DYNAMICS OF EKG CHANGES DURING RESUSCITATION OF DOGS BY DIRECT AND EXTERNAL CARDIAC MASSAGE AFTER CLINICAL DEATH DUE TO VENTRICULAR FIBRILLATION

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V. Ya. Tabak

Laboratory of Experimental Physiology of Resuscitation, USSR Academy of Medical Sciences, Moscow

(Presented by Academician V. V. Parin)
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The restoration of effective cardiac contractions after ventricular fibrillation, as has been demonstrated by a number of investigators [3, 5, 7], depends on the depth of the preceding hypoxia. A single defibrillation may be successful, if the period of effective cardiac standstill does not exceed one to $1^1/_2$ minutes. With longer periods of cardiac standstill it is necessary first to eliminate hypoxia with massage and artificial respiration before defibrillation [3].

In determining the degree of myocardial hypoxia and the work capacity of the heart, analysis of EKG data is of greatest importance. Using this method it is possible, in particular, to resolve the question of the expediency of continuing cardiac massage or the necessity for instituting rapid defibrillation. There is only a single report in the literature which concerns the question of EKG changes during ventricular fibrillation at the time of cardiac massage. Several authors indicate that upon elimination of hypoxia an increase in the amplitude and frequency of the fibrillation waves is observed on the EKG; others note more "lively" fibrillation in these circumstances [1, 2, 5-11].

Our work was designed to study the nature and consequences of EKG changes during ventricular fibrillation occurring while cardiac massage was being carried on, and also to elucidate the optimal electrocardiographic indices for effecting defibrillation.

METHODS

The experiments were performed on 25 adult dogs weighing 12.5 to 20 kg. Before the start of the experiment the animals were given subcutaneous pantopon (calculated from 2 ml of 2% solution per kg of weight). Ventricular fibrillation was induced by a brief (two to three sec) application of alternating current to the heart (127 V).

Direct cardiac massage, without opening the pericardium, at a rate of 50-60 compressions per min was used to resuscitate 15 dogs. After seven to eight min of ventricular fibrillation, direct massage was performed in five dogs for 15 min and in five others for 30 min. In the remaining dogs, in which ventricular fibrillation lasted 10 min, cardiac massage was performed for 15 min.

Ten dogs were resuscitated with external cardiac massage (50-60 compressions of the chest per min); in five dogs after seven min, and in five other dogs after 10 min of fibrillation. Duration of massage was 15 min.

To sustain the arterial pressure above the critical level, 10-15 ml of blood containing adrenalin (0.2-0.5 ml of 0.1% solution) were injected periodically into the artery during direct and external massage. The total amount of injected blood did not exceed 200-250 ml.

Simultaneously with cardiac massage, artificial respiration with a DP-1 apparatus using air mixed with oxygen (40% O_2) is given. Artificial respiration is only stopped after stable, spontaneous respiration is re-established: in

