OXYGEN CONSUMPTION OF THE MYOCARDIUM IN CONDITIONS OF NORMO- AND HYPOTHERMIC CORONARY PERFUSION AND A GENERAL ARTIFICIAL CIRCULATION

(UDC 616.12-78-07:616.127-008.922.1]:[616.151:612.59])

D. D. Bershadenko and E. N. Ashcheulova

Division of Acquired Heart Lesions (Head, Professor S. A Kolesnikov)
and Artificial Circulation Laboratory (Head, Docent V. S. Raevskii),
Institute of Cardiovascular Surgery (Director, Professor S. A. Kolesnikov,
Scientific Director, Academician A. N. Bakulev), Academy of Medical Sciences
of the USSR, Moscow
(Presented by Active Member of the Academy of Medical Sciences of the USSR A. N. Bakulev)
Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 61, No. 6,
pp. 23-27, June, 1966
Original article submitted May 25, 1965

The open correction of acquired defects of the aortic valve is dependent on the possibility of excluding the heart from the circulation. The problem of maintaining the viability of the heart when excluded from the circulation is thus of extreme importance. Most surgeons favor the isolated perfusion of the coronary arteries. However, authors differ in regard to the conditions in which they perform coronary perfusion [3, 9, 13, 14].

To determine the influence of temperature, the state of the heart, and other factors on the energy expenditure of the heart muscle, a comparative experimental study was made of the oxygen consumption of the myocardium of different conditions of cardiac activity, in the conditions of normothermic and hypothermic coronary perfusion and of a general artificial circulation.

EXPERIMENTAL METHOD

Two series of experiments were carried out. In series I (12 experiments) isolated coronary perfusion was performed for 60 min in artificial circulation conditions and general hypothermia (22-27°), with hypothermic arrest (fibrillation) of the heart. In series II (10 experiments) isolated coronary perfusion of the same duration was carried out in artificial circulation conditions and in normothermia, with electrical arrest (fibrillation) of the heart.

Experiments were conducted on dogs of both sexes and different ages, with a mean weight of 20 kg. The animals were given a subcutaneous injection of omnopon (2.5-3.5 mg/kg) 40 min before the operation. Anesthesia was induced with thiopental sodium (100-200 mg) and maintained with ether and oxygen, given by intubation, together with the use of short-acting relaxants in the course of the operation. An artificial circulation was provided by a Crawford-Senning apparatus, giving a volume perfusion velocity of 80-110 ml/min/kg body weight. Oxygen was supplied to the oxygenator at the rate of 10 liters/min, and carbon dioxide at the rate of 3-5% of the volume of oxygen. The artificial circulation apparatus was connected by means of separate cannulas to the venae cavae and the right femoral artery. Both ventricles were drained. With the pulmonary artery occluded, all the coronary venous blood was drained from the right ventricle, and from the left—all the blood entering through the aortic valve, the bronchial arteries, and the vessels of Vieussens and Thebesius. Electrical cardiac arrest (in the experiments of series II) was carried out by a galvanic current with a strength of 10-25 mA for 3-5 sec. After arrest of the heart (hypothermic or electrical), the ascending aorta was clamped and, by means of a simple suspended perfusion system, perfusion of the isolated coronary circulation was started by puncture of the ascending aorta below the clamp.

Blood samples were taken from the femoral artery and coronary sinus or the right ventricle (in the course of coronary perfusion and a complete artificial circulation) at the following stages: I—before the artificial circulation

