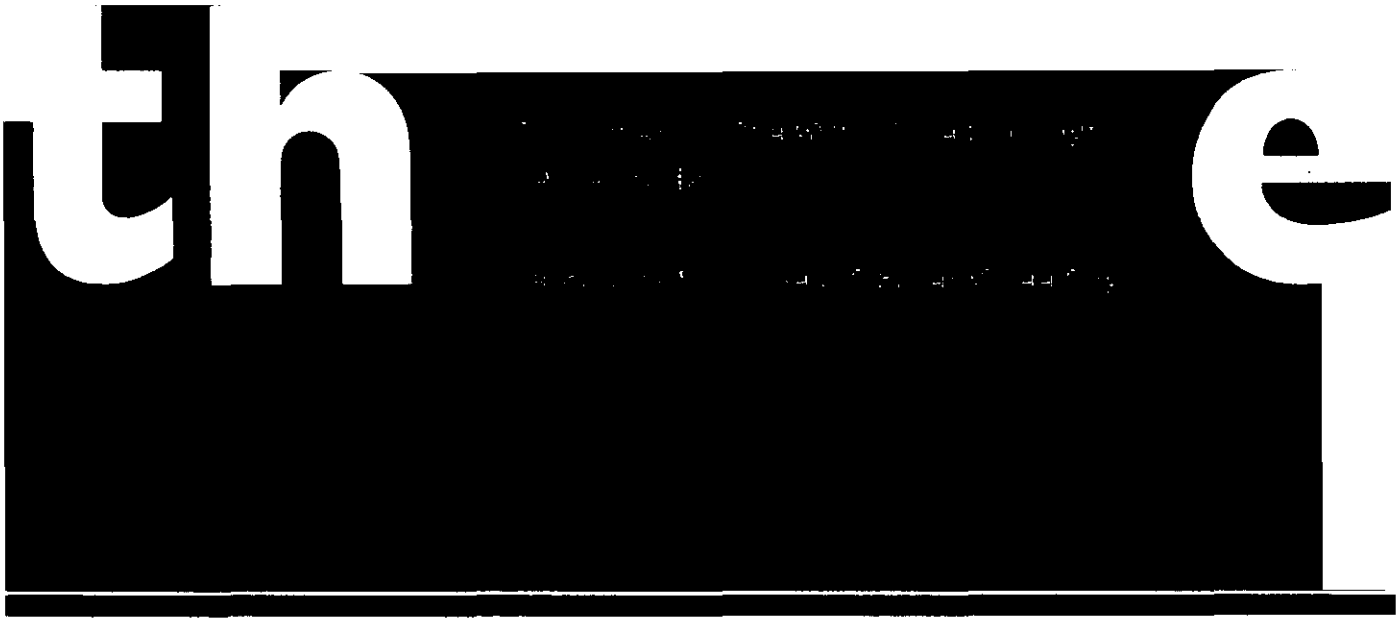
The design of a mock circulation system



THE DESIGN OF A MOCK CIRCULATION SYSTEM

By

W. H. Leliveld

T.H. Report 74-E-47

June 1974

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Abstract

A mock circulation system, consisting of an artificial left ventricle, an aorta and a termination, is presented.
This model, which can serve several purposes, such as testing artificial heart valves, was specially built for the purpose of testing parameter estimation techniques on the human aorta under realistic conditions.

The circulation system also offers the possibility to evaluate the measuring methods, as well as the possibility to verify the results obtained by estimating parameters of the aorta such as the elasticity of the aortic wall.

Contents

MOCK CIRCULATION SYSTEM

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1. Introduction.

For the purpose of parameterestimation on the human aorta, this physical model has been built.
A flexible tube, with dimensions and material constants which are as similar as possible to the real human aorta, is a quite realistic model for this aorta.
It is the aim, to estimate the parameters of this tubelike aorta under conditions which are as realistic as possible. In order to avoid extra troubles with the measurement of flow quantities and pressures and the estimation of parameters on a real aorta in future, it is also desirable to have realistic flow and pressure patterns in the artificial aorta. To implement this, one has to build a special pump which serves as an artificial left ventricle. This artificial left ventricle has to produce a pressure wave-form which is similar to that produced by the real human left ventricle.
Furthermore the fluid impedances of this pump and the artificial aorta must have the correct values in order to get a correct flow pattern when the pressure wave form is correct.
For the same reason the artificial aorta has to be terminated by the correct load impedance.
This termination impedance must have the correct values for modulus and argument as a function of frequency.
In addition to all these precautions, the fluid which is pumped around in the circuit must have a specific density and a viscosity equal to blood in the large blood vessels.

Literature describes several complete mock circulation systems and many types of artificial hearts.
E.H. Weber was one of the first investigators who published in 1850 his work about an artificial circulation system.

During the last years there are many publications about artificial hearts and artificial heart valves and some about mock circulation systems.
These complete circulation systems are mostly built for the purpose of testing artificial hearts and artificial heart valves.
The most important artificial hearts are driven by pneumatical, mechanical, electromechanical, hydraulical or piezoelectrical forces.
Some publications about mock circulation systems or artificial hearts are:

- A circulation model,
  by L.E. Bayliss,
  J. Physiol. (1940) 97, 429-432.
  It describes one of the simplest circulation models.

- Testing of artificial hearts in a circulation model,
  by K.H. Leitz, M. Klain , P. Phillips and W.J. Kolff,
  Proc. Biomedical Symposium on Engineering in Medicine.
  Marquette University.
  June 1966, pp. 287-290.
  This paper presents a circulation system for testing artificial heart valves.

- Variable parameter hemodynamics systems,
  by J. Melbin,
  Medical Research Engineering.
  Fourth Quarter 1968, volume 7, number 4.
  It describes a complete circulation system for studying the effects of changing the parameters of that system.

- A servomechanism to drive an artificial heart inside the chest.,
  by K.W. Hiller, W. Seidel and W.J. Kolff.
  Trans. American Soc. for artificial internal organs.
  Vol. 8 1962, pp. 125-130.
  It describes a pneumatically driven artificial heart.

