PATHOLOGICAL PHYSIOLOGY AND GENERAL PATHOLOGY

CHANGES IN THE DURATION OF THE VENTRICULAR COMPLEX OF THE ELECTROCARDIOGRAM DURING INTERRUPTION AND RESTORATION OF THE WORK OF THE HEART

D. G. Maksimov

The Laboratory of Experimental Physiology of Resuscitation (Head – Prof. V. A. Negovskii)

of the Academy of Medical Sciences USSR, Moscow

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In the opinion of the majority of workers, the duration of the ventricular complex of the electrocardiogram (Q – T interval) reflects the duration of the ventricular contraction of the heart and is an important indication of its activity [9, 11, 14]. In the case of weakening of the contraction of the heart, the duration of the ventricular complex as a rule increases [13, 19, 20, 22]. There are, however, indications of the possibility of shortening of the ventricular complex during apparent death, both clinically [15, 18, 26, 28] and in experiments on the hearts of warm- and cold-blooded animals [3, 7, 21, 24]. Restoration of cardiac activity is accompanied by the converse sign – lengthening of the Q – T interval. Later on however, concurrently with strengthening of the contraction of the heart, the duration of the ventricular complex gradually decreases [1, 3, 7, 21].

In a previous report [3] the hypothesis was proposed that differences in the duration of the ventricular complex during apparent death and resuscitation were due to changes in the number of myocardial cells exhibiting bioelectrical activity.

In the present paper we give the results of further investigation of conditions affecting the duration of the ventricular complex: the influence of the character and duration of the process of apparent death, the duration of clinical death, the type of anesthesia used in the experiment and various other factors.

EXPERIMENTAL METHOD

Experiments were performed on 115 adult dogs weighing from 6 to 25 kg. In the majority of the experiments the animals were given a preliminary subcutaneous injection of pantopon (8 mg per 1 kg body weight) and in about 50% of the experiments general ether anesthesia was used also. Clinical death was brought about in 85 dogs by exsanguination, in 20 dogs by asphyxia and in 10 dogs by electrical trauma. Resuscitation measures began at various intervals of time after the onset of clinical death (from 1 to 16 min). These consisted of artificial respiration and intra-arterial transfusion of blood under pressure with the addition of glucose and adrenalin by a method devised by V. A. Negovskii and his co-workers [4, 5]. In some experiments adrenalin was not given.

The electrocardiograms were recorded on a portable electrocardiograph. The action currents of the heart were tapped by means of needle electrodes inserted into the skin at the level of the upper and lower ends of the body of the sternum.

EXPERIMENTAL RESULTS

Analysis of the electrocardiographic records made during various forms of apparent death showed that changes in the duration of the ventricular complex during clinical death and subsequent resuscitation were determined mainly by the degree of anoxia of the heart, i. e., by the duration of the lethal process and of the state of clinical

Relationship Between the Duration of the Ventricular Complex and the Degree of Anoxia of the Heart

Expt. no.	Cause of death	Duration of		Duration of ventricular complex		Time of restoration of the work of the heart	No. of expts.
		lethal process	clinical death	during clinical death	during resuscitation		
		minutes		seconds			
1	Electrical trauma	2—7	1—3	0.14—0.19	0.23—0.35	25—32	10
2	Asphyxia	6—9	1—4	0.15—0.18	0.25—0.36	24—31	12
3	Exsanguination	4—10	3—5	0.16—0.22	0.27—0.37	22—37	10
4	»	6—12	7—10	0.16—0.27	0.30—0.55	30—48	4
5	»	8—14	15—16	0.26—0.31	0.45	Did not recover	2

death (see table). After a rapid process (less than 10 min) the duration of the ventricular complex was reduced in the first minutes of clinical death to 0.14-0.22 sec (before the experiment the Q – T interval was 0.20-0.27 sec, and after injection of pantopon 0.24-0.30 sec; Figs. 1, 2, 3). Changes in the form of the ventricular complex at this time consisted of the appearance of a deep S wave, and displacement of the S – T interval and its merging with a high T wave. After a more prolonged lethal process (15-30 min) the shortening of the ventricular

