University of Nebraska Medical Center
BREAKTHROUGHS FOR LIFE

University of Nebraska Medical Center
DigitalCommons@UNMC

MD Theses

Special Collections

1964

Cardioversion : its development, uses and techniques

Ralph J. Stearman
University of Nebraska Medical Center

This manuscript is historical in nature and may not reflect current medical research and practice. Search PubMed for current research.

Follow this and additional works at: https://digitalcommons.unmc.edu/mdtheses

 Part of the Medical Education Commons

Recommended Citation

Stearman, Ralph J., "Cardioversion : its development, uses and techniques" (1964). *MD Theses*. 60.
https://digitalcommons.unmc.edu/mdtheses/60

This Thesis is brought to you for free and open access by the Special Collections at DigitalCommons@UNMC. It has been accepted for inclusion in MD Theses by an authorized administrator of DigitalCommons@UNMC. For more information, please contact digitalcommons@unmc.edu.

CARDIOVERSION
IT'S DEVELOPMENT, USES, AND TECHNIQUES

Ralph J. Stearman

Submitted in Partial Fulfillment for the Degree of
Doctor of Medicine

College of Medicine, University of Nebraska

February 3, 1964

Omaha, Nebraska

CONTENTS

Introduction 1
Theory of Cardioversion. 2
AC Cardioversion 3
The Vulnerable Phase 7
Known Causes of Ventricular Fibrillation 8
Pathology in Hearts Following Cardioversion. . . . 8
Closed Chest Massage 10
DC Cardioversion 10-20
Instrument for DC Discharge. 16
Indications 17
Anesthesia. 17
Premedication 18
Site of Electrode Application 18
Electrode Material. 19
Electrode Size. 19
Requirements of an Ideal Defibrillator 20
Cardiac Mechanisms Following Countershock. 20
Experiment 22
Summary. 24
Conclusions. 26
Acknowledgment 27
Bibliography

500951

Introduction

Cardioversion represents a milestone in Medical management. For years medicine has sought some method of effective control of ventricular fibrillation and pre-fibrillatory arrhythmias. It has been over 40 years since the introduction of quinidine. During this time, quinidine has been a valuable drug, along with procaineamide and digitalis, in controlling various arrhythmias. They have, unfortunately, been of no value in the treatment of ventricular fibrillation. The major disadvantage of drug therapy lies in its necessity to titrate, which requires valuable time and introduces the possibility of toxicity.

DC cardioversion offers a new, effective, safe, simple tool for the treatment of ventricular fibrillation and ectopic arrhythmias. Discussed in this thesis will be the evolution of the method, the advantages the method offers, and the present day techniques employed as well as results which have so far been forthcoming.

Theory of Cardioversion

In 1940 Wiggers[46-49] laid down the fundamental principles which have been considered to be the basis for the theory of cardioversion as practiced today. In effect, there are three principles which must be fulfilled if defibrillation or reversion of an ectopic tachycardia is to be accomplished. They are:

(1) Every trace of fibrillation must be abolished. If any fibrillating muscle remains, the heart will not be defibrillated. This means one must have electrodes of proper size and sufficient current. Lown[33] states it in another way. "All fibers of the heart are depolarized at once, extinguishing the ectopic focus. Then normal pacemaker may resume."

(2) The natural pacemaker of the heart must survive the electrical discharge.

(3) Reasonably vigorous contractions must ensue after defibrillation.

AC Cardioversion

The first work done on the conversion of ventricular fibrillation to a normal sinus rhythm was done in 1899 by Prevost and Batelli. In 1936, Ferris reported that in 60% of experimental animals, almost any countershock would arrest fibrillation.[33] In the early 1930's, Kouwenhoven, Hooker and Langworthy established early information about electrical defibrillation. They used 1.5 to 2.0 Amps at 120-130 volts. Paddles were applied directly to the heart in these experimental animals.[22-26] It was in 1936 that Wiggers developed the concept of open chest cardiac massage as a step in the treatment of ventricular arrest which might occur during the course of experiments on various animals. This was later developed more fully by Kouwenhoven who demonstrated that ventricular massage was possible through the closed chest.[26]

In 1940 Wiggers and Wegria[46-50] suggested defibrillation by means of 3-7 serial discharges at one second intervals. Approximately two (2) Amps would be delivered at each discharge. The former practice had been to use 25-30 Amps in one discharge. This, they claimed, defibrillated the cardiac muscle in small units making the sections of fibrillating muscle coarser and coarser until finally a final discharge in the series would depolarize the whole mass simultaneously and the whole muscle would again beat as a unit. Throughout the 1930's and early 1940's there were sporadic reports of work in AC cardioversion but all reports showed a high percentage of failures and dangerous side effects even in experimental animals. For these reasons, no one was willing to try such a dubious method on humans. Finally in 1947, Beck, Pritchard, and Feil[4] reported the first successful electrical defibrillation of a human heart. In this article they reported the use of AC defibrillation attempts in 6 humans in extremis. In all

cases the use seemed justified inasmuch as all these patients were very near death and this seemed the only possible method for saving these individuals, and certainly nothing could be lost by trying. Of the 6 patients, the first 5 expired. Finally, however, a 14 year old boy had begun to fibrillate during a thoracic operation. As the chest was already opened, there was no difficulty in applying the electrodes and administering the shock. One hundred ten volt AC current was used at 1.5 Amps. Large electrodes were used and the interval between two serial shocks was 0.1-0.5 seconds.

The first shock produced no effect. The heart continued to fibrillate. The second shock put the heart in standstill. Aftera few seconds, feeble but regular contractions were established and the blood pressure began to rise. The patient recovered completely with no after effects.

In 1951, Guyton and Satterfield[18] and Mackay and Leeds[36] found that serial shocks were more effective in defibrillation of experimental animals than were single shocks. Mackay and Leeds[36] also demonstrated the need for 3-5 times as much current for closed as for open chest defibrillation.

Some work was done on the effectiveness of capacitor discharge, but results in this field were not nearly as good as those obtainable with AC shock. In view of the results obtained later with DC discharge by Lown, this is difficult to explain.

Kouwenhoven, in 1954[28] in testing various capacitor discharges, reports results which in all instances were worse than results obtainable with AC. He stated that the difference was especially noticeable if the heart had been in fibrillation for greater than 30 seconds. Beyond this time no capacitor discharge of any setting would produce defibrillation. AC was then used to rescue these animals. In 1957, after further work with capacitors, Kouwenhoven did report that defibrillation was

possible, but that bulky equipment was needed which, from a practical standpoint, was not feasible. Serial defibrillation was not possible because of the time required for the capacitors to acquire a charge. He also reported that prohibitively high voltage was necessary.[29] Zoll and co-workers[51-55] from 1952 through 1960, contributed a considerable amount of literature relating to AC defibrillation. In 1956, these men demonstrated the effectiveness of AC discharge in transthoracic defibrillation of a human heart.[53] With their instrument, which delivered 60 cps AC for 0.15 seconds at 180-720 volts, they terminated atrial, nodal and ventricular tachycardias as well as ventricular fibrillations. Frequently, multiple shocks were required to achieve defibrillation. Deaths were common following the use of AC countershock but since many of the patients were in extremis, it was felt the fault was probably not in the cardioverter.

By 1956, the use of defibrillators using AC current was common in hospitals and recommended. The usual machine delivered from 200-700 volts. It was equipped with large copper electrodes used across the chest and delivered its current for a duration of about 0.15 seconds.

In an editorial in the British Medical Journal[11] in 1956, successful use of the Zoll machine was reported in four (4) patients. Reports on patient use were slow in coming in at first because of the scarcity of patients and the severe restrictions of the indications for its employment. These restrictions were proper because of the relative unknown nature of the instrument and the few data which had been collected. As an example of this, in 1956 Zoll[53] et al. reported the use of the AC defibrillator in 11 occasions in 4 patients. Only one of these patients lived for more than five days following defibrillation. The only patient who lived was defibrillated three (3) times and lived for three (3) months.

Other results from Kortz and Swan[24] were also poor with respect to survivals. In their experiment, 30 animals underwent hypothermia and thoractomy and ventriculotomy. Of these 30 animals, 16 fibrillated. Of these, 15 were defibrillated with a Zoll type machine. Of these 15, 11 died in one week. Such results certainly were not good enough to make clinicians inclined to use this type of apparatus on their patients.

In 1957, Kouwenhoven[29] used 60 cps AC, using 5 amps for 0.25 seconds. Of the 107 total attempts at defibrillation in 72 animals, 42 animals survived. Here, again, we see almost two-thirds of the experimental animals dying. Experiments beginning in the early 1930's and continuing up to the present, all show a high mortality rate following the use of AC for defibrillation.