- The piezoelectric artificial heart,
  by M.L. Loehr, W.F, Kosch, M. Singer, W.S. Pierce and C.K. Kirby.
  Trans. American Soc. for artificial internal organs.
  Vol. 10, 1964, pp. 147-150.
  It describes an artificial heart driven by bimorphs.

2. The artificial left ventricle.

2.1. Choice of the left ventricle system.

It was the aim to build a simple artificial left ventricle which can be controlled in an easy way.
For several reasons a pneumatically driven system with electrical input is choosen.
The pneumatic part consists of a position transmitter (Foxboro-type CP) which proportionally converts the position of a shaft into a pressure.
This pressure is transmitted to a double piston system which is sealed with rolling diaphragms.
This system forms the actual artificial left ventricle.
The position of the shaft of the above-mentioned position transmitter is controlled in an electrical way.

2.1.1. The position transmitter.

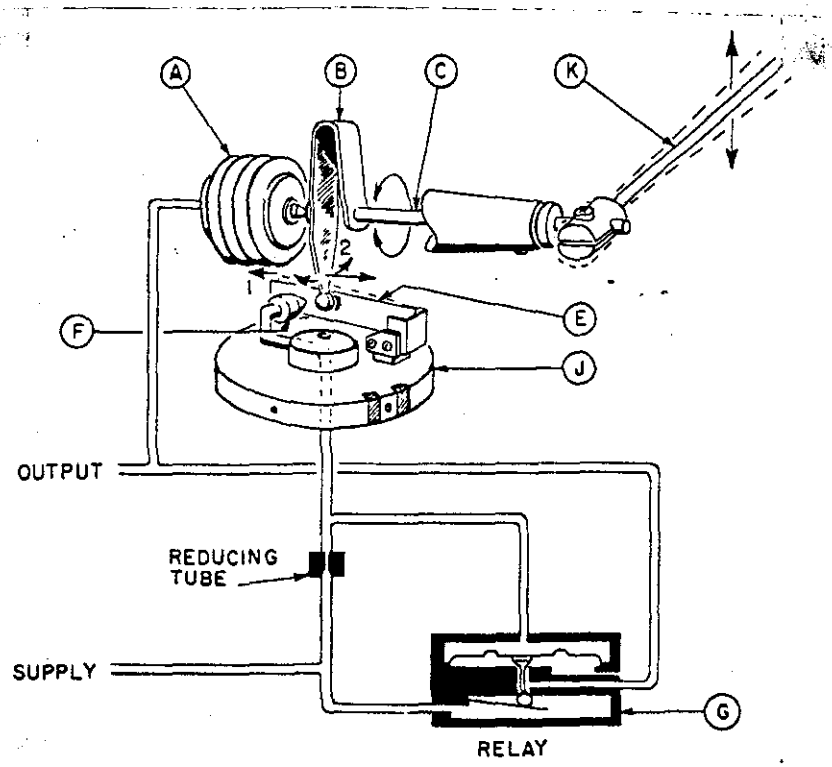


Fig. 2.1
Position transmitter

When the actuating lever K is not in motion, the system is in balance and the feedback bellows A opposes flexure assembly B. With the flapper-nozzle in the position shown in the drawing, clockwise rotation of the

actuating lever rotates the flexure assembly B and the ball tip in such a direction that the flapper E tends to cover the nozzle F.
Pressure established by the flapper-nozzle is amplified by the relay G and led to the feedback bellows A and to the output.
Bellows A expands pushing the ball tip in such a direction as to restore the flapper to its detector position.
For any given position of disc J, the amount of rebalancing motion in direction 1 is exactly proportional to the amount of unbalancing motion in direction 2. Thus the pressure in bellows A, and hence the output pressure, is directly proportinal to the rotation of the actuating lever.
The output pressure is transmitted to the system consisting of two pistons which forms the actual left ventricle.

2.1.2. The electrical input.

For reasons of flexible controllability an electric input signal is choosen which can be changed in shape in a rather convenient way.
For this purpose a d.c. amplifier (with feedback control) has been built to control an electromechanic transducer. (fig. 2.2). This transducer changes the position of the input lever K of the position transmitter (fig. 2.1).
These changes in position are directly proportional to the electric input signal.

