DC pulse defibrillation

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Defibrillation was first produced, with the use of a discharging capacitor, by the Frenchmen, Prevost and Batelli, in 1899.[8] Kouwenhoven[4] has conducted extensive experiments employing capacitor discharges to produce defibrillation. He concluded that capacitors defibrillated in an unpredictable way. R. S. MacKay,[6] in 1960, made the important observation that the presence of a one-henry inductor in series with a capacitor would roughly halve the required energy for defibrillation. This suggested to us that the design of the inductor might be the critical element for capacitor defibrillation and not just the watt second charge on the capacitor.

Method

Fig. 1 is a circuit diagram of the method employed to study the wave forms generated when a capacitor discharge is effected across the heart with a closed or an open chest. Closing of the switch permits the charge which has accumulated on the capacitor "C" to pass through inductor "L" and then through the electrodes, returning to ground through a one-ohm resistance.

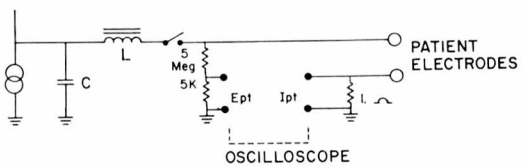

Fig. 1. Circuit used to measure simultaneous current (Ipt) and voltage (Ept) generated through and across electrodes by capacitor *C* discharging through inductance *L*.

By the use of a dual-beam oscilloscope,* it is possible to measure simultaneously the current through the body and the voltage imposed across the electrodes. From this, one can determine the exact inter-electrode resistance by the use of Ohm's Law, $E = I \times R$. Certain well-established relationships are known in an arrangement such as this. The energy which accumulates on the capacitor prior to its discharge is equal to $\frac{1}{2}$ CV^2, where C is the capacitance in farads and V is the voltage. This energy stored on the capacitor may be expressed as watt seconds (w. s.), or joules, both units being interchangeable. The DC pulse defibrillator so used consists of capacitor C in series with inductance L, discharging through the patient electrodes.

Using the circuit of Fig. 1 with the dual-trace oscilloscope for measuring and photographing the discharging wave forms, various combinations of capacitance and inductance were used across the chest. For internal defibrillation on the dog, electrodes

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2 inches in diameter were employed. Externally, the same electrodes were used, with the impulse applied between the suprasternal notch and over the apex of the heart on the left lower chest. When electrodes other than 2 inches in diameter are used, it will be so stated. In patients so defibrillated, the internal electrodes employed were 3 inches in diameter.

Results

In our initial experimentation, we attempted to reduplicate the work of MacKay which indicated that the introduction of an inductor in series with the discharging capacitor could reduce the amount of energy required by one half. We, therefore, took a one-henry inductor and placed it in series with a 16-microfarad capacitor and discharged this across a dog's chest. This initial experiment nearly resulted in our abandoning the experiment. Massive body response occurred to the animal, manifested by his breaking the restraining ropes to his forepaws. Violent respiratory movement ensued along with ventricular fibrillation which had to be corrected by a thoracotomy and internal defibrillation with a conventional 60 cycle AC internal defibrillator. At that time the oscilloscope used to visualize the wave form had a limited response time and the configuration of the wave form could not be determined with certainty. We then obtained our present oscilloscope and repeated the experiment with the same results, which again caused fibrillation. The current wave form so generated is shown in Fig. 2. This shows that an initial very sharp current peak occurred, followed by ringing (oscillation above and below the base line) of the current for a period of 40 milliseconds. More careful analysis of the wave form reveals that the inductor saturated at approximately one ampere of current density. This produced the sharp current spike which then, returning to one ampere, resulted in continuous ringing producing the ventricular fibrillation and the violent body response described. Sufficient discharges of this type were produced to demonstrate the extreme undesirability of such a wave form, as manifested by frequent production of ventricular fibrillation and massive muscular contraction.

