

RESUSCITATION OF ANIMALS WITH EXPERIMENTAL MYOCARDIAL INFARCTION

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A perfectly viable and apparently healthy organism may die from a number of causes (electric shock, asphyxia, blood loss). Advances in the theory and practice of resuscitation, particularly during recent years, have provided a means of treatment of unexpected death from these causes, and the vital functions of the dying organism may sometimes be restored. Moreover, cases have now been described, still only few in number, in which clinical death developing as a result of a lesion of vital organs, including the heart, has been successfully overcome [5, 11, 17, 19, 22]. Nowadays cardiovascular diseases are those most widely occurring, and a foremost place among them is held by coronary arterial disease, especially myocardial infarction [18]. The mortality from this disease remains very high, and amounts to 7-15% among patients admitted to hospital [1, 9, 10].

The possibility of restoring the vital functions, and notably the cardiac activity, in patients with myocardial infarction remains a largely unsolved problem, and is the subject of much discussion. A textbook of physiology published in 1954 contains the statement that the restoration of adequate cardiac activity is generally impossible if the myocardium is affected by pathological changes [3]. Wiggers [24, 25] concluded from his experiments that ventricular fibrillation arising after occlusion of a coronary artery is an irreversible condition until the occlusion has been relieved. Nevertheless, the further study of this problem in experimental and clinical conditions [6, 7, 8, 14, 15, 16, 21, 23] has shown that such statements are largely meaningless, although the restoration of vital functions in such cases is, in fact, attended with considerable difficulty. In itself, the presence of a myocardial infarct cannot be a criterion of the expectedness or irreversibility of the terminal state, for the pathological changes found in this condition may be minimal [1, 4, 10, 26], and there is no doubt about the vast powers of compensation of the heart muscle [1, 2, 9, 12, 13]. The situation of the lesion must, of course, be taken into account.

The object of this experimental study was to investigate the possibility of restoration of the cardiac activity and the other vital functions of animals dying in the acute and subacute periods and in the period of recovery after an extensive experimental myocardial infarct.

EXPERIMENTAL METHOD

Nineteen experiments were performed on 10 dogs of both sexes weighing from 11 to 18 kg. Thoracotomy was performed in the fifth left intercostal space under intravenous nembutal anesthesia with controlled respiration (3 dogs) or ether-oxygen intubational anesthesia (7 dogs). The pericardium was incised parallel to the phrenic nerve. The descending branch of the left coronary artery was mobilized and ligated at a distance of 0.5-0.7 cm from the border of the left auricle (roughly at the site of origin of the septal branch of this artery). Almost at once a well-defined cyanotic area developed in the myocardium, corresponding in size to the area supplied by the branches of the ligated artery and occupying a considerable part of the anterolateral region of the left ventricle. Immediately after ligation of the artery, administration of the anesthetic ceased, and the dogs' lungs were forcibly ventilated for a few minutes with pure oxygen. Three of the 10 dogs spontaneously developed ventricular fibrillation 1.5-3 min after application of the ligature. If fibrillation had not developed 15-20 min after ligation, the ligature was removed and reapplied. Another two dogs developed ventricular fibrillation when the ligature was removed. Fibrillation was produced in the other 5 animals by electric shock from the 127 V alternating current mains supply after application of the ligature, for the repeated removal and reapplication of the ligature did not cause the development



Fig. 1. Preservation of individual aberrant complexes during ventricular asystole following defibrillation. Resumption of fibrillation at the beginning of cardiac massage. The arrow denotes the beginning of massage.

of fibrillation. Resuscitation measures were put in hand on the day the ligature was applied, and also 10 and 30 days thereafter; these consisted of direct and indirect cardiac massage, artificial respiration with oxygen or the ordinary air of the room by means of a Pesty RPR volume respirator, condenser pulse defibrillation (by N. L. Gurvich's method), and intra-arterial injections of small volumes of a 5% solution of glucose with adrenalin in the experiments involving indirect cardiac massage. Defibrillation was performed after restoration of the tone of the heart muscle and the appearance of active ventricular fibrillation in the ECG.