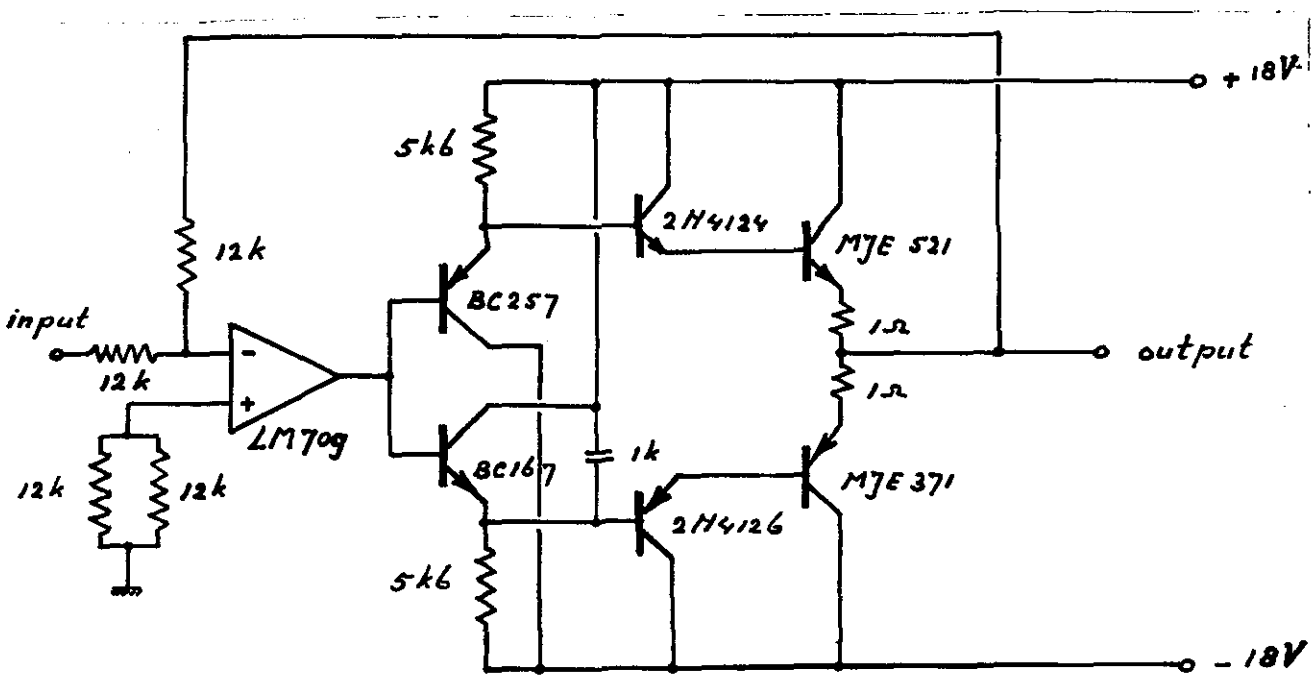


Fig. 2.2. D.C. amplifier.

2.1.3. Matching electrical and pneumatic system.

To convert the output signal of the d.c. amplifier to a directly proportional position change of lever K of the position transmitter (fig. 2.1.) a woofer loudspeaker is used as an electromechanical transducer.
From this loudspeaker the moving system is coupled to the lever K of the position transmitter.
To prevent that friction and inertial forces determine the position of the lever, a stiff spring is used to keep the lever in its rest position.
In this way the position is determined only by the input signal of the d.c. amplifier and the elasticity of the spring.

2.1.4. The double-piston system.

The normal output pressure of the position transmitter has a value between 0.2 and 1.0 atmosphere above atmospheric pressure.
Dividing this pressure by a factor 4 we get a pressure which is maximal 190 mmHg. This divided output pressure serves as the left ventricular pressure.
The above-mentioned pressure dividing takes place in the double piston system, which is schematically drawn in fig. 2.3.

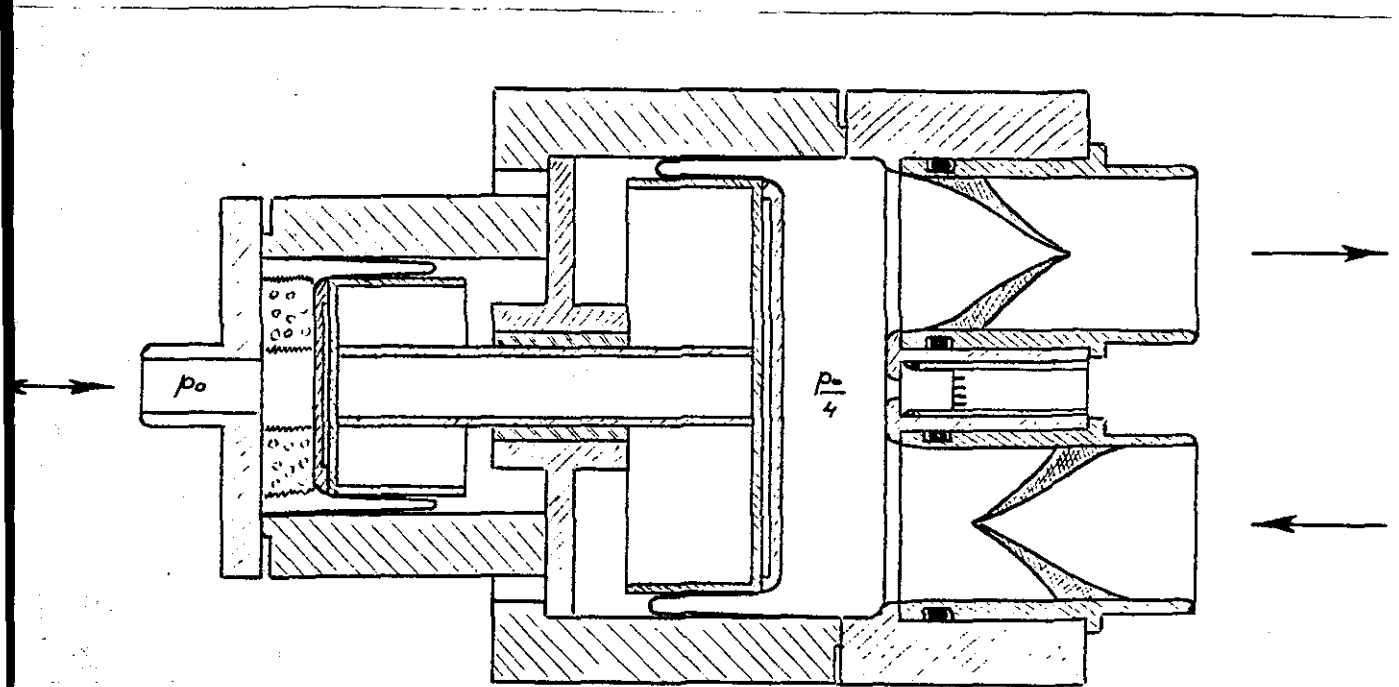


Fig. 2.3. Double-piston system.

The diameter of the right hand piston is twice the diameter of the left hand piston. The pistons are stiffly coupled by a pistonrod. To prevent friction forces as much as possible the pistonrod is sliding in a nylon bearing. To minimize the mass, the whole piston system is made of aluminum. Sealing is provided by means of selfmade thin rolling diaphragms which also minimizes the friction.

The output pressure of the position transmitter is transmitted to the left hand piston-cylinder combination. The same pressure, divided by a factor four, is present in the right hand combination, which forms the actual left ventricle.

