PULSATILE FLOW TESTING AND DEVELOPMENT OF PROSTHETIC HEART VALVES IN CONDUITS

SIMON LEEFE

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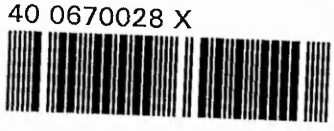

ABSTRACT

The work presented herein documents the development of a prototype valved conduit, building on previous work indicating that significant improvements in conduit haemodynamics were possible. Original contributions to this effort made by the author include an analysis of leakage data from which valve design guidelines are presented, the analysis of occluder instability during forward flow through an early prototype ball valve conduit and the development of an outlet profile to overcome this problem.

The problem of performance evaluation is addressed. Since the fluid dynamical situation of a conduit-mounted valve differs from that for which a "hydraulic efficiency" or some similar performance indicator based on transvalvular pressure drop may be derived, an ideal orifice pressure drop does not represent a theoretical limit in conduit design. This consideration, together with the question of how to present data when the conduit is used as a second ventricular outflow tract prompts a rigorous enquiry into energy loss methods as an alternative criterion for conduit evaluation, demonstrating, for the first time, the particular suitability of this approach to valved conduits both as valvular replacements and, particularly, when used to provide a second ventricular outflow tract.

It is demonstrated experimentally that the promising results of steady flow tests on the ball valve prototype may be realised under pulsatile flow conditions, for both modes of usage. The performance data are compared with those from a commercially available device in identical circumstances, constituting the first pulsatile test data for valved conduits.

Dedication

This thesis is dedicated to my dear daughter, Jessica,

who keeps me in touch with the less serious things in life,

and to all those who may benefit from the work described herein.

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Thanks must go first to Dr. C. R. Gentle, my supervisor and Director of Studies. As well as providing the framework and starting point for much of the work presented herein, his advice and comments have been most helpful. I am also enormously indebted for his practical assistance. Thanks are also extended to Mr. M. Keysell, second supervisor.

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AIMS

- To discuss the design of valved extracardiac conduits and their *in vitro* and *in vivo* performance in the context of published literature

- To form a rational choice between two contending generic design types for further development and to refine the design of the chosen conduit

- To establish suitable evaluation criteria for pulsatile testing of conduits, both as valvular replacements and as second ventricular outflow tracts

- To establish baseline data on pulsatile flow testing of a commercially available conduit incorporating a mechanical valve in both valvular replacement and shunt mode

- To demonstrate that performance improvements may be attained by attention to haemodynamic design

- Specifically, to demonstrate that the promising results of steady flow tests may be realised under pulsatile flow conditions

- To make suggestions concerning constructional detail of a prototype for further *in vitro* and *in vivo* studies

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Nomenclature

b	breadth of leakage gap
c_c	correction factor for orifice area to account for *vena contracta*
c_v	loss coefficient
d	diameter
d_i	duct diameter at section i
d_c	conduit diameter
dA	area element of boundary
$\mathbf{dA}$	vector of boundary area element
e	specific internal energy
f	friction factor ($= \tau_w/(1/2.u_m^2)$
	frequency of cardiac cycle
g	acceleration due to gravity
h	elevation above a horizontal datum plane
	offset of piston and cylinder axis from crank drive centreline
l	characteristic length scale of turbulence
n	exponent in turbulent velocity distribution relationship
	distance along normal direction
p	pressure
p_i	pressure at section i down a duct
p^*	piezometric pressure ($= p + \rho gh$)
p^*_i	piezometric pressure at inlet or outlet section i of control volume
p^*_v	ventricular piezometric pressure
q	magnitude of local velocity vector
$\mathbf{q}$	velocity vector
$\mathbf{q_r}$	local velocity vector of the fluid relative to the boundary
r	radial coordinate
	ratio d_3/d_c
	crank throw
r_o	conduit radius
s	length of flow path
t	time
t_1	time elapsed from instant when crank is horizontal to when piston is at its greatest extent
t_2	time elapsed from instant when crank is horizontal to when piston is at its least extent