In Zoll's 1960 report, he states that of 8 patients treated 532 times with AC, 5 survived.[55] The survival times ranged from 19 hours to 2½ years, but the mortality is significantly almost 50%. Again the usual 60 cps current for 0.15 seconds from 150-450 volts was used. There have been reported successful cases of defibrillation without side effects using 60 cps current for 0.15 seconds, but they are in the minority. Crehan et.al.[9] reports a successful cardioversion of ventricular fibrillation with a Zoll machine in 1962.

Alexander, Kleiger and Lown in 1961, used a Zoll type machine on a patient in standstill. He improved initially, then went downhill and started fibrillating again three days later. The machine was used again and it returned the patient to a normal sinus rhythm which persisted.

AC continued to be used throughout the 1950's, mainly because there was nothing else that could be used or that was available which could produce a better record of successes. Occasionally, at least, AC was successful in saving a life and therefore warranted being kept around for occasional use. It was not until DC cardioversion was introduced, that electrical

conversion of all types of arrhythmias became feasible.

Vulnerable Phase

In 1939, Wiggers and Wegria[50] found that only electrical shocks which were applied during the "refractory" period of the cardiac cycle would produce ventricular fibrillation. This was defined by Wiggers at that time, as being "late systole." No attempt was made to accurately delineate the time boundaries of this period.

No further work was published on the location and duration of this suprasensitive period, until Lown et.al.[5] worked out the exact location of the vulnerable period.

Using capacitor discharge, lasting 2.5 milliseconds, Lown systematically explored the cardiac cycle for the position of the vulnerable period. Two periods of sensitivity were discovered. The first was found to last about 30 milliseconds and began just before the peak of the T wave of the EKG. Any electric shock during this period would likely produce ventricular fibrillation.

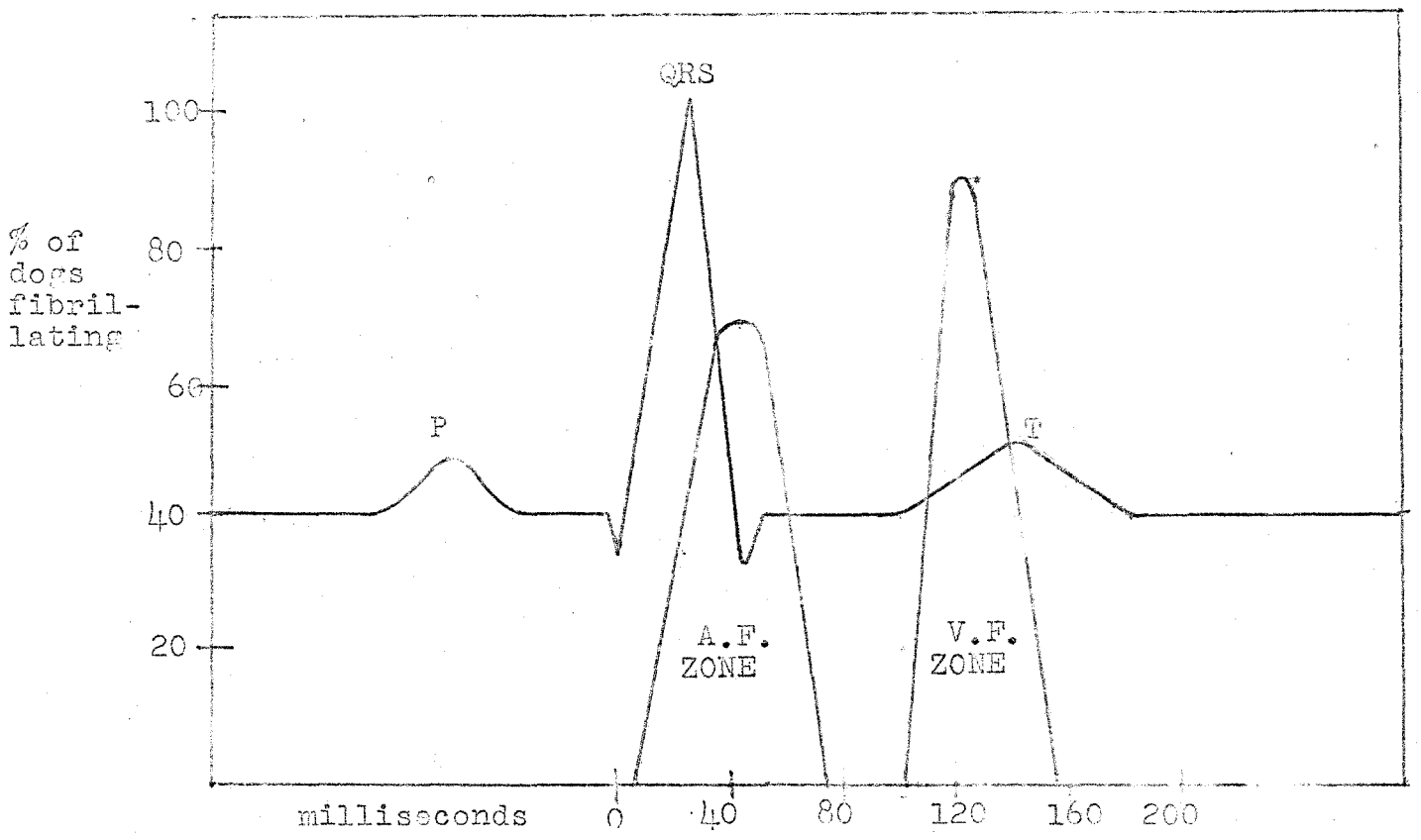
The second vulnerable period was located just after the peak of the R wave. Shocks delivered during this time would likely produce atrial fibrillation.

No difference has been found in the vulnerable period if the shock is given extra or intra thoracically. The energy required to produce fibrillation varies from 0.5 to 10 Watt-seconds. See Diagram I.

Wiggers;[50] found that ventricular fibrillation occured in approximately 2% of experimental animals, following random DC shocks. This figure was considerably too high to allow a true clinical test of defibrillation

Lown, et al[33] have invented a synchronizing device which automatically triggers a DC discharge 5 milliseconds after the peak of the R wave. This prevents the discharge of the current during a vulnerable period.

In 1955, Hoffman, etal[19] demonstrated that shocks given



Electrical discharges given during these zones (Here shown with respect to the cardiac cycle) will produce atrial and ventricular fibrillation respectively.

Diagram I

outside of the vulnerable period, elicit only extra systoles but not ventricular fibrillation. His study also showed that the relative refractory period is the only time one can produce ventricular fibrillation with an electric shock.

Known Causes of Ventricular Fibrillation[37]

(1) Old myocardial infarctions and coronary occlusion
(2) Hypothermia
(3) The post hypercapnic state
(4) Electrocution
(5) Drowning in fresh water
(6) Cyclopropane and epinephrine when administered simultaneously
(7) An uncommon toxic reaction to Digitalis, Quinidine, and Procaineamide
(8) A rare complication of cardiac catheterization
(9) Hooker, et al[20] found that large currents do not produce fibrillation, but rather initiate an asystole which is temporary. (e.g. 2-5 seconds after the discharge, the beat is picked up again) Ventricular fibrillation was not produced in dogs using 2,200 volts for from $\frac{1}{2}$ to 5 seconds, but was produced with 110 volt, 0.8 amp current, open heart.

Bellet[5] states that a current just below threshold, about 0.1 amp, for 1 second is about all one should impose on a human subject.

Pathology in Hearts Following Cardioversion

In 1954, Tedeschi[44] published an article which was the first major morphological study performed on hearts which had been subjected to cardioversion. It is interesting to note that he observed burns in all animals receiving AC countershock. These injuries were related in degree, to the voltage and number of shocks received by the heart and consisted of coagulative

necrosis and subepicardial hemorrhage. Only one of twenty-two dogs had changes in the heart when shocked with capacitor discharge.

No damage was found when only cardiac massage had been performed. Padding electrodes with saline sponges apparently did not change the degree of necrosis.

Lape and Maison[30] had observed myocardial burns after the direct application of AC discharge, but not after defibrillation with single capacitor discharge. In 1957, Kaiser, et al[23] noted burns on hearts with voltages in excess of 230 volts AC. Kortz ans Swan[22] noted at this time, that hearts would be seared if bare electrodes were used, but not if the defibrillating electrodes were padded with saline soaked sponges. This information is in direct disagreement with information presented above by Tedeschi.[44]

In 1932, Kouwenhoven observed that hearts were damaged to some extent by electric current and that the damage was confined to that area of the body in which the current density was higher.

In 1951, Guyton and Satterfield[18] reported that electric currents passing through a heart definitely increased its temperature and weakened it. MacLean and van Tyn[37] were more quantitative in the efforts and discovered that myocardial temperatures rose as high as 149° F (65°C) when voltages in excess of 170 volts AC were used in serial shock. They also found 100-170 volt AC shocks were safe if the time was under 0.15 seconds.

Mackay[36] observed no spinal cord effects in dogs following cardioversion.

These results indicate that heart pathology will result with AC discharge used in greater than 230 volts. Temperature rises in myocardial tissue have been excessive with voltages greater than 170.