This right hand combination is filled via the lower leaflet valve from a reservoir with overflow. In this way it is possible to get a constant filling pressure.

In the filling period the pistons are driven to the left by the pressure of the above-mentioned reservoir. To prevent collision with the cylinder at the end of the stroke there is a stop covered with foam rubber.

During this filling period , the upper leaflet valve (the aortic valve) is closed, because the aortic pressure, which is higher than the ventricular pressure on that moment, keeps that leaflet valve closed.

When the piston system is driven to the right by the increasing output pressure of the position transmitter, the lower leaflet valve (mitral valve) is closed by a starting backflow through the mitral valve.

On the moment the ventricular pressure exceeds the aortic pressure, the aortic valve is opened and the blood aequivalent fluid is expelled into the aorta. When at the end of this period the ventricular pressure falls below the aortic pressure level, the aortic valve is closed by the starting back flow from aorta to left ventricle.

When the ventricular pressure falls below the pressure level of the atrium, which is a constant pressure in this case, the mitral valve opens and the filling periode starts again.

3. The artificial aorta.

3.1. Some properties of blood and bloodvessels.

3.1.1. The blood.

Blood is a suspension of cells in a colloidal solution. Normally the volume fraction of red blood cells is in the order of 45 %. The other elements, white blood cells and platelets, represent less than 2 % of the total blood volume.
The mass density equals 1.05 gr/cm^3. The apperent viscosity $\breve{\eta}$ decreases as the tube diameter or hematocrit decreases and varies inversely with the rate of flow. However, this viscosity reaches an asymptotic value when the diameter of the vessel is larger then about 0.8 mm.
The viscosity also depends on the temperature. The value of η is 3.3×10^{-3} Ns/m^2 at 37°C for the aorta.

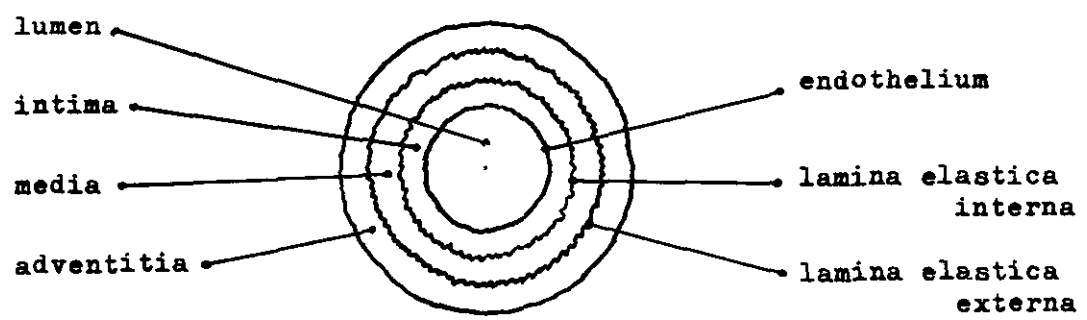
3.1.2. Dimensions of the aorta.

The dimensions of the aorta are not constant. The greater the distance from the heart, the smaller are diameter and wall thickness. In our tubelike model we neglect the branches and the above mentioned dimension changes. For the average dimensions of the tubelike aorta we assume:

diameter $d = 2$ cm
wall thickness $h = 1.5$ mm
length $l_{tot} = 40$ cm

3.1.3. The elasticity of the wall.

A cross-section of an artery is given in fig. 3.1.



Cross-section of an artery

fig. 3.1.

The most important tissues in the arterial wall, which may contribute to the wall elasticity are:

1. The endothelium. It consist of flat cells which form a smooth inner wall. The cells are easily extensible and do not contribute to the elasticity.
2. The elastine fibres. They form a layer (elastica intima) and are also present in media and adventitia. They are about six times more easily extensible as rubber up to several times their unstretched length.
3. The collagen fibres. They form a network throughout media and adventitia. They are stiffer than elastine fibres and the value of their modulus of elasticity is several hundred times greater.
   Because of their structure the collagen fibres only contribute to the elasticity of the wall after the wall has been stretched already to a certain degree.
4. The smooth muscles. They probably contribute less to the passive elasticity, even if they are contracted. Anyway, in the aorta very few smooth muscles are present.
   So they do not play an important role in the case of the aorta.
   The conclusion is that only elastin and collagen fibres are responsible for the elastic stress in the wall of the aorta.

3.1.4. Static elastic behaviour of the wall.

Hook's law for elastic materials expresses a linear relationship between pressure and strain; the ratio is given by the constant Young's modulus of elasticity E.
The elasticity of the wall is included in the compliance C, which is defined as:

$$C = \lim_{\Delta V \to o} \frac{\Delta V}{\Delta p} \qquad (3.1)$$

in which V denotes the volume of a blood vessel and p represents the hemodynamic pressure in the vessel.
However, Young already noticed the fact that the stress-strain relations of the vascular wall material are nonlinear. If the strain of the wall material increases because of increased pressure, the resistance against further strain becomes more than predicted by Hooke's law.

This is caused by the heterogeneity of the wall, which consists of elastin and collagen fibres. The individual fibres obey Hooke's law, but with increasing strain, more and more collagen fibres will reach their unstretched length and will contribute to the elastic resistance with further elongation; that is, Young's modulus E will increase when pressure increases which is shown in fig. 3.2. From this figure we see that $E \approx 2.10^6$ dyn $cm^{-2} = 2.10^5$ Hm^{-2} (at p = 100 cm H_2O).
According to eq..(3.1) we may interprete C as the slope of the pressure-volume curve.

