Experimental Coronary Artery Occlusion: Ventricular Fibrillation and Survival as Affected by Selected Drugs and Ionic Alterations*, **

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The effect of drugs and ions upon the normal heart and upon isolated myocardium has been thoroughly investigated and adequately documented. Increased interest in the application of these substances in a manner which approaches the abnormal clinical situation—that is, following coronary artery occlusion and in various arrhythmias—has been demonstrated during the past few years. The stimulus for this recent investigative trend has come primarily from three sources: (1) Fibrillation following coronary artery occlusion and the efforts to devise means of permanently increasing collateral blood supply to the myocardium; (2) Cardiac arrest or ventricular fibrillation during surgery; and (3) Ventricular fibrillation during hypothermia.

This report concerns experimental myocardial ischemia produced by acute coronary artery occlusion, the influence of drugs and inorganic ions on survival rates and early fibrillation and the influence on certain ions upon other ions.

Materials and Methods

Adult mongrel dogs were used as the experimental animals. Following anesthetization with intravenous nembutal, endotracheal intubation was accomplished and the chest opened by an incision in the fourth inter-space. The left coronary, circumflex coronary, and left anterior descending coronary arteries were carefully demonstrated. A heavy silk ligature was placed beneath the anterior descending branch immediately adjacent to its origin. The loose ligature was threaded through a small plastic tube which was anchored to the pericardium at one end and brought out through the chest wall at the other end. Following closure of the chest wall, the ligature ends and plastic tube were placed subcutaneously, the animal was given penicillin and returned to his cage.

The following day the animal was again anesthetized, control electrocardiograms obtained, and control blood samples drawn for plasma pH, potassium, sodium, chloride, calcium and carbon dioxide tension. The animals were then divided into the following groups depending upon the drug or ion which was administered: (1) Control, one day postopera-

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tive—10 dogs—no infusion. (2) Control, 20 days postoperative—three
dogs—no infusion. (3) Acidosis—five dogs—100-140 mEq. hydrochloric
acid in 400 cc. water. (4) Alkalosis—five dogs—140-150 mEq. sodium
bicarbonate in 400 cc. distilled water. (5) Hyperkalemia—five dogs—
30-45 mEq. potassium chloride in 400 cc. distilled water. (6) Hypercal-
cemia—five dogs—1 gm. calcium chloride in 400 cc. 5 per cent dextrose
in distilled water. (7) Excess sodium chloride—five dogs—4½ gm.
sodium chloride in 500 cc. distilled water. (8) Procaine—five dogs—
500 mgm. "Novacaine" in 400 cc. 5 per cent dextrose. (9) Papaverine
—five dogs—30 mgm. papaverine intravenously. (10) Quinidine—five
dogs—100 mgm. quinidine intravenously. Immediately following the in-
fusion, electrocardiograms were again obtained and blood samples drawn
for the determinations listed above. The ligature was tightened, com-
pletely occluding the left anterior descending coronary artery and electro-
cardiograms were obtained at frequent intervals to one hour postocclusion.
If the animal still lived at the end of this period he was returned to his
cage without further care. Early fibrillation as used in this communi-
cation denotes fibrillation occurring during the sixty minutes following
coronary artery occlusion. Mortality was determined at the end of the
first 24 postoperative hours. pH was determined on the Beckman model
G pH meter. Calcium was determined by titration with EDTA. Sodium
and potassium were analyzed on the Beckman direct reading flame pho-
tometer. Chlorides were determined by a modification of the Volhard
method. Carbon dioxide tension was measured by the Van Slyke manom-
metric technique.

Results

(1) Control. The mortality following ligation of the left anterior
descending coronary artery has been reported on large series of dogs by
others so that we considered it necessary to use only 10 animals occluded
on the first postoperative day. The mortality in these 10 dogs was 80
per cent with 30 per cent of the deaths due to early fibrillation which oc-
curred during the first 12 minutes following ligation.

