THE UNIVERSITY OF MANITOBA

THE PERFORMANCE OF PROSTHETIC HEART VALVES AND ARTERIAL IMPLANTS

A THESIS BY

PAULINO JOSE LOZADA

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE OF:

MASTER OF SCIENCE

DEPARTMENT OF MECHANICAL ENGINEERING
WINNIPEG MANITOBA

April, 1979

THE PERFORMANCE OF PROSTHETIC HEART VALVES AND ARTERIAL IMPLANTS

BY

PAULINO JOSE LOZADA

A dissertation submitted to the Faculty of Graduate Studies of the University of Manitoba in partial fulfillment of the requirements of the degree of

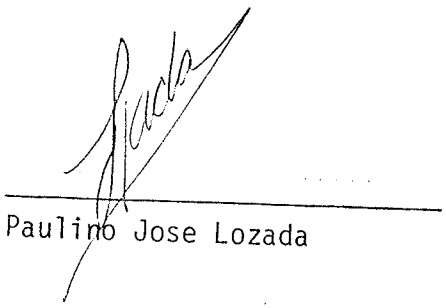
MASTER OF SCIENCE

© 1979

Permission has been granted to the LIBRARY OF THE UNIVERSITY OF MANITOBA to lend or sell copies of this dissertation, to the NATIONAL LIBRARY OF CANADA to microfilm this dissertation and to lend or sell copies of the film, and UNIVERSITY MICROFILMS to publish an abstract of this dissertation.

The author reserves other publication rights, and neither the dissertation nor extensive extracts from it may be printed or otherwise reproduced without the author's written permission.

I hereby declare that I am the sole author of this thesis. I authorize The University of Manitoba to lend it to other institutions or individuals for the purpose of scholarly research.



Paulino Jose Lozada

ABSTRACT

The commercially available arterial prostheses when implanted in the cardiovascular system produce reflected pulse pressure waves as a result of their inextensibility relative to the artery material. The result of these reflections could be higher non-physiological pressure waves that could damage the host organism. Turbulence and high shear stress in the blood flow induce thrombosis and hemolysis. Prosthetic heart valves that are not properly hydrodynamically designed can produce turbulence and high shear stress which is reflected as high energy losses in the blood flow when passing through the valves.

A mechanical model that simulates the human circulation in the left ventricle and ascending aorta was developed in order to reproduce and measure the reflected wave phenomenon and to evaluate a possible means of reduction of the reflected wave. One such means consisted of placing an elliptical insertion in the artery instead of the inextensible round insertion. The mechanical model was also used for evaluating the energy losses in prosthetic heart valves when physiological flow is induced.

The performance of the simulated heart-valve-artery system was satisfactory in efficiency, durability, frequency control, flow generation, valve behaviour and in data reading and data recording aspects. Some unwanted wave reflection was always present, and some viscoelastic parameters of the tubes were not known. These factors were constant, however, thus the results' comparative behaviour are valid. Round insertions in the elastic tube produced refected waves, thus modifying the pressure pattern, causing higher pressure peaks. The elliptic insertion did not produce as high pressure peaks as did the round rigid insertions.

Reflection factors due to rigid insertions were measured and the results showed some disagreement with the predictions based on the available idealized theory.

It was found that the Bjork-Shiley prosthetic heart valve was hydrodynamically superior to the Starr-Edwards. Fourier techniques proved to be valuable for analysing pressure pulse waves.

Experimentation with this model proved to be an inexpensive guide to the determination of variables which should be recorded and controlled. The results appear to make experimentation with animals regarding the variables measured here appear worthwhile.

ACKNOWLEDGEMENTS

The author would like to thank his supervisor, Professor A.B. Thoronton-Trump for his guidance and financial assistance through NRC Grant 8920 during the course of this research. He would also like to thank The University of Manitoba for its support and facilities.

The author thanks Professors K. McLachlan, J. Tinkler and E. Shwedyk for their help and advice. Finally, the author extends his warmest thanks to Jim Sewell for technical support and to all the other students with whom he shared work and enjoyment.