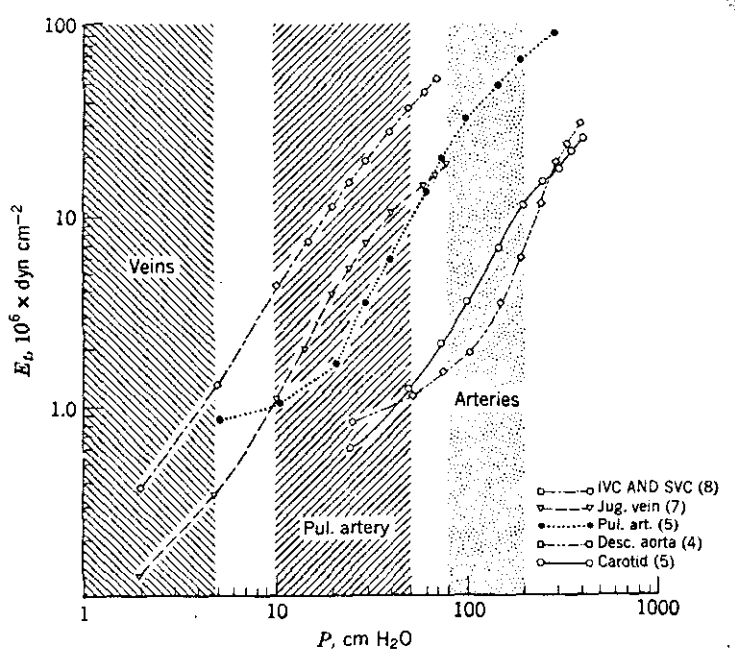


Fig. 3.2. Pressure dependence of the elastic modulus E_t. Shaded areas represent the normal ranges of pressures.

As a consequence of increasing E at increasing p, the pressure-volume curve becomes more parallel to the p-axis at higher pressures because the compliance C is inversely proportional to the Young's modulus E.

3.1.5. Dynamic elastic behaviour of the wall.

The description of the properties of the vessel wall is more complicated if dynamic behaviour is included.
If the volume of a vessel that is occluded at both ends is increased suddenly, the pressure will increase instantaneously.
The new pressure level, however, will start to decrease immediately,

which means that compliance is increasing.
This so called delayed compliance is caused by a slowly changing of stress in the smooth muscles although probably other fibres may also show this effect of stress relaxation. It mainly occurs in veins and only slightly in the aorta.
The vessel wall, therefore, is not perfectly elastic; that is, the stress-strain ratio depends on the duration of the applied stress.

3.2. The blood analog fluid.

A satisfactory blood analog fluid which has at room temperature the same viscosity as blood with a temperature of 37°C is an aqueous-glycerol solution of 21°C which contains 36,7 percent glycerol by volume. This solution has a viscosity

$$\eta = 3.3 \times 10^{-3} \text{ Ns/m}^2$$

3.3. The latex aorta.

The artificial aorta is made according to the dimensions assumed in 3.1.2.
As a first approach curves, branches and tapering are neglected and by doing this we get a straight tube with a length of 40 cm, a diameter of 2 cm and a wall-thickness of 1.5 mm.
The tube is made of latex. This is done in order to avoid non-linear effects which would be introduced if the tube-wall is built of different materials. In this way the elasticity of the wall and therefore the compliance of the tube is independant of the pressure.
The avoidance of non-linearities is important from the point of view of parameter estimation techniques, for which purpose this physical model has been built.
When good results can be obtained in estimating the parameters of this linear model, the artificial-linear aorta will be replaced by a non-linear one, which will be built up of several layers of latex and textile.

3.4. The silastic aorta with extra compliance.

During the test periode of the artificial left ventricle and valves and also during testing and estimating the termination or load impedance at the end of the aorta, the latex aorta can be replaced by a mechanical

stronger one.
This replacement can be effected by a silastic tube which is stronger than the latex one. However, this silastic tube is too stiff and therefore the aortic valve closes too late.
To compensate for this an extra compliance is introduced at the beginning of the silastic tube.
This extra compliance consist of a bottle which is placed upside down and connected to the system.
By varying the amount of compressed air in this bottle it is possible to control the value of the compliance. The way in which this value depends on the amount of air will be derived in 4.2.

4. The artificial termination.

4.1. Choice of the termination configuration.

In ref. [5], describing vascular impedance in man, one can find the modulus of the fluid impedance as a function of frequency along the aorta. These impedances are derived from blood pressure and velocity wave forms recorded in a series of patients at cardiac catheterisation.
Looking at the modulus of the impedance and the phase angle as a function of frequency in the abdominal aorta, which place we consider as the end of the aorta or the beginning of load impedance, one can try to describe this impedance in an electrical analog.
The modulus of the impedance and the phase angle of the electrical network given in fig. 4.1. are quite the same as those of the termination if the electrical components are well dimensioned. R_{SA} represents the resistance of the system arteries, C_{SA} represents the compliance of the system arteries and R_{per} represents the resistance of the periferal arteries.

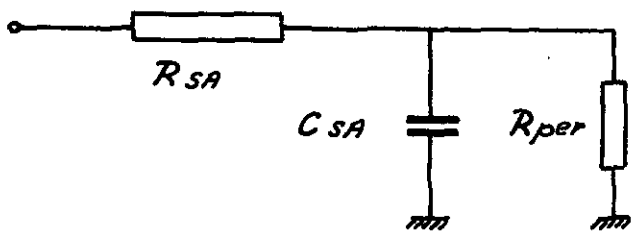


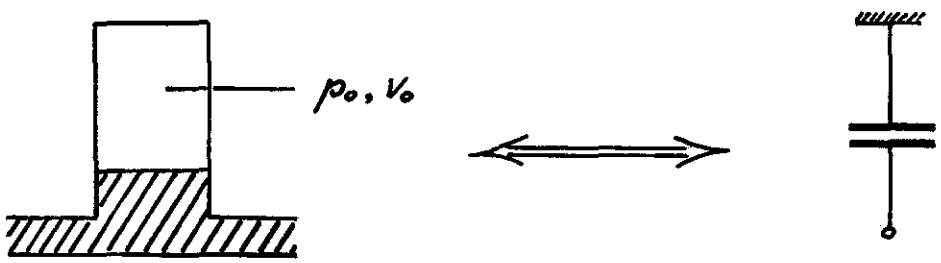
Fig. 4.1. Electric analog of the termination.