In 5 dogs resuscitation was carried out more than once: in the period of acute myocardial ischemia, and then on the 10th (in all five) and 30th days (in three of them) after application of the ligature to the left coronary artery.

EXPERIMENTAL RESULTS

In the first group (10 experiments) the possibility of restoration of the cardiac activity and of the other vital functions of animals dying in the acute period of myocardial ischemia was studied. The period of circulatory arrest varied from 1 to 4.5 min in the first five experiments and 5 min in the subsequent experiments. In three experiments in which the circulation was halted for 5 min, after fibrillation for 3.5 min a condenser discharge of 2500-3000 V was passed through the heart in order to produce experimental ventricular asystole against the background of experimental myocardial ischemia. It should be noted that although no cardiac contractions were present under these circumstances, individual aberrant complexes were recorded in the ECG (Fig 1.).

Resuscitation measures consisted of a combination of those described. In a series of experiments small doses of adrenalin (0.1-0.5 ml of a 1 : 1000 solution) were injected directly into the cavity of the right ventricle during direct massage to reactivate the tone of the myocardium. Fibrillation was abolished after the first electrical discharge in only two experiments; in the remaining 8 experiments from 3 to 17 discharges were required for this purpose. As a rule the cardiac activity was well restored after this procedure, and only in isolated cases was it necessary to give intravenous injections of small doses of calcium chloride to strengthen the contractions. Complete restoration of all the vital functions was obtained in all the dogs, and only one animal died 24 h after the experiment from sudden ventricular fibrillation (during strapping the dog to the bench for recording the ECG).

In the second group (5 experiments) on dogs with myocardial infarction of 10 days' duration, when the animals' condition was fully restored after the operation and resuscitation an electric shock from the mains supply was applied in order to produce a second attack of ventricular fibrillation. After the circulation had been arrested for 5 min, indirect massage of the fibrillating heart was begun, together with artificial respiration and intra-arterial injection of frequent small volumes of 5% glucose solution with adrenalin. External electrical defibrillation was carried out after indirect massage lasting 1-4 min, by means of the application of 1-3 condenser discharges of 3500-5000 V each. It was impossible to restore the cardiac activity in only one dog, on which cardiac massage was continued for 1 h 8 min (direct massage for the last 5 min) and defibrillation was repeated 36 times. Full recovery of all the vital functions was obtained in the remaining four dogs.

In the third group of experiments on 4 dogs with myocardial infarction of 1 month's duration and two previous courses of resuscitation, in 3 of the animals ventricular fibrillation was again produced by electric shock. The duration of the circulatory arrest in all cases was 5 min. The resuscitation measures were similar to those used in the



Fig. 2. Heart of a dog resuscitated twice -- on the 1st and 10th days of development of a myocardial infarct.

animals in the second group of experiments. In 3 dogs complete recovery of all the vital functions was obtained after indirect cardiac massage for 1-4 min and after 1-3 application of external defibrillation. In one dog, despite the successful abolition of ventricular fibrillation, adequate cardiac activity could not be restored and the experiment was terminated after a further 30 minutes' cardiac massage (during which the tone of the heart remained good).

Pathological investigation (N. P. Romanova) of the hearts of dogs sacrificed at periods of between 7 days and 2 months after resuscitation revealed, in every case, an extensive infarct of the myocardium of the left ventricle with the formation of a cardiac aneurysm in two dogs (Fig. 2). Isolated hemorrhages at the borders of the zone of myocardial ischemia could be either the result of trauma during massage or a sign of the development of the pathological process itself.