(2) Control, 20 days postoperative. Of passing interest is the fact
that this experiment was designed originally to allow the animal to re-
cover from thoracotomy and dissection around the coronary artery. We
soon found that if ligation were delayed following pericardiotomy we
failed to achieve any mortality. This point is borne out in three cases
in which ligation was delayed for 20 days after thoracotomy. In this
group there was no mortality and in two of the three animals there was
no change in the electrocardiogram pre and postocclusion.

(3) Acidosis. Hydrochloric acid in the amounts used in this study
produced an acidosis with a mean pH of 7.22 as opposed to a mean con-
tral pH of 7.37. This group of five animals suffered an 80 per cent mor-
tality and 40 per cent fibrillation rate.

(4) Alkalosis. The mean pH was elevated to 7.62 from a mean control
of 7.37 by the infusion of sodium bicarbonate. The mortality rate was
80 per cent and fibrillation rate 60 per cent.
(5) Hyperkaliemia. A mortality rate of 60 per cent and a fibrillation rate of 60 per cent resulted when the mean potassium was elevated to 6.24 mEq. per liter from a mean control of 3.7 mEq. per liter by infusion of potassium chloride.

(6) Hypercalcemia. The infusion of 1 gm. of calcium chloride elevated the serum calcium from a mean control of 5 mEq. per liter to 7.7 mEq. per liter. Mortality 80 per cent, fibrillation 60 per cent.

(7) Excess Sodium Chloride. Only 500 cc. of 0.9 sodium chloride was infused and this hardly represents an excess as demonstrated by the minimum rise in sodium and chloride. However, 100 per cent mortality and 80 per cent fibrillation resulted.

(8) Procaine. A mortality rate of 100 per cent and fibrillation rate of 100 per cent resulted when 500 mgm. of “Novacaine” was infused.

(9) Papaverine. The intravenous administration of papaverine, 30 mgm. yielded mortality and fibrillation rates of 60 per cent.

(10) Quinidine. A mortality of 60 per cent, and fibrillation of 60 per cent followed the intravenous administration of Quinidine, 100 mgm.

(11) Ions. The influence of infusion of certain ions and drugs upon the blood level of other ions is charted in Figure 1.

Discussion

One of the earliest stimuli for investigation of ventricular fibrillation arose as the result of this lethal arrhythmia following coronary artery occlusion. Unfortunately, medical attempts at preventing fibrillation have met with no success despite the tremendous amount of investigative work devoted to the problem. Wiggers1 in 1940 in discussing the mechanism of ventricular fibrillation following coronary occlusion found that a significantly smaller amount of electric current was required to fibrillate the heart following experimental coronary occlusion than normally. He theorized that spontaneous fibrillation during or following coronary occlusion was precipitated because usually innocuous ectopic stimuli became of precipitating level for the hyperirritable myocardium. Beck2 employed the term “electrically unstable heart” in discussing the mechanism of ventricular fibrillation. He found that 90 per cent of all the people who die of coronary artery disease die because the heart becomes electrically unstable and fibrillates. The electrically stable heart is one in which oxygen saturation is uniform throughout, and this stability persists when the oxygen tension is reduced provided the reduction is uniform throughout the myocardium. The heart becomes electrically unstable and may fibrillate when the oxygen tension is not uniform throughout the myocardium; that is, when an area with decreased oxygen tension is surrounded with well-oxygenated myocardium or vice versa. If some substance could be given which would appreciably decrease the hyperirritability of the myocardium and the incidence of fibrillation following coronary occlusion, substantial progress would have been achieved. This was one of the clinical applications in mind when this project was undertaken.
The consistent level of coronary artery ligation in any study of mortality following coronary occlusion has been emphasized repeatedly. Our control mortality rate of 80 per cent is similar to that of others who have used the same location for coronary occlusion. Hahn\textsuperscript{8} reported a mortality rate of 70 per cent following ligation of the descending ramus of the left coronary artery. Bakst\textsuperscript{4} reported a mortality rate of 60 per cent when the same artery was ligated but mentioned that the higher survival rate might have been due to the high incidence of an accessory left anterior descending coronary artery which arose from the circumflex branch of the left coronary artery. Vineberg\textsuperscript{5} noted a mortality rate of 90 per cent. McAllister\textsuperscript{6} showed an immediate mortality rate of 70 per cent following ligation of the anterior descending branch in a group of 100 dogs. The incidence of early fibrillation is not so widely reported but Beck\textsuperscript{2} noticed early fibrillation in 50 per cent of his animals at normothermic temperatures.