DC discharge apparently does not result in the damage to the myocardium that is accociated with AC.

Closed Chest Massage

In cardiac arrest, from whatever cause, a cardioverter may be used to restore normal sinus rhythm and maintain the circulation.

The use of a cardioverter is of no value, however, if cardiac anoxia has persisted for too long a time (e.g. greater than 3 minutes 20, 26, 27). Because of this, a method of maintaining the circulation for a limited period of time, (until a cardioverter could be obtained) was developed. This is a closed chest method of massage in which the operator places his hands on the sternum and approximately 60 times per minute, leans on the chest of the prone patient and applies firm, vertical pressure. The sternum should move 3-4 cm. with each push. With this method, mean circulatory pressure of from 60-100 mm Hg can be maintained up to 65 minutes.

The heart is limited in its motion; anteriorly, by the sternum; posteriorly, by the vertebral bodies; and laterally, by the pericardium. Thus, cyclic pressure on the sternum will produce a pump like action. If this above method is used along with mouth to mouth respiration, it is possible to transport a patient in arrest to a location where a cardioverter is available.

DC Cardioversion

The first work on electrical defibrillation was done by Prevost and Batelli in France in 1899. In their experiments, both AC and capacitor discharge were tested and found effective in defibrillating hearts. Other subsequent work in the United States by Kouwenhoven and Milnor[28] and Guyton and Satterfield[18] showed results which were at best, inconsistent. Kouwenhoven[28] tested defibrillators with capacitors ranging from 25 to 250 microfarads charged to voltages from 450 to 4,000 volts with various values of resistance and inductance in the circuit. Successful defibrillation with any type was considerably less than with only one application of AC. The difference was

especially noticeable of fibrillation has lasted longer than 30 seconds, in which case capacitor discharge could never produce a successful defibrillation. AC current could, however, defibrillate up to 14 minutes following beginning of fibrillation.

These early results, although inconclusive, tended to discourage workers in DC cardioversion because there seemed to be no clinical future for DC discharge.

In 1954, Kouenhoven et al[28] experimented with open and closed chest defibrillation in dogs. Fibrillation was produced with varying doses of current and then the dogs were given DC shocks. If the first shock failed to defibrillate the animal, then subsequent shocks would be correspondingly higher until defibrillation was produced.

Results in this series of experiments showed 32 defibrillations of 55 attempts in the open chest preparation. With the chest closed, there were 13 recoveries and 22 failures. These results indicated that DC had little future in cardioversion. The experiment also showed that of 119 successive discharges delivered with respect to the cardiac cycle, 18 ventricular fibrillations resulted. There have been several which have been more successful since Kouwenhoven's experiments in 1954.

It was probably because of these results that further experimental work on capacitor discharge was discouraged.

In 1962, Bernard Lown and co-workers published two papers which appeared within several month of each other. Both of these articles delt with DC instead of AC cardioversion. Both articles contained results of experiments with DC which indicated that it had more clinical potential than anyone had heretofore suspected. The results of his experiments will be discussed in some detail because of their important effects in this field.

The first article to be discussed dealt with ventricular fibrillation, which was the initially intended use of cardioversion.

It had been, prior to this article, that AC countershock

was the only treatment for ventricular fibrillation and this was only rarely administered. This procedure was usually performed in the operating room. If a patient could not be readily transported to cardioverting equipment in time, he simply died.

With the advent of closed chest cardiac massage in 1960 by Kouwenhoven,[26] it became possible to combine closed chest massage with mouth to mouth respiration and thereby maintain blood pressure and respiration while the patient was transported to cardioverting apparatus. This could be done by relatively untrained persons. For these reasons it became, according to Lown,[33] necessary to determine the optimum method of defibrillation. The following experiments have this as their objective.

In the first experiment, 65 dogs were divided into 3 groups. In the first group of 10 dogs, the minimal levels necessary to produce defibrillation 65% of the time, were found for AC andDDC. Results showed AC to require about 350 volts to revert 65% of the fibrillations produced. DC required 70 watt-seconds. Using AC current, 26% of defibrillation attempts showed subsequent ventricular tachycardia and 87% of the dogs showed atrial fibrillation. Transient ventricular and atrial arrhythmias were commonly observed. Currents of injury were frequently seen on the EKG. With the use of AC, atrial fibrillation was found in 174 of 200 reversions (87%). Following DC shock, the incidence of atrial fibrillation was only 0.9% or 5 episodes in 550 reversions. ST segment changes were found in 32% of DC and 50% of AC reversions.

A second group of 25 dogs received fixed levels of 350 volts AC and 70 Watt-seconds DC. Ten dogs were given AC, and of these, 5 died. Of 15 dogs receiving DC, none died.

A third group of 30 dogs were subjected to varying intensity levels of AC and DC. Ten dogs received 158 AC shocks from 25 to 750 volts. Ventricular fibrillation occured after 27% of the

shocks and 2 of the 10 dogs died. The incidence was inversely related to the voltage. i.e. 45% of shocks at 50 volts but only 13% of shocks at 750 volts caused ventricular fibrillation. These findings are consistant with earlier results by other workers.

Atrial fibrillation occurred after 80% of AC shocks and was found to be directly related to the voltage employed.

Of the 20 dogs receiving 458 DC shocks, ventricular fibrillation was observed in 6, for an incidence of 1.3%. The most frequent arrhythmia was ventricular tachycardia which was observed in 22% of DC shocks.

Comparison of above results indicates an almost overwhelming superiority of DC because of practically complete lack of side effects with the exception of ventricular tachycardia. We see, however, AC discharge producing atrial fibrillation on 80-87% of defibrillation attempts. One-fifth of dogs treated with AC, died but none died with DC. Ventricular fibrillation was observed with 27% of AC shock, but only 1.3% of DC attempts. These results would certainly indicate that DC must be more valuable than previously thought.

Lown's second major experiment involved the use of capacitor discharge in treatment of cardiac arrhythmias.[33] In this experiment, 12 patients received AC shock; 8 for ventricular tachycardia; 2 for ventricular arrhythmias; 1 for nodal tachycardia; and 1 for atrial fibrillation. Of these 12 patients, 4 suffered ventricular fibrillation following the use of AC, and additional shocks were necessary for defibrillation. One of these patients died. Without taking into account all the various nuances which could be pointed out regarding the health of these patients, it will be observed that the statistics here presented regarding use of AC in humans, correlates closely to results found in experimental animals.

Five patients with 9 episodes of ventricular tachycardia, were given DC shocks. Of these, all 9 reverted with one

synchronized shock.

These results were so encouraging that the DC cardioverter was tried on 10 patients with atrial fibrillation. Of these 10 patients, 8 reverted to a normal sinus rhythm. All had been refractory to quinidine management. One chronic atrial flutter was reverted to normal sinus rhythm, as well as two "double tachycardias."

These results would certainly seem to indicate that DC perhaps has some superiority over AC in cardioversion.

Almost a year elapsed before any further data were published comparing AC and DC results. In August1963, Lown et al[35], published an article relating the further use of DC cardioversion in the treatment of atrial fibrillation.

Case studies were reported of 65 episodes of atrial fibrillation observed in 50 patients. Of these, 58 were successfully reverted (89%). All but three of these patients had rheumatic heart disease with mitral stenosis or insufficiency. To date, the best reported percentage of cures with drug therapy (Quinidine) is about 47%.

In May 1963, at Atlantic City, Lown presented results of the use of cardioversion in the treatment of ectopic tachycardias.[34] Twenty-five episodes of ventricular tachycardia were successfully treated in 11 patients. None of these showed untoward effects. It can be seen that some of these patients must have reverted to their arrhythmia several times, therefore cardioversion does not necessarily offer a permanent solution to arrhythmia, but rather is of use in correction of an individual episode while drug therapy, used in conjunction, offers the best hope of maintaining permanent reversion.

Of 103 episodes of atrial fibrillation in 85 patients, 91% were successfully reverted. Quinidine had been tried in 38 of these patients andhad been successful only in 6 (15.8%).

Reversion to atrial fibrillation in these patients in reported as common. Again this suggests the need for a more permanent

method of maintenance. Probably best supplied by drug therapy.

In this study, Lown reports 15 patients with atrial flutter were all successfully reverted. Five episodes of supraventricular tachycardia were all reverted.

Treatment for ectopic tachycardias prior to 1962 consisted either of quinidine, procainamide, or a digitalis product. Reversion of an ectopic tachycardia using these drugs, is a time consuming and even dangerous process which is often unsuccessful. Of 611 cases reported by Thomson,[45] the death rate which he ascribes to quinidine alone was 3.3%. He further observed embolism episodes in 2.3% of 418 patients. Drug therapy as seen offers toxicity, a time consuming process, and intensive observation. Cardioversion is fast, safe, and simple. As mentioned, DC cardioversions main discrepancy is lack of permanency. Perhaps a combination of the best of both methods would supply patients needs, i.e. the safe rapidity of cardioversion and maintenance of drug therapy.