TABLE OF CONTENTS

Page

LIST OF FIGURES

Page

LIST OF TABLES

NOMENCLATURE

A	pressure gradient, real or complex.
A', A_1', A_2'	pressure - gradient modulus.
a	damping constant.
a_1	inner radius.
a_2	outer radius.
b	phase constant.
B	$E/(1 - \sigma^2)$ relation of the elastic constants.
c	complex wave velocity. Real part represents phase velocity and imaginary part represents wave attenuation.
c_o	$c_o = (Eh/2\ \rho_c\ a_1)^{1/2}$ phase velocity for the perfect fluid.
c_1, c_2, c_3, ...etc.	phase velocity, general.
D_1	arbitrary constant.
E	Young's modulus of elasticity.
E_1	arbitrary constant.
F	function of.
F_{10}	$F_{10} = \dfrac{2\ J_1\ (\alpha\ i^{3/2})}{\alpha\ i^{3/2}\ J_o\ (\alpha\ i^{3/2})} = 1 - M_{10}'\ e^{i\ E_{10}'}$ variable related to the complex wave velocity.
f	rotational frequency in revolutions per unit time.
H_1, H_2	total energy in a fluid.
H_{1-2}	total energy losses due to friction in a fluid.
h_{10}	$h_{10} = \dfrac{2M_1}{\alpha M_o}$ constant related to the modulus of Bessel functions.
h	$h = a_2 - a_1$ wall thickness.

I_v $\quad I_v = \dfrac{(1 - i)\, x'\, J_0\{(1 - i)\, x'\}}{J_1\{(1 - i)\, x'\}}$

variable related to the complex wave number in visco-elastic tubes. This function is tabulated in reference (19) for values of x' from 0.1 to 110. I_v approaches infinity if μ approaches zero.

I_o $\quad I_o = \dfrac{i\, Ka_1\, J_0\,(i\, Ka_1)}{J_1(i\, Ka_1)} \approx 2$

variable related to the complex wave number in visco-elastic tubes.

I_1 real part of I_v.

I_2 imaginary part of I_v.

i $\quad i = \sqrt{-1}$.

$J_0\,(\quad)$ Bessel function of zero order.

$J_1\,(\quad)$ Bessel function of first order.

K $\quad K = ia + b$ complex wave number.

k $\quad k \simeq h/a_1$ constant that relates wall thickness to inner radius.

L physical constant related to damping.

M'_{10} $\quad M'_{10} = \sqrt{1 + h_{10}^2 + a\, h_{10} \cos \delta_{10}}$ constant related to modulus of Bessel functions.

M''_{10} $\quad M''_{10} = |1 + \eta\, F_{10}|$ constant related to modulus of Bessel functions.

M_o modulus of the Bessel functions of the zero order.

M_1 modulus of the Bessel functions of the first order.

N $\quad N = \dfrac{(I_v - I_o)\,\rho}{\rho_o} + I_o\, I_v\,(\tau + 1 + \sigma) + \tau\,(1 = 2\, I_o \sigma)$

variable related to the complex wave number in visco-elastic tubes.

N' $\quad N' = I_o\,(1 + \sigma)\,(\tau + \dfrac{I_v\,\rho}{\rho_o})\,\{1 + \tau\,(1 - \sigma)\}$

variable related to the complex wave number in visco-elastic tubes.

n	$n = 2\pi f$ angular velocity in radians per unit time.
p	pressure, real or complex.
p_o, p_1, p_2	modulus of complex pressure.
r	radial axis in cylindrical coordinates, or position along the radial axis.
t	time.
u	fluid velocity component in the radial direction, real or complex.
u_1	radial complex - velocity modulus of the fluid.
$\overline{V}(r, z, t)$	displacement vector.
w	fluid velocity component in the axial direction, real or complex.
w_1	axial complex-velocity modulus of the fluid.
x	$x = R(1 - \sigma_2)$ variable related to fluid velocity in thin walled elastic tubes.
x'	$x' = a_1 \left(\frac{\rho_o n}{2\mu}\right)^{1/2}$ non-dimensional constant that relates some properties of tube and fluid.
y	$y = r/a_1$, relation between radial position and inner radius.
z	axial axis in cylindrical coordinates, or position along the axial axis.
Z	any real variable.
α	$\alpha = a_1 \left(\frac{n}{\nu}\right)^{1/2}$ non-dimensional constant that relates some properties of tube and fluid.
γ	the propagation constant.
γ'	$n \mu_{vi}/\mu_s$ relation of some viscoelastic constant (Voigt solid).
∇	vector differential operator.
ζ	inner wall displacement in the z - direction.
λ	wavelength.
λ_s	$\lambda_s = E\sigma/[(1 + \sigma)(1 - 2\sigma)]$ Lame's elasticity parameter.