Because of the nature of this study utilizing various ions and drugs, it was important to determine the incidence of early fibrillation. Because of space considerations, these results are not discussed in the text and form the basis of a separate report.

\textbf{FIGURE 1}: Plasma pH, calcium, sodium, potassium, chloride, and CO\textsubscript{2} content as affected by the ions and drugs employed in the study. The base line, from which the deviations are charted, represents the mean of the control determinations. Because of space considerations, these results are not discussed in the text and form the basis of a separate report.
these substances were administered only once immediately before coronary occlusion and were not administered to the surviving animals, a clear picture of the worth of the substance can be drawn from the early fibrillation rates rather than the overall mortality rates when the drug may have lost its effect. Our early fibrillation rate of 30 per cent is somewhat lower than that reported by Beck although the ultimate mortality rate is similar.

During the past several years efforts have been made along several lines to devise some procedure which would materially and consistently increase collateral blood supply to the myocardium. One of the earliest and still one of the relatively most successfully employed is the principle of irritation of the pericardium and myocardium to encourage collateral vessel growth to the myocardium. The three animals in this series whose coronary artery ligation was delayed 20 days following pericardotomy come under this category although no deliberate attempt was made to stimulate collateral vessel growth. It has been shown previously that simple pericardotomy offers significant protection to the animal when coronary occlusion is delayed for several days. In our series, in addition to opening the pericardium, a polyethylene tube and a large silk suture were left within the pericardium for 20 days. An additional stimulus for collateral vessel growth may have been bacterial pericarditis resulting from the clean, but not sterile, technique and propagated by the intrapericardial foreign bodies. In any event, we were surprised that the collaterals were of sufficient content as to prevent electrocardiographic evidence of coronary occlusion in two of the three animals, in addition to the survival of all three animals in the group. This group is of insufficient size to furnish any conclusions and, of course, was not originally designated for that purpose. However, it does point out the difficulty in evaluating some of the experimental results of other forms of myocardial vascularization.

During the past few years cardiac arrest or ventricular fibrillation occurring during an operation has been the subject of an ever increasing investigative effort. During the early years of these studies various anesthetic agents or combinations thereof were thought to be contributing factors toward ventricular fibrillation. However, during recent years carbon dioxide retention, acidosis and anoxia have been implicated as the contributing factors. With the advent of hypothermia, ventricular fibrillation attained even greater notoriety. It was essential that the incidence of ventricular fibrillation be greatly reduced or abolished if hypothermia were to continue in use as a procedure with acceptable risk. Few projects have been the subject of such intense investigation as has ventricular fibrillation in hypothermia. Certain investigators suggested that ventricular fibrillation might be used as an ideal state during surgery in the heart employing hypothermia and extracorporeal circulation. However, the majority of surgeons consider ventricular fibrillation a hazard worthy of avoiding. Cahn and Melon reported the use of xylocaine anesthetization of the sinoauricular node as a means of preventing ventricular
fibrillation during hypothermia. Riberi, Siderys, and Shumacker\(^9\) have effectively utilized procaine injection of the sin-auricular node. Webb\(^9\) by injecting the auriculoventricular node with procaine, has achieved even greater protection against fibrillation.

