## MAINTENANCE OF THE CIRCULATION IN VIVO BY AN IMPLANTED AUTOMATICALLY CONTROLLED ARTIFICIAL HEART

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An artificial heart in which the output is automatically controlled by the venous return, when implanted into dogs in the orthotopic position was found to be highly sensitive to changes in the hemodynamic conditions (a decrease in the venous return, hypo- and hypervolemia, increased peripheral resistance) and prevented the development of critical situations. For 24 h the principal vital functions of the experimental animals were maintained.

KEY WORDS: artificial circulation; artificial heart with automatic control; pneumatic drive.

Considerable progress in the design and construction of prostheses intended to replace the natural heart has been achieved in recent years [2-7]. However, many important aspects of this problem still remain unsolved.

The object of this investigation was to create a model of an implantable heart capable of changing its working conditions in accordance with changes in the venous return and to investigate the hemodynamics during the working of such a heart.

## EXPERIMENTAL

Experiments were carried out on adult dogs weighing 23-30 kg. After transsternal thoracotomy, with the aid of an artificial circulation the heart was removed from the animals and the artificial heart implanted in the orthotopic position.

The artificial heart had two atria and two ventricles with inlet and outlet valves. The ventricles were compressed by means of an alternating pressure applied by the pneumatic drive. The stroke volume of the artificial heart was about equal to the stroke volume of the natural heart. The artificial heart was connected by means of cannulas to the venae cavae and pulmonary veins, the pulmonary artery, and the aorta (Fig. 1). An automatic control system was developed for the heart in which its output was made to depend on the venous return [1]. The contraction rate of the right pump was determined by the rate of filling of the chamber with blood. The filling gauge consisted of a noncontacting pair of sealed magnetic switches located outside the blood flow. The frequency of the left pump was determined by the frequency of the right, and its stroke ejection volume was controlled by a signal of disparity between the assigned and true duration of diastole.

The response of the artificial heart to a reduction in the venous return to the right atrium, to hypoand hypervolemia, and to injection of noradrenalin was studied in 17 experiments. In 10 experiments the work of the artificial heart was studied for 6-24 h after closure of the thorax without any additional procedures.

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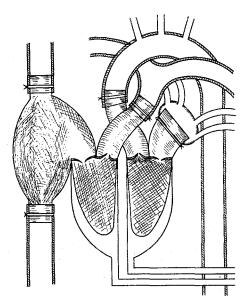


Fig. 1. Scheme of connection of the artificial heart to the vascular system.

TABLE 1. Mean Pressure in Right and Left Atria, Pulmonary Artery, and Aorta and Volume Blood Flow along Ascending Aorta with Natural and Implanted Hearts ( $M \pm m$ )

Heart	Mean pressure (in mm Hg)				Heart rate,	Volume blood
	right atrium	pulmo- nary artery	left atrium	aort a	beats per minute	flow along ascending aorta (in ml/kg/min)
Natural Artificial	1±1 2±1	16±2 21±3	5±1 7±2	110±12 103±9	140±6 89±5	80±7 82±8

The EEG, the blood flow and pressure in the aorta, and the pressure in the pulmonary artery and atria were recorded on the Mingograf-81 polygraph; the acid-base balance and composition of the blood gases were determined by the micro-Astrup method.

## RESULTS

As Table 1 shows, after thoracotomy and catheterization of the chambers of the heart and blood vessels, the indices of the central hemodynamics were within normal physiological limits. The artificial heart maintained the minute volume with a slower rate of contraction. The pressure in the pulmonary artery and aorta and the central venous pressure also remained within normal physiological limits.

A sharp decrease in the venous return to the right atrium (occlusion of the venae cavae) led to a drop in pressure in the left atrium, pulmonary artery, and aorta, a decrease in the minute volume of the heart, and slowing of the ventricular contractions. Removal of the occlusion was followed by a rapid increase in the heart rate and restoration of the normal central hemodynamic indices (Fig. 2A). With an increase in the venous return to the right heart by intravenous infusion of physiological saline, the artificial heart increased the frequency of its ventricular contractions and its minute volume. The pressure in the right atrium returned to its initial value. Reducing the circulating blood volume by bleeding led to a decrease in the central venous pressure, accompanied by a slowing of the rate of ventricular filling and a decrease in the minute volume of the heart until the normal pressure in the atria was restored.