It should be noted that the failures of cardioversion in atrial fibrillation were in patients who had multivalvular disease or significant mitral valve regurgitation. Of 352 discharges required to achieve 89% success of atrial defibrillation, not one case of ventricular fibrillation, ventricular tachycardia or cardiac standstill resulted. Cardioversion then, shows better than 90% total success with the various forms of ectopic tachycardia, while drug therapy alone produces only a 47% success rate with considerable danger.

Another experiment was published by Lefemine et al[31] in 1962 in which AC and DC were compared under hypothermic-conditions. Such conditions are frequently found in open heart surgery where many hearts go into fibrillation. In this study, defibrillation was attempted 172 times in dogs with DC and 137 times with AC. The temperatures ran from 20 degrees to 37 degrees Centigrade. Failure of the AC unit was defined as inability to defibrillate with a series of three serial shocks of 1,000 volts each, delivered

for 0.15 seconds. DC failed to defibrillate the dogs twice in this study for an indicence of 1.2%, whereas AC failed 20 times for a failure incidence of 15%. It also happened that DC discharge restored a normal sinus rhythm in all 20 of the AC failures.

The average energy levels required for defibrillation were higher for AC than for DC and it was also found that DC discharge produced no temperature rise in the myocardium whereas AC discharge consistently produced a 1-2 degree rise in temperature.

Use in patients in open heart surgery hasshown DC consistently effective at temperatures from 24-34 degrees Centigrade.[31]

The Instrument for DC Discharge

The usual DC defibrillator available on the market today consists of four parts:

(1) A DC defibrillator which delivers a single, monophasic pulse of 2.5 milliseconds duration. Energy levels are available from 0-400 Watt-Seconds.

(2) A synchronizing device which permits discharge which will avoid the vulnerable period. In the American Optical device, the discharge is timed to deliver just after the peak of the R wave. To deliver the capacitor discharge at this period necessitates

(3) A cardiac monitor (EKG)

(4) An oscilloscope for direct observation of the type of arrhythmia being dealt with.

The discharge burst is administered by means of heavily insulated, large, electrode paddles. The chest is covered with conductive paste. (See Application of Electrodes.) The discharge is controlled by either an automatic control which delivers the current after the peak of the R wave (lead 2 is usually used) or, in the case of ventricular fibrillation where there is no R wave, the burst may be given manually at any desired point.

Generally, low levels of approximately 70 Watt-Seconds are

used at first, and increased progressively, as necessary, for reversion.

Indications for Cardioversion

The early indications for cardioversion were rather more serious than those which have evolved within the last two years. Zoll,[52] in 1956, described countershock as being indicated in any desperate or intolerable situation, e.g. when the patient is moribund, comatose or in advanced decomposition with no chance for extensive drug therapy.

In 1962, Master and Rosenfeld[39] describe the use of cardioversion only in "life threatening situations," i.e. ventricular tachycardia, ventricular fibrillation and acute arrhythmias due to digitalis toxicity. Master and Rosenfeld added a few limited situations to the indications for cardioversion and as experience with its uses and results has grown, the list of indications has expanded. At the present time, this device has been used with ventricular fibrillation, ventricular tachycardia, supraventricular tachycardia, paroxysmal atrial tachycardia with block, atrial flutter and atrial fibrillation. All of the above have been treated with beneficial results.

Cardioversion is not particularly effective in cases of recurring bouts of tachycardia. In cases like these, it may be used on the individual attack, but drug therapy should be used in conjunction, Bellet.[5]

Anesthesia Prior to Cardioversion

In early reports, patients were generally given a barbiturate for sedation one hour before the expected time of cardioversion. At the time of the cardioversion, thiopental was given IV which lasted 3-5 minutes. This was just enough time to attach the necessary leads and perform the cardioversion. Recently, Stock[8] indicated that thiopental may not be necessary with additional mortality attendant upon general anesthesia.

In his patients, he has been administering meperidine prior to cardioversion and believes no thiopental is necessary. His patients have been questioned about a sensation which accompanies cardioversion, and they report it as not being entirely painful even though there is considerable muscle spasm associated with the procedure. Some patients have undergone the procedure a second time with only meperidine as analgesia.

Premedication

It is now being generally accepted that the use of quinidine prophylactically, before the attempted cardioversion of ectopic tachycardias, is valuable.

There are difficulties which attend the use of quinidine, however, which are not found with cardioversion:

(1) The effective dose of quinidine cannot be accurately predicted.

(2) Titration is an essential procedure which is time consuming. In the acutely ill patient, this time lag may be sufficient to be fatal.

(3) There are dangers of toxicity with quinidine which are not minimal.

Even with these difficulties for patients in atrial fibrillation, 300 mgms of quinidine is given 2 hours before cardioversion.[35]

Site of Electrode Application

Placement of electrodes should obviously be of such advantage as to allow the passage of the must current through the myocardium. Kouwenhoven, et al[25] in 1932, in experiments on dogs, found that in using transverse thoracic leads, the myocardium received only 3% of the total current provided, whereas with longitudinally placed leads, the myocardium received 7.8% of the total provided dose. In the later study, Kouwenhoven showed that 8-12 amps were required for defibrillation using transverse thoracic electrodes, whereas only 3.5 to 6.7 amps were needed for longi-

tudinal electrodes.[29] These findings would seem to indicate that a longitudunal placement of electrodes would defibrillate with less, total current.

The only difficulty with a longitudinal placement of electrodes is that a considerable amount of tissue other than the myocardium is supplied with current.

The presently accepted method of electrode application, on humans, is something of a compromise of both methods. Electrodes are placed at the second right intercostal space and in the midaxillary line at the level of the fifth intercostal space on the left.[33]

Material in the Electrodes

Zoll has used a round copper electrode 7.5 cm in diameter and held to the chest with an encircling band.[54] Kouwenhoven has used 4" x 6" soft, sheet copper electrodes.[29] Lately, conductive jelly has been used in conjunction with the paddles.[33]

Electrode Size

The size of electrodes necessary or optimum in defibrillation was only of moderate interest to early workers and small mention is made in early literature. Guyton and Satterfield[18] were the first to accurately describe the value of electrode size on defibrillation.[20]

Using 110 volt, AC current, they found that in using 8 cm diameter electrodes, they could stop ventricular fibrillation practically at will. If electrodes 3 cm in diameter were used, 1 dog in 5 would not stop defibrillating. If electrodes less than 3 cm in diameter were used, the dogs could not be made to stop fibrillating at all. They also found that with large electrodes, less total voltage was needed.

McLean and van Tyn[37] discovered in 1961, that large electrodes were more advantageous than small ones. They state that to terminate fibrillation, it is necessary to depolarize

the entire heart at one and that such a thing is not possible with small electrodes. In the use of small paddles, both the voltage and current must be increased with resultant increased damage to the myocardium.

In their experiment, 20 dogs were subjected to artificial induction of ventricular fibrillation 115 times. Using the open chest defibrillatory technique, and 110 volts for 0.1 second, with paddles 5 centimeters in diameter, only 5 dogs failed to revert to a normal sinus rhythm. When paddles 7.6 centimeters in diameter were applied, even these previous 5 reverted to normal sinus rhythm without use of higher voltage. These authors also found that with small paddles, even single AC shocks produced dangerous temperature rise in the myocardium.

The above evidence then indicates a need for electrodes which are, optimally, at least as large as the myocardium.

Requirements of an Ideal Defibrillator

Lown set forth the requirements of an ideal defibrillator in 1962. "The ectopic mechanism should be controlled instantly and consistently without damage to the myocardium, impairment of conractility, or depression of normal pacemakers." Inasmuch as can be determined with the present available evidence, DC cardioversion seems to offer this.

Cardiac Mechanisms Following Countershock

Following the DC shock, which lasts approximately 2.5 milliseconds, the heart will be observed to be in a period of asystole for several seconds. (See accompanying record) Following this, the heart rate may revert directly to regular sinus rhythm. Frequently, nowever, a transient arrhythmia is found which may consist of multiply premature beats, coupled rhythm, and escape beats. These arrhythmias are transient as a rule and usually only persist for a few seconds up to a minute. With AC discharge, ST segment changes may be seen for several seeks. In most cases,

by 10-15 minutes following cardioversion, the patterns have returned to normal.

Following cardioversion, breathlessness, dyspnea, congestive phenomena, precordial pain or discomfort will disappear. Quantitatively, there is better oxygen saturation, a normal A-V difference, increased stroke volume and increased cardiac output.[5]

The chief hazard is, of course, ventricular fibrillation. With properly synchronized discharge (see Vulnerable Period) it is a fairly simple procedure to apply current outside these vulnerable periods.

Experimental evidence of the efficacy of the DC Cardioverter is presented as well as a typical example of cardiac response following countershock. See Diagram II.