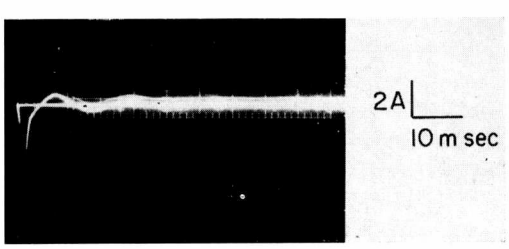

Fig. 2. C = 16 ufd; L = 1 Henry, 10 watt seconds. Current wave form shows 40 milliseconds ringing which produces violent muscle jerk and frequently ventricular fibrillation.

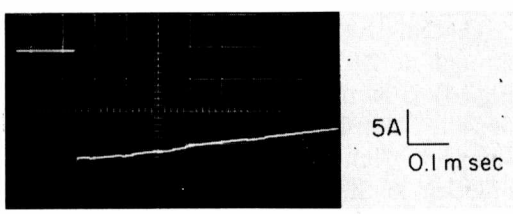

Fig. 3. Thirty watt second capacitor discharge through dog's chest shows current wave form.

We then removed the inductor completely from the circuit and observed wave forms generated by the capacitor alone discharging through the electrodes. Such a wave form is shown in Fig. 3. Under these circumstances it was noted that the rise time was virtually instantaneous (in the order of 5 microseconds). An exponential decay of the current resulted which was a function of the inter-electrode resistance and the capacitance. It is to be noted that the instantaneous rise time with exponential decay is the type of wave form which Milnor[7] and Kouwenhoven[4] investigated so thoroughly.

Milnor, in studying the response to transthoracic capacitor discharge in the dog, used capacitors of 25 or 50 microfarads. The threshold for inducing ventricular tachycardia and atrioventricular block related more to peak voltage than to the energy levels of the capacitor. The level for inducing such arrhythmias was 2,000 volts, even though at any given voltage on the two

capacitors, the energy on the larger would be twice the smaller. The threshold for inducing ventricular fibrillation was 2,700 volts and again related more to the voltage than to the energy level of the capacitor. Since the inter-electrode resistance of the large 4 inch by 6 inch electrodes used was fixed between 37 and 55 ohms, the peak current would also relate to the induction of the arrhythmias.

Kouwenhoven, in his prior work, observed no effect on the heart when discharges below 50 watt seconds were discharged through the chest of the dog with the use of the same large electrodes, and 200 watt seconds were required to defibrillate. In our own studies, 50 watt seconds discharging from a 25 microfarad capacitor through a 24 millihenry inductor[3] (*A* of Fig. 4) is more than enough energy to defibrillate consistently; all of this shows simply the importance of the design of the inductor in the DC pulse-type defibrillator.

The body may be viewed as a resistance and capacitance in parallel, an analogy which has been very well documented by Schwann,[9] as long as frequency is also stated. The two wave forms cited can be analyzed in this way. With the use of a simple capacitor, during the first interval most of the current flows into the body capacitance and little into the resistance. An inductor placed in the circuit permits a proportionately higher portion of the current to affect the body resistance. This suggests that there must be some desirable level of inductance which will slow the rising current wave forms sufficiently to permit a cellular response and yet not produce ringing with the undesirable side effects of massive muscle response and the production of ventricular fibrillation. Using a simple capacitor, we, as Kouwenhoven, observed that defibrillation was produced in an extremely irregular and unpredictable way.

