A Study of the Usefulness and Limitations of Electrical Countershock, Cardiac Massage, Epinephrine and Procaine in Cardiac Resuscitation from Ventricular Fibrillation

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The efficacy of electrical countershock, cardiac massage, epinephrine and procaine in stopping ventricular fibrillation and restoring a competent ventricular contraction was studied in anesthetized dogs. It was found that countershock is a reliable means of stopping fibrillation. However, it must be preceded by cardiac massage if not applied promptly after the initiation of fibrillation. Epinephrine helps restore a competent ventricular contraction once fibrillation has been stopped by countershock, but it increases the incidence of recurrence of fibrillation. The doses of procaine which constitute a reliable means of stopping fibrillation depress the rhythmicity of the heart to such an extent that the cessation of fibrillation is followed by prolonged periods of cardiac standstill.

Ventricular fibrillation is a cardiac mechanism in which, because of the lack of coordination in the activity of the ventricular fibers, no blood is expelled from the heart and this results in generalized anoxia and death. Fibrillation can be caused by coronary occlusion, electrocution, various drugs or combination of drugs such as digitalis and digitalis-like drugs, chloroform, chloroform-adrenalin, benzol-adrenalin and cyclopropane-adrenalin. The fibrillation induced by digitalis and digitalis-like drugs is of a somewhat particular type however. Ventricular fibrillation can also be initiated by mechanical, chemical or thermal trauma to the heart. Isolated instances have been noted during various surgical procedures, especially those involving the thorax and the heart. It may also occur as an agonal event. Although in certain species of mammals, such as the cat, ventricular fibrillation may stop spontaneously and a normal cardiac mechanism be resumed, in dogs as well as humans the usual type of true fibrillation is essentially a cardiac mechanism which is not spontaneously reversible. It usually persists for as much as 30 minutes until terminal cardiac arrest ensues. It can be stopped, however, when it is not induced by an organic lesion of the myocardium such as myocardial infarction.* The present study was undertaken in an attempt to estimate the respective usefulness and limitations of several procedures and drugs which have been recommended to stop ventricular fibrillation and restore a competent ventricular contraction.

* For a review of the subject of ventricular fibrillation, see references 1 and 2.
Electrical countershock, cardiac massage, epinephrine and procaine were studied.

METHODS

Seventy-five dogs weighing between 6 and 31 Kg., 53 of which weighed between 9 and 15 Kg., were anesthetized by the intravenous administration of 25 mg. of sodium pentobarbital per kilogram of body weight. The chest was opened by a midsternal incision, and under artificial respiration the heart was suspended in a pericardial cradle. The mean arterial blood pressure was recorded optically from a cannulated carotid artery. Ventricular fibrillation was induced electrically with an alternating current stimulus of minimal intensity applied through two fishhook electrodes hooked into a gauze pledget soaked with isotonic sodium chloride solution and resting on the left ventricle. Electrocardiograms in the three standard limb leads were recorded at appropriate times.

Cardiac massage consisted of rhythmic compressions of both ventricles with one hand at a rate ranging from 40 to 60 per minute. The descending aorta was compressed and partially constricted during massage to force more blood into the coronary and cerebral circulations. There were marked variations in the arterial pressure reached during massage, the mean pressure ranging between 50 and 100 mm. of mercury.

The technic used to apply electrical countershock in order to stop ventricular fibrillation was essentially that used by Wiggers and Węgria.\(^1\)\(^2\)\(^3\) It consisted in enclosing the whole ventricular mass between two copper electrodes padded with cotton soaked with isotonic sodium chloride solution and sending through the heart short bursts, estimated to last about 0.1 second, of a 60 cycle per second alternating current of 110 volts, passing through a variable resistance. Sometimes one such shock was effective and sometimes a series of shocks had to be applied. During all the different procedures used for resuscitation, artificial respiration was maintained, whether cardiac massage was applied or not.

one-half minute after the induction of ventricular fibrillation, two countershocks stopped ventricular fibrillation and regular sinus rhythm was resumed. The arterial blood pressure rose progressively and reached a maximum of 165 mm. Hg, 2 minutes and 20 seconds after the initiation of fibrillation. In a part of the record not reproduced in figure 1, the blood pressure was seen to decrease progressively from its peak of 165 mm. Hg to values of 105, 98, 87 and 50 mm. Hg 15, 25, 45 and 60 minutes, respectively, after the initiation of fibrillation. In figure 2 are reproduced electrocardiographic tracings (lead II) recorded during the same experiment. Section A is the control electrocardiogram showing regular sinus rhythm. Section B was taken at the end of the period of ventricular fibrillation. The record of section C, taken one minute after the initiation of
fibrillation, that is, 22 seconds after fibrillation had been stopped, demonstrates that regular sinus rhythm had been resumed. The S-T segment of the electrocardiogram was markedly elevated. Section D, recorded seven minutes after the initiation of fibrillation, shows that the electrocardiogram had resumed its control form. Essentially similar observations were made in all five experiments of this type. Countershock was effective in stopping fibrillation in all five dogs. Immediately thereafter, the ventricles began to beat spontaneously. In three dogs regular sinus rhythm was resumed immediately; in the other two dogs, auricular fibrillation lasting 10 and 90 seconds, respectively, preceded the resumption of regular sinus rhythm. It is believed that these bouts of atrial fibrillation were induced by the electrical countershocks which stopped the fibrillation in the ventricles. In all five dogs, the blood pressure rose rapidly to or above control as soon as ventricular fibrillation was stopped. In three, the blood pressure remained above 100 mm. Hg for one hour; in the fourth dog the blood pressure was 80 mm. Hg during the control period, and was 80, 77, 68 and 70 mm. Hg, respectively, 5, 15, 40 and 60 minutes after the initiation of fibrillation. The fifth dog is the one described in figure 1. Electrocardiograms were recorded in four of the five experiments. In three of these there was marked displacement of the S-T segment immediately after the re-establishment of a coordinated ventricular beat. This displacement disappeared within three minutes.

