# VENTRICULAR FIBRILLATION DUE TO SINGLE, LOCALIZED INDUCTION AND CONDENSER SHOCKS APPLIED DURING THE VULNERABLE PHASE OF VENTRICULAR SYSTOLE[1]

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It is generally believed that in order to induce ventricular fibrillation by electric currents they must not only have a minimal intensity but must act for a fair interval of time (cā, several seconds). Indeed, the variation in duration of a current has been used as a criterion of the sensitivity of the heart (1).

In 1934 King (2) and in 1936, Ferris, King, Spence and Williams (3) reported that shocks as short as 0.03 second are effective in fibrillating the ventricles provided they are applied during the occurrence of the T wave, which they interpreted as the partial refractory phase. Previous to this, de Boer (4) had shown that a process similar to fibrillation in mammalian hearts can be induced in the frog's ventricle by induction shocks applied near the end of the systole, but he believed only during a hypodynamic state. Andrus, Carter and Wheeler (5) found that an induction shock similarly introduced into normal auricles of dogs caused auricular fibrillation. During 1923–24 the senior author (6) in studying the response of the dog's ventricles to strong induction shocks demonstrated that the mammalian ventricle is not refractory to stimuli for a considerable, though apparently variable, interval of systole (last 0.03–0.09 sec. of systole). In order to produce premature contractions by shocks applied during systole it was necessary to utilize very strong break shocks, and in the zeal to elicit such contractions ventricular fibrillation all too often terminated the experiment.

A survey of many records has shown that this was due to single shocks and that all were delivered somewhere during the non-refractory phase of ventricular systole. In view of the importance of observations that a very brief shock is capable of inducing fibrillation even when the exciting current traverses only a small area of the ventricle, it seemed important

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to repeat these observations more methodically, by using condenser shocks of exceedingly brief duration.

METHODS. Dogs were lightly anesthetized with morphine and sodium barbital, the chest was opened, the heart suspended in a pericardial cradle and intraventricular pressure was recorded by a Wiggers optical manometer.

In order to introduce shocks at appropriately timed portions of late systole, a condenser discharge was released by the aid of a photocell and time delay circuit.

By focussing a single slit lamp (filament lamp of constant brilliancy) on the manometer membrane, a long narrow beam was reflected back to the photokymograph,[3] about $1\frac{1}{2}$ meter away. Another part of the beam moved past the slit of a housing surrounding the photoelectric cell. The flow of current from cathode to anode occasioned when the photocell was momentarily illuminated was properly amplified and activated by a thyratron trigger tube. The voltage thus developed was impressed on a resister-condenser which operated as a variable delay circuit. This circuit was designed so that its critical voltage developed after a predetermined time interval and then overpowered the negative bias on a thyratron-condenser. A high resistance in parallel with the condenser made the thyratron tube a self-extinguishing circuit. When the bias voltage was reduced sufficiently on the discharge tube, it conducted and discharged a 4-micro-farad condenser charged with 300 volts. Any fraction of the voltage discharged through a voltage divider could be used as the stimulus. For stimulation two stigmatic $Ag \cdot AgCl_2$ electrodes were directly applied to the heart. The distance between the electrodes was about 1 cm.

The time of the condenser-discharge was recorded by shunting a small fraction of the discharge to a General Electric oscillograph, recording on the photokymograph with the pressure curve.

During each stimulation the quantity of current used for stimulation was determined by a ballistic galvanometer previously calibrated with an external shunt of 2000 ohms in circuit. Since the effective resistance of the heart could not be determined each time, these values are approximate only, but serve as relative values if it be assumed the heart resistance did not change.

In order to repeat observations on the fibrillating effectiveness of shocks on dogs, the ventricles of which do not recover spontaneously, provision for prompt revival by countershock needed to be made. The procedure of Hooker, Kouwenhoven and Langworthy (7) was adopted. To assure prompt revival without manipulation, massage, or use of drugs—all of which might alter irritability, two large metal electrodes were kept permanently in place, one on each lateral aspect of the ventricles. By throw-

[3] The optical system utilizing double slit lamps or the projection system for Hamilton or Gregg manometers does not lend itself to such projection.

ing two switches in quick succession, the stimulating apparatus was protected and the countershock was applied. Thus, revival was achieved in 15 seconds to 1 minute.

The only unfortunate feature of the arrangement was that the presence of large metal electrodes deformed standard e.c.g. leads; indeed, this necessary feature for revival prevented use of the R wave to set off the stimulus, a method so effectively used by others.

RESULTS. Repeated tests on seventeen different dogs demonstrated consistently that stimuli falling during the rise of intraventricular pressure are without effect, that those applied during the summit at various intervals up to about 0.06 second before the end of contraction, marked by the incisura, produce either a small premature contraction early in following diastole, or ventricular fibrillation, and that those given during isometric relaxation or subsequent diastole cause premature contractions only. It may be added that strong break induction shocks generated by a Harvard induction with core removed again yield similar results.