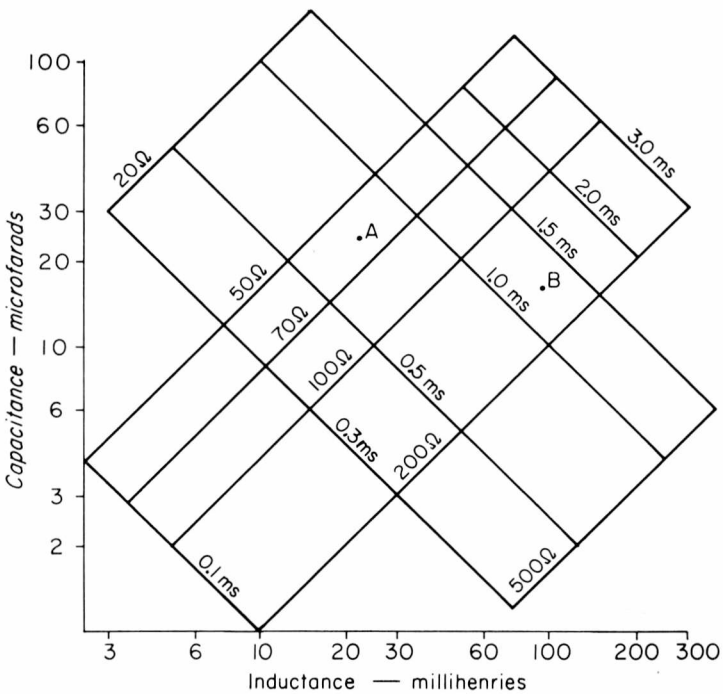

Fig. 4. Nomogram takes into consideration the four important variables relating to DC pulse defibrillation. These are the capacitance of the condensor in microfarads, the value of the inductance in millihenries, the critical inter electrode resistance in ohms, and current time to maximum in milliseconds.

Given a certain frequency to be reproduced by a combination of inductance and capacitance, it is quite true that an infinite number of combinations are obtainable. However, if one further qualifies this statement so that in the circuit consisting of capacitance, inductance, resistance in series (body impedance) is added such that the wave form generated should not ring or be overdamped, but should be just critically damped; then for any given one value of critical external dampening resistance and a given rise time only one value of capacitance and inductance will fulfill this requirement. This concept is illustrated in the nomogram of Fig. 4.[11] It is significant that any DC pulse-type defibrillator employing an inductor and capacitor in series may be represented by a single point on this nomogram.

Fig. 5A illustrates a wave form generated by a capacitor and inductor combination chosen to produce a small amount of ringing and represented by point *B* of the nomogram. Note that this type of wave form does not ring for longer than 6 milliseconds. Critical external dampening with such a system is approximately 156 ohms. The inter-electrode resistance was less than this amount and produced 22.5 per cent ring. Ringing is expressed as the per cent of overshoot after the initial wave form. This wave form is currently and very effectively being used for cardioversion by Lown.[5] Fig. 5B (point *A* on the nomogram) is the critically dampened wave form used by us and on the 16 patients to be subsequently cited. Such a wave form is capable of producing a consistent defibrillation in the dog with external settings of 20 to 30 watt seconds and internal settings of less than 5. As such, the amount of energy required to externally defibrillate the experimental animal is only 10 per cent the amount necessary with the use of only a capacitor and represents a considerable increase in efficiency. The inductor, of course, primarily influences the rise time of the current wave form as can be seen by mere inspection of the wave forms described.

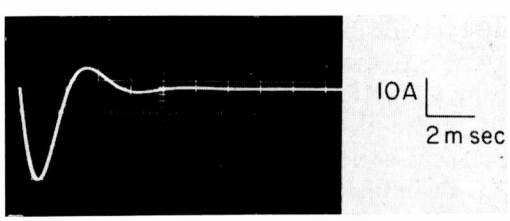

Fig. 5A. Current (Ipt) noncritically dampened wave form generated during external dog defibrillation at 100 watt seconds. L = 93 mh, C = 16 mfd. Ringing 22.50 per cent.

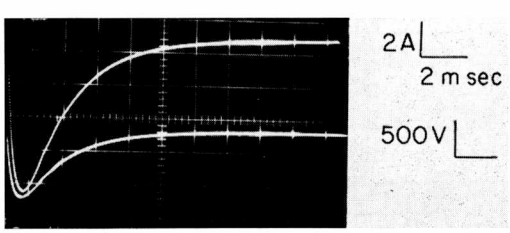

Fig. 5B. Critically dampened 30 watt second discharge externally defibrillating a dog shows current *(top)* and voltage *(bottom)* wave forms.