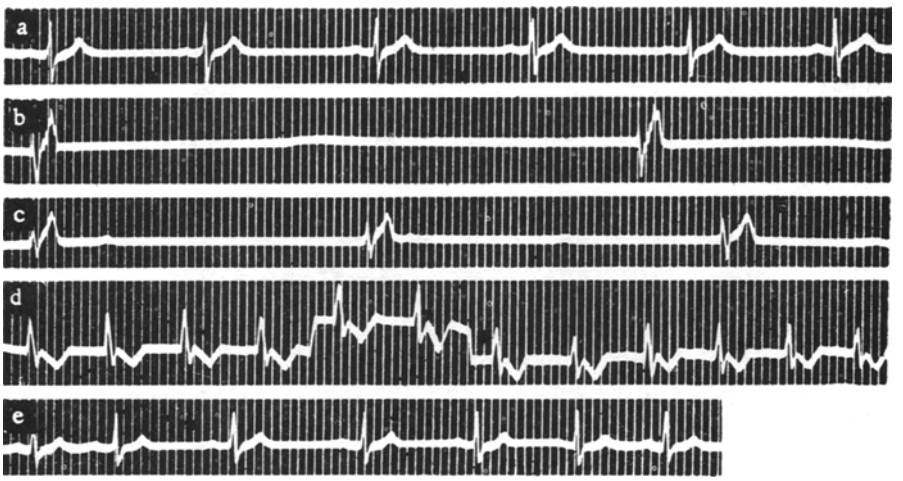

Fig. 1. Changes in the electrocardiogram during clinical death lasting 5 min (caused by hemorrhage lasting 8 min). a) Before hemorrhage Q – T was 0.26-0.27 sec; b) 3 min of clinical death: diminution of Q – T to 0.17-0.18 sec; c) beginning of resuscitation: lengthening of Q – T to 0.19-0.22 sec; d) 30 sec from the beginning of resuscitation: appearance of effective contractions of the heart, lengthening of Q – T to 0.28-0.30 sec; e) 20 min from the beginning of resuscitation: Q – T equal to 0.22-0.24 sec.

complex during clinical death was less pronounced or even could not be found (see Figs. 1,b-3,b). The form of the ventricular complex was changed considerably more in these experiments: often total merging of the initial and terminal parts of the complex was observed with the formation of a typical monophasic deflection (see Fig. 3,c).

In the later stages of clinical death (7-10 min) the duration of the ventricular complex was increased. Subsequently, a secondary reduction in the duration of the complex could be seen with an alteration in its shape and diminution of its amplitude (see Fig. 3, b, d, f). A similar feature – increase in the duration of the ventricular complex and its subsequent shortening with increasing anoxia – was observed also in the case of secondary interruption of the work of the heart following its temporary restoration (see Fig. 3, i).

