A Morphologic Study of Canine Hearts Subjected to Fibrillation, Electrical Defibrillation and Manual Compression

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The pathologic effects on the heart of fibrillation, countershock and manual compression have been studied in canine hearts. Survival experiments have permitted evaluation of the permanence of the damage produced. Massage of the normally beating heart produced minimal nonspecific damage. Manual compression on the fibrillating heart was seen to elicit changes more severe than those observed after fibrillation alone, including occasional focal myocardial necrosis. Alternating-current countershock produced both epicardial and myocardial damage at the site of application of the electrodes. In view of their localization, these changes would not be expected to alter cardiac function appreciably. Comparable changes, but less severe in type, were found in the hearts of animals subjected to condenser discharge countershock. Paddling of the electrodes with saline-soaked gauze did not appear to influence the degree of damage.

The application of electrical currents to the heart in order to stop ventricular fibrillation has been studied by several investigators. A number of reports have appeared describing alternating current devices for defibrillation of the human heart in the operating room, and at least two such machines are commercially available. Although electrical defibrillation has been employed clinically, the damage which may result from this procedure has not been assayed.

The opportunity to examine the hearts of animals subjected to ventricular fibrillation, countershock defibrillation and rhythmic manual compression has been afforded by experiments on cardiac resuscitation carried on by Lape and Maison. Gross and microscopic examination of such organs is important, first, to demonstrate whether or not countershock and resuscitation are productive of visible changes, and second, to establish the type of procedure which will allow the optimum resuscitative result with the least damage to the heart.

In the present study the morphologic changes in the hearts of dogs resulting from ventricular fibrillation, defibrillation by alternating current or condenser discharge countershock, and manual compression are presented and discussed.

**METHOD**

In order to separate, insofar as possible, the different factors in the sequence of experimental events, fibrillation, defibrillation, manual compression and excision of the heart and to demonstrate the individual significance of each, several series of animal studies were carried out. They included: (1) basic control experiments involving the examination of hearts excised from animals at the termination of unrelated experiments; (2) fibrillation control experiments; (3) experiments on the effects of manual compression on both fibrillating and normally beating hearts; (4) alternating current countershock defibrillation studies; and (5) condenser discharge countershock defibrillation studies.

In the animals of groups 3, 4, and 5 varying periods of survival were allowed in order to obtain information regarding the restoration of normal structure. Ether anesthesia was used except in the animals of group 1 which received injections of veratrum viride where pentobarbital was used.

* The devices used for alternating current and condenser discharge countershock are described elsewhere.
Intravenous pentobarbital was also used to kill the animals in which survival had been permitted, before excision of the hearts.

After excision, the hearts were placed in a 10 per cent solution of formaldehyde in isotonic saline. Following gross examination of each heart, several blocks of the myocardial tissue were cut, imbedded in paraffin, sectioned at different levels and stained with hematoxylin and eosin.

**Results**

The hearts from control animals subjected to unrelated procedures showed minimal pathologic changes. Among four hearts from dogs in which extracts of veratum viride had been injected intravenously during pentobarbital anesthesia, three showed no apparent damage either grossly or microscopically. The fourth showed epidermal venous congestion and focal red blood cell extravasation on microscopic section. No changes were found in three animals subjected to thoracotomy during ether anesthesia; a fourth revealed marked microscopic congestion of the myocardium microscopically. The hearts from five dogs in which fatal anemic hypoxia was produced by severing major arteries were unremarkable in three cases; while one showed granulomatous foci and eosinophil infiltration suggesting incidental infestation and one showed some vacuolization of muscle fibers. Except for the incidental observation of granulomatous foci in the one heart, changes found in this series may be regarded as agonal. Congestion was seen in 2 of 13 hearts.

