ON THE RESTORATION OF THE NEUROREFLEX REGULATION OF THE CARDIOVASCULAR SYSTEM DURING RESUSCITATION AFTER CLINICAL DEATH

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The stability of the restoration of the cardiovascular system during resuscitation is closely connected with the restoration of the central nervous system and the resumption of the neuroreflex regulation [10, 12, 19]. In the beginning of the resuscitation after clinical death only elementary reflex reactions can be observed in the cardiovascular system [12, 18, 19]. A higher level of neuroreflex regulation can be observed after the function of the respiratory center has been restored [10, 11, 12].

It was the aim of the present paper to study the restoration of the neuroreflex regulation in the cardiovascular system during resuscitation after clinical death, caused in various ways. We paid particular attention to changes in the regulation of the cardiovascular system, connected with the restoration of the respiratory function.

EXPERIMENTAL METHOD

The experiments were carried out on adult dogs, weighing 10-15 kg. Before the actual experiment we prepared the femoral blood vessels, under pantopon-ether narcosis or under local anesthesia, with 0.5% novocain solution. In 35 dogs the clinical death was brought about by bleeding, in 18 dogs by mechanical asphyxia and in 15 dogs by electric trauma (induction of cardiac fibrillation by connecting the heart with the alternating current mains at a tension of 127 V). Death occurred in the dogs which were bled out within ~38 rain, and in cases of mechanical asphyxia or electric trauma within 3-10 min. In various experiments the state of clinical death lasted between 1 and 15 min. The dogs were resuscitated by the method developed by V. A. Negovskii and co-workers [7, 10, 12]. During the experiments we recorded the arterial blood pressure, the electrocardiogram and the respiratory movements of the chest. The early stages in the stimulation of the respiratory center, which became manifest in movements of the tongue and neck muscles were marked on the kymogram with crosses.

EXPERIMENTAL RESULTS

The appearance of the first respiratory movements during the resuscitation was accompanied by reactions of the cardiovascular system, reactions which varied depending on the speed at which the death had occurred and on the duration of the state of clinical death. In this context the cause of death was of secondary importance. If the death had occurred slowly (more than 15 min) or if the state of clinical death had lasted for a long period (over 6 min) the excitation of the respiratory center became initially manifest only in movements of the tongue and neck muscles. The response of the cardiovascular system to these movements became manifest in a shortlasting rise in the arterial blood pressure without changes in the cardiac rhythm or in the shape of the electrocardiogram. As soon as the respiratory movements of the chest had been resumed, the character of the cardiovascular response to the act of inspiration changed: instead of an increase a temporary fall could be observed in the arterial blood pressure (Fig. 1 b). In the electrocardiogram a slowing down of the pulse rate instead of the usual inspiratory tachycardia could be observed, the atrial P wave was flattened and the atrio-ventricular conduction time was prolonged (Fig. 2 c). Parallel to the increase in the depth of the respiratory movements the above signs became more marked. Simultaneously the mean arterial blood pressure rose and the heart rate became less frequent.

3-7 min after the resumption of the respiration the mean arterial blood pressure decreased and the respiratory variations in the blood pressure became less intensive (Fig. 1 c, d). The inspiratory bradycardia became gradually
less marked and was later replaced by the usual inspiratory tachycardia (Fig. 2 d). Simultaneously with the restoration of the normal type of respiratory arrhythmia the electrocardiographic changes connected with the respiration reverted to their normal character (greater amplitude of P-wave and shortening of the P-Q interval during inspiration). The normal response of the cardiovascular system to the act of inspiration was restored within 15-30 min after the beginning of the resuscitation.

If the hypoxia had been less marked (death occurring within 10 min and clinical death lasting not longer than 6 min) the initial excitation of the respiratory center became immediately manifest in chest movements. In this case the response to inspiration began directly with a fall in the arterial blood pressure and a slowing down of the heart rate. The subsequent changes were similar to those described above. The usual cardiovascular response to inspiration was restored 7-15 min after the beginning of the resuscitation.

As soon as the reflexes to pain and the tendon reflexes were restored—particularly after prolonged hypoxia—external stimuli elicited, in addition to the adequate response, an unusually strong cardiovascular response, consisting in a marked increase of the arterial blood pressure and of the heart rate. After the reappearance of the pupillary reflex, i.e., with the beginning restoration of the mesencephalic function, the pathological character of the cardiovascular response to external stimuli became less marked. The inadequate character of the response, however, persisted for several days after the experiment.

Full restoration of the nervous regulation of the cardiovascular system occurred, after a short dying process and a shortlasting state of clinical death, within 1-2 weeks; if death occurred more slowly, or if the state of clinical death was prolonged, within 4 weeks or later. Other authors observed similar periods [9, 21].