Theory of defibrillation

The exact mechanism by which a defibrillator operates is not known. Kouwenhoven set down well-accepted rules regarding the 60-cycle defibrillators in which high currents (greater than one ampere) internally would defibrillate; however, smaller amounts of current would paradoxically cause fibrillation internally. Externally, the values of current were multiplied by a factor of three to ten times.

Some light might be shed on how a defibrillator functions by examining an individual cell in the ventricle, observing its action potentials as the heart spontaneously reverts into ventricular fibrillation. Such an occurrence is illustrated in Fig. 6. Here we see the action potentials developed by a single cell in the ventricle—as the ventricle spontaneously goes into ventricular fibrillation. The animal was maintained on total heart-lung bypass at 26° C. and then the pH was gradually elevated, by means of sodium hydroxide, until spontaneous fibrillation occurred.[2] These recordings were

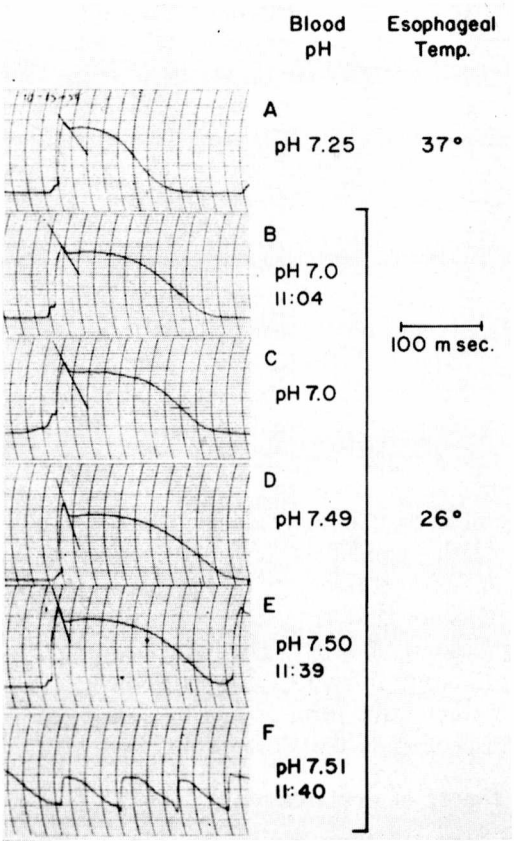

Fig. 6. Action potential recorded from within a single cell of the ventricle while ventricular fibrillation produced by gradual elevation of blood pH at an esophageal temperature of 26° C.

made in our laboratory by Dr. John Lee and Mr. Sam Kirk after a technique described by Dr. J. W. Woodbury.[10] Note that (A) the depolarizing action potential of a single cell is approximately 120 milliseconds' duration, corresponding to the normal Q-T interval. This interval is prolonged by hypothermia in which the heart becomes more vulnerable to fibrillation. Surprisingly, the circus movement theory of fibrillation would predict that, by prolonging the action potential, the incidence of ventricular fibrillation should be less. In this respect, the increased incidence of ventricular fibrillation at reduced temperatures directly contradicts the circus movement theory of ventricular fibrillation. Increasing the blood pH

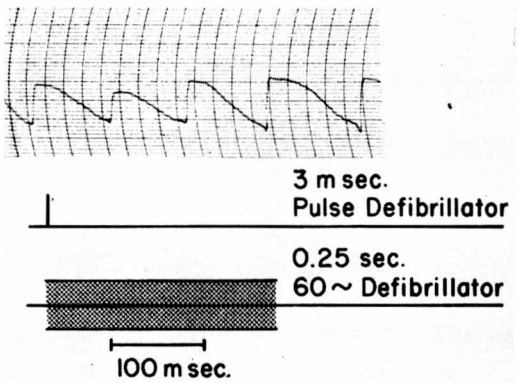

Fig. 7. Upper trace is intracellular action potential (same as V-F) produced by fibrillating cell in ventricle to show time base in relation to defibrillating impulse duration.

during hypothermia increases the susceptibility to fibrillation, as is illustrated from "B" to "E." Spontaneous fibrillation occurs at a pH of 7.51 and the action potential duration within the individual cell shortens to approximately one third the pre-fibrillation duration. The action potential amplitude decreases by one half.