B. Countershock Applied after Five Minutes of Fibrillation. In four dogs countershock was applied five minutes after the initiation of ventricular fibrillation. During the five minutes of fibrillation, the character of the fibrillation changed markedly. Direct observation of the ventricles revealed that the initial fine, rapid quivering became progressively coarser and slower. A similar evolution in the fibrillatory process can be seen on the electrocardiogram of such an experiment, reproduced in figure 3. A, the control electrocardiogram, shows the cardiac mechanism to be regular sinus rhythm. Between A and B, ventricular fibrillation was induced. Sections B, C and D, recorded after 20 seconds, 1½ minutes and 4½ minutes of fibrillation, respectively, illustrate that the rate of fibrillation slowed from about 450 per minute to approximately 300 per minute. Between D and E, electrical countershock was applied after five minutes of ventricular fibrillation. It was effective in stopping ventricular fibrillation. Section E, recorded 20 seconds after the counter-

![Fig. 2. Sections of electrocardiogram, lead II, recorded during the experiment of figure 1; A, during the control period; B, at the end of the period of fibrillation; C, one minute and D, seven minutes after the initiation of fibrillation.](image-url)
made: ventricular fibrillation was stopped by electrical countershock; sinus rhythm was resumed, first with ventricular standstill then with idioventricular rhythm. Such ventricular beats were ineffective in raising the arterial blood pressure. During the 10 to 20 minute period of observation which followed the cessation of ventricular fibrillation, the heart deteriorated further and the ventricular rate remained very low.

II. Cardiac Massage

As will be shown by the observations reported in section III, cardiac massage alone did not stop ventricular fibrillation. However, it prevented the progression of the fibrillatory process from the early fine, fast type to the late, slow, coarse type. Massage even caused the late type to revert to the early type at least within the limits of time which were studied.

III. Cardiac Massage and Electrical Countershock

A. Cardiac Massage–Electrical Countershock Repeatedly Applied after Five Minutes of Fibrillation. In a group of five dogs, ventricular fibrillation was induced and observed for five minutes. Phenomena similar to those described in section I, part B were observed. After this five-minute period of fibrillation, cardiac massage was instituted for 30 seconds. Massage was seen to change the fibrillation from the late type (slow and coarse) to the early type (rapid and fine). It was also noted that under the influence of massage the ventricles which first felt flabby to the massaging hand became rather suddenly much firmer. After the 30 seconds of massage, electrical countershock was applied and stopped ventricular fibrillation in all experiments. In this group also, the ventricular contractions were ineffective in raising the blood pressure. When it became apparent that the ventricular contraction was going to deteriorate further, massage was administered again although the danger of inducing ventricular arrhythmias by massaging ventricles contracting in a coordinated manner was fully realized. Indeed, massage resulted in ventricular fibrillation in all five dogs. This sequence of maneuvers (massage resulting in ventricular fibrillation followed by countershock stopping fibrillation) was repeated from two to five times in each of these five dogs. The maneuver, massage-countershock, was stopped when it was noted that the ventricles were beginning to beat more vigorously and the arterial blood pressure was rising. At this time the blood pressure rose above control level in four of the five dogs. In one of these four dogs it remained above 100 mm. Hg for one hour and in the other three it ranged between 60 and 80 mm. Hg. In the fifth dog, the blood pressure remained around 50 mm. Hg. In four of the five dogs electrocardiographic changes were slight, consisting of some S-T deviation, T-wave changes or both. These changes persisted throughout the period of observation but tended to decrease. In the
remaining dog, the changes in the S-T segment were marked, but tended to decrease also.

B. Countershock Applied after 9 to 18 Minutes of Fibrillation during which the Ventricles Were Massaged Intermittently. In five dogs countershock was applied after ventricular fibrillation had persisted for 9 to 18 minutes. During fibrillation, cardiac massage was administered continuously for 7 to 10 minutes except for interruptions of about 30 seconds every one to two minutes to allow the recording of electrocardiograms. After this period of 7 to 10 minutes, massage was discontinued for one and one-fourth to three minutes and in only one dog for four and three-fourths minutes, then the massage-countershock procedure was instituted. In all dogs, fibrillation was stopped by the countershock, and coordinated ventricular beats were restored. The arterial blood pressure rose rapidly within the control range of about 100 mm. Hg. In the dog in which fibrillation was allowed to persist for 18 minutes and no massage given for four and three-fourths minutes, the blood pressure did not rise above 75 mm. Hg; the control blood pressure of this dog was 90 mm. Hg. Figure 4 illustrates the electrocardiograms recorded during one of these experiments. Section A recorded during the control period shows regular sinus rhythm. Section B reveals that after seven minutes of fibrillation during which cardiac massage had been applied, fibrillation was still of the early type, but as seen in section C, it became slow two minutes after massage had been discontinued. Section D illustrates that fibrillation regained the characteristics of the early type of fibrillation after massage. Section E is the tracing after fibrillation had been stopped, regular sinus rhythm restored and the heart allowed to recover so that the electrocardiogram was similar to the control tracing. Electrocardiograms were recorded on four of the five dogs after ventricular fibrillation had been stopped. None was recorded on the dog in which no massage was given for the longest time. The electrocardiographic changes were essentially similar to those described in the group of experiments in which countershock was applied after 30 seconds of fibrillation (paragraph I, part A) and they evolved similarly.

