

# Testing Of Artificial Hearts In A Circulation Model

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## INTRODUCTION

This paper presents some of the results of our experimental studies with a new circulation model. The advantage of the new model is evaluated, particularly in relation to testing of artificial hearts.

In 1912, Krokh<sup>1</sup> and in 1940, Bayliss<sup>2</sup> described the forerunners of such a model. In 1961 Rushmer<sup>3</sup> also gave an example of such a model, which consisted of a pump, a compression chamber, a variable resistance, and a reservoir. In 1959 Kolff<sup>4</sup> showed the importance of using such a system to test artificial hearts. He called it a "mock circulation". In a mock circulation the output of an artificial heart in relation to various resistances, the response of the heart to various venous pressures, the function of the atria and of the artificial valves, and the best positive and negative driving pressure can be tested. The question of equilibrium of output of the right and left ventricles can be studied. In long testing flaws, such as leaks of the artificial heart becomes apparent, and can be corrected so that subsequently the lives of animals, as well as money will be saved.

## METHOD AND MATERIALS

The new circulation model was designed and constructed by Hydro-space Research Company in Rockville, Maryland. The entire system is built of medical grade Silastic. It is comprised of two discrete parts: (1) the circulatory system which is called the circulator, and (2) the controller (Fig. 1 and 2).

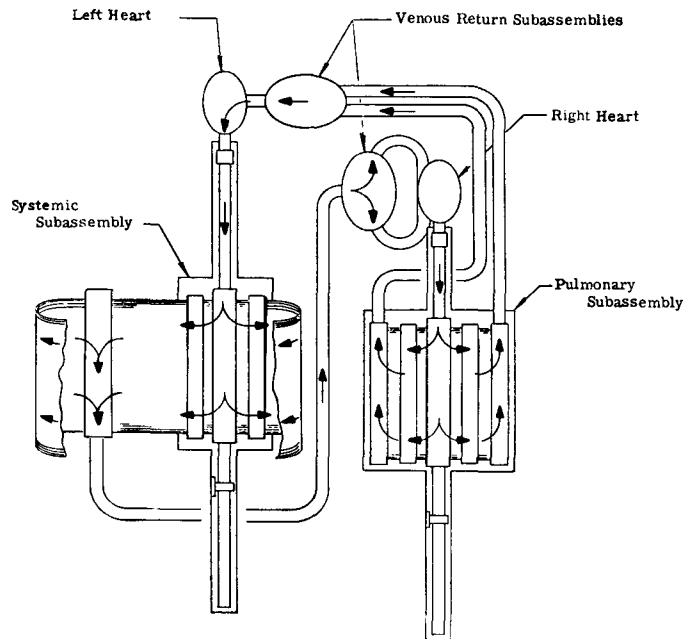


Figure 1.