In Fig. 7, the duration of the impulse from a pulse defibrillator is compared with the 0.25 second duration of a conventional 60-cycle defibrillator. This tracing is interesting in several respects. It diagrammatically shows that the pulse duration from a DC pulse defibrillator is approximately 1 per cent as long as that of a conventional AC defibrillator. The extremely short duration of the DC pulse defibrillator impulse suggests that with this wave form the status of any individual cell in the heart must not be important. Considering the millions of cells that are fibrillating within a ventricle, such a short duration would find the random-oriented cells in all phases of fibrillatory action. In regard to a conventional 60-cycle defibrillator, it has often been stated that the duration of the impulse should be 0.25 seconds for maximum efficiency. It can be seen that a defibrillating impulse of this duration encompasses more than one of any single cell action potential and, as such, it may be important that the cell be defibrillated in some phase of the action

Table I. *Internal pulse defibrillation**

Case no.	Patient age (yr.)	Diagnosis	Operation	Number of impulses		Comment
				No defib.	*Defib.*	
1	10	P. S. A. S. D. cyanotic	Repair	2 - 10 w. s.	1 - 20 w. s.	32° C.
2	32	A. S. A. I.	Valve replacement	2 - 30 w. s.	1 - 40 w. s.	Small 2 inch electrodes Massive hypertrophy
3	38	A. I.	Annuloplasty Aortic graft		1 - 30 w. s.	
4	60	A. I.	Valve replacement		1 - 30 w. s.	35.9° C.
5	37	M. S. M. I.	Repair		1 - 30 w. s.	27° C. pH control
6	45	M. I.	Repair	1 - 20 w. s.	1 - 30 w. s.	34° C.
7	40	M. I.	Repair	1 - 20 w. s.	1 - 30 w. s.	34° C.
8	48	A. S. A. I.	Repair	2 - 30 w. s. 2 - 40 w. s. 3 - AC, 0.1 sec., 140v.	1 - 40 w. s. 1 - 40 w. s.	35° C. Very anoxic during first tries 36° C.
9	49	A. I.	Repair		1 - 35 w. s.	35° C.
10	40	M. S.			1 - 35 w. s.	
11	18	Tetralogy	Repair		1 - 40 w. s.	33° C. One blade on heart other on chest
12	35	M. I.	Repair	1 - 50 w. s. One electrode on heart, one on chest	1 - 30 w. s.	35° C. Defibrillated with both electrodes on heart
13	45	A. I.	Repair		1 - 50 w. s.	33° C. Enormous heart
14	30	A. S. D. Anomalous venous return	Repair		1 - 40 w. s.	32.5° C. Large heart
15	55	A. S. A. I.	Repair	1 - 40 w. s. 32° C.	1 - 40 w. s. 33° C.	Very large heart
16	5	I. V. S. D.	Repair		3 - 50 w. s.	One electrode on heart, one on chest

Legend: P.S., Pulmonary stenosis. A.S.D., Atrial septal defect. A.S., Aortic stenosis. A.I., Aortic insufficiency, M.S., Mitral stenosis. M.I., Mitral insufficiency. I.V.S.D., Interventricular septal defect.

*DC pulse defibrillation in the first 16 patients. No. 1, Dec. 7, 1961. Critically dampened wave form of Fig. 5B used in all instances.[12]

potential which is less refractory to defibrillation, although this exact point is not known. The mechanism for pulse defibrillation may then differ from that of the conventional 60-cycle defibrillator.