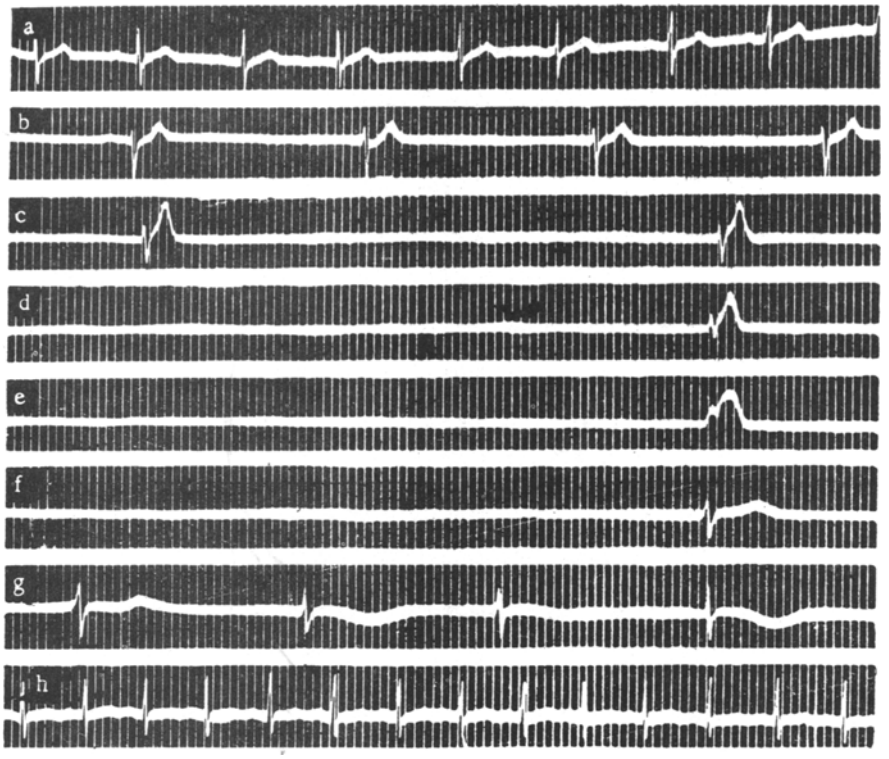

Fig. 2. Changes in the electrocardiogram during clinical death lasting 10 min (caused by hemorrhage lasting 7 min). a, b) Before hemorrhage and 4 min after its start: duration of Q – T 0.20 and 0.22 sec, respectively; c, d, e) 3rd, 5th and 8th min of clinical death: duration of ventricular complex in the region of 0.2 sec; f) beginning of resuscitation: lengthening of Q – T to 0.42 sec; g) 50 sec from the beginning of resuscitation: appearance of effective contractions of the heart, lengthening of Q – T to 0.54 sec; h) 28 min from the beginning of resuscitation: Q – T equal to 0.20 sec.

During the performance of resuscitation measures, simultaneously with restoration of the form of the ventricular complex, its duration increased. The degree of this increase was variable, depending on the duration of the anoxia. After rapid production of apparent death (less than 10 min) and a short period of clinical death (less than 5 min) the duration of the ventricular complex rose to 0.23-0.37 sec. On restoration of the cardiac activity after a more lengthy lethal process (10-15 min) or a more prolonged period of clinical death (7-10 min) the duration of the complex increased to 0.3-0.55 sec (see table and Figs. 1 and 2). The absence of a rapid and significant lengthening of the ventricular complex during resuscitation was a bad prognostic sign. This feature was usually observed after prolonged periods of agony and clinical death, when the cardiac activity was restored extremely slowly or not at all (see table and Fig. 3, g and h).

No connection was established between the changes in the duration of the ventricular complex during clinical death and the subsequent restoration of cardiac activity and the frequency of the rhythm.

Slowing of the electrocardiographic complexes after the onset of clinical death was accompanied in most cases by shortening of their duration; increase in the rate during resuscitation, on the other hand, was accompanied by lengthening of the complexes. The absence of any relationship between the duration of the ventricular complex and the rate of the rhythm is shown clearly by comparing the electrocardiograms recorded during production of apparent death and resuscitation: with the same rate of the rhythm the duration of the Q – T interval is considerably longer in resuscitation than in production of apparent death (see Fig. 3, b, g and Fig. 2, b, g).

The method of anesthesia used during the experiments did not have any essential influence on the changes in the action currents of the heart during clinical death and subsequent resuscitation. Only with deep and prolonged ether anesthesia were more persistent anoxic changes produced in the electrocardiogram, which were superimposed upon the changes arising during agony and clinical death and enhanced the corresponding disturbances of form and duration of the ventricular complex.