The pathologic effects of ventricular fibrillation during ether anesthesia were studied in 16 dogs. After plane 2 surgical anesthesia was obtained, the chests of these animals were opened at the fourth left intercostal space and ventricular fibrillation was produced by stimulating the ventricles with 4 volts alternating current through the intact pericardium. In four animals the state of fibrillation was allowed to persist for one minute; at the end of this interval the heart was quickly excised. Periods of fibrillation of 2, 5, 10 and 20 minutes were permitted in three animals each. Gross inspection of these hearts revealed no abnormalities, while microscopic damage was found in nine cases. The pathologic findings consisted chiefly in fragmentation of muscle fibers, engorgement of blood vessels and disseminated microscopic hemorrhages. These changes were present both at the area of application of the fibrillating shock and at distant sites in varying degree. In seven hearts no morphologic changes were found, and the presence of calcified granulomata in one was incidental. The changes were absent or minimal in animals which were sacrificed after five minutes or less of fibrillation, while the hearts in which fibrillation had persisted for 10 or 20 minutes uniformly showed more pronounced changes. In the hearts of two of the animals subjected to 20-minute experiments subepicardial necrosis of muscle and fat was also seen. The fact that the severity of change increased in relation to the duration of fibrillation leaves little doubt that fibrillation itself, and/or the accompanying hypoxia, was responsible for the congestion, hemorrhage, muscle fragmentation and focal subepicardial necrosis.

Since in the experiments involving defibrillation and resuscitation repetitive manual cardiac compression at a rate of 25 to 35 compressions per minute was uniformly applied, the effects of compression by itself, and superimposed on ventricular fibrillation, were studied in 13 animals. In four dogs, fibrillation was induced, artificial respiration was instituted, and manual compression was applied for three minutes. Two of the hearts revealed no morphologic change; a third showed vascular engorgement and disseminated focal hemorrhages; and the fourth displayed subepicardial muscle necrosis. Four hearts were subjected to manual compression for three minutes after ether anesthesia was deepened to a fatal level. In two of these no change was observed; while focal subepicardial and myocardial hemorrhages were present in one, and focal myocardial hemorrhage and necrosis were observed in another. In addition manual compression and artificial respiration were carried out for a period of 10 minutes on five normally beating hearts; three of the hearts were excised immediately following the procedure, while two were excised after the animals had subsequently survived for a period
of two weeks. The normally beating hearts which were excised immediately after compression revealed minimal damage consisting in slight fragmentation of muscle fibers; occasional fragmentation of muscle was observed also in the hearts of the surviving animals indicating that this change was not related to compression itself.

The effects of alternating current countershock defibrillation and subsequent resuscitative procedures were studied in 18 dogs. Fibrillation was induced and allowed to persist for two minutes, manual compression and artificial respiration were then carried out for a period of three minutes. Several alternating current volleys* were then applied. Compression was resumed following these volleys. If an orderly heart beat did not return, further countershock was employed. These procedures were repeated as necessary until defibrillation and restoration of cardiac function were obtained. The range of alternating current voltage was 100 to 300 volts applied by means of self-retaining electrodes with stainless steel plates 35 to 47 mm. in diameter. In some instances the electrode surfaces were covered by saline-soaked gauze pads while in others the bare metal electrodes were used. The total time of fibrillation and compression and the number of countershock volleys required for restoration of cardiac function varied widely. Fibrillation time varied from 5 to 30 minutes; the number of countershock volleys ranged from one to as many as 65. Eleven animals survived from 1 to 15 days following the experiment,† six of the dogs survived less than 24 hours, and one was not defibrillated.

In spite of the wide variation in the experimental procedures, all the animals in this group showed a grossly detectable burn at the site of electrode application and various degrees of microscopic morphologic cardiac change which fell into two principal categories: (a) vascular disorders, and (b) regressive changes in muscle fibers accompanied by reactive phenomena. These changes varied quantita-

* By “volley” is meant a stimulus consisting of a series of spikes.
† These animals were sacrificed intentionally at the end of the survival period.

tively according to length of survival of the animals.

The vascular disorders were observed in the animals which died, or were sacrificed, early in the experiment. These changes were seen both at the area of application of the electrodes and at distant areas. They consisted in diffuse vascular engorgement, focal perivascular hemorrhage, and edema extending among the muscle fibers. This alteration, with variation in intensity, was quite similar to that seen in the animals in which ventricular fibrillation was the essential experimental procedure.