The amount of current passed through the heart is the important variable for 60-cycle defibrillation, according to all available reports. This appears not to be the case with the DC pulse defibrillator, although peak current may be important. Indeed, the watt second setting apparently is not the

absolute determining factor since a proper inductor will enable external defibrillation with 30 watt seconds in the dog and yet 500 watt seconds may be required in the absence of an inductor. The rise time of the increasing current may well be the most important factor and one which we are currently investigating in our laboratory.

Clinical experience

Having determined in the laboratory that the DC pulse defibrillator was as efficient as the 60-cycle unit and our calculations indicating that considerably less energy was expended in the heart than with the conventional 60-cycle unit, we began the clinical application of the DC pulse defibrillator (Table I). This was first applied by us on Dec. 7, 1961, to a 10-year-old girl undergoing open repair for a cyanotic pulmonary stenosis with atrial septal defect. Table I shows that two 10 watt second impulses were applied at 32° C. without producing defibrillation. A third impulse at 20 watt seconds subsequently produced prompt defibrillation.

In the remainder of the patients, there was defibrillation in all maximum settings of 50 watt seconds. Of importance is the fact that in patients who are reoperated upon, in whom only limited mobilization of the heart is possible, defibrillation employing one blade on the heart and one blade on the chest wall can be a lifesaving measure, as shown by Case 16. In the first 23 patients so defibrillated at temperatures down to 27° C., defibrillation took place at settings between 15 and 50 watt seconds, in children and adults, respectively. Recently, in a 61-year-old man with mitral insufficiency undergoing open repair with blood pH control,[1] defibrillation was achieved with a single 10 watt second discharge at an esophageal temperature of 28° C.

Comparison of 60-cycle and DC pulse defibrillator

The difference in construction of the DC pulse defibrillator gives it certain advantages over the 60-cycle unit. The DC pulse defibrillator will not blow Operating Room fuses since it draws a current under one ampere from the power line. Because of the small current drain and reduced weight, it may be used as a portable unit in an ambulance with a converter, should this be necessary.

A comparison of the electrical wave forms generated can be done in three different ways—by comparing the relative amounts of energy, power, and heating produced during an average defibrillation.

With respect to energy or work performed during the defibrillation, this may be defined as Power × Time. An internal 60-cycle defibrillator develops 110 volts across an average heart resistance of 50 to 70 ohms, passing a current of greater than one ampere and usually about 2.2 amperes for 0.25 seconds (Kouwenhoven). Energy dissipated is thus:

$$(I^2R)\ t = (2.2)^2 \times 50 \times 0.25 = 61 \text{ watt seconds.}$$

The DC pulse defibrillator requires about 10 to 30 joules (watt seconds) or about one sixth to one half as much energy to defibrillate. Since heating is a direct function of energy or work expended, calories dissipated in the heart by the DC pulse defibrillator would also be one third to one half as much as a 60-cycle unit.

With respect to power, which may be defined as the Time/Rate at which energy is expended, in the 60-cycle unit, 61 joules are expended over 0.25 seconds, producing 244 watts of power. The pulse defibrillator expends its energy of 30 joules in about 3 milliseconds, producing about 10,000 watts of power during the short interval of the pulse expenditure. The DC pulse defibrillator, as such, develops forty-one times as much power as the 60-cycle unit while expending one half as much energy.

Discussion and summary

1. In 23 cases of congenital and acquired heart disease, internal DC pulse defibrillation, with the use of a critically dampened wave form, was effective at settings between

15 and 50 watt seconds in children and adults, respectively.

2. The first such defibrillation in a human being was on Dec. 7, 1961.

3. The DC pulse defibrillator, internally, expends one half to one third as much energy to defibrillate while developing forty-one times as much power as a 60-cycle defibrillator.

4. It is suggested that the superiority of this type of defibrillator will result in its eventual replacement of all 60-cycle defibrillators.

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11 Johnson, Douglas, of our laboratory evolved these relationships by similation of wave forms on an analog computer.

12 Personal communication: Information on certain of these cases, such as Number 2, was supplied by Dr. K. A. Merendino of Seattle and on Number 11 by Drs. Frank Gerbode and John Osborne of San Francisco.