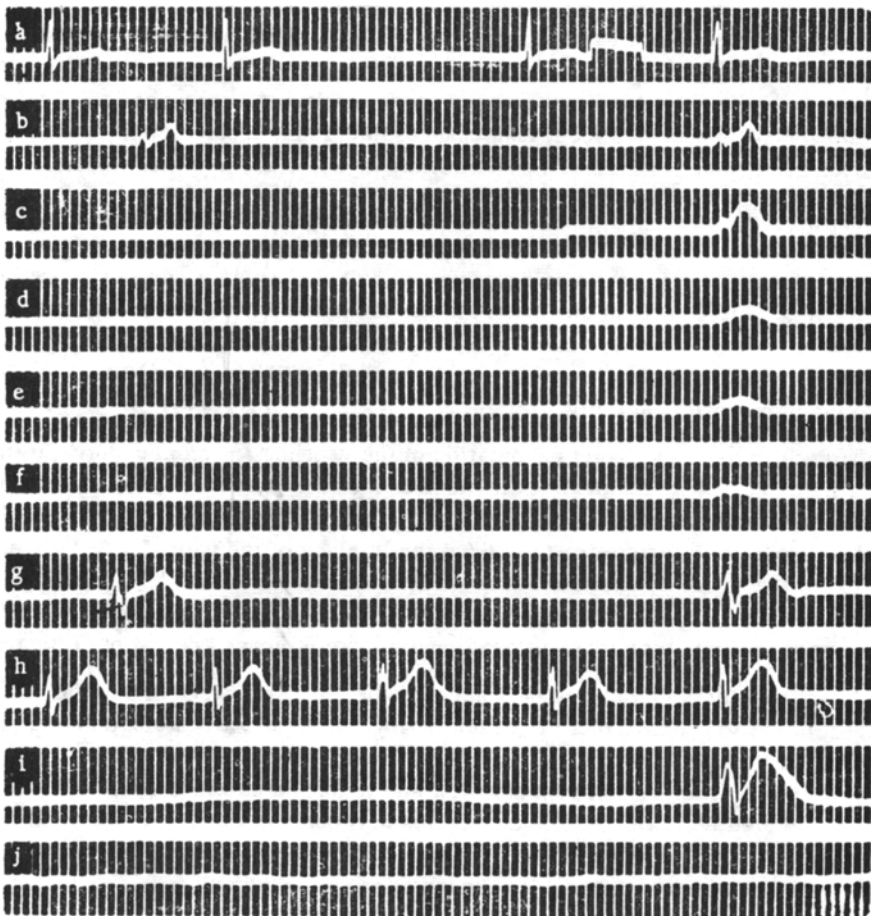

Fig. 3. Changes in the electrocardiogram during clinical death lasting 16 min (caused by hemorrhage lasting 14 min). a) Before hemorrhage Q – T was 0.3 sec; b, c, d, e, f) 3rd, 5th, 7th, 9th and 12th min of clinical death: duration of ventricular complex 0.23, 0.25, 0.30, 0.27 and 0.20 sec, respectively; g) 1.5 min from the beginning of resuscitation: lengthening of Q – T to 0.37-0.40 sec; h) 9th min of resuscitation: temporary restoration of weak cardiac activity, Q – T 0.35-0.40 sec; i) secondary interruption of the work of the heart; j) appearance of ventricular fibrillation.

On infusion of blood under pressure without the addition of adrenalin the cardiac activity was restored more slowly or not at all. In these experiments the changes in the electrocardiogram were produced more slowly and the Q – T interval was lengthened to a lesser degree. This shows that the injection of adrenalin at the time of resuscitation brings about restoration of the form of the ventricular complex, lengthening of its duration and the more rapid restoration to normal of the effective work of the heart.

In view of reports in the literature on the relationship between the Q – T interval and the filling of the heart with blood [10, 19, 23, 25], it might be thought that reduction in the duration of the ventricular complex during production of apparent death from exsanguination was due to reduction in the volume of the heart. In this case, lengthening of the Q – T interval during resuscitation might be explained by increase in the ventricular volume as a result of the infusion of blood. However, the analogous reduction in the duration of the ventricular complex observed during production of apparent death by asphyxia or electrical trauma, in spite of the considerably greater volume of the ventricles, does not support such a hypothesis.

These changes in the duration of the ventricular complex which have been described are most likely to be connected with the qualitatively distinguishable changes in conductivity during production of apparent death and

resuscitation. The reduction in the duration of the ventricular complex during clinical death can be explained, in the view of N. L. Gurvich [2], by the smaller number of myocardial fibers taking part in the excitation, in consequence of the development of a block to the conduction of the excitation. This hypothesis is confirmed by the changes in the form of the ventricular complex observed at this time: the splitting up and widening of its initial part (QRS); displacement of the S – T interval and the formation of a monophasic deflection (see Figs. 1, 2, 3). According to the generally accepted idea, such changes in the electrocardiogram result from disturbance of intraventricular conductivity and the exclusion of part of the myocardium from the process of excitation [8, 10, 11, 30].