While these measures have been successful, investigation along other lines has strongly implicated potassium as the excitatory agent for ventricular fibrillation. Harris\(^10\) et al. injected potassium chloride solutions in varying concentrations into coronary arteries of dogs and noted that the intensity of ectopic ventricular activity depended upon the amount of potassium chloride present. Following coronary ligation, there was a large increase in the potassium content of venous blood from the ischemic area, increasing from a control level of 12.75 mgm. per cent to 24.5 mgm. per cent. The potassium concentration showed a positive correlation with the ectopic activity. In addition, potassium concentration re-approached control levels during the periods that corresponded to the times of disappearance of ectopic activity. At that time potassium content of the infarcted muscle was greatly reduced. Montgomery, Prevedel, and Swan\(^11\) likewise obtained convincing evidence relating potassium and plasma pH to ventricular fibrillation. Hooker\(^12\) found that ventricular fibrillation in the isolated perfused heart could be converted by the addition of potassium to the perfusing medium. Brown and Miller\(^13\) produced ventricular fibrillation in 11 of 15 dogs by a rapid reduction in alveolar carbon dioxide tension following 4 hours of breathing 30 to 40 per cent carbon dioxide.

Our studies suggest that there is no correlation between serum potassium

**EFFECTS UPON MORTALITY AND FIBRILLATION RATES**

![Graph showing mortality and fibrillation rates](Downloaded From: http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21314/ on 04/28/2017)
level and ventricular fibrillation. When Figs. 1 and 2 are inspected it is noted that when the serum potassium was lowest, as in the sodium bicarbonate and calcium chloride series, a fibrillation rate of 60 per cent occurred. When the serum potassium was considerably increased by the infusion of potassium, fibrillation again occurred in 60% of the animals. Thus it would appear that if potassium is the excitatory agent for ventricular fibrillation that myocardial potassium is unaffected by plasma concentration and is on a metabolic basis.

Grumbach et al., using isolated rabbit hearts, studied ventricular fibrillation as related to calcium and potassium. Their results indicated that the initiation of fibrillation was dependent upon the calcium content in the tissue fluid which in turn was determined by the potassium content in the fluid. In our studies in which the serum levels of calcium and potassium were considerably increased, the 60 per cent fibrillation rate was the same in each although the ultimate mortality in the hypercalcemia group was slightly higher.

Bellet in discussing the possible modes of action of 0.5 molar sodium lactate in preventing or correcting cardiac arrhythmias considered that one of the beneficial actions of this solution might be due to the production of alkalosis. In this study, employing sodium bicarbonate rather than sodium lactate to obtain a significant alkalosis, the mortality rate following coronary occlusion was the same as the control but the fibrillation rate of 60 per cent was twice the control level. Thus, alkalosis as obtained with sodium bicarbonate, does not reduce the fibrillation rate which follows the demanding stimulus of coronary occlusion.

Montgomery’s observation showed that blood pH influenced the concentration of potassium in the myocardium, demonstrating that with a low pH the heart takes up potassium whereas with a high pH the heart maintains potassium balance. In our series the mortality and fibrillation rates in the acidosis group most closely approached the control levels whereas the alkalosis group demonstrated fibrillation rates twice the control levels although the ultimate mortality was the same. Again, if potassium is the agent inciting ventricular fibrillation, it would appear that myocardial metabolism of potassium in myocardial ischemia is unaffected by blood pH insofar as decreasing early fibrillation is concerned.

The mortality rate of 100 per cent and fibrillation rate of 80 per cent obtained when coronary occlusion followed infusion of 500 cc. 0.9 per cent sodium chloride is difficult to explain. It can be seen from Figure 1 that the elevation of sodium and chloride were minimal and that the other ions measured were not appreciably changed. In addition, the volume of fluid employed in the infusion was similar to that used in the other series.

The studies of Long and associates show that the intravenous administration of procaine in normal dogs in increasing dosages produced successively, bundle-branch block, slowing of conduction through the A-V node, ventricular tachycardia, and ultimately ventricular fibrillation.
Their studies also suggested that in hearts with muscle damage cardiac changes occurred with therapeutic doses of procaine. Van Dongen\textsuperscript{17} found that "Novacaine" was active against electrical fibrillation and its after effects and against heterotopic rhythms caused in other ways. Wiggers and Wegria,\textsuperscript{18} employing cats' hearts and electrical stimulation, concluded that procaine raised the resistance of the ventricles to fibrillation but did not prevent its occurrence. In our study, in which coronary ligation rather than electric current was used as a stimulus, intravenous procaine was the most lethal agent used insofar as fibrillation was concerned with a 100 per cent mortality and 100 per cent fibrillation rate resulting.