μ dynamic coefficient of viscosity of the fluid.

μ_{vi} viscous constant of the wall material (Voigt solid).

μ_s $\mu_s = E/[2\ (1 + \sigma)]$ Lame's elasticity parameter.

υ kinematic coefficient of viscosity of the fluid.

ξ tube inner wall displacement in the r direction.

ρ wall density.

ρ_o fluid density.

σ Poisson's ratio

τ $\dfrac{2a_1/a_2}{a_2/a_1 - a_1/a_2}$ constant that relates inner and outer radii.

E'_{10} $E'_{10} = \arctan\left(\dfrac{h_{10} \sin \sigma_{10}}{1 + h_{10} \cos \sigma_{10}}\right)$, constant related to the phase of Bessel functions.

E''_{10} E''_{10} = phase of $(1 + F_{10})$, constant related to the phase of Bessel functions.

σ_{10} $\sigma_{10} = \dfrac{3\pi}{4} - \theta_1 + \theta_0$, constant related to the phase of Bessel functions.

CHAPTER I INTRODUCTION

From theory and experience it is known that prosthetic arteries and heart valves produce alterations in the hydrodynamic characteristics of the blood flow when implanted in the cardiovascular system [8, 16, 20, 27, 35] as well as other side effects. Since the mortality figures for the users of the Bjork-Shiley and the Starr-Edwards prosthetic heart valves appear significantly different [11, 13, 18], measurements of the alterations of the pulse pressure waves caused by each type of valve and comparison of pressure patterns produced by the Bjork-Shiley tilting-disc and Starr-Edwards ball-and-cage prosthetic heart valves should be made to determine whether basic hydrodynamic differences could possibly contribute to the decreased life of the users of the one prosthetic valve. The design of the Bjork-Shiley tilting-disc heart valve has hydrodynamic characteristics closer to those known to be the best for the blood, heart and arteries than does the Starr-Edwards ball-and-cage heart valve [8, 27]. Confirmation, evaluation and quantization of such improvements is necessary.

Alterations of pulse pressure are also caused by an arterial prosthesis as a result of its rigidity relative to the artery itself. The extensibility of most knitted prostheses is only about 10% of that of the host vessel [16, 35]. Reflected pressure waves from a prosthesis may be added to the incident waves from the heart, thus producing higher non-physiological pressure pulses on the artery itself and on the heart valves if the insertion is in the aorta, close enough to the heart valves so the refected waves are not significantly damped

before reaching the valves.

In order to investigate the pressure wave characteristics, the development of a heart-valve-artery model is necessary since "in vivo" measurements are beyond the scope of this work. The model must be designed to reproduce as closely as possible the flow characteristics of the human circulation in the aorta, which may be simulated by an elastic tube. It is possible to insert inextensible sections into the elastic tube which play the role of the inextensible prosthesis and, with pressure-transducers, to measure their effect on the pulse pressure wave. A possible method to reduce the magnitude of the reflected wave is to place in the test circuit an insertion with elliptic cross-section, instead of the round inextensible cross-section often used. In response to the pressure the elliptic insertion would approach a circular configuration, allowing greater flow cross-section thus reducing the pressure peak. The magnitude of this pressure wave reduction depends on the characteristics of the ellipse and the range over which the pressure excursions take place.

The equipment necessary to allow measurement of the pressure wave characteristics of the prosthetic arterial implants can be easily adapted to measure the effects of the prosthetic heart valves on the simulated arterial flow. The results of such investigations may be related to the characteristics known to be best for the cardio-vascular system.

CHAPTER II REVIEW OF LITERATURE

The significance of changes in the hydrodynamics of blood flow as a result of implanted prostheses lies in the possible disruption of the ability of the blood to perform its functions. The disruption of the blood flow characteristics may also have an adverse effect on the functions of other physiological materials such as high pressures on heart valves or restriction of arterial movement. The importance of these aspects of blood flow can be appreciated through a review of the literature.