The relatively long duration of the ventricular complex after prolonged agony and clinical death and secondary interruption of the work of the heart may be explained by the slowing of conduction which coincides with the increasing anoxia. This slowing of conduction masks the reduction in the number of myocardial fibers taking part in the excitation. With a longer period of clinical death, the secondary shortening of the ventricular complex is evidently due to further disturbance of conduction and the still greater reduction in the number of myocardial cells showing bioelectrical activity.

In accordance with the above, the increased duration of the ventricular complex during restoration of the work of the heart may be explained by removal of the block of intraventricular conduction present during clinical death and by increase in the number of myocardial cells taking part in the excitation. The extremely long duration of the ventricular complex at this time (up to 0.3-0.4 sec in place of the normal 0.2-0.27 sec) can be attributed to the retardation of the spread of excitation through the myocardium which is natural in such a condition. This hypothesis agrees well with the even greater increase in the duration of the ventricular complex (up to 0.55 sec) after a more prolonged production of apparent death and a more prolonged period of clinical death.

This also explains the regular coincidence of changes in the electrocardiogram and the rate of restoration of the effective work of the heart. Where there is a rapid change in the electrocardiogram and pronounced increase in the duration of the ventricular complex, the effective work of the heart is restored rapidly. With slow restoration of the form of the ventricular complex and slow and slight increase in its duration, the restoration of cardiac activity is also observed to be slow. The connection between the alteration in the form of the ventricular complex and its duration with the effective work of the heart is evidently due to the fact that each of these signs depends on the rate of restoration of intraventricular conductivity.

Similar findings were obtained by Bauereisen [12]. By artificial stimulation of the frog's heart with an excessive supply of oxygen this worker observed a diminution in the amplitude and duration of the mechanogram of the heart with increasing exhaustion. In oxygen lack the duration of the contraction was only slightly affected, but the amplitude was reduced as before. It has to be pointed out that these changes in the duration of the contraction of the heart are to some extent analogous to those which we observed in the duration of electrical systole depending on the degree of anoxia.

Weakening of the contraction of the heart by vagal inhibition is also known to be accompanied by reduction in the duration of the ventricular complex [6, 16, 17, 29]. It is very probable that in this case the interference with conduction takes place up to and including total blockade of the terminal branches of the conducting system, thereby isolating the myocardial fibers from the process of excitation. This idea of the possibility of partial asystole during vagal inhibition was expressed by Rothberger and Winterberg [27], who regarded this phenomenon as analogous with the weakening of the contraction in pulsus alternans.

It can be concluded from these findings that the changes in the duration of the ventricular complex during apparent death and resuscitation are determined by the degree of interference with intraventricular conduction. Its slowing after the prolonged production of apparent death causes increase in the duration of the ventricular complex. The extreme degree of disturbance of conductivity arising during clinical death leads to blockade of the terminal branches of the conducting system and to reduction in the duration of the ventricular complex. The combination of these two degrees of disturbance of conduction determines the duration of the ventricular complex during clinical death. Changes in the duration of the ventricular complex during resuscitation depend on the degree of restoration of intraventricular conductivity, more particularly on the number of myocardial fibers taking part in the excitation and on the rate of spread of the excitation throughout the ventricle.

SUMMARY

ECG changes were studied in the agonal condition and during resuscitation of dogs. After quick death the ventricular complex usually shortened to 0.15-0.20 sec during the first minutes of the clinical death, while in re-establishment of the heart work it was increased to 0.25-0.35 sec. In a prolonged agony and clinical death the ventricular complex was not shortened as much,while in resuscitation it was prolonged to 0.4-0.55 sec.

Various changes which take place in the length of the ventricular complex in the processes of dying and resuscitation may be explained by the relationship of two factors: the velocity of the spread of excitation and the number of the myocardial elements which are excited.

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