In contrast to the vascular disorders which were diffuse, the regressive changes and associated reactive phenomena were localized at and near the area of application of the electrodes. Both epicardium and myocardium were involved. The epicardial changes consisted chiefly in the loss of cellular lining from the serosal surface, often accompanied by subepicardial fat necrosis. Fibrinous hemorrhagic exudate with widespread granulocyte infiltration was present in one animal which died three hours after defibrillation, while in animals which were sacrificed later in the experiment fibroplastic tissue proliferation, progressively poorer in cells and richer in fibers, became apparent. Changes in the underlying myocardium extended to a depth of 2 to 3 mm. and were characterized by vacuolization of muscle fibers with frequent loss of striations and outlines and fusion of adjacent fibers into blocks of amorphous eosinophilic material as a result of necrosis. There was concurrent inflammatory reaction with a predominance of polymorphonuclear leukocytes in the early phases of the process and of connective tissue cells and fibers in the later stages. The cardiac tissue change was fundamentally the same regardless of the type of electrode used. The formation of pericardial adhesions at the area of electrode application and a moderate degree of superficial myocardial fibrosis are the expected end results of this damage; but these changes were not sufficiently extensive to warrant the anticipation of appreciable alteration of cardiac function.

Defibrillation by the application of con-
Fig. 1. (A) Perivascular edema extending between muscle fibers, from an animal subjected to fifty 300-volt alternating current volleys. Defibrillation was not achieved. (B) Fragmentation of muscle fibers, minimal granulocytic cell infiltration and tissue edema, from an animal which was defibrillated by ten 250-volt alternating current volleys. The dog survived for 12 hours. (C) Fibrinous epicarditis with moderate granulocyte infiltration, from a heart subjected to one 300-volt alternating current volley. The animal survived for one day. (D) Early thrombus in a subepicardial vein. Defibrillation was accomplished with one alternating current volley. The animal survived one day. (E) Interstitial myocardial edema and granulocyte infiltration in an area of muscle necrosis. The animal received thirty alternating current volleys at 300 volts and survived two days. (F) Focal myocardial necrosis with calcification and inflammatory reaction. Defibrillation resulted after ten 100-volt alternating current volleys; three and one half days of survival were allowed. (G) Epicardial fibroblastic proliferation extending into the myocardium which shows necrosis, interstitial edema and hemorrhage; from an animal which survived for five days after defibrillation by ten alternating current volleys at 300 volts. (H) Subepicardial fat necrosis with mesenchymal cell infiltration. The heart was defibrillated by a single 300-volt alternating current volley and the animal was sacrificed at 15 days.
denser-discharge countershock was carried out in the same way as by the alternating current countershock. It should be noted, however, that the condenser-discharge shock is a single spike in contrast to the alternating current volley which is a series of spikes. Twenty-two animals were included in the studies of condenser-discharge countershock. Defibrillation was accomplished 6 to 34 minutes after fibrillation was induced. From 1 to 14 condenser discharges at 1500 volts were required to restore an orderly heart beat. Unpadded electrodes were used in this series of animals. The survival period varied from 12 hours to 6 days, most of the animals being sacrificed at the second or third day. In contrast to the hearts of animals in which alternating current countershock was used, only one heart of the dogs which received condenser-discharge countershock revealed a grossly detectable burn. Epicardial and myocardial damage were seen, however, in microscopic sections. The damage, once more, may be placed in two classes, vascular disorders and regressive changes. The former again were characteristically diffuse, consisting in dilatation and engorgement of blood channels, perivascular edema and hemorrhage; these changes were seen in the animals which died within 24 hours after defibrillation. In one instance early thrombosis of a middle-sized epicardial vein was observed, and in five hearts recent thrombi in the mural endocardium of the left ventricle were present. At the area of application of the electrodes the epicardium showed varied patterns of inflammatory reaction, chiefly fibrinous hemorrhagic exudation in animals sacrificed early in the experiment and fibroblastic tissue proliferation in those allowed longer survival periods. Less severe and less extensive epicardial damage was apparently produced by the condenser discharge technique than by alternating current countershock. The regressive changes in the myocardium were likewise less severe, consisting in minimal muscle necrosis with occasional reactive phenomena and deposition of lime salts after longer periods of survival. In 6 of these 22 animals, neither epicardial nor myocardial lesions could be found by gross or microscopic observation.