II.1 Principles of human circulation

The blood circulation duty is to maintain the constancy of the internal environment of the organism. In doing so, it must: bring oxygen to the tissues, exchange it for the metabolic CO_2, carry this gas back to the lungs, release it to the atmosphere and replace it with oxygen; at the same time the blood picks up nutrients from the digestive tract, brings them to be processed and stored in the appropriate organs and performs the final distribution of nutrients and transport of the waste products to the organs of excretion. It also plays an important role in the regulation and coordination of the body by distributing the hormones of the endocrine glands. It participates in the regulation of water and electrolytes as well as regulation of body temperature. Another duty is the production and distribution of antibodies for protection against foreign particles [5, 10].

The circulation from the heart to the lungs and back to

the heart is called Pulmonary Circulation. The circulation from the heart to the body and back to the heart is called Systemic Circulation. In order to pump the blood through both systems the heart has a set of conveniently located valves. Figure II.1 is a schematic diagram of the double circulation and the heart with its valves [10].

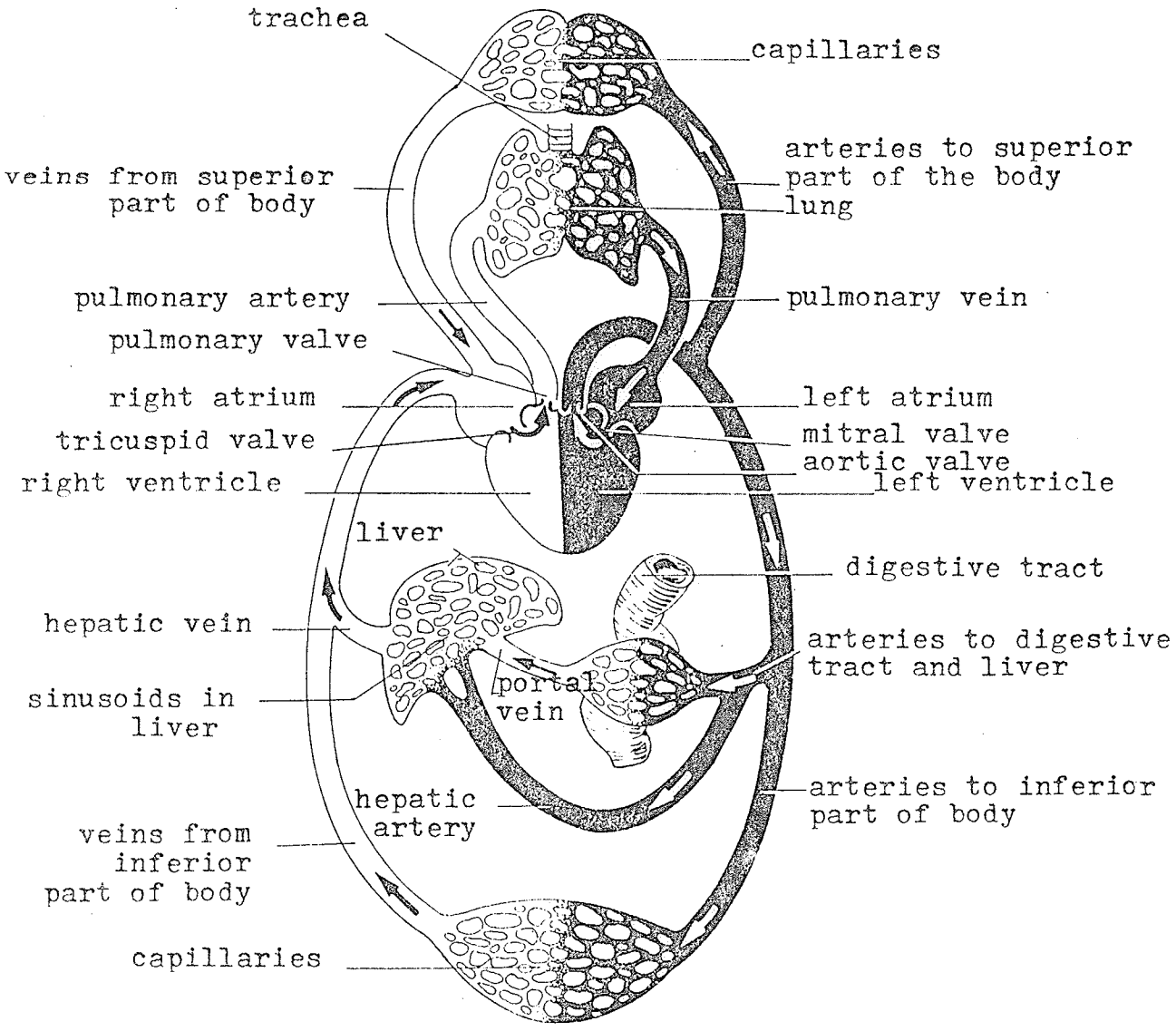


Fig. II.1: Scheme of the double system of circulation. Oxygenated blood is indicated in black, the nonoxygenated in white. Arrows show the direction of the blood flow. (Reproduced from [10]).