In discussing antifibrillatory drugs, DiPalma and Schultz\textsuperscript{19} showed that papaverine raised the threshold for ventricular fibrillation. In addition, papaverine intravenously was demonstrated to be a marked coronary dilator which was considered to be one of the reasons for its beneficial action in fibrillation. However, it was stated that papaverine might cause ventricular fibrillation in large doses. Elek\textsuperscript{20} studied the effects of increasing levels of papaverine on the animal electrocardiograms. In addition he demonstrated that the favorable action of papaverine in reversing artificially induced ventricular fibrillation was due to depression of conductivity and irritability and to prolongation of the refractory period of the ventricles. In this present series, using 30 mgm. of papaverine intravenously, the fibrillation rate was twice the control although the mortality rate was slightly reduced.

Hess and Haugaard\textsuperscript{21} studied the effect of quinidine on the carbohydrate metabolism of rat heart slices and homogenates. In varying concentrations quinidine produced marked inhibition of glucose utilization and oxygen uptake by homogenates. In some of the older medical literature\textsuperscript{22-23} quinidine was believed to be beneficial in ventricular tachycardia following coronary thrombosis. Moissette,\textsuperscript{24} studying the action of quinidine following occlusion of the descending branch of the coronary artery concluded that quinidine did not prevent ventricular fibrillation but on the contrary often favored it in the presence of coronary occlusion. Smith,\textsuperscript{25} investigating the action of quinidine by a method similar to the one employed in this study, concluded that the dog's myocardium was rendered more susceptible to the development of cardiac irregularities by quinidine sulfate. The mortality rate in his series following occlusion of the left circumflex coronary artery was reduced from 75 to 55 per cent when large doses of quinidine were administered intravenously. The mortality and fibrillation rate of 60 per cent in our quinidine series would suggest that it favors the development of ventricular fibrillation.

**SUMMARY**

1. The effect of selected drugs and various inorganic ions on ventricular fibrillation and mortality following experimental coronary artery occlusion has been investigated.

2. Control mortality of 80 per cent and early fibrillation of 30 per cent
was obtained following ligation of the left anterior descending coronary artery immediately at its origin.

3. When coronary occlusion was delayed for 20 days post-pericardotomy in three animals there was no mortality and two of the three animals failed to demonstrate any change in the pre and postocclusion electrocardiogram.

4. Mortality of 80 per cent and fibrillation of 40 per cent resulted when acidosis was produced by hydrochloric acid.

5. Mortality of 80 per cent and fibrillation of 60 per cent resulted when alkalosis was produced by the infusion of sodium bicarbonate.

6. A mortality rate of 60 per cent and a fibrillation rate of 60 per cent resulted when hyperkalemia was produced.

7. Hypercalcemia produced a mortality of 80 per cent and fibrillation of 60 per cent.

8. Following the infusion of a relatively small amount of sodium chloride an unexpected 100 per cent mortality and 80 per cent fibrillation rate resulted.

9. A mortality rate of 100 per cent and a fibrillation rate of 100 per cent followed the infusion of procaine.

10. The intravenous administration of papaverine produced mortality and fibrillation rates of 60 per cent.

11. Quinidine administered intravenously yielded a mortality rate of 60 per cent and fibrillation rate of 60 per cent.

12. The influence of infusion of certain ions and drugs upon the concentration of other ions was determined.

13. It would appear from this study that if potassium is the excitatory agent for ventricular fibrillation that myocardial potassium in ischemia is unaffected by the plasma concentration of potassium and is on a metabolic basis.