IV. Epinephrine

The effect of epinephrine alone on ventricular fibrillation was studied in four dogs. Ventricular fibrillation was induced electrically as previously described and after a period of at least 30 seconds of fibrillation, 0.05 mg. of synthetic 1-epinephrine per kilogram of body weight was injected, half of the amount in each ventricular cavity. The total amount of the drug given was dissolved in 1 cc. of a 0.9 per cent sodium chloride solution. To be sure to submit the ventricles to the effect of the drug, after the injection the ventricles were massaged 10 times within about one-half minute, the descending aorta being partially occluded with the fingers. After this last maneuver, the effects of the drug were observed without any further interference. In none of the four experiments was it observed that epinephrine stopped fibrillation. However, the drug modified the fibrillatory process. Fibrillation became very fine and rapid and remained so for some time. Figure 5 illustrates the electrocardiogram of such an experi-

![Figure 4](http://circ.ahajournals.org/)
ment. The record of section A shows ventricular fibrillation after 30 seconds of fibrillation. The tracing in section B, recorded 3 minutes and 40 seconds after the administration of epinephrine, that is five minutes after the initiation of fibrillation, shows that the fibrillation was still very rapid and fine; this is very striking when this tracing is compared with the tracing of figure 3, section C, which shows how much coarser and slower fibrillation had become after 1 minute and 30 seconds when not modified by epinephrine. The record of section C in figure 5 shows that even after 11 minutes and 30 seconds of fibrillation, that is, 10 minutes and 10 seconds after the administration of epinephrine, the fibrillation was still very fine and rapid, and this is especially striking when the tracing of figure 5, section C is compared with the tracing of figure 3, section C.

In these four experiments the control arterial blood pressures were 125, 110, 160 and 160 mm. Hg, respectively. Massage-countershock was applied respectively 9 1/2, 10, 11 and 10 1/2 minutes after the injection of epinephrine. Ventricular fibrillation was stopped and a coordinated ventricular beat restored. However, because of the recurrence of ventricular fibrillation, massage-countershock had to be used five and six times, respectively, in the first two experiments. The highest arterial blood pressures reached were 135, 135, 130 and 195 mm. Hg, respectively. At the time the observation was discontinued, that is, 24, 23, 19, and 17 minutes after the injection of epinephrine, the blood pressures were 65, 70, 130 and 40 mm. Hg, respectively.

V. Procaine

To study the effect of procaine on ventricular fibrillation a technic essentially similar to that used for the study of epinephrine was employed. Ventricular fibrillation was induced electrically as previously described; then after 30 seconds of fibrillation, variable amounts of a 20 per cent solution of procaine hydrochloride were injected, half into each ventricular cavity. This was followed by 10 massages of the ventricles given within one-half minute and with the aorta partially occluded. After the massage the heart was observed.

A. Dose of 200 mg. of Procaine Hydrochloride per Kilogram of Body Weight. In all four dogs which received this dose of procaine, ventricular standstill occurred during or immediately following massage. One of these experiments is illustrated by figure 6. Two dogs remained in ventricular standstill for the 10 minutes and 30 seconds and 14 minutes and 40 seconds following the injection of procaine that they were observed. In the other two dogs, five and seven minutes, respectively, after the inception of fibrillation ventricular standstill was followed by slow, localized, irregular ventricular contractions which did not raise the arterial blood pressure. The electrocardiographic deflections corresponding to those ventricular contractions were continuous, slow, irregular undulations very variable in size and form, essentially similar to those seen in the late type of fibrillation previously described. After a period of 10 to 20 minutes of observation, all four hearts were massaged whether they were in ventricular standstill or in what will be referred to from now on as coarse ventricular fibrillation. All four dogs remained in or developed ventricular fibrillation. In three of the four dogs, 1 mg. of epinephrine injected into the ventricles at this time increased the frequency of the fibrillatory waves.

B. Dose of 150 mg. of Procaine Hydrochloride per Kilogram of Body Weight. In all of seven dogs this dose of procaine produced a slowing
and coarsening of the ventricular fibrillation from a frequency of 600 to 800 per minute to a frequency of 80 to 150 per minute. In four of the seven dogs, coarse fibrillation was followed by ventricular standstill in one to three minutes after the administration of procaine. All four dogs remained in ventricular standstill for a 10 to 15 minute period of observation, after which cardiac massage and 1 mg. of epinephrine resulted in coarse ventricular fibrillation. In the other three dogs, procaine made the fibrillation coarser and slower without producing ventricular standstill. After a period of observation of 10 to 15 minutes, cardiac massage alone in one of the three dogs and cardiac massage—epinephrine in the other two modified the character of the fibrillation without stopping it.