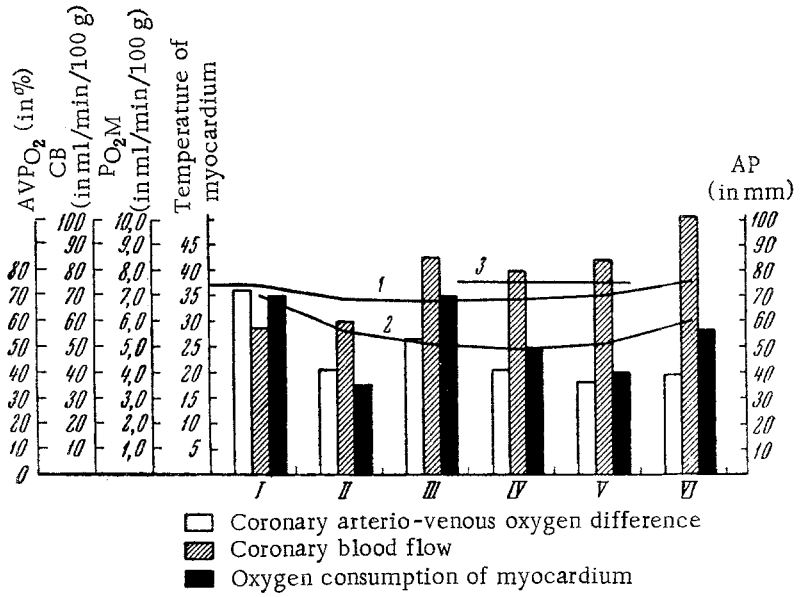

Fig. 1. Arterio-venous oxygen difference, volume velocity of the coronary blood flow, and oxygen consumption of the myocardium in the conditions of an artificial circulation and hypothermic coronary perfusion (hypothermic arrest of the heart). Here and in Fig. 2: I) before artificial circulation; II) during artificial circulation; III) at limit of hypothermia (after cardiac arrest); IV) after coronary perfusion for 30 min; V) after coronary perfusion for 60 min; VI) at the height of reheating; VII) after restoration of cardiac contractions; 1) temperature of the heart; 2) arterial pressure; 3) pressure in the coronary perfusion system; AP) arterial pressure.

began; II—against the background of the artificial circulation with a normal temperature and a rhythmically contracting heart; III—after arrest of the heart; IV—30 min after the start of coronary perfusion; V—60 min after the start of coronary perfusion; VI—at the height of reheating (in the experiments of series I); VII—after restoration of the cardiac activity, and VIII—after disconnecting the apparatus (in some experiments).

To measure the coronary blood flow, the coronary sinus was cannulated. Since only part of the coronary venous blood was collected in this way, in fact about 60%, the result was converted to the equivalent of 100%.

Since the metabolism of the myocardium is mainly an aerobic process, the oxygen consumption may be used as an index of the intensity of the energy-forming processes in the heart when in different functional states.

To determine the oxygen consumption of the myocardium, the degree of oxygen saturation of the hemoglobin of the arterial and venous coronary blood, the volume velocity of the coronary blood flow, and the hemoglobin concentration in the blood were investigated.

The oxygen consumption of the myocardium was calculated by the formula suggested by I. E. Kisin [1]: $P_{O_2}M = 1.34 \cdot Hb \cdot AVP_{O_2} \cdot CB \cdot 10^{-4}$, where $P_{O_2}M$ is the oxygen consumption of the myocardium (in ml/min/100 g weight of the heart); 1.34 is the volume of oxygen bound with 1 g hemoglobin (in ml); Hb is the hemoglobin content of 1 ml of blood; AVP_{O_2} is the arterio-venous oxygen difference (in % HbO_2); and CB is the coronary blood flow (in ml/min/100 g weight of the heart).

EXPERIMENTAL RESULTS

The results of the experiments in which coronary perfusion was carried out in hypothermic conditions are given in Fig. 1. These results show that the value of $P_{O_2}M$ for normal working of the heart before connection of the artificial circulation apparatus, with the chest open, and under general anesthesia was 7 ml/min/100 g, when the coronary blood flow was 57.3 ml/min/100 g and the value of AVP_{O_2} was 72.4%. With a complete artificial circulation, the value of $P_{O_2}M$ of the empty, rhythmically contracting heart, in the absence of drainage of the left ventricle, fell by comparison with the first stage to 3.5 ml/min/100 g, i.e., to 50% of the initial level, with a slight