Experiment

Object: Recent experimental evidence indicated that capacitor discharge is very effective in the treatment of ventricular fibrillation and ectopic tachycardias. This experiment attempts to verify these findings.

Material and Methods: A 9 kilogram dog in good condition was anesthetized with thiopental. Initial heart rate was 120 beats per minute. (See Strip I) The animal was put into fibrillation with 130 volt AC applied over 0.5 second, using the small tip electrodes across the shaved, intact chest. (See Strip II)

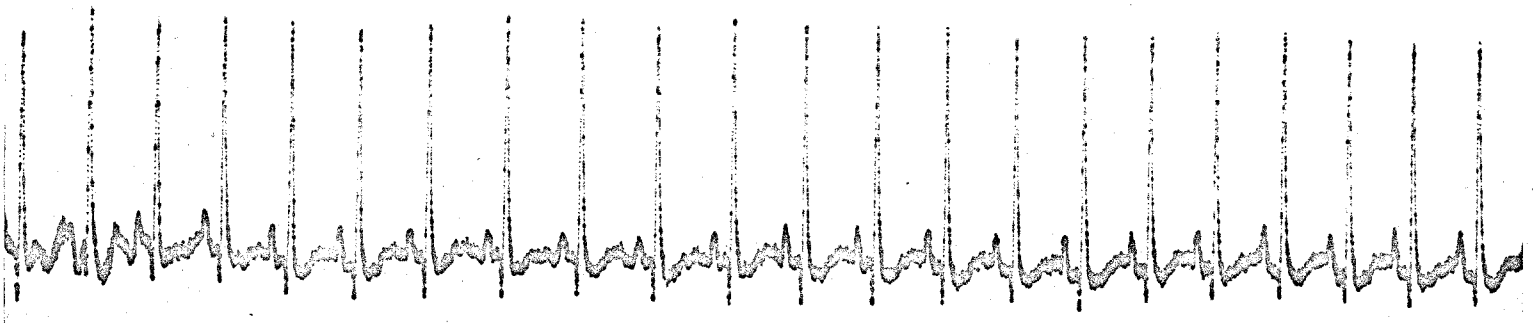
The instrument used for defibrillation is described on pages 16-17, and is the type apparatus used by Lown.

Results: The initial defibrillatory attempt was done at 170 Watt-Seconds for 2.5 milliseconds. Applications of the electrodes were transverse across the chest. Only one attempt was needed to defibrillate this animal. After application of the countershock, the recording oscilloscope deviated completely and no recording was made for approximately 0.1 seconds. (See Strip III) Following this, the heart activity showed multiphasic activity with no recognizable pattern for 4.0 seconds. (StrippIII) The heart then returned to normal sinus rhythm. In this part of the record, (Strip IV) we see negative displacement of the S-T segment, probably representing an injury vector. Heart rate at this time was about 60 beats per minute.

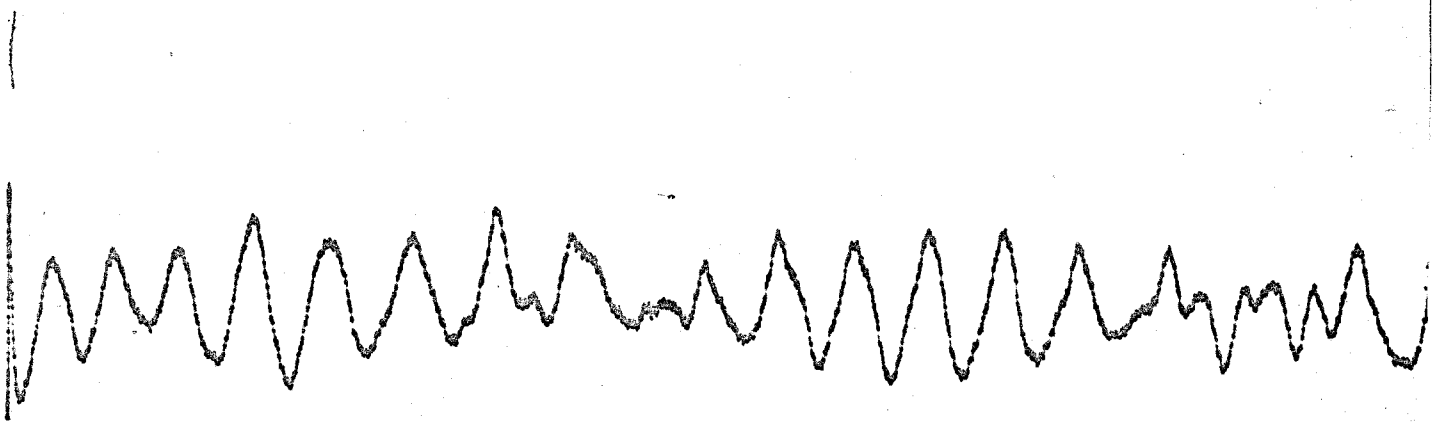
At this time, the animal's respirations began to slow and after approximately one minute, stopped. Artificial respiration was begun immediately. (See Strip V) The animal resumed control of his own respirations within two minutes. Following this, the heart began to show premature beats and normal beats, both appearing regularly. The heart appeared to be responding to both the normal sinus pacemaker and an ectopic focus. (Strip VI)

This arrhythmia lasted less than a minute. Following this, the heart returned to normal sinus rhythm, and remained as such. The animal showed no ill effects after the experiment.