The electrocardiographic deflections were so constant in shape and frequency that this cardiac mechanism may be called ventricular flutter. The frequency of the flutter waves was between 95 and 140 per minute. After three to four minutes of ventricular flutter, slow fibrillation returned and persisted for a 10 to 15 minute period of observation.

D. Dose of 50 mg. of Procaine Hydrochloride per Kilogram of Body Weight. At this dosage, procaine slowed fibrillation from a frequency of 550 to 850 per minute to 250 to 450 per minute and fibrillation persisted for a 10 minute period of observation in four out of six dogs. In the fifth dog, ventricular fibrillation slowed, then was followed by ventricular standstill seven minutes after the inception of fibrillation. Then

C. Dose of 100 mg. of Procaine Hydrochloride per Kilogram of Body Weight. With a dose of 100 mg. of procaine per kilogram, results essentially similar to those observed with the dose of 150 mg. per kilogram were observed in a group of seven dogs. Three of these seven dogs went into ventricular standstill during or immediately following massage. One of the three reverted to slow ventricular fibrillation after 30 seconds of ventricular standstill. Massage did not stop ventricular fibrillation. The other two remained in standstill for a 10-minute period of observation after which cardiac massage resulted in ventricular fibrillation. In the other four dogs, procaine slowed fibrillation considerably from a frequency of 600 to 850 to a frequency of 100 to 250 per minute, and all four hearts went through a period during which regular sinus rhythm of a rate of 30 per minute with prolonged atrioventricular conduction and intraventricular block developed. These ventricular contractions did not raise the aortic blood pressure. As the heart did not seem to improve dynamically after three minutes of sinus bradycardia, the ventricles were massaged, which resulted in ventricular fibrillation. Massage-countershock was instituted 20 minutes after the beginning of the first bout of fibrillation and restored regular sinus rhythm. At this time 1 mg. of epinephrine resulted in sinus tachycardia and the arterial blood pressure reached 220 mm. Hg. The preparation was not followed any further.

In the sixth dog, procaine slowed fibrillation; then ventricular flutter of a rate of 330 per minute was observed. Four minutes after the

![Figure 6. Continuous electrocardiographic tracing, lead II, showing ventricular fibrillation becoming coarser and slower and then ending in ventricular standstill after the intracardiac administration of 200 mg. of procaine per kilogram of body weight and cardiac massage. Procaine was administered 30 seconds after the initiation of fibrillation and the recording of the tracing was begun immediately after the injection of procaine.](http://circ.ahajournals.org/doi/10.1161/01.MAX.105.2.746)
inception of fibrillation, ventricular flutter of a rate of 85 per minute was present and three minutes later ventricular standstill occurred. It persisted for four minutes, then irregular, coordinated ventricular beats were resumed, at which time the atria were noted to beat infrequently and irregularly; the ventricular contractions were idioventricular in origin. As the ventricular beats did not raise the arterial blood pressure, cardiac massage was administered for one and one-half minutes, which resulted in sinus tachycardia with prolonged atrioventricular conduction. The arterial blood pressure rose progressively, reaching a peak of 130 mm. Hg 17 minutes after the inception of the first bout of ventricular fibrillation, then it declined progressively and was 70 mm. Hg one hour after the initiation of fibrillation; the control blood pressure was 120 mm. Hg. The electrocardiogram was similar to the control tracing 16 minutes after the beginning of ventricular fibrillation.

E. Dose of 20 mg. of Procaine Hydrochloride per Kilogram of Body Weight. In all eight dogs which received this dose, ventricular fibrillation became coarser and slowed from a rate of 500 to 750 per minute to a rate of 150 to 400 per minute. In three of these dogs, fibrillation persisted for a 10, 12 and 16 minute period of observation, after which massage-countershock resulted in the return of a regular coordinated ventricular beat. However, the arterial blood pressure in all three dogs either did not rise, or rose but then decreased within approximately five minutes to levels of 30 to 50 mm. Hg. In the remaining five dogs, ventricular standstill occurred 6, 8½, 9½, 10 and 14 minutes, respectively, after the inception of fibrillation. The dog which developed standstill after nine and one-half minutes remained in ventricular standstill for six minutes at which time massage resulted in ventricular fibrillation. Massage-countershock restored a coordinated ventricular beat and the blood pressure reached a peak of 120 mm. Hg, but it declined to 20 mm. within five minutes. In the other four dogs, ventricular standstill was immediately followed by a slow idioventricular rhythm of from 10 to 50 beats per minute in three dogs and regular sinus rhythm with a 4:1 atrioventricular block in the other dog. These coordinated beats were weak and unable to raise the aortic blood pressure. Cardiac massage at this time resulted in ventricular fibrillation. Massage-electric countershock caused the resumption of regular sinus rhythm in all four dogs. In three dogs the blood pressure rose and remained above 90 mm. Hg and was still 90 mm. Hg or more 45 to 60 minutes after the initiation of fibrillation. In the fourth dog in which ventricular fibrillation persisted for 14 minutes, the blood pressure rose to 75 mm. Hg but decreased markedly within a few minutes.