Small sections from a continuous series of observations are reproduced in figure 1, the strength of stimulus being expressed in coulombs. It may be noted that the current variations are not great.

The first curve, A, shows the typical premature contraction starting at $P$ resulting from a shock at the beginning of isometric relaxation. Curve B shows that a slightly weaker shock delivered at the summit of the pressure curve, or 0.064 second preceding end of mechanical systole (at $X$), resulted in fibrillation. Close inspection reveals a slight tendency to alternation in this record and the stimulus fell during the smaller beat. Alternation or other hypodynamic states are not a *sine qua non* for induction of fibrillation, as was amply demonstrated on other dogs. If the forms of the first and third beats are carefully compared, we note the same tendency to start a premature contraction at $P$ as in the curve of record A. This was followed by several inefficient contractions ($P_1$, $P_2$) that only produce slight variations of intraventricular pressure. The heart was promptly revived by countershock and in less than one minute was beating normally. Approximately five minutes later, record C was obtained. A shock administered toward the end of isometric relaxation caused a larger premature contraction than that shown in curve A. Very shortly thereafter a similar shock was delivered to the left ventricle during the peak of systole, or 0.027 second before its termination and fibrillation was again induced. As the experiment continued, the ventricular beats became more hypodynamic. The other records show that fibrillation was consistently produced by shocks during late systole, and it may be added, never by shocks delivered during any other phase of the heart cycle. Fibrillation and recovery were thus produced ten times; the animal did not die in fibrillation. This again emphasizes the efficiency of the method of countershock.

DISCUSSION. The results reported confirm previous investigations in

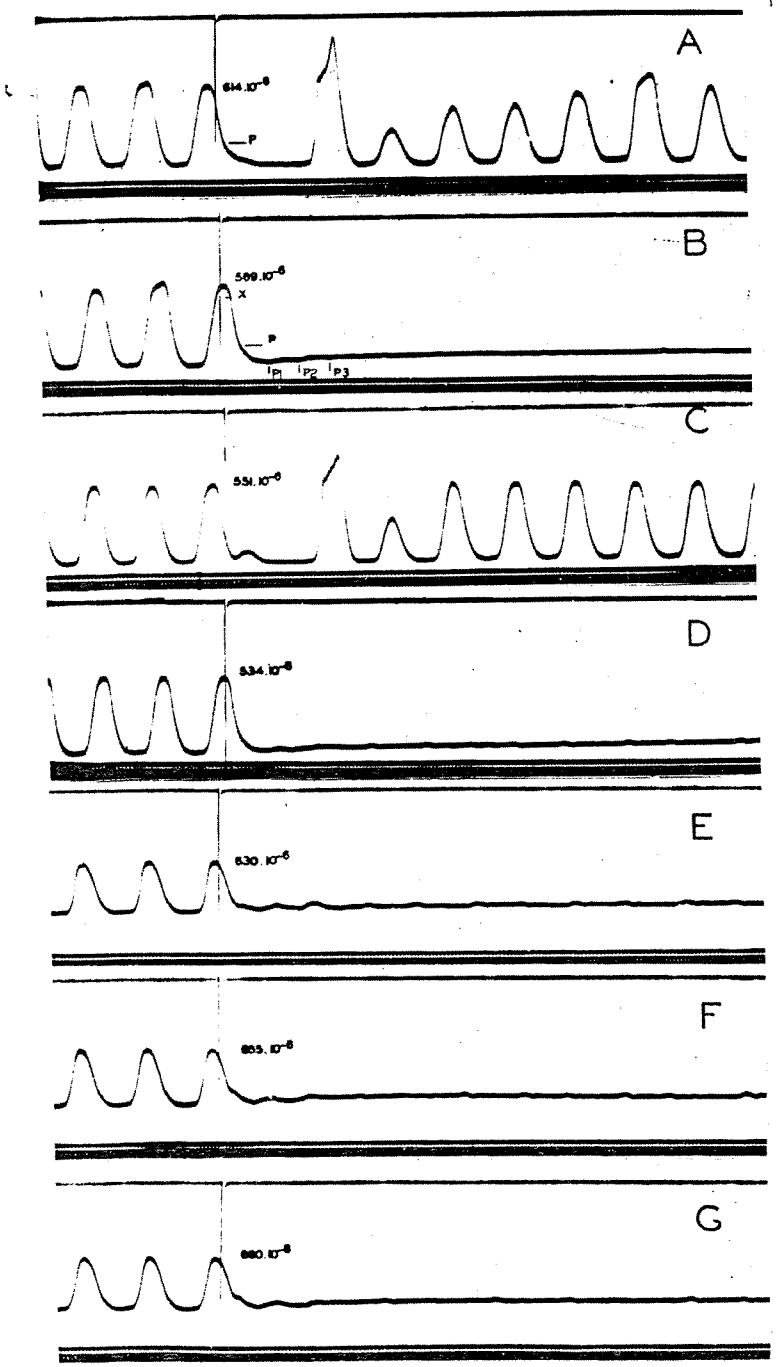
A
614.10⁻⁶
P
B
569.10⁻⁶
X
P
I_P1 I_P2 I_P3
C
551.10⁻⁶
D
534.10⁻⁶
E
630.10⁻⁶
F
665.10⁻⁶
G
660.10⁻⁶

