ECG CHANGES DURING PROLONGED CLINICAL DEATH AND SUBSEQUENT RESUSCITATION

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An artificial circulation is a more effective means of resuscitation after prolonged clinical death (8-12 min) than intra-arterial blood transfusion. General anesthesia also promotes the more rapid recovery of cardiac functions.

Changes in the ECG during resuscitation of dogs after clinical death for 5 min have been described previously [1]. In particular, the phenomenon of a gradual increase in duration of the ventricular complex before restoration of efficient working of the heart has been demonstrated.

The object of this investigation was to study changes in the ECG during prolonged clinical death (10 min) and subsequent resuscitation by various methods.

EXPERIMENTAL METHOD

Experiments were carried out on 33 dogs. Before the experiment, all animals received a subcutaneous injection of pantopon in a dose of 8 mg/kg, and 14 dogs in addition were anesthetized with nembutal (10-20 mg/kg). Clinical death was induced by blood loss. Nine unanesthetized and one anesthetized dogs were resuscitated by intra-arterial blood transfusion, and the other 23 dogs (10 unanesthetized and 13 anesthetized) by an artificial circulation using the AIK-RP-64 apparatus. A type RPR apparatus provided artificial respiration.

EXPERIMENTAL RESULTS

ECG changes during blood loss and clinical death were determined by the effect of the general anesthesia and by individual differences between the animals. After a period of sinus tachycardia at the beginning of blood loss, sino-atrial block developed, leading to the appearance of nodal and idioventricular rhythms. In unanesthetized dogs this period coincided with the respiratory (terminal) pause and it ended at the beginning of agony. In the anesthetized dogs, inhibition of the sinus activity frequently continued until the second minute of clinical death.

The second and final cessation of sinus rhythm occurred in most animals at the third to fourth minute of clinical death. Usually it was preceded by a transient incomplete, followed by complete, atrio-ventricular block. After the cessation of the sinus rhythm, ventricular complexes of nodal origin continued to be recorded on the ECG, and gradually assumed the shape of monophasic waves. Some animals developed asystole at the fifth to seventh minute of clinical death. In others, atrial complexes continued to be recorded until the beginning of resuscitation, along with ventricular complexes, although the two were completely dissociated.

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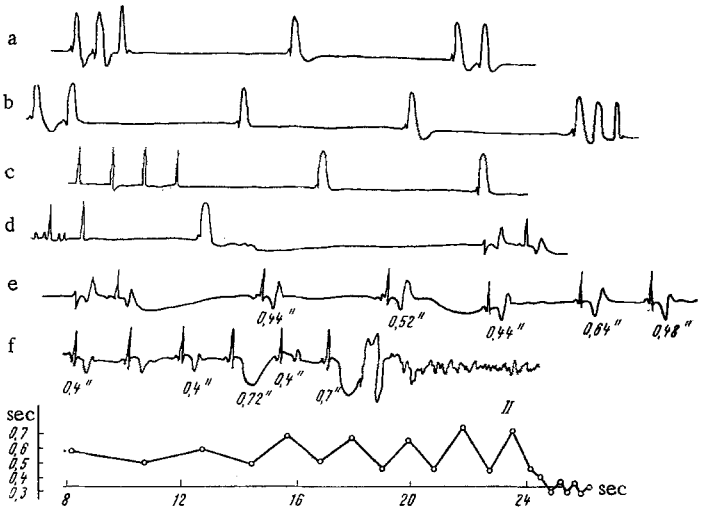

Fig. 1. Changes in type and duration of ventricular complex on ECG during resuscitation after prolonged clinical death: I) ECG in lead II from experimental dog No. 5480 during clinical death and beginning of resuscitation (intra-arterial blood transfusion); a, b, c) at 6th, 7th and 9th min of clinical death respectively. Monophasic and more prolonged biphasic ventricular complexes recorded on ECG; sometimes groups of two and three complexes appear (recording speed 25 mm/sec; in cuts c and d, on the left, 5 mm/sec); d, e, f) continuous recording from beginning of intra-arterial blood transfusion (marked by arrow). Increasing alternation of duration of ventricular complex to 0.4-0.72 sec visible on ECG, before beginning of fibrillation. II) Duration of 13 ventricular complexes before beginning of ventricular fibrillation. Abscissa, time from beginning of resuscitation (in sec); ordinate, duration of ventricular complex (in sec).