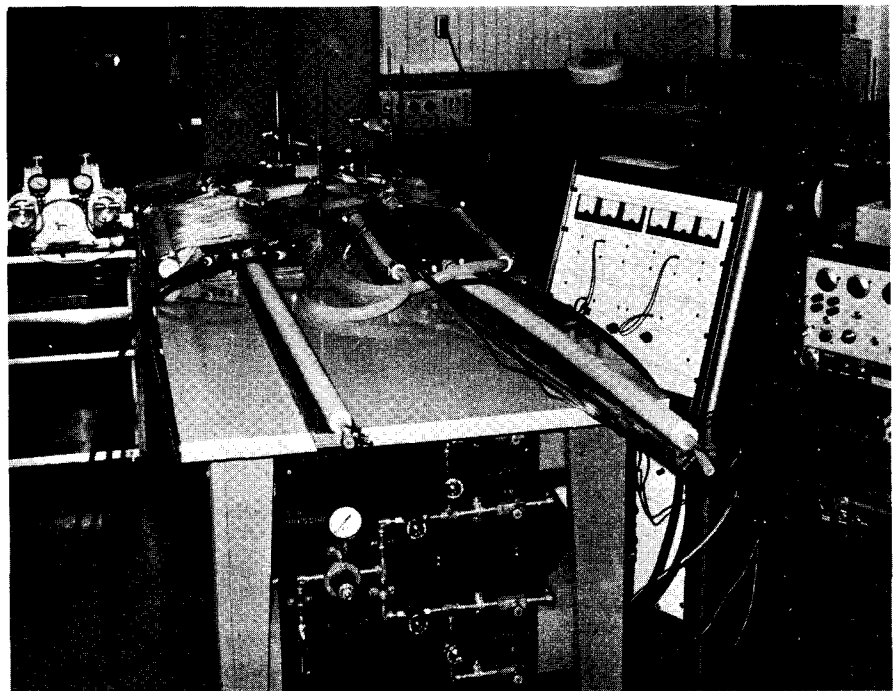


Figure 2.

**The Circulator.** Each ventricle is connected to a long tube that is the artificial aorta and the pulmonary artery. Approximately midway between the heart and the end of this tube there is a manifold area where 260 small tubes (Fig. 4) each having an internal diameter of 0.058 inch emerge and pass under the two bags that serve as air chambers. These bags can then exert a pressure on the small tubing, forcing

them against an aluminum plate, thus constricting the lumen and thereby increasing the resistance to flow. These small tubing then empty into the artificial vena cava or into the pulmonary vein, which returns the fluid to the atria. The aorta and pulmonary artery have tails (Fig. 1 and 2) comprised of tubes 1-inch internal diameter; they are surrounded by polyurethane foam. The foam is then covered with 0.020 inch thick Dacron reinforced Silastic. The basic difference between the systemic and pulmonary circulation is the length of the small tubes. That is, the tubing on the systemic side is 36 in. long, while on the pulmonary side it is 4 in. long.

**The Controller.** The controller can be described as two subpackages, one being the air supply, the other the electronic control system (Fig. 2). The air supply system consists of two large air reservoirs that supply compressed air to the compressing bags. The electric controller consists of two controlling systems, one for the systemic and one for the pulmonary side. Each side of the controller adjusts the peripheral resistance of each side of the circulatory system independently. The controller is capable of measuring venous and arterial pressure and aortic flow. It can be set to control one of the three variables. This is accomplished by means of 4 linear core pressure transducers, two per side, and an electromagnetic flowmeter for each side. All the signals from the system are read as mean values, and the pulsatile measurements can be recorded. The console is capable of controlling one variable per side of the circulator. The other two variables can be considered as dependent variables, e.g. if arterial pressure is controlled in each side, the venous pressure and the variation in flow must depend upon the set arterial pressure. The console at present can predetermine or regulate either the arterial pressure or the arterial flow. As yet it can hardly maintain the venous pressure.

The electronic console simply controls only one function, i.e. the amount of air that is admitted to the air reservoir that compresses the small tubing or the quantity of air released from the tubing. We added pressure manometers to the air bags

to have some indication of the resistance produced.

The circulatory system is filled with normal saline to a capacity of approximately 10 liters. Saline is used because of the dependency of the flowmeter upon a conductive fluid. In order to continuously check the measurements of the electromagnetic flowmeter we put a mechanical rotometer (Fischer and Porter) in the circuit. The artificial hearts used in this experiment were mainly the Holter hearts<sup>5</sup>. The Detroit Coil timer<sup>5</sup> or the Thompson Ramo Wooldridge driving system<sup>6</sup> (Fig. 2) were used as the driving systems for the artificial heart. The pressure and flow recordings were made with a Sanborn 2-channel recorder and a Honeywell 12-channel recorder.

## RESULTS

The reaction of the new circulatory system to changes of the peripheral resistance was traced (Fig. 3). The tracings are of the aortic and venous pressures and of the aortic flow. In the medium section of the figure the peripheral resistance was increased and a significant reduction of aortic flow occurred. The venous pressure fell slightly; the aortic pressure increased. At the right side of the figure the starting values are achieved again by decreasing the peripheral resistance, that is by allowing the air to escape from the air bags.