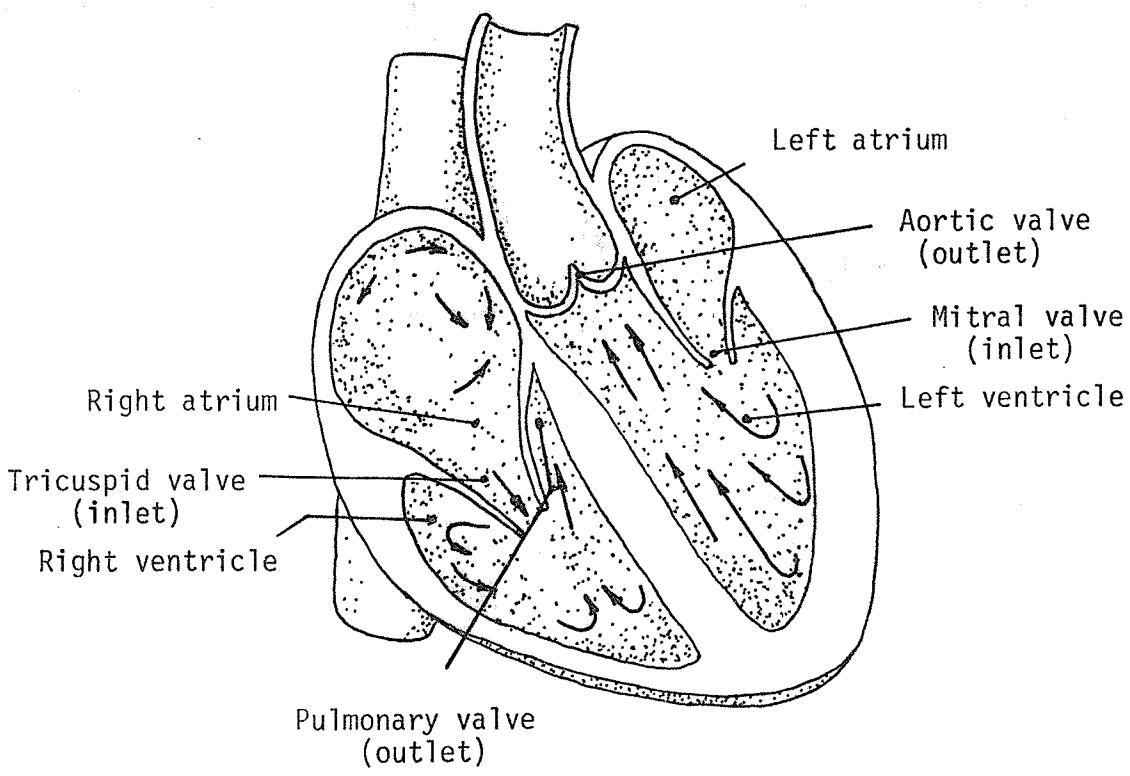


Fig. II.2: Schematic figure of the heart (Reproduced from [26]).

The heart is a pump with four chambers whose function can be visualized with the help of Fig. II.2.

The chambers are classified in accordance with their position: right atrium, right ventricle, left atrium, and left ventricle. The contraction of the heart chambers is called systole and the relaxation is called dyastole. The atria are chambers that have a minor function since they only help filling their respective ventricle. The major pumping action is exerted by the ventricles. The valves control the flow by means of a passive function [26, 29]; they close or open depending on which side the pressure is higher. The blood from the systemic system is received in the right atrium and pumped to the right ventricle where it is, in turn, ejected to the pulmonary system, received by the left atrium, passed to the left ventricle and finally ejected to the systemic circulation. The atria do not have double valves, so when they contract some blood refluxes. Since the ventricles have double valves, the regurgitation is negligible in the healthy heart.