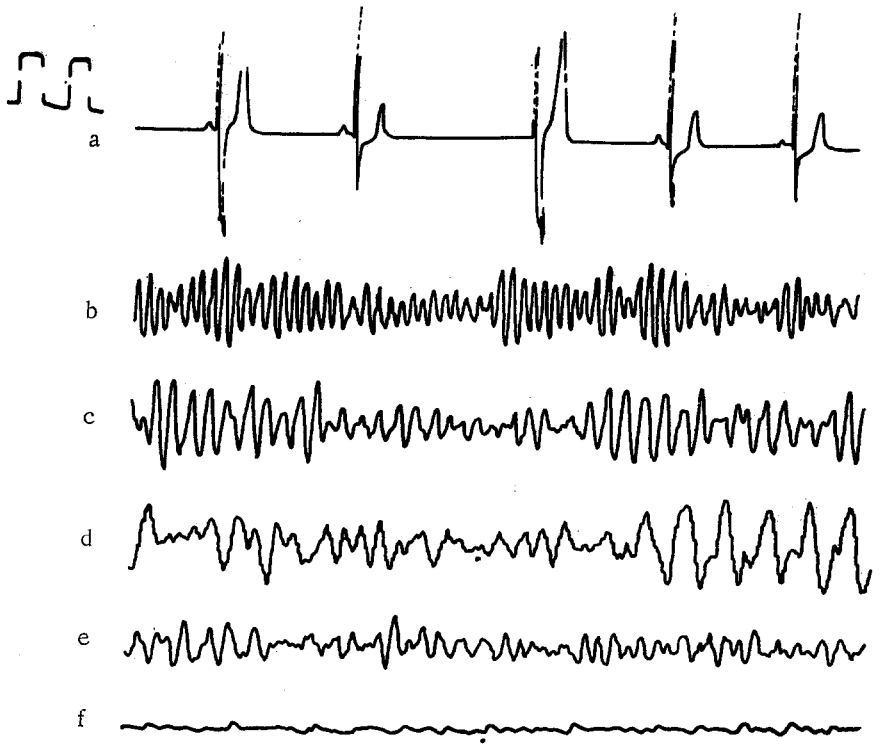

Fig. 1. Dynamics of changes in fibrillatory oscillations on the EKG with death provoked by electroshock. a) At one h after pantopon injection; b) 30 sec. of ventricular fibrillation; c) $1^1/2$ min, ventricular fibrillation; d) $2^1/_2$ min of ventricular fibrillation; e) 4 min of ventricular fibrillation; f) 7 min of the same.

animals subjected to direct massage, after the thorax is closed surgically, in animal resuscitated by external massage, in 2-3 min after defibrillation.

Ventricular fibrillation was converted by discharging a condenser of 24 microfarads and voltage 1500-2500 V (with placement of electrodes on the exposed heart) or 3000-45000 V (placement on the intact thorax).

Arterial pressure and respiration during the experiment were measured with a mercury manometer and pneumatic cuff and were recorded on a moving kymograph tape. Note: EKG was taken on Standard lead II of ink-writing "Al'var" and ELKAR-3' apparatus with standardization of 1 mV = 1 cm.

RESULTS

After pantopon injection, a moderate sinus arrhythmia with cardiac rate of 60-80 per min was recorded on the EKG. In some of the animals an atrioventricular conduction disturbance was observed with single or groups of complexes of multifocal origin (Fig. 1,a).

After electrotrauma, ventricular fibrillation occurred and circulation ceased. During the next 20-30 sec the arterial pressure fell to 20-40 mm, and the corneal reflex disappeared. Respiration ceased at 1-3 min after circulatory collapse.

As hypoxia set in, acute regular changes in the form of ventricular fibrillatory activity was observed in the EKG of all dogs. In the first 25-40 sec after the electroshock, groups of rhythmic sinusoidal waves (oscillations) were recorded, which in short order (0.2-0.5 sec) became transformed into a complete arrhythmia. The frequency of rhythmic oscillations during this period was 800-600 per min, the EKG revealing groups of rhythmic oscillations of moderate amplitude which formed distinctive "spindle" figures (Fig. 1. b).

From the end of the first to the beginning of the third minute the frequency of the rhythmic oscillations gradually decreased (to 400-500 per minute), and the periods of total arrhythmia lengthened (0.5-2 sec). The "spindle" figures disappeared from the EKG (Fig. 1, c). During the third to fourth minute the frequency of rhythmic

oscillations fells to 280-300 per minute. The EKG recorded groups of tall (up to 2 mV) isomorphic mono- or diphasic waves, interspersed with prolonged periods of low polymorphic and arrhythmic oscillation (Fig. 1 d). In the next 1-2 min (that is, at the fifth and sixth min of fibrillation) the amplitude of the rhythmic oscillations decreased to 0.5-0.7 mV, groups of rhythmic waves were recorded more rarely, and the picture of arrhythmia predominated in the EKG (Fig. 1, e). Starting from the fifth to seventh minute of fibrillation the bioelectric activity of the ventricles almost completely disappeared; sometimes the EKG registered rhythmic P waves (Fig. 1 f).

The dynamics of the disappearance of fibrillatory oscillations on the EKG in all animals, without exception, showed that this process proceeds in a regular and sequential manner. The data we obtained confirmed that of earlier investigations [3, 4] on the sequential nature of the development of fibrillation.