Figure 4 demonstrates that aortic flow was a function of the aortic pressure. The heart was driven by 5 and 8 psi; the vacuum for both

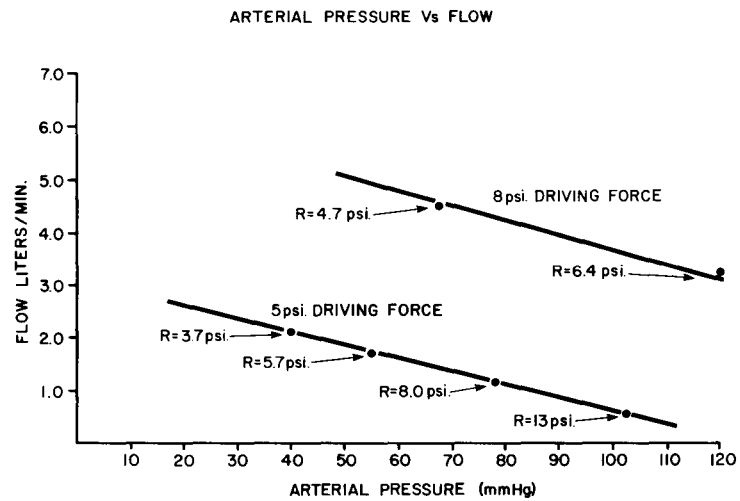
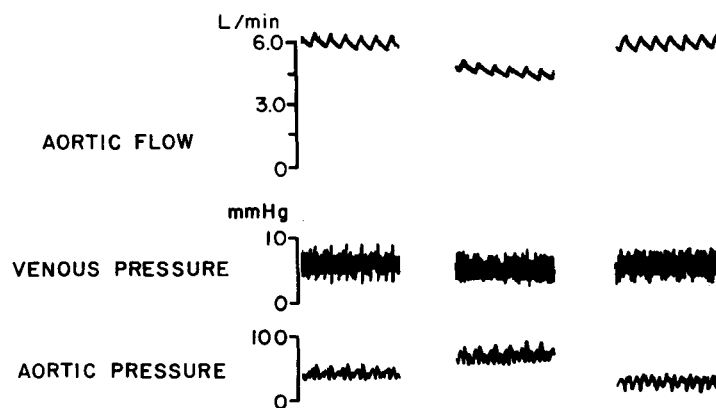


Figure 3.



INCREASE AND DECREASE OF PERIPHERAL RESISTANCE

Figure 4.

curves in 5 inches of mercury. The setting for the arterial pressure was increased manually. The peripheral resistance varied steadily as indicated by the pressure in the air bags. With an increase of aortic pressure there was a decrease of aortic flow. When the console showed, for example a mean arterial pressure recording of 65 mm. of mercury, flow rates of 1.5 liters per minute and 4.5 liters per minute could be read for the two driving forces. The machine changed the air pressure in the air bag, which produced the change in peripheral resistance from 6.8 to 4.7 psi.

In order to get reproducible measurements it was important to always have the same amount of fluid in the system. The determination of the mean circulatory pressure was a good gauge to control this amount. According to Guyton<sup>7</sup> the mean circulatory pressure is the pressure that would be measured at all points in the entire circulatory system if the heart were stopped suddenly and the blood were redistributed instantaneously in such a manner that all pressures were equal. The mean circulatory pressure is an expression of the volume contained in the circulatory pressure. Figure 5 shows a typical example of how to determine the mean circulatory pressure. In this case it was found to be 9 mm. of mercury. The balance of pressures, the increase of venous pressures and the decrease of aortic pressure can clearly be seen.

Figure 6 shows the influence of the venous pressure on the cardiac output. In this certain experiment a twin sack heart was used. The increase of venous pressure was achieved by infusion of 300 ml. of saline in the venous system. There were two distinctly different reactions of the artificial heart. In the two sets of curves to the left the heart was driven without negative pressure during diastole. One realizes an increase of cardiac output of about one liter per minute. In the two curves to the right the heart is driven with negative pressure during diastole; there was no increase in cardiac output. In Figure 7 the flow measurements of the aortic are shown when the artificial heart was driven by various pressures. The air bag pressure to affect the peripheral resistance was set at zero, which is as low as possible. Increasing the driving pressure showed an increase of aortic flow. After having