Hence, in animals with extensive myocardial ischemia and infarction in the region of the anterolateral wall of the left ventricle, it is possible to restore the cardiac activity, followed by all the other vital functions, by timely and properly applied measures. After short periods of both direct and indirect cardiac massage, no significant traumatic lesions of the affected area of heart muscle were observed so that this method of restoring cardiac activity may be recommended unreservedly for use in clinical death due to coronary disease. The dimensions of the ischemia and of the myocardial infarct themselves cannot, it seems, be used as criteria for the indication or contraindication of resuscitation measures, particularly because in emergency it is usually impossible to determine with any accuracy the character and degree of the lesion of the heart muscle.

SUMMARY

A study was made of the possibility of restoring the vital functions in dogs with experimental extensive myocardial infarction of the left ventricle, cause by preliminary ligation of the descending branch of the left coronary artery. As demonstrated, restoration of the cardiac activity with the aid of massage of the heart (direct and indirect), artificial respiration and defibrillation is quite possible at various stages of the development of infarction.

LITERATURE CITED

1. A. N. Berinskaya, N. V. Kalinina, and T. I. Meerzon, Outcome and Prognosis of Myocardial Infarction [in Russian], Moscow (1958).
2. I. M. Gol'dberg and E. M. Komina, Problems in Shock and Terminal States [in Russian], p. 45, Voronezh (1958).
3. G. P. Konradi, Textbook of Physiology [in Russian], p. 76, Moscow (1954).
4. T. A. Naddachin, Abstracts of Proceedings of a Jubilee Scientific Session of the Skliforsovskii Institute [in Russian], p. 36, Moscow (1960).
5. V. A. Negovskii, Klin. med., 2, 3 (1963).
6. V. S. Ryshkin, Abstracts of Proceedings of a Scientific Session on the Problem: Cardiovascular Diseases, Their Prevention and Treatment [in Russian], p. 168, Moscow (1962).
7. A. I. Smallis and T. Norkus, Okhrana zdorov'ya, 6, 29 (1961).
8. Yu. S. Chechulin, Abstracts of Proceedings of the 7th Scientific Session on the Problem of Cardiovascular Pathology [in Russian], p. 136, Tbilisi (1961).
9. S. V. Shestakov, Angina Pectoris and Myocardial Infarction [in Russian], Moscow (1962).
10. L. Adelson and W. Hoffman, J.A.M.A. (1961), 176, p. 129.
11. P. R. Brass and R. E. Kendall, Brit. med. J. (1961), 1, p. 18.
12. B. Ersten and S. Proger, J.A.M.A. (1955), 159, p. 845.
13. A. Kagan, Circulation (1952), 5, p. 816.
14. J. I. Kessler and E. Senderoff, J. clin. invest. (1962), 41, p. 1531.
15. P. Kokkalis and G. Schwarz, Thoraxchirurgie (1961), 9, p. 400.
16. E. Milch et al., Am. Heart. J. (1955), 50, p. 483.
17. S. N. Nickel and H. H. Gale, J.A.M.A. (1959), 170, p. 23.
18. M. Plotz, Koronarnaya bolezni', Moscow (1961).

19. L. B. Reagan, K. R. Young, and J. W. Nicholson, *Surgery* (1956), 39, p. 482.
20. E. Senderoff, A. E. Welberry, M. Kaneko et al., *Ann. Surg.* (1960), 151, p. 193.
21. S. E. Stephenson et al., *Am. J. Cardiol.* (1960), 5, p. 77.
22. W. M. Straight, R. Litwak, and J. C. Turner, *Ann. intern. Med.* (1961), 54, p. 566.
23. F. M. Weiser, L. N. Adler, and L. A. Kuhn, *Am. J. Cardiol.* (1962), 10, p. 555.
24. C. J. Wiggers, *Am. Heart, J.* (1950), 20, p. 399.
25. C. J. Wiggers, R. Wegria, and B. Pinera, *Am. J. Physiol.* (1940), 131, p. 309.
26. W. M. Uater, P. P. Welsh, J. F. Stapleton et al., *Ann. intern. Med.* (1951), 34, p. 352.

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.