14. If potassium is the agent inciting ventricular fibrillation it would appear from this study that myocardial metabolism of potassium in myocardial ischemia is unaffected by blood pH insofar as decreasing early fibrillation is concerned.

15. None of the drugs or ions employed in this study showed any beneficial effect on reducing mortality or ventricular fibrillation following coronary artery occlusion.

We gratefully acknowledge the electrocardiographic interpretation of Dr. Thomas M. Blake.

RESUMEN

1. Se investigó el efecto de drogas seleccionadas, y de varios iones inorgánicos sobre la fibrilación ventricular así como sobre la mortalidad después de la oclusión coronaria experimental.

2. Se obtuvo el control de la mortalidad en el 80 por ciento y fibrilación temprana en el 30 por ciento después de ligadura de arteria coronaria descendente izquierda inmediatamente en su origen.

3. Cuando la oclusión coronaria fue retardada por 20 días después de la pericardiotomía en tres animales no hubo mortalidad y en dos de
los tres animales no se observó cambio alguno en el electrocardiograma antes y después de la oclusión.

4. Resultó una mortalidad de 80 por ciento y fibrilación de 40 por ciento cuando hubo acidosis provocada por ácido clorhídrico.

5. Una mortalidad de 80 por ciento y fibrilación de 60 por ciento resultó cuando se produjo alcalosis por infusión de bi carbonato de sodio.

6. Cuando se provocó hiperkalemia hubo una mortalidad de 60 por ciento y fibrilación de 60 por ciento.

7. La hipercalcemia produjo una mortalidad de 80 por ciento y fibrilación de 60 por ciento.

8. Después de una infusión de una cantidad relativamente pequeña de cloruro de sodio, resultó una inesperada mortalidad de 100 por ciento y fibrilación de 80 por ciento.

9. La mortalidad fue de 100 por ciento y la fibrilación de 100 por ciento, después de la infusión de procaina.

10. La administración intravenosa de papaverina produjo mortalidad y fibrilación en 60 por ciento.

11. La quinidina intravenosa dió una mortalidad de 60 por ciento y fibrilación de 60 por ciento.

12. Se determinó la influencia de la infusión de ciertos iones y drogas sobre la concentración de otros iones.

13. Parecería según este estudio que si el potasio es el agente excitador para la fibrilación ventricular, el potasio miocárdico en la isquemia no es afectado por la concentración del potasio en el plasma como lo es sobre base metabólica.

14. Si el potasio es el provocador de fibrilación ventricular parecería según este estudio, que el metabolismo miocárdico del potasio en la isquemia del miocardio no es afectado por el pH sanguíneo en lo referente al decremento de la fibrilación temprana.

15. Ninguna de las drogas o iones empleados en este estudio mostró efecto benefico alguno para reducir la mortalidad o la fibrilación ventricular después de la oclusión coronaria.

RESUME

1. L’auteur a examiné l’effet de médications choisies et de différents ions inorganiques sur la fibrillation ventriculaire et sur la mortalité consécutive à l’occlusion expérimentale de l’artère coronaire.

2. Il obtint de ramener le taux de mortalité à 80% et la fibrillation précoce à 30% après ligature de l’artère coronaire descendante antérieure gauche, immédiatement à son origine.

3. Lorsque l’occlusion coronarienne ne survint que 20 jours après la péricardotomie chez trois animaux, il n’y eut aucune mortalité, et chez deux des trois animaux on ne put mettre en évidence la moindre altération de l’électrocardiogramme avant et après l’occlusion.

4. Un taux de mortalité à 80% et 40% de fibrillation furent obtenus quand on créa une alcalose par acide chlorhydrique.

5. Un taux de mortalité de 80% et de 60% de fibrillation fut obtenu lorsque on produisit une alcalose par injection de bicarbonate de soude.
6. Un taux de mortalité de 60% et un taux de fibrillation de 60% furent obtenus par l’hyperkaliémie.