u	fluid mean velocity
	component of local velocity in the x-direction
	quasi-steady local velocity in the x-direction in turbulent flow
u'	fluctuating component of local velocity in the x-direction in turbulent flow u_m .
	mean flow velocity across a section
u_o	centreline flow velocity for a symmetric velocity distribution
u^*	friction velocity ($= \sqrt{(\tau_w/\rho)}$)
v	fluid mean velocity
	component of local velocity in the y-direction
v'	fluctuating component of local velocity in the y-direction in turbulent flow
w	component of local velocity in the z-direction
x	Cartesian coordinate
x_1	horizontal distance from crank drive centreline to con-rod/piston pivot with piston at greatest extent
x_2	horizontal distance from crank drive centreline to con-rod/piston pivot with piston at least extent
y	Cartesian coordinate
	distance from the wall
z	Cartesian coordinate
A	area
	constant
	drive-piston and cylinder cross-sectional area
A'	corrected area
A_i	cross-sectional area at section i down a duct
B	constant
C	constant
K	figure of merit (see Viggers *et al* - 1969)
K_1	constant
Q	volumetric flowrate
Q_d	diastolic (retrograde) flowrate
Q_i	volumetric flowrate at inlet or outlet section i of a control volume
Q_s	systolic (forward) flowrate
R_b	radius of ball occluder
R_o	spherical polar coordinate of point on profiled inner surface of conduit outlet duct, with origin defined by centre of ball occluder in fully-open position
Re	Reynolds number ($= u_m.l/\nu$ where l is a typical geometric length scale)
S.V.	stroke volume
T	cardiac cycle time

W	work done per unit volume by a fluid element on its surroundings
W_{walls}	work done per cycle by part of control volume boundary formed by walls
W'_{walls}	rate of work done on the fluid by the solid boundary
α	fraction of the forward flow taken by the conduit in shunt configuration
	multiplier representing variation of systolic to cycle time from 50% with introduction of piston offset, h
β	systolic fraction of the cardiac cycle time
δ	leakage gap width
δV	elemental volume
ϕ'	dissipation function
$\phi'_{K.E.}$	change in convected kinetic energy between inlet and outlet to a control volume (inlet - outlet)
κ	Von Karman's constant
μ	dynamic viscosity
μ_t	apparent turbulent viscosity (defined by $(\tau_R)_{xy} = \mu_t \partial u/\partial y$)
ν	kinematic viscosity ($= \mu/\rho$)
θ	angle from conduit centreline
ρ	fluid density
σ_{jj}	normal stress on fluid element in direction j
σ_n	outward normal direct stress on boundary
τ	in-plane shear stress on boundary
τ_{ij}	shear stress in direction j on the face of a fluid element normal to direction i
$(\tau_R)_{xy}$	Reynolds shear stress in the x-y plane ($= -\rho.\langle u'.v' \rangle$)
τ_w	wall shear stress
ψ	multiplier representing deviation of stroke volume from twice crank throw times piston area with introduction of piston offset, h
ω	drive crank angular velocity
Δh	drop in hydrostatic head (upstream - downstream)
Δp	drop in static pressure (upstream - downstream)
Δp_d	diastolic (retrograde) pressure drop
Δp_d	systolic (forward flow) pressure drop
Δp^*	drop in piezometric pressure from inlet to outlet
	difference in piezometric pressure between a stagnant point in the ventricle and the control volume outlet section
Φ	total energy dissipated per cycle inside control volume
Φ'	rate of energy dissipated inside control volume
Φ_d	energy dissipated inside control volume over ventricular diastole

Φ_s	energy dissipated inside control volume over ventricular systole
D/Dt	total derivative (= $\partial/\partial t + u.\partial/\partial x + v.\partial/\partial y + w.\partial/\partial z$)
<>	time average

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1 Introduction

1.1 General Introduction

Described in engineering terms, the human heart is a dual circuit positive displacement pump operated by the squeezing action of its muscular walls. Each side contains a suction and discharge non-return valve. The left side pumps oxygenated blood to the systemic circulation. Its suction valve, between atrium and ventricle, is the mitral valve, whilst discharge into the aorta is via the aortic valve. The right side pumps deoxygenated blood to the lungs. Its suction valve is the tricuspid valve, whilst discharge to the pulmonary arteries is via the pulmonic valve. This is illustrated in Fig. 1.1

Being biological structures, cardiac valves are prone to disease or malformation and hence may require replacement. This is usually undertaken by excising the defective valve and sewing in an artificial replacement. In a small number of cases, however, for a variety of reasons this cannot be done, or the condition requires an alternative treatment. One alternative involves the use of a length of conduit tubing containing a prosthetic valve. Typically, this replaces an aortic valve plus a length of the aorta (Fig. 1.2) , or provides an alternative outflow tract from the left ventricle to the aorta (Fig. 1.3), or from the right ventricle to the pulmonary arteries (Fig 1.4).