VI. Procaine and Continuous Massage for Ten Minutes

Because it was thought that procaine might be more effective in stopping ventricular fibrillation and restoring a competent ventricular contraction if myocardial anoxia were prevented, a series of experiments was conducted in which cardiac massage was continued for 10 minutes after the injection of procaine into the ventricles. Under such circumstances, a dose of 50 mg. of procaine hydrochloride per kilogram of body weight immediately slowed the fibrillation from a rate of 500 to 650 to a rate of 250 to 300 per minute, and the fibrillation became slower and coarser. However, it persisted continuously in three of the five dogs; in the other two dogs there was a brief period of ventricular standstill lasting 30 and 60 seconds, one and one-half minutes after the injection of procaine. These episodes of ventricular standstill were followed by the resumption of ventricular fibrillation on continuing cardiac massage. After 10 minutes of massage, ventricular fibrillation was stopped with the electrical countershock in all five dogs and regular sinus rhythm with coordinated ventricular beats was resumed. One-half hour after the initiation of fibrillation, that is, 20 minutes after fibrillation had been stopped by countershock, the mean arterial blood pressures in the five dogs were 60, 75, 20, 40 and 20 mm. Hg, respectively, their control blood pressures being 135, 110, 120, 80 and 105 mm. Hg, respectively. In another five dogs, a dose of 20 mg. of procaine per kilogram and continuous massage for 10 minutes, resulted in a gradual slowing of the
fibrillation from a rate of 600 to 700 to a rate of 300 to 400 per minute, but fibrillation persisted in all five dogs. After the 10 minute period of massage, massage-countershock resulted in the resumption of regular sinus rhythm. In three dogs the blood pressure rose to 170 to 180 mm. Hg and was still 75 to 85 mm. Hg 30 minutes later. In the other two dogs, the blood pressure rose only slightly when fibrillation was stopped and fell to around 40 mm. of mercury within 5 minutes.

VII. Epinephrine and Countershock after Five Minutes of Fibrillation

It has been reported in previous paragraphs that, when electrical countershock was applied after fibrillation had been allowed to evolve spontaneously for approximately five minutes, electrical countershock still stopped ventricular fibrillation, but the effect of the ischemia due to the five-minute period of fibrillation was such that the ventricular contractions, although coordinated, were ineffective in raising the aortic blood pressure, and the intracardiac conduction (atrioventricular and intraventricular) as well as the rhythmicity of the ventricles was much depressed. Cardiac massage under such circumstances was shown to improve the dynamics of the ventricles as well as the intracardiac conduction and the rhythmicity of the ventricles. However, when, under the circumstances of these experiments, massage was applied to ventricles beating weakly but in a coordinated manner, it frequently resulted in the inception of ventricular fibrillation. It was thought of interest to investigate whether, by injecting epinephrine into the ventricular cavity just prior to applying the electrical countershock, it might not be possible to avoid massage and thereby the return of ventricular fibrillation and yet to obtain immediately upon stopping fibrillation with the electrical countershock coordinated ventricular contractions strong enough and frequent enough to restore an adequate circulation without any appreciable latent period. For this purpose ventricular fibrillation was induced electrically in 10 dogs. In 3 of these 10 dogs 0.05 mg. of epinephrine per kilogram of body weight was injected, half of the amount in each ventricular cavity after five minutes of fibrillation. In the other seven dogs, 0.01 mg. of epinephrine per kilogram of weight was similarly injected after five minutes of fibrillation. The total amount of the drug given was dissolved in 1 cc. of a 0.9 per cent sodium chloride solution. After the injection of epinephrine into the ventricles, the ventricles were massaged 10 times over a 30-second period; then electrical countershock was applied. The first one of the three dogs which received 0.05 mg. of epinephrine per kilogram of body weight had a control blood pressure of 110 mm. Hg. After five minutes of ventricular fibrillation, epinephrine was administered; then ventricular fibrillation was promptly stopped by electrical countershock and a coordinated ventricular contraction immediately supervened. However, within a few seconds spontaneous ventricular fibrillation recurred. It was stopped immediately by countershock and an effective coordinated ventricular beat immediately followed the termination of fibrillation. After a period of 2 minutes and 10 seconds during which an effective ventricular contraction persisted and the blood pressure rose above 100 mm. Hg, ventricular fibrillation again recurred spontaneously. It was stopped by countershock and immediately followed by effective ventricular beats, the blood pressure rising above 160 mm. Hg, but after 20 seconds, ventricular fibrillation recurred once more. It was stopped again promptly by countershock and immediately followed by effective ventricular beats, the blood pressure rising above 160 mm. Hg. The blood pressure was 170, 90, 100, 120, 115, and 100 mm. Hg 10, 15, 20, 30, 45, and 60 minutes, respectively, after the induction of ventricular fibrillation. Twenty minutes after the inception of the initial ventricular fibrillation, the electrocardiogram was similar to the control tracing. The second of the three dogs which received 0.05 mg. of epinephrine per kilogram of weight reacted essentially in a similar manner except for the important fact that ventricular fibrillation recurred spontaneously only once 30 seconds after it had been stopped. In the third and last dog of this series, 10 electrical countershocks over a period of 45 seconds had to be applied before ventricular fibrillation was stopped. Fibrillation was then followed by ven-
tricular standstill, then by weak ventricular contractions. The blood pressure remained around 10 mm. Hg for about two minutes, then it rose as high as 230 mm. Hg, i.e. 70 mm. Hg above the control blood pressure. It then decreased and was 170, 115, 115, 120 and 140 mm. Hg 10, 20, 30, 40 and 60 minutes, respectively, after the inception of the initial bout of ventricular fibrillation. Because of the frequent recurrence of ventricular fibrillation in these three dogs, similar experiments were conducted with a smaller dose of epinephrine.