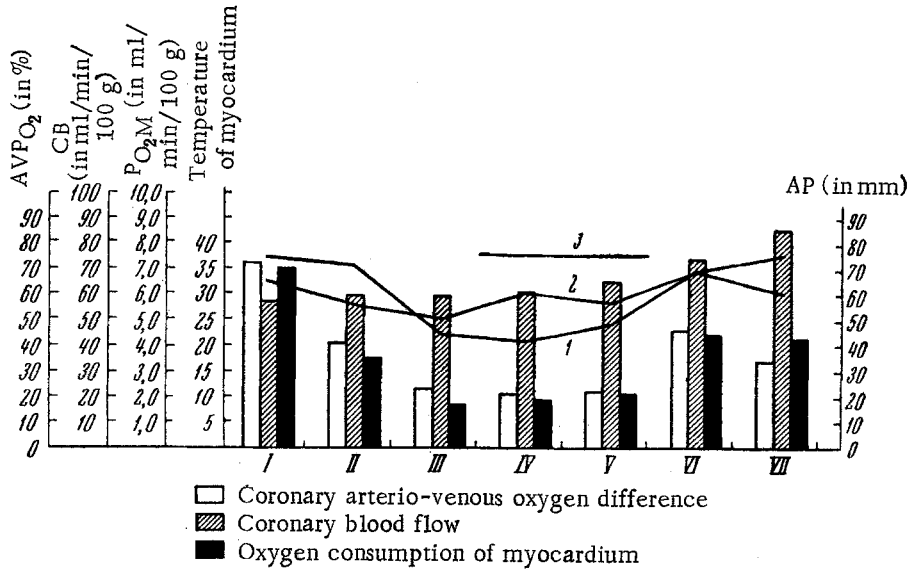

Fig. 2. Arterio-venous oxygen difference, volume velocity of the coronary blood flow, and oxygen consumption of the myocardium in the conditions of an artificial circulation and normothermic coronary perfusion (electrical arrest of the heart).

increase in CB to 59.1 ml/min/100 g and a considerable fall of AVP_{O_2} to 40.8%. In stage III, after the onset of hypothermic cardiac arrest (fibrillation), when the temperature of the heart was 22° on the average, P_{O_2M} fell to 1.67 ml/min/100 g, i.e., approximately to 24% of the initial level. In these circumstances the value of AVP_{O_2} fell to 22.6%, while the coronary blood flow remained unchanged—58 ml/min/100 g.

The value of P_{O_2M}, AVP_{O_2}, and CB remained almost unchanged 30 and 60 min after the start of coronary perfusion. The slight increase in the coronary blood flow and in the oxygen consumption of the myocardium towards the end of coronary perfusion was due to a very small increase in the temperature of the heart to 24.4°.

Hence, the factor determining the oxygen consumption of the myocardium at these three stages was the temperature of the myocardium and not the duration of the artificial circulation or coronary perfusion.

At the height of reheating, against the background of violent fibrillation of the heart, with a complete artificial circulation and at a mean temperature of 34.8°, the value of P_{O_2M} rose to 4.4 ml/min/100 g and that of AVP_{O_2} to 46.1%. Consequently, the level of P_{O_2M} was higher than that of the empty, rhythmically contracting heat at stage II.

After restoration of the cardiac activity, the value of P_{O_2M} remained practically unchanged from the preceding stage. It was 4.3 ml/min/100 g, while at the same time AVP_{O_2} had fallen to 34.2% and CB had risen to 85.2 ml/min/100 g.

The results of the investigations of series II with isolated coronary perfusion in the conditions of a normothermic artificial circulation and of electrical asystole are given in Fig. 2.

In stages I and II, before connection of the apparatus and against the background of complete artificial circulation, when the empty heart was contracting rhythmically, because of the identical experimental conditions there was no difference in the values of CB, P_{O_2M}, and AVP_{O_2} found then and in the experiments of series I.