If the cardiac output is 5 l/min = 83 ml/sec. the flow in the abdominal aorta will be about 60 ml/sec.
The main pressure is 100 mmHg.
From the modulus of impedance as a function of frequency the ratio between R_{SA} and R_{per} which is 1 : 20 can be derived. Now it is possible to conclude that $R_{SA} = 0.08$ mmHg $ml^{-1}s$
and $R_{per} = 1.58$ mmHg $ml^{-1}s$
The determination of the exact value of C_{SA} from the data in this article mentioned, is more difficult. The results vary from 0.8 ml $mmHg^{-1}$ to 1.2 ml $mmHg^{-1}$. For that reason we simulated the aorta and the termination on an analog computer. For all components we chose the nominal value and for the input signal a physiological pressure curve.

The value of C_{SA} in this model was varied in such a way that the input flow of the analog computer model was also a physiological curve. By this procedure we determined the most correct value of C_{SA} which was 1 ml $mmHg^{-1}$.

4.2. How to realize a compliance.

If we consider a reservoir partly filled with fluid and above this fluid a certain amount of air, like in fig. 4.2, we can derive a formula for the compliance of this bottle, which will be the hemodynamic aequivalent of an electric capacitor.



$C = \lim_{\Delta V \to 0} \frac{\Delta V}{\Delta p}$ $\qquad$ $C = \frac{\Delta Q}{\Delta V}$

V = volume $\qquad$ Q = charge

p = pressure $\qquad$ V = voltage

Fig. 4.2.

Assuming that compression and expansion of the air in the reservoir is an adiabatic proces, it is possible to use Poisson's law for adiabatic state variations.

This assumption is quite real, because air is a bad heat conductor and compression and expansion take place in a rather short time, so that heat conduction to the environment is negligible.

Poisson's law states:

$$p\, V^{K} = \text{constant} \qquad (4.1)$$

where $K = \frac{C_p}{C_V}$

p = pressure

V = volume

and C_p = specific heat at constant pressure

C_V = specific heat at constant volume.

For air at room temperature and at a pressure of about 1 atmosphere, the value of K = 1.4.
Keeping in mind, that an increase in fluid volume corresponds to a decrease in the air volume, it is possible to write:

$$p_o V_o^K = \text{constant} = (p_o+\Delta p)(V_o-\Delta V)^K$$

where: P_o = mean pressure

V_o = mean volume

$$p_o V_o^K = (p_o+\Delta p)V_o^K \left[1 - \frac{K}{1!}\frac{\Delta V}{V_o} + \frac{K(K-1)}{2!}\left(\frac{\Delta V}{V_o}\right)^2 - \ldots\ldots \right]$$

neglecting terms of higher order yields:

$$p_o V_o^K = (p_o+\Delta p)V_o^K(1-K\frac{\Delta V}{V_o})$$

$$\frac{\Delta V}{\Delta p} = \frac{V_o}{p_o}\cdot\frac{1}{K} - \frac{\Delta V}{p_o}$$

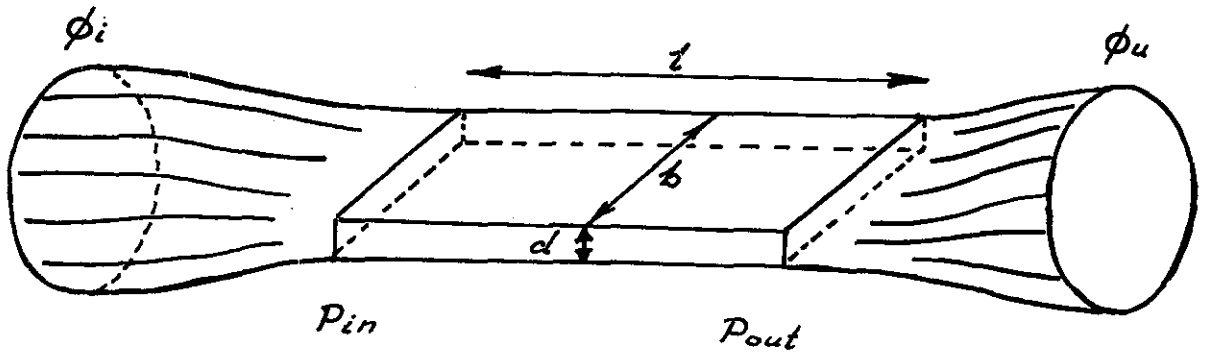
$$C = \lim_{\Delta V\to o}\frac{\Delta V}{\Delta p} = \frac{V_o}{p_o}\cdot\frac{1}{K} = \frac{5}{7}\cdot\frac{V_o}{p_o} \qquad (4.2)$$

The nonlinearity, as a result of volume changes, which in their turn result from the pressure changes in the reservoir, may be neglected, since the changes in pressure (arterial pulse pressure) are much smaller than the mean pressure in the reservoir (atmospheric pressure plus mean arterial pressure).

4.3. How to realize a resistance.

The most elaborated method to realize a variable hemodynamic resistance which is the analog of the electric resistance in the simulated termination, is a silastic tube, which is pressed flat between two metal plates in such a way that the crosssection becomes a slit.
By changing the width of this slit it is possible to vary the value of the resistance.
If the fluid is Newtonian and the flow is laminar (which is garanteed if the Reynolds nummer is less than Re_{kr} = 2300 for such a slit) it is possible to derive a formula for the hemodynamic resistance of such a slit which will be defined as the quotient of pressure drop and flow.

$$R = \frac{P_{in} - P_{out}}{\overset{o}{V}} = \frac{12\eta l}{bd^3} \qquad (4.3)$$



P_{in} = input pressure
P_{out} = output pressure
$\overset{o}{V}$ = flow
η = viscosity of the fluid
l = length of the slit
d = width of the slit
b = breadth of the slit
] $b >> d$
ϕ_i = cross sectional area at the input side
ϕ_u = cross sectional area at the output side

Fig. 4.3.