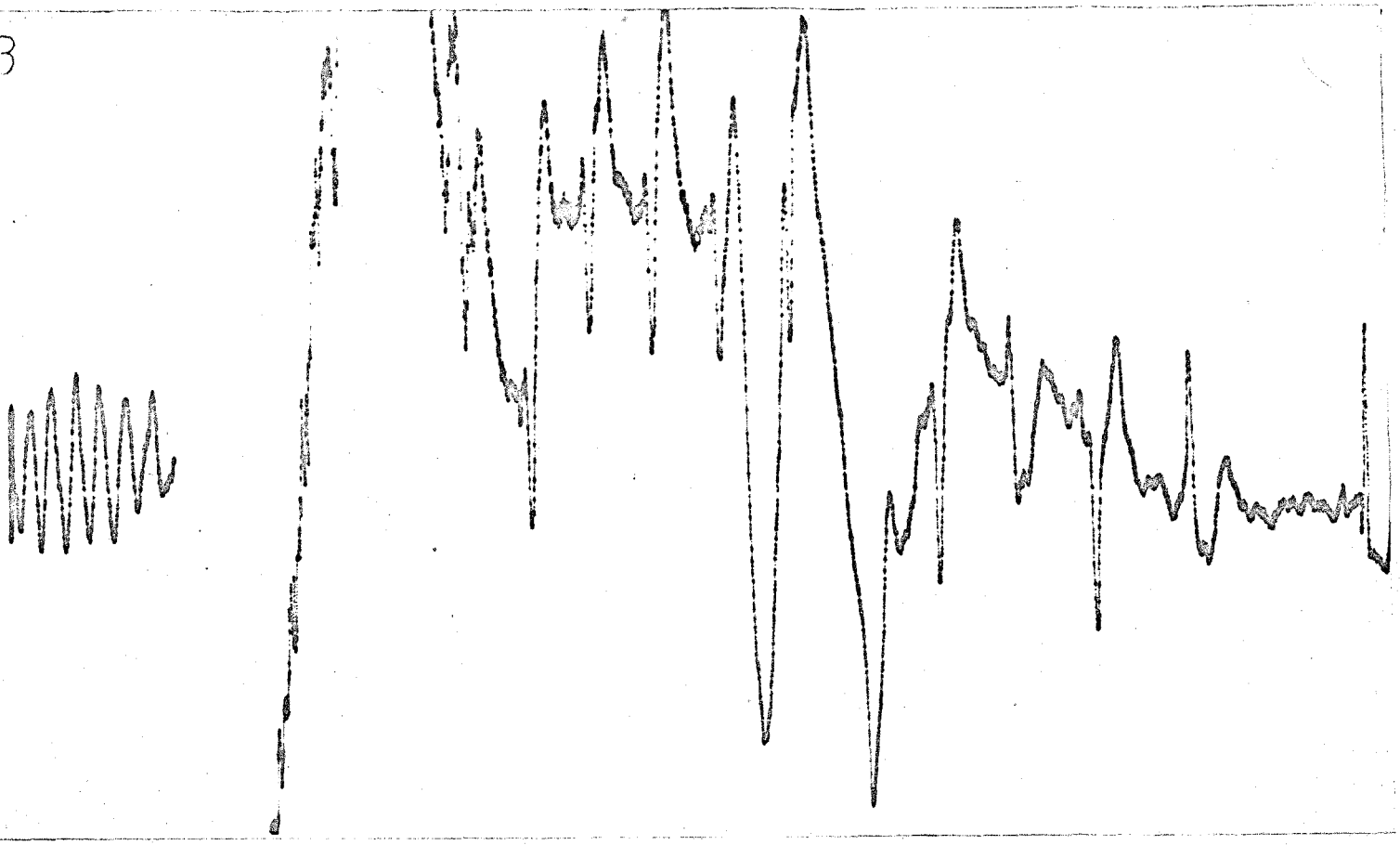
Strip I



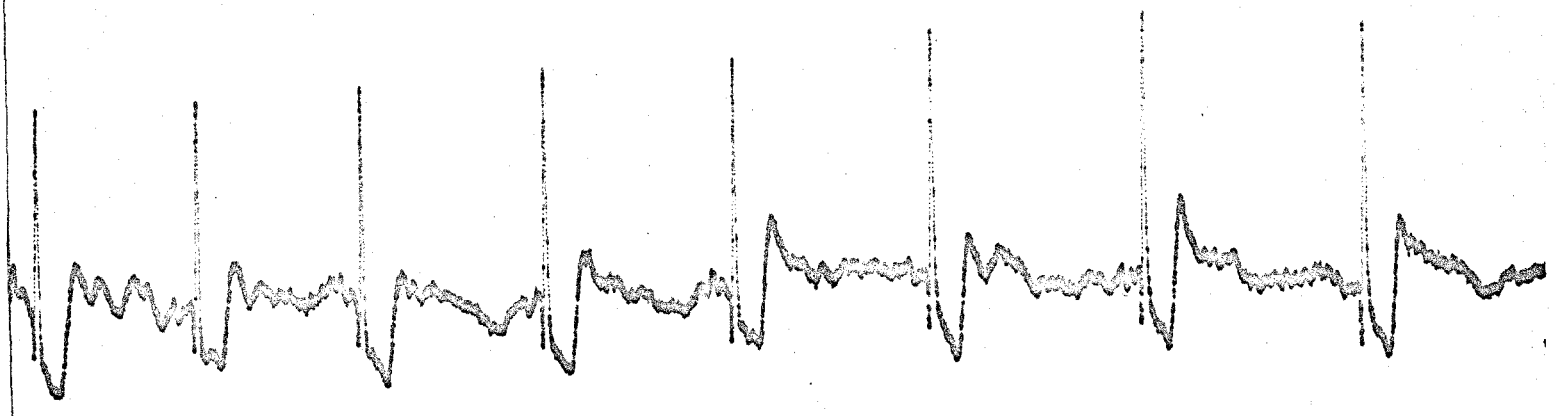
Fibrillation
Strip II



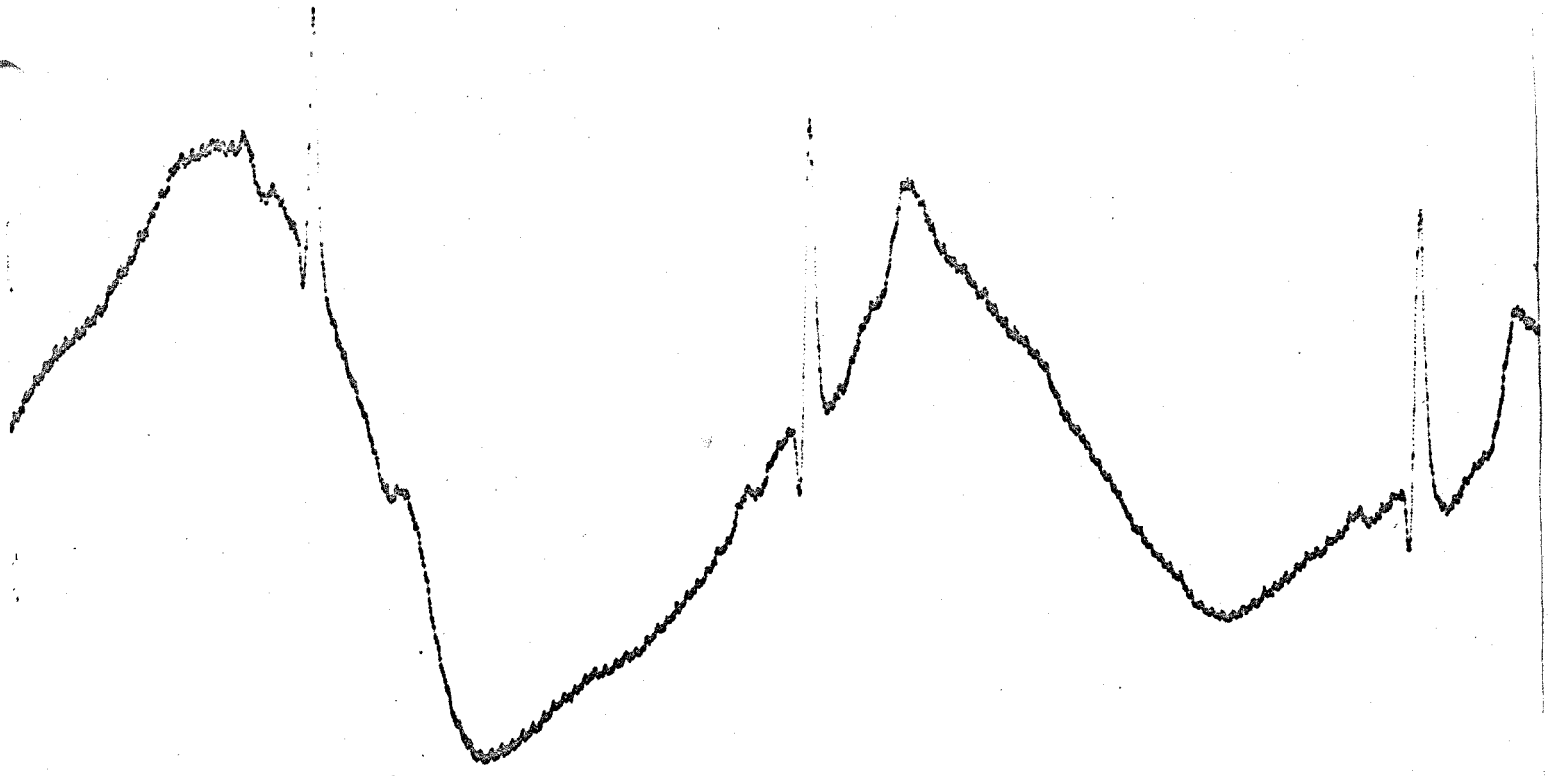
3



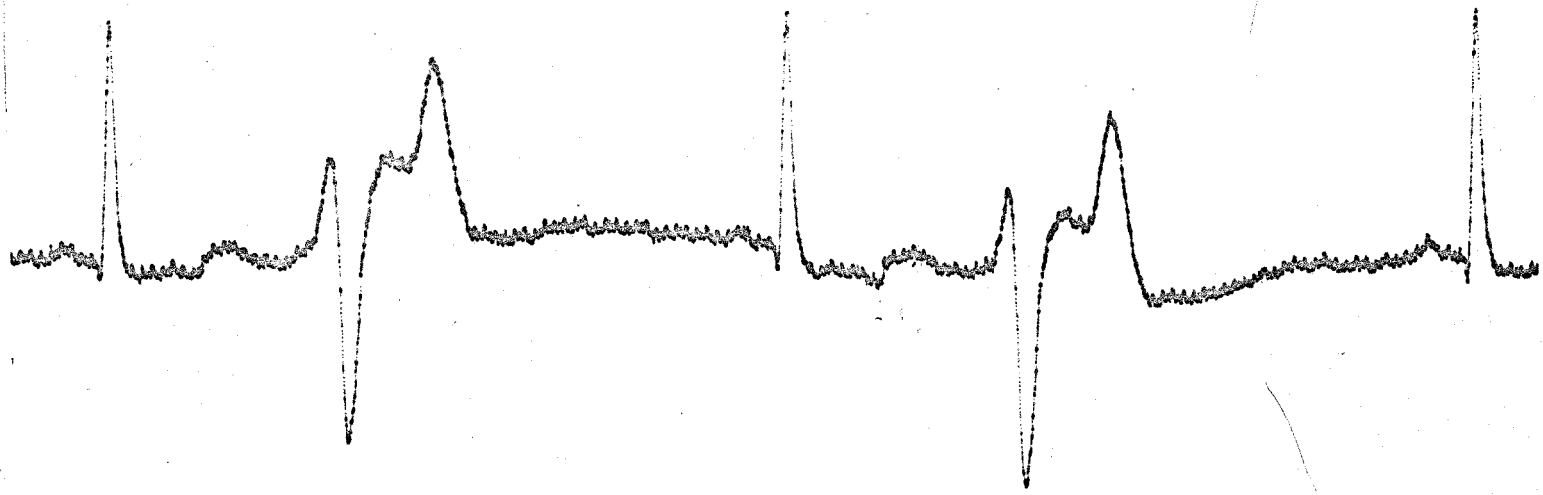
4



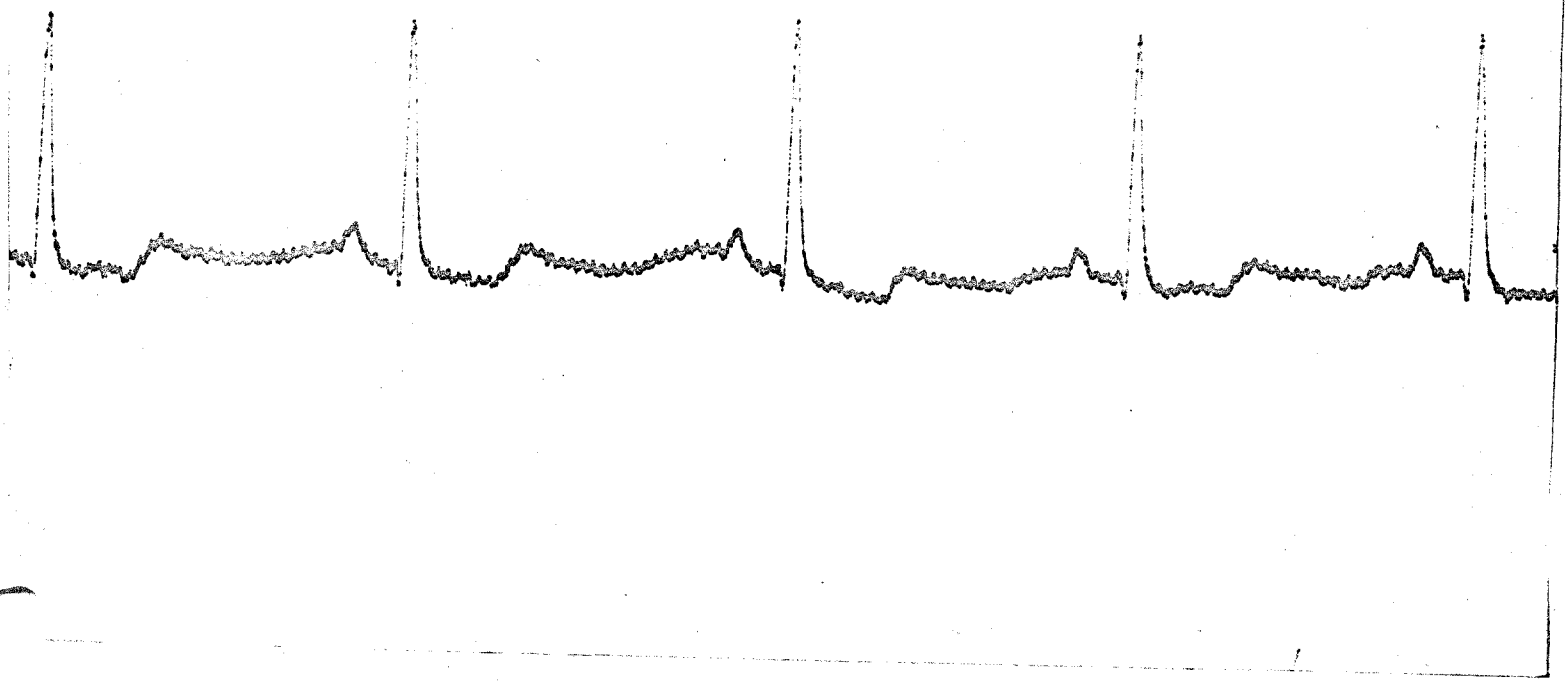
5



6



7



Summary

Cardioversion is based on theories proposed by Wiggers in the early 1930's. At that time, he stated that in order to achieve defibrillation of cardiac muscle, it was necessary that every fiber of myocardium be simultaneously depolarized. When this was done, the ectopic focus would be eliminated, re-entry would be obliviated and the normal sinus mechanisms would again resume control.

Prevost and Batelli, in 1899, were the first to notice the value of electrical discharge on the myocardium and the possible use in the treatment of various disorders. Since their initial experiments, work has proceeded steadily. At first, in the early 1900's, most emphasis was put on the development of alternating current devices which could be used in the therapy of ventricular fibrillation. There were a few attempts to develop a capacitor discharge device which might also prove to be effective.

Early experiments produced data which lead many to believe that devices employing capacitor discharge were very ineffectual. Consequently, most effort was spent in trying to develop an alternating current device which would be valuable.

Many different voltages for varying durations of time were employed. It was discovered early, that small voltages seemed, paradoxically, to be more consistent in producing fibrillation, while large voltages were more effectual in stopping fibrillation.

Experimental results in the use of alternating current always had large mortality rates and this type of current seemed to produce a large number of incidental serious arrhythmias, such as ventricular tachycardia, in about one-third of attempts. Atrial fibrillation was observed as high as 70% of the time following alternating current shocks of almost any voltage or any duration. Capacitor discharge seemed to be even less effectual than alternating current discharge.

Incidental findings which were of value, such as the discovery of a suprasensitive period of the heart, were made during

these early experiments. Any electric shock delivered during these periods produces ventricular or atrial fibrillation. Also discovered, were the various things which could produce ventricular fibrillation. During the use of all the electrical apparatus on myocardial tissue, much pathology was studied on the various effects of current on tissue.

Because cardioversion was not really used much, it didn't develop rapidly until Kouwenhoven published the results of two experiments he had run, in which people were kept alive for as long as 65 minutes and blood pressures maintained between 60 and 100 mm Hg with closed chest cardiac massage. This then, made it possible for untrained people to keep anyone alive who happened to have a Stokes-Adams attack on the street, and transport them to where they could get to a defibrillator or vice versa.