The cardiac function already described can be classified by a separation of five events based on the closed or opened position of the ventricular inlet and outlet valves (see Fig. II.3). The duration and timing of these events are of particular concern in the design of prosthetic heart valves. Notice that the inlet and outlet valves are never opened at the same time [26, 29]. The five events specified in appropriate order for analytical purposes are:

(1) Filling (inlet open, outlet closed)

(2) Atrial Systole (inlet open, outlet closed)

(3) Isovolumetric Contraction (both closed)

(4) Ejection (inlet closed, outlet open)

(5) Isovolumetric Relaxation (both closed)

(1) Filling. The outlet valves have closed before this even starts A rapid increase in the volume of the ventricular chambers produces a fast drop of pressure in these chambers resulting in a rapid rate of filling through the atrioventricular valves. This is followed by a more moderate rate of increase of ventricular volume that produces a reduced rate of filling.

(2) Atrial Systole. Atrial Systole concludes the filling of the ventricular chambers. At the end of this event there is a drop of pressure in the atria that produces the closure of the atrio-ventricular valves.

(3) Isovolumetric Contraction. Inlet and outlet valves of the ventricles are closed when the heart starts its contraction so pressure increases very fast in the ventricles. The ventricular pressure soon reaches and exceeds the arterial pressure which has been decreasing due to the runoff of blood through the peripheral arteries. When the ventricular pressure exceeds the arterial pressure, ventricular outlet valves open, ending the isovolumtric contraction.

(4) Ejection. Initially there is a high rate of ejection due to the strong contraction of the ventricle. The ventricular pressure remains above the aortic pressure, reaches its maximum then drops slightly below the aortic pressure. At this point a more gradual

contraction of the ventricle produces a reduced ejection and the flow continues due to the inertia of the mass of the blood pumped. Finally, the inertial flow is reduced and by the end of the event there is a slight backwards flow that perhaps helps in the closure of the ventricular outlet valves. This closure produces a small secondary peak in the aortic pressure wave, as shown in Fig. II.3.

(5) Isovolumetric Relaxation. Inlet and outlet valves are closed again so the ventricular volumes can not change but the orientation of the musculature does so in order to relax. This relaxation produces a fast pressure drop in the ventricles that soon falls below the atrial pressure and the atrioventricular valves open to start the next cycle.

The sequence of events is similar in both sides of the heart, except that the peak systolic pressure in the left ventricle is about five times higher than that in the right (120 mm Hg left, 24 mm Hg right), and isovolumetric relaxation and isovolumetric contraction are of slightly shorter duration in the right side [26].

Since the aim of this investigation deals with the events in the left heart, the aorta and its main branches, attention is concentrated on this part of the circulatory system. Fig. II.3 is a graph where the events in the left heart and ascending aorta can be visualized in a quantitative way. Instantaneous flow measured in the ascending aorta of dogs and adapted to values expected in man is also shown in Fig. II.3 [29].