In anesthetized dogs the ECG changes during clinical death, as well as during the period of its induction, were somewhat reduced in frequency. The ventricular complex in these dogs retained its specific form until the seventh to eighth minute of clinical death, and sometimes until the beginning of resuscitation.

The character of the ECG changes during resuscitation depended both on the anesthesia and on the method of resuscitation. In unanesthetized dogs resuscitated by means of the artificial circulation, the sinus rhythm was restored after 7.5 ± 1.3 sec, compared with 10.5 ± 1.8 sec in animals resuscitated by intra-arterial blood transfusion. In anesthetized dogs resuscitated by means of the artificial circulation, sinus rhythm was restored even later, after 12.4 ± 1.2 sec, although efficient working of the heart was restored sooner in these animals (after 37 ± 3.4 sec) than in the unanesthetized dogs (after 61 ± 10.7 sec).

Ventricular fibrillation developed in eight of the 10 dogs during intra-arterial blood transfusion, and cardiac activity was restored late, 1.5-4.5 min after defibrillation. Of the 23 dogs resuscitated by the artificial circulation (with or without anesthesia), fibrillation occurred in only two.

In dogs in which sinus rhythm did not cease during clinical death, atrio-ventricular conduction was restored quicker than in other dogs (after 5-7 sec). It is interesting to note that the two dogs in which sinus rhythm persisted until the beginning of resuscitation were the only ones from the group resuscitated by intra-arterial blood transfusion which did not develop ventricular fibrillation.

From the beginning of resuscitation until restoration of cardiac activity there was a considerable increase in duration of the ventricular complex although it exhibited definite alternation. During resuscitation of dog No. 5480 the following changes occurred in the duration of the ventricular complex: 0.48, 0.44, 0.52,

0.44, 0.64, 0.48, 0.64, 0.4, 0.6, 0.4, 0.72, 0.4, and 0.7 sec (Fig. 1). Alternation of this type was observed in nearly all dogs regardless of the method of resuscitation, and it was evidently due to the disturbance of intraventricular conduction after the prolonged hypoxia. During resuscitation of dogs after clinical death for 5 min, the increase in duration of the ventricular complex took place gradually from one complex to the next without alternation [1].

The results of these experiments show that the artificial circulation is a more effective method of resuscitation after prolonged clinical death than intra-arterial blood transfusion. The reason for the frequent onset of ventricular fibrillation during intra-arterial blood transfusion was that this method does not provide an adequate blood supply to the coronary vessels (about 50 ml/mg/min compared with 100-120 ml/mg/min in the case of resuscitation by the artificial circulation). As a result of this, the chambers of the heart become overdistended with blood before myocardial function is restored, thus leading to the development of fibrillation. This explanation does not contradict the well known fact that successful resuscitation can be achieved by intra-arterial blood transfusion after brief (5-6 min) clinical death: if the degree of hypoxia is slight, effective contractions of the heart begin to take place early, and it does not become over-distended with blood.

Another factor leading to the frequent onset of fibrillation during intra-arterial blood transfusion of the prolonged clinical death is the disturbance of intraventricular conduction, as shown by the well-defined alternation in shape and duration of the ventricular complex. The comparatively rare occurrence of ventricular fibrillation during resuscitation by the artificial circulation is comparable with the frequency of this complication (in about 10% of cases) during resuscitation by intra-arterial blood transfusion after a shorter period of clinical death (5 min) [2, 3].

The action of anesthesia was not only to delay the disturbance of intraventricular conduction during clinical death, but it also had a favorable effect on the outcome of resuscitation. Of the 14 anesthetized dogs resuscitated by the artificial circulation, 10 survived, compared with only two of the 10 unanesthetized dogs resuscitated in the same way ($P < 0.01$). To this it must be added that, of the 10 dogs of the control group, one survived, the only one of the group which was anesthetized during blood loss.

Changes in the ECG during clinical death thus accurately reflect the degree of myocardial hypoxia and their prognostic significance as regards the outcome of resuscitation is not less than that of the chronometric measurements of the duration of clinical death.

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