephrina,
o phenylephrina eseva usate in le resuscitation
que in casos ubi acetylstrophanthidina o solu-
tiones isotonic de sal eseva usate, nulle dif-
ferentias del procentages de resuscitation o de
superviventia eseva associabile con un o
altere agente o con un o altere via de admin-
istration.
6. Essea attingite un total de 84 pro cento
de resuscitaciones.

Acknowledgments

Liberal amounts of L-norepinephrine (Levophed)
were supplied by Winthrop-Stearns, Inc.; acetyl-
strophanthidin was supplied by Eli Lilly and
Company.

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