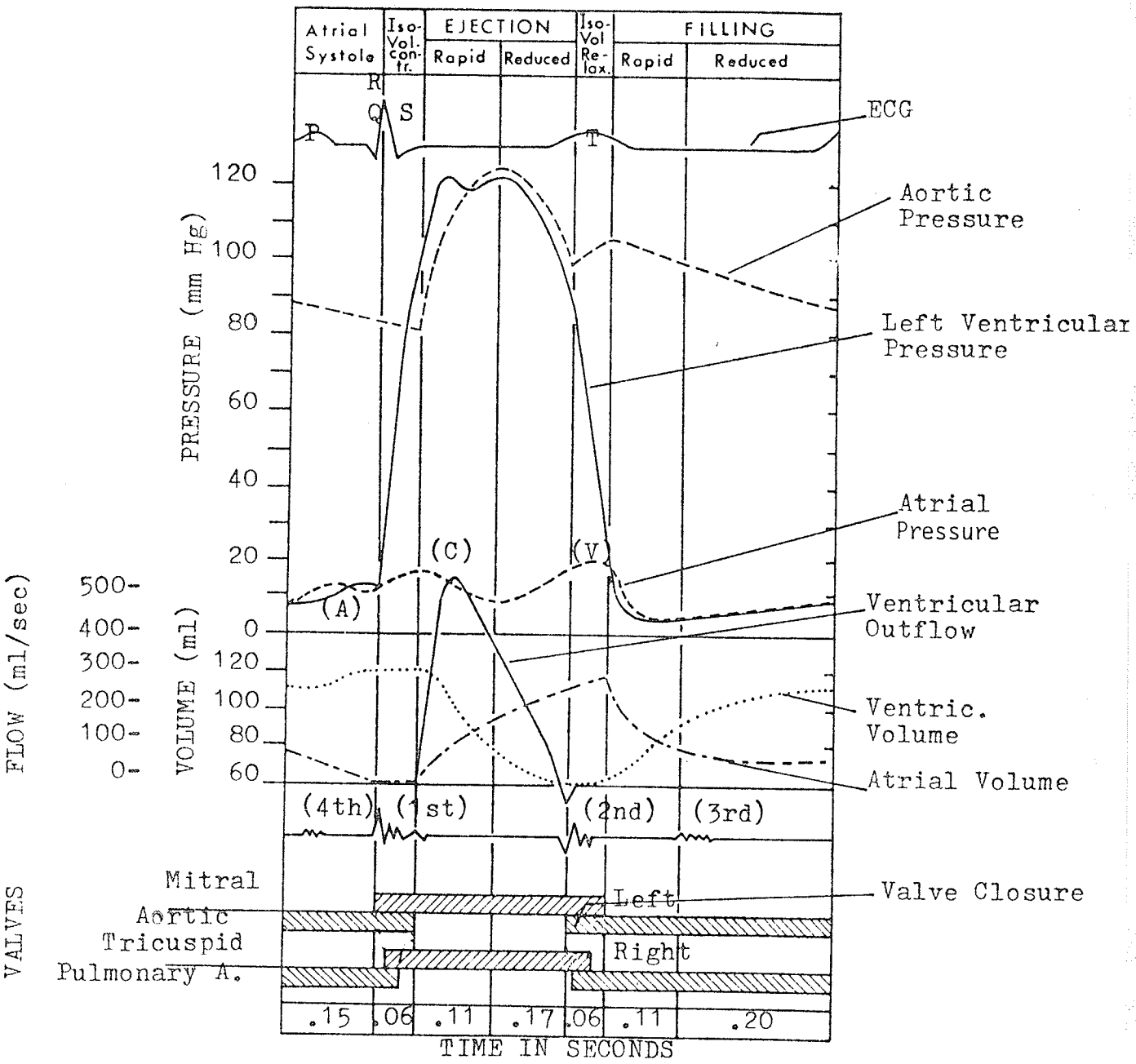


Fig. II.3: Pressure and volume events in the left heart during the cardiac cycle (Reproduced from [29]).

II.2 History of arterial implants

The use of arterial implants is recommended when the arteries have been blocked by emboli (blood clots circulating in the blood), occlusion or severe stenosis (narrowing of the artery), when they have been damaged by trauma (injury by the application of external force or violence), and when they are diseased, such as by advanced arteriosclerosis (narrowing, hardening and loss of elasticity) or aneurism (abnormal dilation).

The first attempts at implantation were done between 1914 and 1947 using rigid materials such as tubes of silver, glass and aluminum; but the success was negligible because the surfaces of these materials are highly thrombogenic (produce blood clots that remain attached to its place of origin) so most grafts were occluded within hours. The first major advances were obtained by Hufnagel in 1947 [21, 22] using tubes made of the thermoplastic polymethyl methacrylate, for implants in the thoracic aorta of dogs.

Homografts and heterografts have been tried also, with different degrees of success but these kinds of implants will not be studied in this work. However, the main problem with artificial grafts was that when the blood came in contact with the surface, spontaneous thrombus (blood clots that remain attached to its place of origin) were produced. The modern design of grafts was initiated by Voorhees et al in 1952 [21, 22]. It was based on the assumption that a porous arterial graft would be sealed with fibrin (fibrous protein formed in the clotting of the blood), thus avoiding leakage and providing an organic layer for

the interface between blood and foreign material. It was expected that this organic layer would be less susceptible to thrombosis. The material used for the first experiments was cloth mesh of Vinyon "N" fashioned into tubes. Implanted in the abdominal aorta of dogs and with anticoagulant therapy it remained patent for 153 days [21, 22]. It was found that the porous prosthesis could be preclotted with host blood before implantation thus reducing or avoiding seepage.