In seven dogs, ventricular fibrillation once induced was allowed to evolve for five minutes, at which time 0.01 mg. of epinephrine per kilogram of body weight was injected into the ventricles. The ventricles were then massaged 10 times and electrical countershock applied. In two of these seven dogs, although ventricular fibrillation was stopped by the countershock, 10 and 19 countershocks were necessary. The ventricular beats never were strong enough to raise the arterial blood pressure significantly.

In the other five dogs, ventricular fibrillation was stopped by the countershock, but, within a few seconds to two and one-half minutes after the first countershock, it recurred once in four of the five dogs and twice in the fifth dog. Every time countershock was used successfully to stop ventricular fibrillation. After fibrillation was stopped, the blood pressure rose above its control level then decreased progressively. In these five dogs the control mean arterial blood pressures were 180, 180, 130, 115 and 150 mm. Hg, and one hour after the initiation of fibrillation pressures had progressively decreased to 120, 140, 85, 80 and 110 mm. Hg.

**Discussion**

From our observations as well as those of others,\(^1\) it is clear that electrical countershock when applied to the ventricles of dogs which have been fibrillating for about 30 seconds was very effective in stopping electrically induced ventricular fibrillation. A few seconds after the termination of ventricular fibrillation, regular sinus rhythm was resumed, this being sometimes preceded by an episode of atrial fibrillation induced by the countershock. The mean arterial blood pressure promptly rose above control, then progressively decreased to approximately its control level. It might then progressively decline over the next hour; this might occur even without a period of ventricular fibrillation in dogs with open chest. The electrocardiogram returned to its control form soon after the ventricular fibrillation had been stopped. If the ventricles were allowed to fibrillate for five minutes, fibrillation was seen on direct observation of the heart to become coarser and slower and the electrocardiographic tracing revealed that the rate of the fibrillation became progressively slower. Electrical countershock applied after five minutes of fibrillation still stopped fibrillation, but the myocardium had been so damaged during this five minute period of fibrillation that atrioventricular block and temporary ventricular standstill were observed. The ventricular contractions remained ineffective in raising the arterial blood pressure, and the heart progressively deteriorated further as previously observed by Dow and Wiggers.\(^9\) Manual massage of the ventricles alone was never observed to stop ventricular fibrillation, which is in agreement with the findings of Stearns, Maison and Stutzman.\(^?\) However it ensured a good enough coronary circulation to prevent the fibrillatory process from deteriorating from the early fine, fast type to the late, coarse and slow type, and it even made the late type revert to the early type.

When, after the ventricles had been allowed to fibrillate for five minutes, the ventricles were massaged for 30 seconds, cardiac massage of even such a short duration changed the character of the fibrillation. The fibrillation which had become of the late type, slow and coarse, by the end of the five minute period of fibrillation reverted to the early type, fine and rapid. Electrical countershock applied after five and one-half minutes of fibrillation, cardiac massage being applied for the last 30 seconds, stopped fibrillation, but the ventricular contractions were weak and did not seem to improve. When it seemed probable that the ventricular contraction would not improve but rather deteriorate further, the ventricles were again massaged although it was realized that massage of the ventricles beating in a coordinated manner would probably induce ventricular arrhythmias.
and possibly ventricular fibrillation.* Indeed, fibrillation was induced. It was then stopped with the electrical countershock. This countershock-massage-countershock maneuver was repeated two to five times in each of the dogs of this series. During massage of the fibrillating ventricles, the ventricular mass which first felt very flabby to the massaging hand, rather suddenly became strikingly much firmer. When the ventricles were noted to beat more vigorously and the arterial blood pressure began to rise after fibrillation had been stopped electrically, massage was not administered again. At this time the blood pressure rose above its control level in most of these dogs, but by the end of one hour it had fallen markedly in most dogs. It seems probable that one longer period of massage right after the five minutes of fibrillation would have given the same or better results without the repeated use of countershock.