When realizing this hemodymical resistance the construction must be such that a resistance value which is flow independant and equal to the value calculated from equation (4.3) is obtained.
By means of Bernoulli's law, which states: $p + \rho gh + \frac{1}{2}\rho v^2 = \text{constant}$ (4.4) and neglecting the gravitation term ρgh, it is possible to derive a formula for the hemodynamical resistance of the complete construction, that is the slit plus the connections to input and output tubes.

$$R = \frac{12\eta l}{bd^3} - \overset{o}{V}.\left[\frac{1}{\phi_i^2} - \frac{1}{\phi_u^2}\right] \text{x const.} \qquad (4.5)$$

This formula shows that if in the construction $\phi_i \neq \phi_u$ the consequence is that $R = f(\overset{o}{V})$, which is an undesired situation.

5. Results.

5.1. Trapezoidal input signal.

The duration of the depolarization of the ventricle muscle is about 80 msec. and the contraction time of a muscle fibre lasts a few hundred milliseconds.

Considering these facts it is possible to conclude that the contraction force of the ventricle muscle is a more or less trapezoidal time function.

Another interesting aspect of the heart muscle is, that the duration of the contraction time depends upon the contraction frequency: the higher the frequency, the shorter the contraction time.

In view of this fact we decided to take a trapezoidal electric input signal with constant duty cycle.

Lit. [3] describes a proper pressure curve shape in pneumatically driven artificial hearts. Several shapes were tried out by the author and empirically it was found that a more or less trapezoidal pressure curve produced a ventricular pressure nearly similar to that obtained from the left ventricle of the natural heart.

5.2. Measured pressure in the artificial left ventricle.

As already described, a trapezoidal input signal for the constructed artificial left ventricle produced a pressure pulse which looks very similar to the natural left ventricular pressure pulse.

Figure 5.1. shows the pressure puls produced by our physical model.

Figure 5.2. shows the natural pulses taken from the textbook of physiology "A Primer of catheterisation 1965" and fig. 5.3. depicts the left ventricular pressure got by catheterisation*).

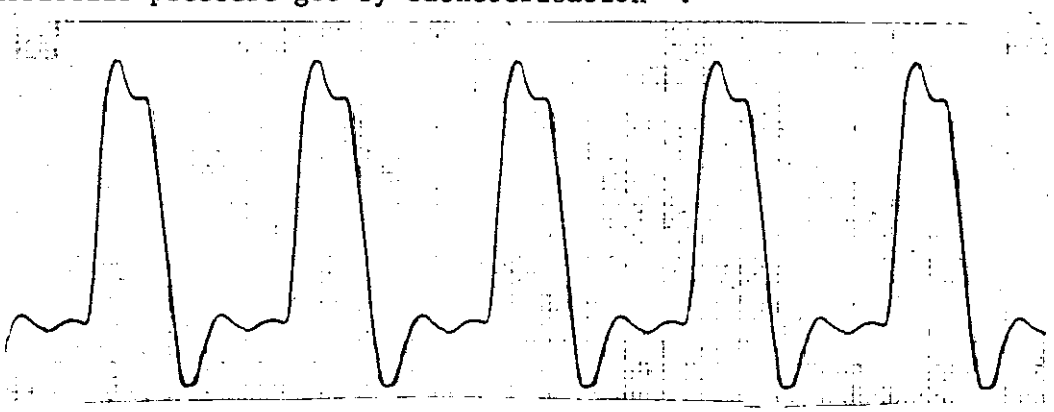


Fig. 5.1. Left ventricular pressure measured at the physical model.

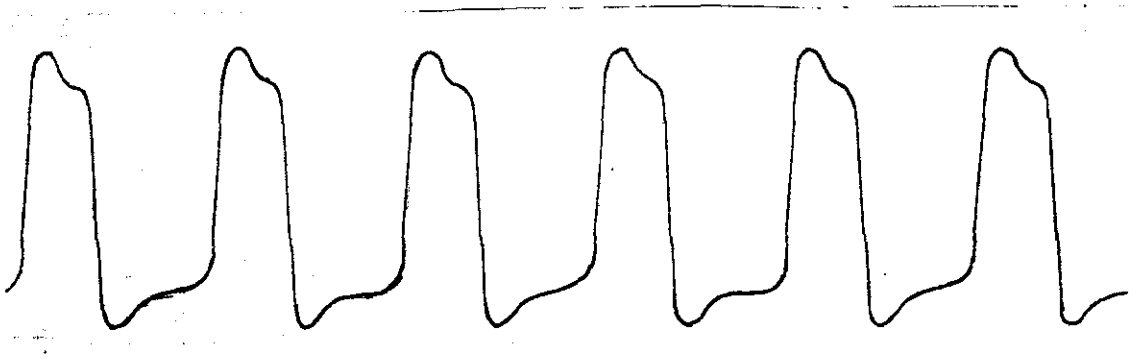


Fig. 5.2. Left ventricular pressure taken from a textbook.

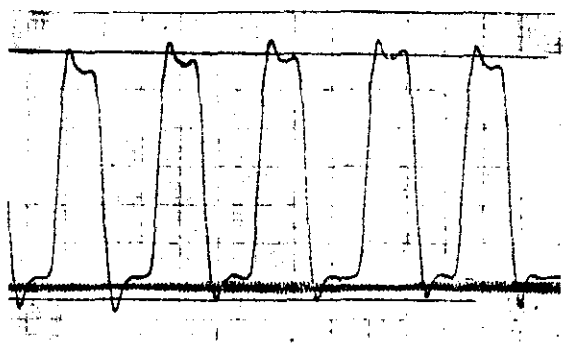


Fig. 5.3. Left ventricular pressure got by catheterisation.*

5.3. Aortic pressure and flow.

Fig. 5.4. shows the aortic pressure, measured just behind the aortic valve in the artificial aorta.

Fig. 5.5. shows the aortic flow in the artificial aorta and fig. 5.6. and 5.7. show the flow in the human aorta and in the aorta of a dog, respectively.