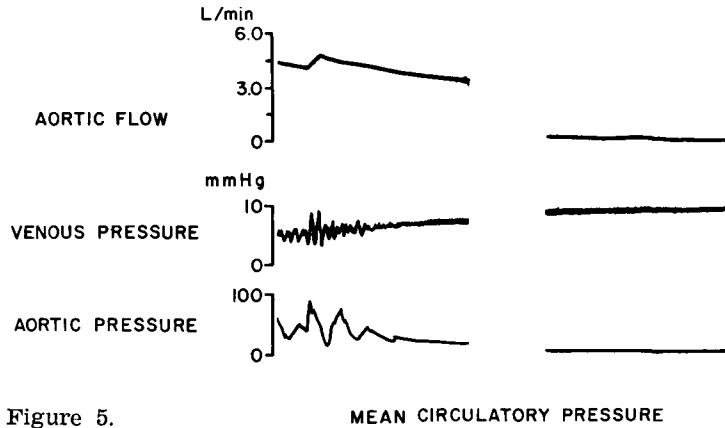
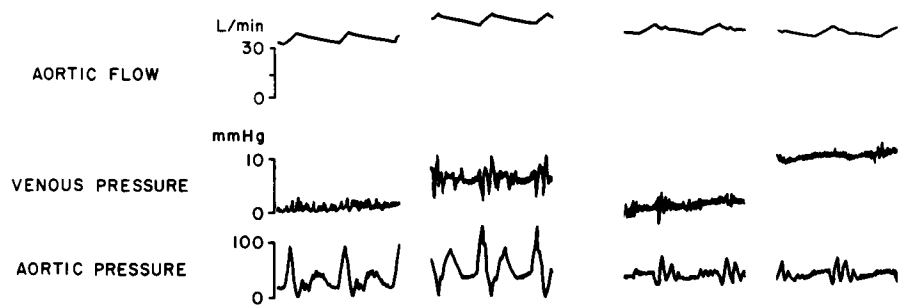


Figure 5. MEAN CIRCULATORY PRESSURE



TWIN SACK HEART, INFUSION OF 300 cc, LEFT SIDE NO SUCTION, RIGHT SIDE 4 in. Hg OF SUCTION TO DRIVE THE ARTIFICIAL HEART

Figure 6.

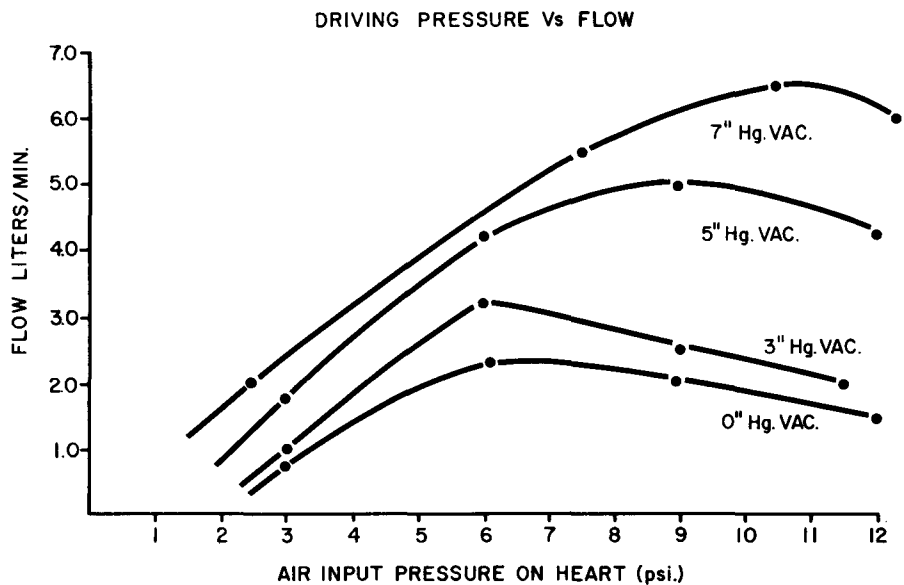


Fig.7 USING: HOLTZ HEART ON MOCK CIRCULATORY SYSTEM (HYDROSPACE RESEARCH) PERIPHERAL RESISTANCE = 0 ELECTRONIC CONTROL OFF VARIABLE = VACUUM LEVEL

Figure 7.

reached a peak the cardiac output decreased. For some types of artificial hearts we realized that the negative phase of the driving pressure during diastole had an important influence on the cardiac output. Driving a heart with a positive pressure of 10.5 psi and a negative pressure of 7 inches of mercury the cardiac output was approximately 7 liters per minute. This was the highest cardiac output obtained with a Holter heart. Other types of artificial hearts had a still higher cardiac output.