The concept of tubes made of porous material has remained as the most suitable concept for arterial substitutes [21, 22]. Fig. II.4 is a cross-section of a prosthesis with the layers that grow after implantation [22].

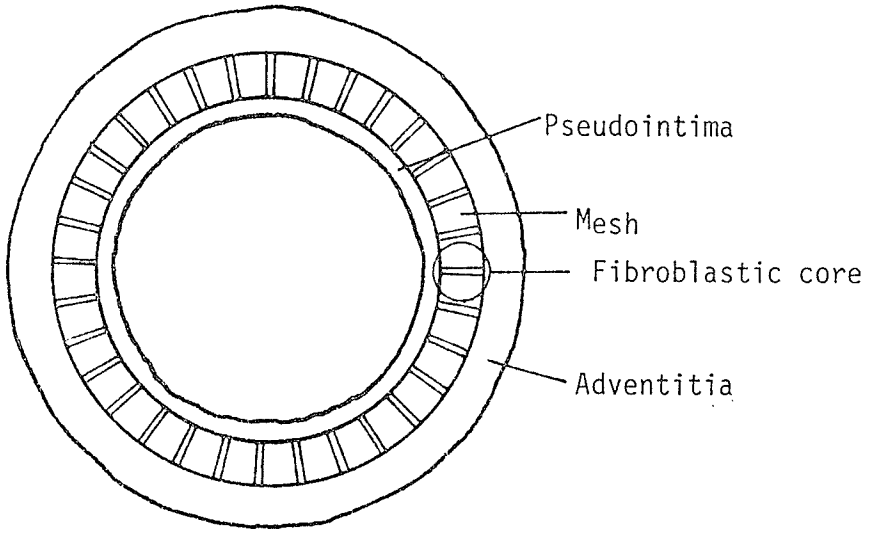


Fig. II.4: Cross-section of a prosthesis with the layers that grow after implantation. (Reproduced from [22]).

After the first work of Voorhees et al, many improvements have been made. Improvements in construction have consisted of refinements which have made the implant more functionally acceptable to the host. Since the pseudointima might thicken as much as 20% of the prosthesis interior diameter, it is convenient to build the graft's internal diameter higher than that of the host vessel. It was found also that a higher porosity would allow a more rapid and better formation of the pseudointima, thus reducing the chances of thrombogenesis. Knitted fabrics have higher porosity and more elasticity than the initial woven ones, (see Fig. II.5) but woven fabrics are difficult to suture since they fray easily. Knitted fabrics do not fray at the suture even if they are cut at different angles. Crimping the prosthesis walls reduces kinking and gives some extensibility which is desirable for prostheses that pass across the joints of the body.

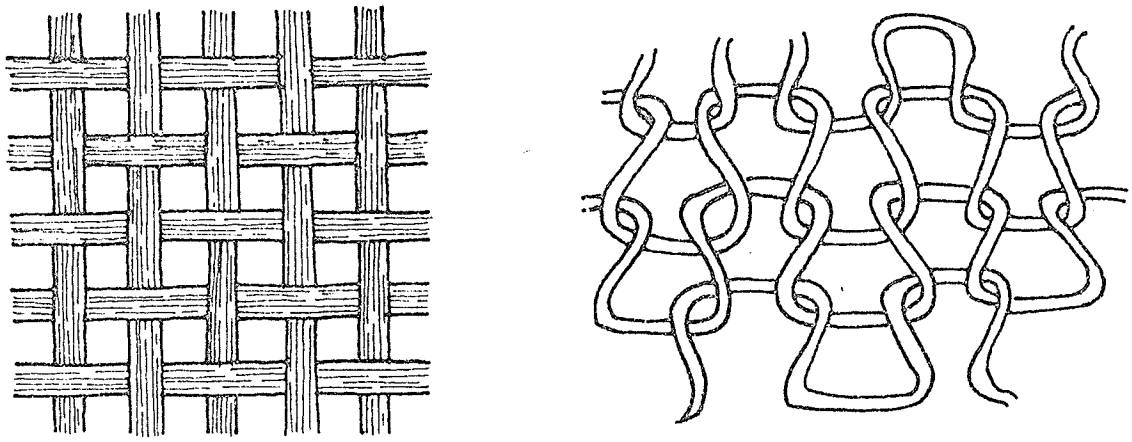


Fig. II.5: Difference between woven and knitted fabrics. (Reproduced from [22]).