This was confirmed to a certain extent by the observations made on a group of five dogs in which fibrillation was allowed to persist for 9- to 18-minute periods during which the ventricles were massaged continuously except for interruptions of 30 seconds every one or two minutes to permit the recording of electrocardiograms. At the end of the period of fibrillation no massage was given for one and one-fourth to three minutes and in only one dog for four and three-fourths minutes. Then countershock preceded by a last short period of massage restored regular sinus rhythm, vigorous ventricular contractions and a level of blood pressure within the control range except in the dog in which massage was interrupted for four and three-fourths minutes. In this last dog the blood pressure rose but remained slightly below control level. It is probable that, when the experimental conditions, especially the duration of the bout of ventricular fibrillation, were such that the arterial blood pressure was not restored to control level, the myocardial damage produced played a role, but it is no less probable that damage to other structures such as the vasomotor center might have been an important determinant of the level to which the blood pressure was restored. In short it seemed that ventricular fibrillation, per se, did not produce any damage to the myocardium and/or other structures in the body and that complete recovery from ventricular fibrillation was possible if fibrillation lasted only a few seconds, or if during longer periods of fibrillation an adequate supply of oxygenated blood to the heart and the whole body was maintained. As far as recovery of the heart was concerned, nothing seemed to be gained by trying to stop fibrillation before adequate massage of the ventricles had restored sufficient coronary blood supply for a long enough period. It must be added that when manual massage of the ventricles was applied, often large epicardial hemorrhages appeared after the blood pressure returned to or toward control level. No attempt was made to estimate either the depth of such hemorrhages or their effects on the dynamics of the heart.

In our experiments epinephrine never stopped fibrillation although it modified the fibrillatory process very markedly. It was observed that epinephrine rendered the fibrillation much more rapid and fine and prevented the progressive slowing and coarsening of the fibrillation. Massage-countershock applied after periods of fibrillation ranging between 10 and 16½ minutes from the beginning of the fibrillation stopped the fibrillation and restored coordinated, effective ventricular beats, but the arterial blood pressure, except in one experiment, fell rapidly.

A dose of 200 mg. of procaine hydrochloride per kilogram of body weight stopped ventricular fibrillation within a few seconds, but after fibrillation had been stopped, the ventricles remained in standstill or reverted to a slow type of ventricular fibrillation. Massage and massage-epinephrine resulted in fibrillation. With doses of 100 and 150 mg. of procaine, similar results were observed, except that ventricular standstill occurred less frequently. In those

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* In our experience, massage of the ventricles contracting in a coordinated manner, administered as far as possible during diastole, induced ventricular tachycardia lasting as long as the massage and very seldom ventricular fibrillation in normal hearts. In hearts which had been allowed to remain in ventricular fibrillation for five minutes, after which fibrillation had been stopped electrically, massage resulted in ventricular fibrillation in almost all cases.
dogs in which standstill was not obtained, procaine slowed the fibrillation. A dose of 50 mg. of procaine slowed the fibrillation without stopping it in four out of six dogs. In the fifth dog, procaine stopped fibrillation seven minutes after the initiation of fibrillation; the ventricles began to beat, the cardiac mechanism being sinus bradycardia with prolonged atrioventricular conduction time and intraventricular block. Because the ventricular contractions were ineffective in raising the aortic pressure and did not seem to improve dynamically, the ventricles were massaged and ventricular fibrillation resulted. Massage-countershock restored regular sinus rhythm, and with epinephrine the arterial blood pressure rose to a peak of 220 mm. Hg. In the sixth dog, ventricular standstill followed the administration of procaine. It occurred seven minutes after the initiation of fibrillation. The standstill was followed by an idioventricular rhythm. Because of the weakness of the ventricular contractions, massage was resorted to. The heart improved greatly and the blood pressure rose to a peak of 130 mm. Hg to decline to 70 mm. Hg by the end of one hour from the induction of fibrillation. With a dose of 20 mg. of procaine, fibrillation persisted but was made slower in three out of eight dogs. Massage-countershock restored coordinated beats, but the arterial blood pressure did not rise very markedly, or fell to very low levels within a few minutes. In the other five dogs, ventricular standstill occurred rather late, 6 to 14 minutes after the inception of fibrillation. One of these five dogs remained in ventricular standstill; massage induced ventricular fibrillation and countershock-massage restored an efficient ventricular beat, the arterial pressure rising to a peak of 120 mm. Hg but declining rapidly. In the other four dogs, ventricular standstill was followed immediately by regular sinus rhythm or idioventricular rhythm. Because of the weakness of the ventricular contraction, massage was applied. It induced ventricular fibrillation. Massage-countershock restored regular sinus rhythm with adequate arterial blood pressure. The blood pressure was still 90 mm. Hg 45 to 60 minutes after the inception of fibrillation; only in that dog in which fibrillation persisted for 14 minutes did the arterial blood pressure decline markedly within a few minutes.

It seems from these experiments on the effect of procaine that the doses of procaine hydrochloride which constituted a reliable means of stopping fibrillation were such that they depressed the rhythmicity of the heart so that the ventricles remained in standstill. Smaller doses of procaine did not seem a reliable means of stopping ventricular fibrillation because too many failures were encountered or because ventricular standstill occurred with a long latent period. Therefore, procaine alone seemed to be not too promising a means of stopping fibrillation. When massage was administered after ventricular standstill had been induced by procaine or after the fibrillation had been made slower, ventricular fibrillation recurred or persisted. Massage-countershock was still effective in stopping fibrillation and restoring a normal cardiac mechanism. However, in most experiments the blood pressure did not rise, or if it did, it promptly declined to very low levels, although in a few dogs which had received 20 mg. of procaine, massage-countershock resulted in the re-establishment of normal cardiac mechanism and a fairly normal blood pressure. It is probable that massage-countershock alone would have given better results. With the hope of preventing myocardial anoxia, cardiac massage was maintained after 20 or 50 mg. of procaine had been administered to 10 dogs, but this did not improve the results observed with procaine. There was no definite indication in these experiments that procaine rendered countershock more effective in stopping ventricular fibrillation, although the possibility was not excluded by our experiments performed with hearts of relatively small size in which electrical countershock alone was eminently effective in stopping ventricular fibrillation. Our observations essentially are in agreement with those of Kay as well as those of Stearns, Maison and Stutzman, except for the fact that procaine alone stopped ventricular fibrillation more frequently in our series of experiments probably because heavier doses were used at least in some dogs.