Intravenous injection of 0.5 ml noradrenalin solution (ampule-packed) was followed by an increase in the pressure in the atria, aorta, and pulmonary artery. As the pressure effect ceased the original values of these indices were restored (Fig. 2B).

The artificial heart thus responded to a change in the circulation and restored the main indices of the central hemodynamics to their original levels; its responses were stable throughout the period of investigation.

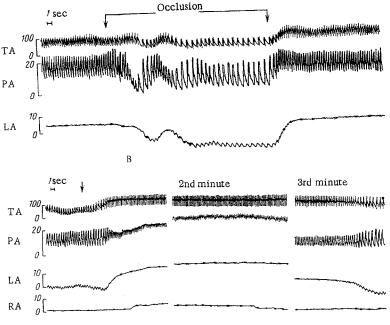


Fig. 2. Response of artificial heart to reduced venous return to the right atrium (A) and to injection of noradrenalin (B). From top to bottom: pressure (in mm Hg) in thoracic aorta (TA), pulmonary artery (PA), left atrium (LA), and right atrium (RA). Arrow indicates time of injection of noradrenalin.

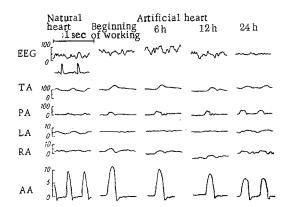


Fig. 3. Some physiological characteristics of the work of the artificial heart during 24 h. From top to bottom: EEG (electroencephalogram) in  $\mu$ V; pressure (in mm Hg) in thoracic aorta (TA), pulmonary artery (PA), left atrium (LA), and right atrium (RA); AA) volume blood flow in ascending aorta (in liters/min).

During prolonged function of the artificial heart the pressure and blood flow were not significantly different from initially (Fig. 3). Changes in the acid-base balance, the oxygen saturation of the hemoglobin, and the partial pressure of oxygen and carbon dioxide in the blood were corrected during the working of the artificial heart for 24 h by changes in the parameters of ventilation of the lungs and by injection of sodium bicarbonate. The ocular reflexes, responses to pain and acoustic stimulation, goal-directed muscular activity, and the bioelectrical activity of the brain were maintained in the animals.

It can be concluded from the results of these experiments that the artificial heart with a system of control by the venous return can maintain effective perfusion of the tissues for a long time. Automatic control of the ventricles of the artificial heart by means of a noncontacting transducer of the sealed magnetic switch type, located outside the blood flow, ensures a rapid increase or decrease in the minute volume of the heart and in the rate of its contraction and the high sensitivity of the heart to changes in the hemodynamic conditions.

## LITERATURE CITED

- 1. A. Guyton, Physiology of the Circulation. The Minute Volume of the Heart and Its Control [Russian translation], Moscow (1969).
- 2. W. F. Bernard, M. A. Bankole, C. La Farge, et al., Surgery, 70, 205 (1971).
- 3. J. H. Kennedy, M. E. DeBakey, W. W. Akers, et al., Biomater. Med. Devices Artif. Organs, 1, 3 (1973).
- 4. L. Kwann-Gett, M. Crosby, A. Schoenberg, et al., Trans. Am. Soc. Artif. Intern. Organs, 14, 238 (1968).
- 5. V. Nose and W. Kolff, J. Cardiovasc. Surg., 9, 22 (1968).
- 6. V. I. Shumakov, E. B. Mogylevsky, A. Kasimov, et al., J. Cardiovasc. Surg., 14, 150 (1973).
- 7. S. K. Topaz, M. M. Ameli, S. S. Morovati, et al., Trans. Am. Soc. Artif. Intern. Organs, 13, 294 (1967).