Commercially available conduits employ a standard cardiac valve prosthesis mounted in flexible conduit tubing. The valves have been designed to separate a chamber from a downstream enlargement (a ventricle or a sinus cavity). However, one can hardly expect optimum performance if the same device is placed in a length of uniform bore tube, since the fluid mechanics differ. In valve replacement surgery, the geometry surrounding the valve is predetermined. However, with a conduit, the designer can take advantage of the freedom from geometrical constraints to develop a more streamlined valve, and thus improve performance,

in terms of the resistance to flow and of the power required to pump the cardiac output past the valve.

The demonstration of this argument represents the main endeavour of the work presented herein. However, original contributions in the fields of evaluation methodology and valve design, particularly in the control of leakage, also form a substantial part of the thesis.

1.2 Review of conduit development at Nottingham Polytechnic

Nottingham (formerly Trent) Polytechnic has been involved in two overlapping areas of heart valve design development: the all-ceramic mitral valve and the valved conduit. Since both programmes have overlapped chronologically and since there has inevitably been collaboration between workers, it is beneficial to outline the historical context of the current work, both to enable the reader to understand its development and to explain the unity behind the seemingly disparate strands of research.

Having demonstrated theoretically that there exists a limit to the reduction of forward flow pressure-drop, for a valve separating two chambers (Gentle 1977, 1977a and 1977b), and hence that a valvular "efficiency" can be derived in terms of a comparison to that ideal standard, Gentle (1982) went on to explain that the different fluid mechanical situation of a conduit-mounted valve offers the potential, with suitable attention to design, for vast improvement to performance. This was demonstrated by Gentle and Benjamin (1984) who tested typical commercially available conduits both of mechanical construction (Bjork-Shiley) and employing a tissue valve (Polystan).

In Gentle (1982), the caged ball principle was highlighted as offering particularly promising forward flow characteristics. Accordingly, a series of prototypes with different inlet and outlet profiles were constructed, and tested under steady flow conditions. The results were published by Benjamin (1986), who concluded that an included angle of 40^{o} for the diverging inlet section of the valve with 30^{o} outlet angle gave the best results out of the models tested in terms of forward flow pressure drop. Comparison was also made with two mechanical valved conduits (Bjork-Shiley with convexo-concave and spherical disc occluder, both 22 mm conduit diameter) and with a tissue valved conduit (Polystan porcine valved conduit, of 24 mm diameter). Despite the smaller diameter (16 mm), the pressure drop obtained with the prototype was comparable with that of the two mechanical prostheses. Tansley (1988) has presented the comparison in the more instructive non-dimensional

form of Euler number, $\Delta p/(1/2\ \rho v^2)$, versus Reynolds number based on conduit tube diameter for tests in both water and blood analogue solution. Significantly, the Euler number of the prototype conduit is the lowest at any given Reynolds number, suggesting haemodynamics superior even to those of the tissue valved conduit.

At the same time as this investigation into conduit design, work was progressing on the development of an all-ceramic mitral valve (Gentle 1980, Gentle *et al* 1981 and 1981a). The two principal innovative thrusts of this work, were: a) the use of porous ceramic to combine the durability of a mechanical valve with the low thrombogenicity of a tissue valve, due to the encouragement of a tissue growth over the prosthetic surface; and b) the use of a central flow valve whose orifice diameter was a large proportion of the tissue annulus diameter, implying low forward flow pressure drop. Both these features also made this valve an obvious choice for incorporation into a conduit design.

Leakage considerations, detailed in Chapter 3, drawing on results previously published by Rief *et al*(1980), Parsons (1981) and Gentle (1982) as part of the ceramic mitral valve development programme, dictated the selection of the caged ball principle as the route for development. However, before proceeding with this prototype, the problem of occluder instability highlighted by Benjamin (1986) and similarly noted by Viggers *et al* (1977) and Uglov *et al* (1984), needed to be addressed. This took the form of a high frequency axial oscillation of the ball. The approach adopted to overcome this problem and the results of this investigation are also outlined in Chapter 3.

The remaining design development was undertaken with the aim of minimising forward flow pressure drop without compromising stability. This work was performed by Tansley both experimentally and with the aid of computational fluid dynamics (CFD), and the results published (Tansley (1988)) as a PhD thesis.