This really created a need for the best method of practical cardioversion. A machine was needed which could convert successfully, a large percentage of the time without the disadvantages of a multiplicity of post shock arrhythmias.

Subsequent experiments by Lown, showed that capacitor discharge as an effective means of cardioversion, had been grossly neglected. First reports, at least, indicated rather greater success than anyone had enjoyed before the early 1900's. Clinical work showed the capacitor discharge device invented by Lown and co-workers, to be almost 100% effective in defibrillating, as well as approximately 90% effective in treatment of all other ectopic tachycardias. If these results are duplicable, DC cardioversion is a very significant advance in the treatment of arrhythmia.

Conclusions

(1) Complete depolarization of all myocardial fibers is necessary before a heart will defibrillate.

(2) A supersensitive period of the heart muscle exists shortly after depolarization of the myocardium. Shocks administered during this period will consistently produce atrial or ventricular fibrillation. A different period exists for both upper and lower chambers.

(3) Large electrodes are necessary to achieve complete depolarization of myocardial fibers simultaneously.

(4) Capacitor discharge offers a more physiologic method of depolarization of myocardial tissue than does alternating current.

(5) DC Cardioversion is excellent for terminating individual eposides of cardiac arrhythmia, but has no capacity to maintain normal sinus rhythm.

(6) Drug therapy is necessary in conjunction with cardioversion for maintaining permanency of normal sinus rhythm.

Acknowledgments

I wish to gratefully acknowledge the assistance of Dr Robert Stratbucker in the preparation of this Thesis.

BIBLIOGRAPHY

1. Abelson, D. S., Samet, P., Rand, G., and Moraca, J., Endocardial Pacemaking and Insertion of A Permanent Internal Cardiac Pacemaker: A Dual Approach in a Case of Repeated Cardiac Asystole, New Engl. J.Med., 265: 792-794 (Oct 19) 1961.

2. Alexander, S., Kleiger, R., and Lown, B., Use of External Electric Countershock in Treatment of Ventricular Tachycardia., J. A. M. A., 177:916-918 (Sept. 30) 1961.

3. Applebaum, I. L., Parsonnet, V., Gilbert, L., and Levine, B., Complications of an Implantable Cardiac Pacemaker, J. Newark Beth Israel Hosp. 13:166-174 (July) 1962.

4. Beck, C. S., Pritchard, W. H., and Feil, H. S., Ventricular Fibrillation of Long Duration Abolished by Electric Shock, J. A. M. A. 135:985, 1947.

5. Bellet, Samuel, Clinical Disorders of the Heart Beat, 2nd Ed., Philadelphia, Lea and Febiger, 1963, p.1040-1059.

6. Burch, G. E., and Winsor, T., A Primer of Electrocardiography, 4th Ed., Philadelphia, Lea and Febiger, 1960.

7. Callaghan, J. C., and Bigelow, B.G., An Electrical Artificial Pacemaker for Standstill of the Heart, Ann. Surg. 134:8-17, (July) 1951.

8. Correspondence, Cardioversion Without Anesthesia, New Engl. J. Med. 269:534-535 (Sept. 5)1963.

9. Crehan, J. P., Nicholson, M. J., and Krocker, E. J., Further Application of Electric Countershock in Ventricular Tachy-Cardia, J. A. M. A. 181:727-728 (Aug. 25) 1962.