Fig. 1

establishing that a considerable portion of late systole constitutes a *vulnerable period*, during which alone stimuli are effective in inducing ventricular fibrillation. This corresponds roughly to the T wave of a standard electrocardiogram, although in our experience great variability exists in the relation of this waye to the end of systole.

On the basis of our experience, this vulnerability is inherent in normal hearts; it occurs in young dogs as well as old, and in vigorously beating as well as hypodynamic or alternating ventricles. The latter is obviously not a requirement for production of fibrillation, as de Boer (4) has postulated for the frog ventricle.

The demonstration that fibrillation is only induced by a single shock delivered to a localized region of the ventricle when it falls during the vulnerable period makes it obvious that the induction of fibrillation—i.e., the physiological condition necessary for converting coördinated into incoördinated contractions—does not require passage of a current through every fraction of the myocardium, as was the case in experiments reported by the Columbia investigators (2, 3). Hence the probability that fibrillation following drugs or myocardial ischemia may likewise be initiated by localized excitation, and does not necessarily require a generalized altered irritability or reactivity.

If fibrillation is only induced by effective stimuli that fall during the vulnerable period of late systole, some *a priori* conclusions may be drawn as to the manner in which more prolonged currents induce fibrillation. A direct current lasting only two seconds would cover four complete heart cycles at a heart rate of 120/min. In accordance with well established laws of stimulation such currents would most probably induce fibrillation if either the moment of opening or closing fell during this period. In the case of an alternating current, say 60 cycles, the closing of the circuit during a vulnerable period would likewise constitute a particularly dangerous event since this represents its highest stimulating value. However, effective changes in intensity still occur several times during each vulnerable period, any one of which could serve as *the* effective stimulus inducing fibrillation (8). This offers an explanation for the generally recognized fact that alternating currents are more dangerous than direct currents at lower ranges of effectiveness, for the chance of a direct current being opened or closed during the brief vulnerable period is not great. However, unless passage of alternating current during one cycle alters irritability for the next it may be anticipated that fibrillation is always induced during the first vulnerable period that it strikes.

The corollary follows that no *a priori* reason exists for supposing that the duration of application of any current bears any relation to its fibrillating efficiency, or that it can be used as a criterion either of the sensitivity of the ventricles to fibrillation or in estmating the value of agents supposed to increase or decrease such sensitivity.

It may be admitted that such conclusions fail to take into account the possible effects that passage of currents during a series of beats have on irritability; but in such event some fortuitous stimulus must operate during some vulnerable period to start the fibrillation. Many possibilities may be thought of, such as pressure changes within the heart, mechanical stimuli with movements of the heart, chance variations in current strength passing through the myocardium during the vulnerable period, etc. But if fortuitous stimuli act to induce fibrillation when currents are applied for various durations of time, the variability of current duration inducing fibrillation is not a scientific criterion of ventricular sensitivity to stimulation, because the intensity of the fortuitous stimulus and time of its action are unknown and probably variable.

## SUMMARY

Experimental evidence is reported which demonstrates that a brief induction or condenser shock applied to normal hearts of old or young dogs by stigmatic electrodes induces fibrillation only when the shocks fall during the vulnerable period of late systole. Since the effect is obtained by use of stigmatic electrodes, passage of the current through the whole or large parts of the myocardium is not necessary to initiate fibrillation.

Several implications are discussed: 1. Fibrillation following use of prolonged electrical currents, drugs, chemicals and ischemia may likewise be induced by some effective stimulus—possibly a fortuitous one—falling during the vulnerable period. 2. One reason why an alternating current at critical voltages is more dangerous than the direct current seems to lie in the fact that effective variations of current strength obviously fall during the vulnerable phase in the former and only during closing and opening of the direct current. 3. The experimental results indicate that the effectiveness of currents or the variations of myocardial sensitivity to fibrillation cannot be tested experimentally by noting variations in the duration of current flow, a criterion frequently employed by previous investigators.

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