It is a little more difficult to evaluate the results obtained in those experiments in which
ventricular fibrillation was allowed to persist for five minutes, after which epinephrine (0.05 or 0.01 mg. per kilogram of body weight) was injected into the ventricular cavities and 10 massages of the ventricular mass given before electrical countershock was applied. Such doses of epinephrine did not prevent the electrical countershock from stopping ventricular fibrillation. However, in three experiments numerous countershocks had to be given before ventricular fibrillation was stopped. Whether or not this was due to epinephrine cannot be determined inasmuch as such a difficulty is occasionally encountered with hearts not under the influence of epinephrine. There seems to be little doubt that such doses of epinephrine given as has been described resulted in the immediate restoration of a strong and frequent ventricular beat without massage as soon as fibrillation had been stopped by countershock. It must be kept in mind, however, that the frequent recurrence of ventricular fibrillation in this series of experiments was a definite drawback. It was not attempted to determine whether doses of epinephrine sufficiently small not to cause a recurrence of ventricular fibrillation would still be useful in restoring promptly the strength of the ventricular contraction.

SUMMARY AND CONCLUSIONS

The usefulness of electrical countershock, massage, synthetic l-epinephrine and procaine in stopping electrically-induced ventricular fibrillation and restoring a competent ventricular contraction was studied in 75 dogs anesthetized with sodium pentobarbital. The following conclusions were reached.

1. Massage alone does not stop ventricular fibrillation although it prevents it from deteriorating from the early fine, rapid type to the late slow, coarse type, or even makes it revert from the late to the early type.

2. Epinephrine alone does not stop fibrillation although it alters it to make the fibrillatory process become very fine and rapid.

3. Electrical countershock is very effective in stopping ventricular fibrillation in its early stage as well as its late stage. It also stops fibrillation when the heart is under the influence of procaine or epinephrine. When shock is applied after a relatively short period of fibrillation of approximately 30 seconds, a coordinated and competent ventricular beat promptly supervenes after fibrillation has been stopped by the countershock, and the arterial blood pressure is restored to its control level. When applied after five minutes of fibrillation, countershock stops fibrillation; a coordinated ventricular contraction is restored but it is incompetent, which, together with probable damage to other structures than the myocardium, such as the vasomotor center, prevents the restoration of an adequate circulation.

4. If the fibrillating ventricles are continuously massaged before the countershock used to stop fibrillation is applied, a coordinated competent ventricular contraction immediately follows the termination of fibrillation even when fibrillation is allowed to persist for as long as 10 minutes and possibly longer. There seems to be no advantage in trying to stop with countershock a fibrillation which has lasted more than one to two minutes before massage has been administered for an adequate period of time. The reason is that, if countershock is prematurely used, only a weak ventricular beat will follow the termination of fibrillation. If massage is then resorted to in order to prevent further deterioration of the circulation, it will again induce fibrillation in such hearts, and a series of massage-countershock-massage maneuvers will have to be administered.

5. Procaine slows the rate of the fibrillary process or even stops fibrillation, depending upon the dose of the drug used. However, those doses of procaine, which constitute a reliable means of stopping fibrillation, depress, among other things, the rhythmicity of the heart to such an extent that after fibrillation has been stopped, long periods of ventricular standstill occur. Massage then applied induces fibrillation.

6. If after a five minute period of fibrillation, epinephrine is administered prior to the countershock, a competent ventricular contraction is resumed as soon as fibrillation has been stopped, and the blood pressure is restored to a level seen only when, under similar experimental conditions, a prolonged period of massage precedes countershock. However, with the
doses of epinephrine studied, fibrillation recurs very frequently, which necessitates the repeated use of the electrical countershock.

**SUMARIO ESPAÑOL**

La eficacia del contrachoque eléctrico, masaje cardíaco, epinefrina y procaina en terminar la fibrilación ventricular y restaurar contracciones ventriculares eficientes en el perro anestesiado ha sido estudiada. Se encontró que el contrachoque es una medida confiable de terminar la fibrilación. Sin embargo, debe de ser precedido de masaje cardíaco si no es aplicado prontamente luego de la iniciación de la fibrilación. La epinefrina ayuda a restaurar contracciones ventriculares eficientes una vez la fibrilación haya sido terminada con contrachoque, pero aumenta la incidencia de la reaparición de la fibrilación. Las dosis de procaina que constituyen una medida confiable para la terminación de la fibrilación deprimen el ritmo del corazón hasta tal extremo que la cesación de la fibrilación es seguida de prolongados intervalos de pausa cardíaca.

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A Study of the Usefulness and Limitations of Electrical Countershock, Cardiac Massage, Epinephrine and Procaine in Cardiac Resuscitation from Ventricular Fibrillation

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