10. Cowley, R. A., Tischler, M., Attar, S., and Tamres, A., Cardiac Defibrillation Above 60 Cycles with a Portable Square-Wave Defibrillator, Surgery 51:207, 1962.

11. Editorial, An External Defibrillator, Brit. Med. J. 2:468, 1956.

12. Editorial, Single Shock Reverses Cardiac Arrhythmias, Medical World News 3:11, 1962.

13. Elmqvist, R., Cardiac Pacemakers and Defibrillators, ACTA Anesthesia Scand, Suppl. 11:95-98, 1962.

14. Furman, S., and Schwedel, J. B., An Intracardiac Pacemaker for Stokes-Adams Seizures, New Engl. J. Med. 261:943, 1959.

15. Grant, R. P., Clinical Electrocardiography, 1st Ed., New York, McGraw-Hill, 1957.

16. Gurvich, N. L., and Yuniev, G. S., Restoration of Regular Rhythm in Mammalian Fibrillating Heart, Am. Rev. Soviet Med. 3:236-239 (Feb.) 1946.

17. Gurvich, N. L., and Yuniev, G. S., Restoration of Heart Rhythm During Fibrillation by Condenser Discharge, Am. Rev. Soviet Med 4:252-256 (Feb.) 1947.

18. Guyton, A. C., and Satterfield, J., Factors Concerned in Electric Defibrillation of the Heart, Particularly Through the Unopened Chest, Am. J. Physiol. 167:81, 1951.

19. Hoffman, B. F., Suckling, E. E., and Brooks, C. McC., Vulnerability of the Dog Ventricle and Effects of Defibrillation, Circulat. Res. 3:147, 1955.

20. Hooker, D. R., Kouwenhoven, W. B., and Langworthy, O. R., Effects of Alternating Electrical Currents on the Heart, Am. J. Physiol. 103:444, 1933.

21. Jude, J. R., Kouwenhoven, W. B., Knickerbocker, G. G., An Experimental and Clinical Study of a Portable External Cardiac Defibrillator, Surgical Forum 13:185-187, 1962.

22. Jude, J. R., Kouwenhoven, W. B., and Knickerbocker, G. G., A New Approach to Cardiac Resuscitation, Ann. Surg. 154: 311, 1961.

23. Kaiser, G. C., Edgcomb, J. H., and Kay, J. H., An Experimental Study Comparing Various Voltages and Durations of Electric Shock in Defibrillation of the Canine Heart, J. Thor. Surg. 33:537, 1957.

24. Kortz, A. B., and Swan, H., Electrical Ventricular Defibrillation, Arch Surg. 74:911, 1957.

25. Kouwenhoven, W. B., Hooker, D. R., and Langworthy, O. R., Current Flowing Through the Heart Under Conditions of Electric Shock, Amer. J. Physiol 100:344-350 (Jan. 12) 1932.

26. Kouwenhoven, W. B., Jude, J. R., and Knickerbocker, G. G., Closed Chest Cardiac Massage, J. A. M. A. 173:1064-1067, (July 9) 1960.

27. Kouwenhoven, W. B., and Kay, J. H., A Simple Electrical Apparatus for Clinical Treatment of Ventricular Fibrillation, Surgery 30:781-786, 1951.

28. Kouwenhoven, W. B., and Milnor, W. R., Treatment of Ventricular Fibrillation Using Capacitor Discharge, J. Appl. Physiol. 7:253, 1954.

29. Kouwenhoven, W. B., Milnor, W. R., Knickerbocker, G. G., and Chestnut, W. B., Closed Chest Defibrillation of the Heart, Surgery 42:550, 1957.

30. Lape, H. E., and Maison, G. L., Cardiac Resuscitation and Survival: Influence of Rate of Manual Compression and Type of Countershock and of Epinephrine, Am. J. Physiol. 172:417, 1953.

31. Lefemine, A. A., Amarasingham, R., Harken, D. E., Berkovitz, B. V., and Lown, B., Defibrillation by Direct and Alternating Currents at Normal and Reduced Temperatures, S. Forum 13:183-185, 1962.

32. Lown, B., Newman, J., Amarasingham, R., and Berkovits, B. V., Comparison of Alternating Current with Direct Current Electroshock Across the Closed Chest, Am. J. Cardiol. 10: 223, 1962.

33. Lown, B., Amarasingham, R., and Neuman, J., New Method for Terminating Cardiac Arrhythmias: Use of Synchronized Capacitor Discharge, J. A. M. A. 182:548, 1962.

34. Lown, B., Bey, S. K., Perlroth, M. G., and Abe, T., Cardioversion of Ectopic Tachycardias, Am. J. of Med. Sciences, v 246, No. 3 (Sept.) 1963.

35. Lown, B., Perlroth, M. G., Bey, S. K., Abe, T., and Harken, D. E., Cardioversion of Atrial Fibrillation, New Engl. J. Med. 269:325-331 (Aug. 15) 1963.

36. Mackay, R. S. and Leeds, S. E., Physiologic Effects of Condenser Discharges with Application to Tissue Stimulation and Ventrucular Fibrillation, J. Appl. Physiol. 6: 67, 1953.

37. Maclean, L. D., and van Tyn, R. A., Ventricular Fibrillation: An Experimental Investigation of Voltage Requirements and Effect of Electrode Size, J. A. M. A. 175:471, 1961.

38. Mansfield, P. B., An Apparatus for Elective Fibrillatory Cardiac Arrest in Experimental and Clinical Cardiopulmonary By-pass Operations, J. Thor-Cardiovasc. Surg. 43:402-405 (Mar.), 1962.

39. Master, A. M. and Rosenfeld, I., The Lown Cardioverter, Dis. Chest 42:213 (Feb.) 1963.

40. Moe, G. K., and Mendez, C., Basis of Pharmacotherapy of Cardiac Arrhythmias, Mod. Conc. Cardiov. Dis., 31:742, 1962.

41. Nahum, L. H., The Cardioconverter, Conn. Med. 27:25-26 (Jan.) 1963.

42. Paul, M. H., and Miller, R. A., External Electrical Termination of Supraventricular Arrhythmias in Congenital Heart Disease, Circulation 25:604-609 (April) 1962.

43. Robicsek, F., McCall, M., Taylor, F. H., Hajob, A., and Sanger, P., The Treatment of Atrial Fibrillation by Direct Current Countershock, No. Carolina Med. J., v 24, No. 4, (April) 1963.

43a Rokseth, R. J. and Storstein, D., Quinidine Therapy of Chronic Auricular Fibrillation: Occurrence and Mechanism of Syncope, Arch. Int. Med. III:184-189, 1963.

44. Tedeshi, C. G. and White, C. W. Jr., A Morphologic Study of Canine Hearts Subjected to Fibrillation, Electrical Defibrillation and Manual Compression, Circulation 9: 916, 1954.

45. Thompson, G. W., Quinidine as a Couse of Sudden Death, Circulation 14:757-765, 1956.

46. Wegria, R., andWiggers, R. J., Factors Determining the Production of Ventricular Fibrillation by Direct Currents, Am. J. Physiol. 131:104-118, 1940-1941.

47. Wegria, R. and Wiggers, C. J., Production of Ventricular Fibrillation by Alternating Currents, Am. J. Physiol. 131:119-128, 1940-1941.

48. Wiggers, C. J., The Mechanism and Natureof Ventricular Fibrillation, Am. Heart J. 21:399-412, 1940.

49. Wiggers, C. J., The Physiologic Basis for Cardiac Resuscitation from Ventricular Fibrillation. Method for Serial Defibrillation, Am Heart J. 20:413, 1940.

50. Wiggers, C. J. and Wegria, R., Ventricular Fibrillation Due to Single, Localized Induction and Condenser Shocks Applied During Vulnerable Phase of Ventricular Systole, Am. J. Physiol. 128:500-505 (Oct. 28) 1939.

51. Zoll, P. M., Resuscitation of the Heart in Ventricular Standstill by External Stimulation, New Engl. J. Med. 247:768-771 (Nov.) 1952.

52. Zoll, P. M. and Linenthal, A. J., Termination of Refractory Tachycardia by External Countershock, Circulation 25:596, 1962.

53. Zoll, P. M., Linenthal, A. J., Gibson, W., Paul, M. H., and Norman, L. R., Termination of Ventricular Fibrillation in Man by Externally Applied Electric Countershock, New Engl. J. Med. 254:727, 1956.

54. Zoll, P. M. Linenthal, A. J., Norman, L. R., Paul, M. H., and Gibson, W., The effects of External Electric Currents on the Heart. Control of Cardiac Rhythm and Induction and Termination of Cardiac Arrhythmias, Circulation 14: 745, 1956.

55. Zoll, P. M., Linenthal, A. J., and Zarsky, L. R. H., Ventricular Fibrillation: Treatment and Prevention by External Electric Currents, New Engl. J. Med. 262:105, 1960.