A group of curves correlating aortic pressure to aortic flow is shown in Figure 8. The driving force of the artificial heart was gradually increased for the construction of each curve. A constant vacuum of 5 inches of mercury was used. The electronic control was set out of action. The peripheral resistance was dictated by a hydrostatic head produced by raising the artificial vena cava above the table level (Fig. 8, curves a and b) or by

the amount of air inflated into the air bag (Fig. 8, curves c and d). After having reached a certain air pressure the air bag was closed so that the pressure remained constant and the compression of the small tubes remained constant. When the driving pressure of the heart increased, both flow and aortic pressure increased. There was a linear relation between flow and pressure, as was to be expected.

In order to compare the new circulation model with the human circulatory system the behavior of the volume pressure of the venous bags was studied. The capacity of one liter showed only a small minor increase of pressure. Further filling of the bags was accompanied a steady increase of pressure.

### CONCLUSION

The new circulation model has a variable resistance. The resistance can be measured as air pressure com-

pressing the artificial arterioles. Kolff took as resistance in his model a column of water of 82cm. which corresponds to 60 mm. of mercury. The disadvantage of a fluid column is the inertia of the fluid mass that must be overcome so that there will be space for the cardiac output. The advantage of Kolff's system was that the constant diastolic pressure determines a great deal of the resistance. The Hydrospace system also has to overcome the inertia of a large fluid volume in the aorta. This results in an unphysiologic pressure curve. The venous bags of the new circulatory system have a good capacitance; filling of the bags causes only a small change in venous pressure. Whether or not the reflection section of the Hydrospace circulation model is of any use must await further experimental work.

The new circulation model is a good experimental method to get further information of the functional behavior of all the artificial hearts now available.

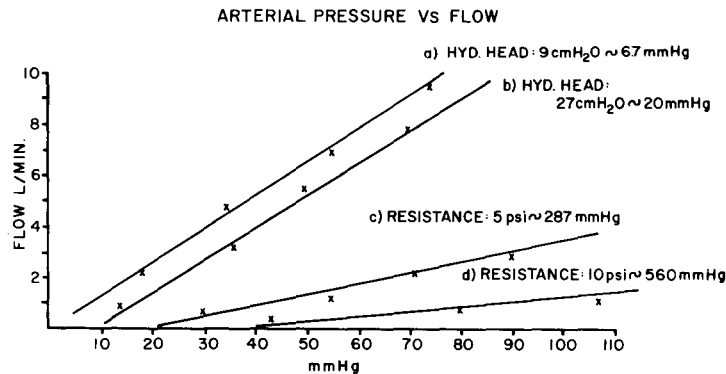


Figure 8.

### REFERENCES

1. Krogh, A., The regulation of the supply of blood to the right heart. *Scan. Arch. Phys.* 27:227, 1912.
2. Bayliss, L. E., A circulation model. *J. Physiol.* 17:423, 1940.
3. Rushmer, R. F., *Cardiovascular Dynamics*, W. B. Saunders, Phila., pp. 166, 1962.
4. Kolff, W. J., Mock circulation to test pumps designed for permanent replacement of damaged hearts. *Cleve. Clinic Quart.*, 26:223, 1959.
5. Sen Gupta, A., et al, Total mechanical replacement of heart in calves. *Cleve. Clinic Quart.*, 32:69, 1965.
6. Panayotopoulos, E. K., et al, A special reciprocating pump to drive an artificial heart inside the chest. *J. Thoracic Cardiovasc. Surg.*, 48:844, 1964.
7. Guyton, C., *Circulatory Physiology: Cardiac Output and Its Regulation*. W. B. Saunders, Phila., 1963.

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