After the start of cardiac massage and artificial respiration the arterial pressure rapidly (20-40 sec) rises to 70-130 mm. In most animals during the subsequent 15-30 min of direct and external massage the systolic pressure was maintained at the level 70-110 mm.

Respiration in the majority of dogs was re-established at the second to fifth minute of massage (single breaths), the corneal reflex returned at the sixth to 15th min. In three dogs artificial respiration was ineffective. The arterial pressure in these dogs was low during massage (40-60 mm) and the first spontaneous breaths appeared late (at the eighth to 12th min).

Toward the end of the first and beginning of the second minute of resuscitation the EKG revealed rare groups of rhythmic oscillations with a frequency of 400-500 per min and amplitude of 0.3-0.8 mV. In this period the picture of total arrhythmia predominated on the EKG (Fig. 2, a). After three minutes of massage the amplitude of the rhythmic oscillations had already risen to 1-1.5 mV and the periods of arrhythmia shortened, but still predominated over periods of regular rhythm (Fig. 2, b). In dogs which had passed the 7-8th min of cardiac standstill, toward the 5-7th min of massage (direct and external) the amplitude of the rhythmic oscillations reached a maximum (1.5-2.5 mV) and their frequency varied from 500-700 per min, a regular rhythm predominated over arrhythmia and the EKG registered "spindle" figures (Fig. 2, c). In animals past the 10th min of cardiac arrest, a similar EKG picture was observed later on—at the 10-12th min of massage. Beginning at this period and lasting until the cessation of massage the total EKG pattern did not change substantially (Fig. 2, d).

In three dogs, in which artificial respiration was ineffective during cardiac massage, the EKG during the first 6-10 min of massage recorded low polymorphic and mainly arrhythmic oscillations: groups of slow, tall waves with a frequency around 300 per min. The low oscillations (0.5 to 1.2 mV) remained on the EKG for the duration of the arrhythmia period in these animals until the cessation of massage.

In these experiments we did not note essential difference in the EKG picture and the dynamics of its development in animals which were revived by direct as opposed to external cardiac massage, except in that circumstance in which the amplitudes of the oscillations reached greater values in direct massage and their frequency was lower. No significant EKG effect of intra-arterial injection of blood containing adrenalin was noted.

Coordinated myocardial contractions in the majority of dogs reappeared after electrical defibrillation of the heart. In three dogs, in which artificial respiration was ineffective, cardiac activity was restored only after prolongation of cardiac massage for 2-3 additional minutes.

Out of 15 dogs which underwent direct cardiac massage, nine subsequently survived: four—after 7-8 min of fibrillation and 15 min of massage, three—after 30 min of massage, and two—after 10 min of fibrillation and 15 min of massage. Among the dead animals were the three in which ineffective ventilation occurred during the massage period. Out of 10 dogs which had 15 min of external cardiac massage, seven lived: four—after seven min and three—after 10 min of ventricular fibrillation.

In the first hours after the experiment varied atrioventricular and intraventricular conduction disturbances, signifying myocardial hypoxia (Fig. 2, e, f) were observed on EKG. The EKG changes in dogs after 15 min of external massage (for a single period of cardiac arrest) were significantly less prominent than in dogs which had direct cardiac massage of an equal duration. The most serious arrhythmias and abnormal complexes were observed in animals after 30 min of direct massage. In the surviving animals normalization of the EKG began in three to seven days (after 15 min of external massage) or 10-15 days (after direct massage).

Analysis of these results showed that correction of hypoxia lead to a regular increase in the frequency and amplitude of the fibrillatory oscillations, the EKG resembling that observed in the first 20-40 sec after electroshock.