In stage III, after electrical asystole, a sharp increase in P_{O_2M} to 7 ml/min/100 g was observed, while CB was 84.7 ml/min/100 g and AVP_{O_2} was 53%. The oxygen consumption of the myocardium reached the level characteristic of the normally working heart. As is clear from Fig. 2, the increase in P_{O_2M} was temporary in character and 30 min after the start of coronary perfusion it had already fallen to 4.93 ml/min/100 g, while at the same time AVP_{O_2} had fallen to 41.0% and CB to 78 ml/min/100 g. Sixty minutes after the start of coronary perfusion a small increase in CB to 83.8 ml/min/100 g and a further fall in AVP_{O_2} to 35.5% were observed.

The values of P_{O_2M} obtained for the normally working heart corresponded approximately to those cited by most investigators who have studied this process in the heart in the same functional state [5, 6, 15, 18].

It has been shown that exclusion of the external work of the heart by a complete artificial circulation lowers the energy expenditure of the heart and, consequently, its oxygen consumption also, to 50% of the original level.

Values of $P_{O_2}M$ similar to those obtained during the present experiments when the heart was cooled to 21-24° have been obtained by other investigators [4, 5, 17]. It should be noted that the values of $P_{O_2}M$ obtained by a number of authors when the temperature of the heart was 8-10° and 15° were only slightly below those obtained now [10, 11]. Consequently, the very slight decrease in $P_{O_2}M$ in profound hypothermia of the heart is evidence of the undesirability of reducing the temperature of the myocardium suddenly, more especially because there are reports in the literature of the harmful effect of deep hypothermia on the heart muscle.

No data on the effect of an electric current on the oxygen consumption of the myocardium could be found in the literature. The sharp increase observed in the present experiments in the oxygen consumption immediately after the onset of fibrillation caused by the action of an electric current and after restoration of the cardiac contractions by the use of a defibrillator was short in duration and was due to the action of the electric discharge, leading to a rapid increase in the intensity of both aerobic and anaerobic metabolic processes in the heart muscle.

The oxygen consumption of the myocardium after restoration of the cardiac activity was higher than at stage II. This was evidently because of the action of the electric current and of the higher temperature of the myocardium at this state.

It may be concluded from these results that both hypothermic coronary perfusion with hypothermic asystole and normothermic perfusion with electrical asystole can be used in clinical conditions.

LITERATURE CITED

1. I. E. Kisin, Byull. éksper. biol., No. 3, 117 (1959).
2. W. T. Bernhard, et al., Ann. Surg., 153 (1961), p. 43.
3. A. Beuren, Ch. Sparks, and K. Bing, Am. J. Cardiol., 1 (1958), p. 103.
4. V. Björk and A. Hallen, Acta chir. scand. 124 (1962), p. 177.
5. H. C. Cheng, Trans. Am. Soc. Artif. Intern. Organs 5 (1959), p. 273.
6. V. Gott, J. Gonzale, et al., Proc. Soc. Exp. Biol. (New York), 94 (1957), p. 689.
7. J. Greenberg, L. Edmunds, and P. Brown, Surgery, 48 (1960), p. 31.
8. H. E. Hoffmeister, H. Kreutzer, and W. Schoeppe, Pflüg. Arch. Ges. Physiol. Bd. 269, S. 407 (1959).
9. Ph. M. Ikins, et al., Surg. Forum., 5 (1958), p. 180.
10. O. Jardetzky, E. Green, and V. Lorber, Circulat. Res., 4 (1956), p. 144.
11. J. R. Jude, L. M. Haraotunian, et al., Am. J. Physiol., 190 (1957), p. 57.
12. E. Kay and D. Mendelson, Ann. Chir. jhorac. cardiovasc., 1 (1962), p. 792.
13. W. Mckeever, D. Gregg, et al., Circulat. Res., 6 (1958), p. 612.
14. J. Prehatny, H. Cheng, et al., Circulation, 27 (1963), p. 691.
15. H. Rheinlander and H. Wallace, Surgery, 52 (1962), p. 47.
16. F. Spencer, D. Merrill, et al., Am. J. Physiol., 160 (1960), p. 149.
17. A. Starr, M. Edwards, et al., Circulation, 27 (1963), p. 779.
18. R. Stoney, L. Langer, and B. Benson, Surgery, 52 (1962), p. 37.

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 the first issue of this year.