7. L’hypercalcémie produisit une mortalité à 80% et 60% de fibrillation.

8. Après injection d’une quantité relativement faible de chlorure de sodium, un taux de mortalité inattendu à 100% et 80% de fibrillation se produisirent.

9. Un taux de mortalité de 100% et de 100% de fibrillation suivirent l’injection de procaïne.

10. L’injection intraveineuse de papaverine produisit des taux de mortalité et de fibrillation de 60%.

11. La quinidine administrée par voie intraveineuse provoqua un taux de mortalité de 60% et un taux de fibrillation de 60%.

12. L’auteur détermina l’influence de l’injection de certains ions et médications sur la concentration des autres ions.

13. D’après cette étude, il apparaîtrait que le potassium est l’agent excitateur de la fibrillation ventriculaire, que le potassium du myocarde dans l’ischémie n’est pas affecté par la concentration plasmatique de potassium et l’est sur la base métabolique.

14. Si le potassium est l’agent provocateur de la fibrillation ventriculaire, il apparaîtrait d’après cette étude, que le métabolisme myocardique du potassium dans l’ischémie myocardique n’est pas affecté par le pH sanguin, au moins autant que le diminution de la fibrillation précoce est intéressée.

15. Aucune des médications ou des ions utilisés dans cette étude ne montra un effet bénéfique sur la réduction de la mortalité ou de la fibrillation ventriculaire après occlusion de l’artère coronaire.

ZUSAMMENFASSUNG

1. Es wurden die Wirkungen von ausgewählten Arzneimitteln und verschiedenen anorganischen Jonen bei Kammerflimmern und Tod nach experimentellen Coronararterienverschlüssen untersucht.

2. Eine Kontroll-Mortalität von 80% und ein frühzeitiges Flimmern von 30% wurden erzielt, nach der Ligatur des vorherigen absteigenden coronararterienastes unmittelbar an seinem Abgang.

3. Wurde der Coronarverschluss bei 3 Tieren aufgeschoben um 20 Tage nach der Pericardotomie, so gab es keine Sterblichkeit und bei 2 von den 3 Tieren war es nicht möglich, irgendeine Veränderung im EKG vor und nach dem Gefäßverschluss nachzuweisen.

4. Eine Mortalität von 80% und ein Flimmern in 40% traten ein, wenn eine Acidose durch Salzsäure erzeugt wurde.

5. Eine Mortalität von 80% und Flimmern in 60% traten ein, wenn eine Alkalose erzeugt wurde durch die Infusion von Natrium-Bicarbonat.

6. Eine Mortalität von 60% und Flimmern in 60% traten ein, wenn eine Hyperkalaemie erzeugt wurde.
7. Hypercalcaemia führte zu einer Mortality von 80% und Fibrillationen in 60%.
8. Im Anschluss an die Infusion einer relativ geringen Menge von Kochsalz ergab sich eine nicht erwartete Mortality von 100% und Fibrillationen in 80%.
9. Eine Mortalitätsziffer von 100% und Fibrillation in 100% folgte der Infusion von Novocain.
10. Die intravenöse Verabfolgung von Papaverin führte zu Mortalitäts- und Fibrillatoraten von 60%.
11. Quinidin intravenös zugeführte erbrachte eine Mortality von 60% und Fibrillation in 60%.
12. Es wurde der Einfluss der Infusion von bestimmten Jonen und Arzneimitteln auf die Konzentration anderer Jonen bestimmt.
13. Es möchte nach dieser Untersuchung scheinen, dass Kalium der erregende Stoff ist für Kammerfibrillationen, dass das Kalium des Myocards bei der Ischämie nicht beeinflusst wird durch die Plasma-Konzentration des Kaliums und zwar auf der Basis des Stoffwechsels.
15. Keine der Arzneimittel oder Jonen, die für diese Untersuchung verwandt wurden, zeigten irgendeine nützliche Wirkung hinsichtlich der Verringerung der Sterblichkeit oder des Kammerfibrillierens im Anschluss an einen Coronar-Arterienverschluss.

REFERENCES