It appears from comparison of these patterns that the flow pattern in the artificial aorta is a quite realistic one.

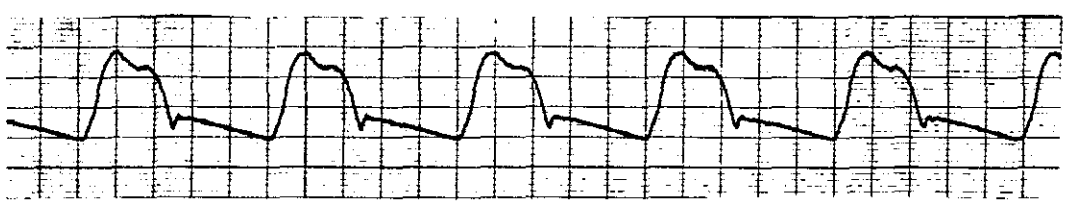


Fig. 5.4. Aortic pressure got from the physical model,

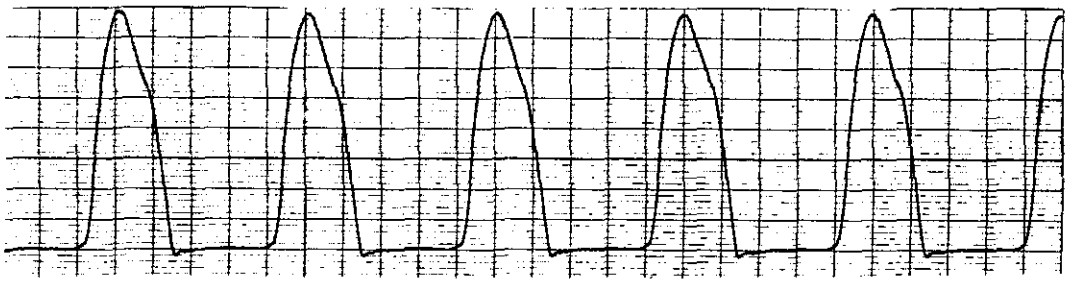


Fig. 5.5. Aortic flow in the arteficial aorta.

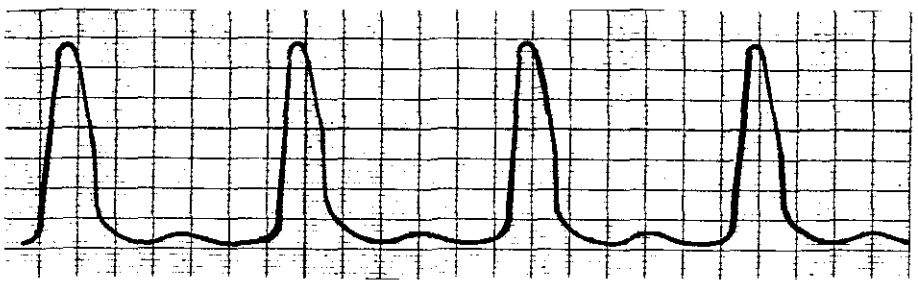


Fig. 5.6. Flow in the descending aorta of a man.

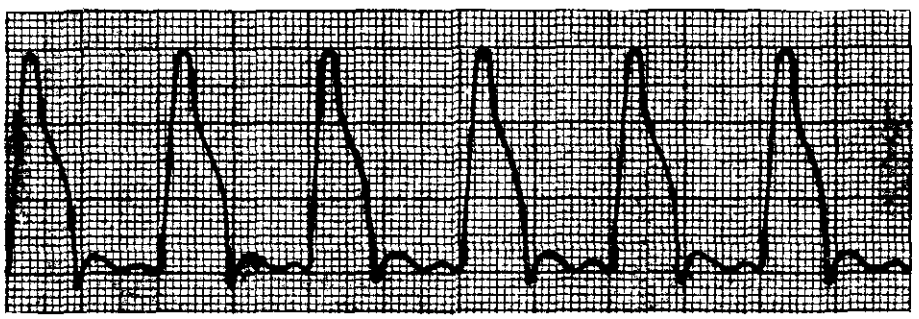


Fig. 5.7. Flow in the aortic root of a dog.

This means that the hemodynamic impedances used in the physical model have the correct values and the aim of building a realistic mock circulation system has been fulfilled to a very satisfying degree.

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6. References.

1. Sumner H. Levine; Advances in Biomedical Engineering and Medical Physics (Volume 1),
   Interscience Publishers, New York, London, Sydney.

2. Yukihiko Nosé, Sumner H. Levine; Advances in Biomedical Engineering and Medical Physics (Volume 3),
   Cardiac Engineering Interscience Publishers,, New York,.London, Sydney.

3. S.D. Moulopoulos, S. Topaz, K. Hiller, W.J. Kolff; Proper pressure curve shape in ventricles of intrathoracle mechanical hearts.
   Journal of Appl. Physiol. 17, 1025 (1962).

4. Knut H. Leitz, Miroslew Klain, Paul Phillips, Willem J. Kolff; Testing of Artificial Hearts in a Circulation Model.
   Proc. Biomedical Symposium on Engineering in Medicine, Marquette Univ. June 1966, pp. 287 - 290.

5. C.J. Mills, I.T. Gabe, J.H. Gault, D.T. Mason, J. Ross, Jr., E. Braunwald, J.P. Shillingford; Pressure-flow relationships and vascular impedance in man.
   Cardiovascular Research, 1970, 4, 405-417.

6. A.C. Burton; Physiology and Biophysics of the circulation.
   Year Book Med. Publ. Inc., Chicago (1968).

7. V. Hardung; Vergleichende Messungen der dynamischen Elastizität und Viscosität von Blutgefäszen, Kautschuk und synthetischen Elastomeren.
   Helv. Physiol. Acta, 11 (1953), 194 - 211.

8. Robert F. Rushmer; Cardivascular Dynamics Second Edition.
   W.B. Saunders Company, Philadelphia and London.