Illustrations in figure 1 show some of the characteristic pathological changes observed in these hearts.

Conclusions

1. The pathologic effects on canine heart of fibrillation, countershock and manual compression have been studied. Survival experiments have permitted evaluation of the permanence of the damage produced.

2. Ventricular fibrillation produced diffuse vascular congestion, focal microscopic hemorrhage, edema and muscle fragmentation.

3. Massage of the normally beating heart produced minimal nonspecific damage. When the fibrillating heart was subjected to manual compression, the changes were somewhat more severe than those observed after fibrillation alone, and occasional focal myocardial necrosis resulted.

4. Alternating-current countershock produced both epicardial and myocardial damage at the site of application of the electrodes. The epicardial change was manifested initially as fibrinous hemorrhagic exudation and subepicardial fat necrosis, ultimately as fibrous tissue formation and pericardial adhesions. The myocardium revealed first necrosis, fragmentation and inflammatory reaction, and finally superficial fibrosis. In view of their localization, these changes would not be expected to alter cardiac function appreciably.

5. In the animals subjected to condenser-discharge countershock, the observed effect was of the same type as that produced by alternating-current defibrillation. Myocardial damage was not apparent in all these animals, however; nor was it as severe as that produced by alternating current. Further investigation would be required to determine the relationship between condenser-discharge countershock and the presence of mural thrombi in the left ventricle of 5 of these 22 animals.

6. The morphologic change resulting from alternating-current countershock was not apparently influenced by the type of electrode surface. Padding of the electrodes with saline-
soaked gauze did not appear to influence the degree of damage.

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SUMARIO Español

1. Los efectos patológicos de la fibrilación, contra choque y compresión manual en el corazón canino, han sido estudiados. Experimentos de supervivencia han permitido la evaluación de la permanencia del daño producido.

2. Fibrilación ventricular produjo congestión vascular difusa, hemorragia focal microscópica, edema y fragmentación muscular.

3. Masaje del corazón normal pulsante produjo cambios mínimos no específicos. Cuando el corazón fibrilante fué sujeto a compresión manual, los cambios fueron algo más severos que los observados luego de fibrilación solamente y ocasionalmente resultó en necrosis focal del miocardio.

4. Contra choque con corriente alternante produjo daño al epicardio y miocardio en el sitio de la aplicación de los electrodos. El cambio en el epicardio se manifestó inicialmente como un exudado fibrinoso hemorrágico y necrosis de grasa subepicardial, finalmente como formación de tejido fibroso y adherencias del pericardio. El miocardio reveló principalmente necrosis, fragmentación y reacción inflamatoria y finalmente fibrosis superficial.

En vista de su localización, estos cambios no se esperarían que alterasen la función cardíaca apreciablemente.

5. En los animales sujetos a contra choque por descarga de condensador, el efecto observado fue del mismo tipo como el producido por la corriente alternante desfibriladora. Daño al miocardio no estuvo aparente en estos animales, sin embargo; ni tampoco fue tan severo como el producido por la corriente alternante. Más investigación sería requerida para determinar la relación entre contra choque por descarga de condensador y la presencia de trombos murales en el ventrículo izquierdo en 5 de 22 de estos pacientes.

6. Los cambios morfológicos resultantes del contra choque por corriente alternante no fueron aparentemente influenciados por el tipo de superficie de electrodo. Empaquetamiento de los electrodos con gasa mojada en solución salina aparentemente no influenció el grado de daño.

REFERENCES