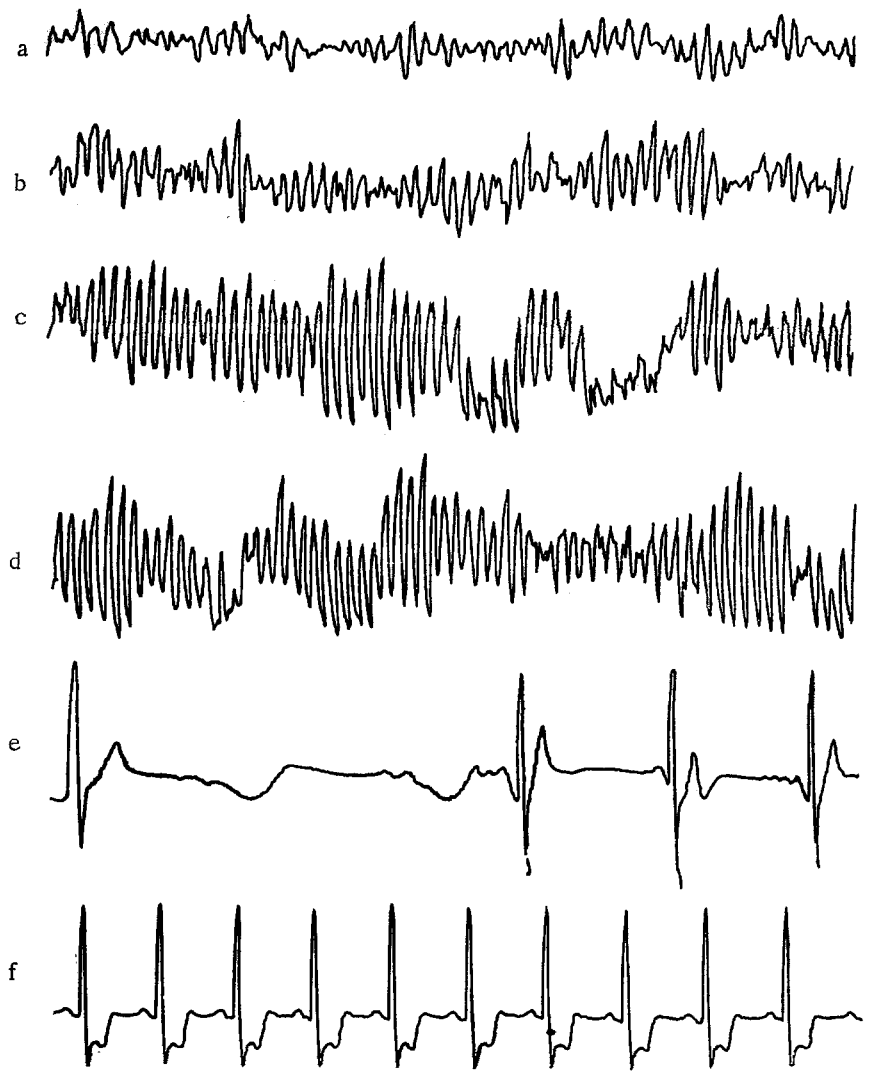

Fig. 2. Dynamics of changes in fibrillatory oscillations on the EKG during direct cardiac massage (15 min) and artificial respiration . a) $1^1/_2$ min of cardiac massage; b) $3^1/_2$ min of cardiac massage; c) 7 min of cardiac massage; d) 15 min of cardiac massage; e) EKG of dog after 10 seconds post defibrillation; f) EKG at one hour post closure of thorax and restoration of spontaneous respiration.

By the 5-12th min of cardiac massage (depending on the period of preceding hypoxia) relative stabilization of the EKG occurred, which did not change further until the end of massage.

This suggests that effective cardiac activity may be re-established, if the EKG directly before defibrillation records mainly regular sinusoidal waves with a frequency of 500-700 per min. This is confirmed by the fact that in animals with uncorrected hypoxia (as a result of ineffective artificial respiration) defibrillation occurred against a setting of relatively low arrhythmic oscillations and cardiac activity was restored by auxiliary massage and arterial pumping of blood.

These data confirm the close dependence of the electrocardiographic picture during cardiac massage on the period of preceding hypoxia, the duration of massage and the effectivity of the entire complex of resuscitative measures.

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All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. *Some or all of this periodical literature may well be available in English translation.* A complete list of the cover-to-cover English translations appears at the back of this issue.