In parallel with this work, preparations were underway for pulsatile flow testing, in order to provide validation of the promising steady flow results under more physiologically realistic conditions. This required the development of an appropriate evaluation methodology - a topic discussed at length in Chapter 4, whilst the apparatus, materials and test procedures are explained in Chapter 5. The pulsatile flow performance of the prototype optimised for steady flow is compared with that of a widely used mechanical alternative, both as an aortic replacement and as a shunt bypassing an outlet tract with a typical degree of stenosis, in which configuration the resulting energy loss is assessed and compared with the loss associated with the stenosed outlet alone. The results are presented and discussed in Chapter 6.

2 Literature Survey

2.1 Introduction

Looking back over the literature on heart valve surgery, it is with surprise that one discovers that successful research with valved conduits pre-dates heart valve replacement by some thirty years. Dembitsky and Weldon (1976) cite Jeger (1923) who successfully implanted an apico-aortic shunt in animal experiments. Indeed, Hufnagel, widely regarded as the pioneer of contemporary heart valve replacement operations, first reports (1948) unsuccessful experiments in which a graft containing an aortic valve is connected between a canine left ventricle and descending aorta. This was three years prior to his 1951 paper in which he demonstrated that dogs could survive with a prosthetic valve inserted into the descending aorta. This work came to fruition when, in 1953, the first prosthetic valve was inserted into a human. This was a caged ball valve, again placed in the descending aorta. Templeton (cited in Norman *et al* (1976)), appears to have made the first clinical use of a valved conduit, used as an apico-aortic shunt, in 1962. However, it is Harken (1960) who claims credit for the first successful clinical implantation of a prosthetic heart valve in the normal anatomical site. This work is briefly outlined in Harken (1977), which also offers a highly readable and succinct account of the early motivation for the development of a heart valve prosthesis and lists ten ideal criteria against which it ought to be designed. All of these, except the dictate that it should be capable of insertion in the normal physiological site, pertain equally to valved conduits and are as relevant now as when they were first enunciated some thirty years ago, and so are reproduced below as representing the context in which the present work should be viewed.

" 1. It must not propagate emboli.

2. It must be chemically inert and not damage blood elements.

3. It must offer no resistance to physiological flow.

4. It must close promptly (less than 0.05 sec.).

5. It must remain closed during the appropriate phase of the cardiac cycle.

6. It must have lasting physical and geometric features.

7. It must be inserted in a physiologic site - generally the normal anatomic site.

8. It must be capable of permanent fixation.

9. It must not annoy the patient.

10. It must be technically practical to insert."

The extent to which progress had been made against these criteria by the early 1970's is well summarised by Swales *et al* (1973). More recently, Black (1986) provides a succinct account of the present state of heart valve prosthesis design, highlighting the *pros* and *cons* of mechanical and tissue types as follows. Mechanical valves are basically durable and consistent, but their unnatural form can present problems of accommodation and pressure drop, whilst the materials of their construction usually require the patient to undergo postoperative anticoagulation treatment. Tissue valves, whilst avoiding these disadvantages, are prone to inconsistency in manufacture and (accumulating data suggests) long-term failure through tissue degradation and calcification (see for example Lawford *et al* (1986)). Recent developments in ceramics as valve materials (see, for example, Gentle's work, discussed in Section 2.5.2, below) indicate that anticoagulation treatment is not a necessary adjunct to the use of mechanical valves, whilst conduit-mounted prostheses do not suffer from the geometrical constraints encountered by the designer of valves for conventional replacement operations. These considerations lead one to the conclusion that there is great scope for the substantial improvement of valved conduits, and that the most appropriate line of research is into mechanical prostheses.

2.2 Conduit operations

This section aims to provide a historical survey of the development of valve-bearing conduits and an outline of the surgical procedures in which they are employed. A discussion of how anatomical and conduit design considerations affect *in vivo* performance is left until Section 2.4. The interested reader might wish to refer to Tuna and Danielson (1989) who provide a list of 45 key medical references.

2.2.1 Historical background and milestones

Credit for the first experimental use of extra-cardiac conduits usually goes to Carrel (1910), who established a bypass from the left ventricle to the abdominal aorta using a vein graft or a "paraffinised rubber tube". To put this work in context, the paper was primarily concerned with exploring the possibilities for thoracic cardiovascular surgery, given the contemporary constraints: asepsis, materials and, most importantly, lack of cardiopulmonary bypass. It was possible to block circulation only for a few minutes (which led the author into an interesting discussion of what actually constitutes death). Thus, the rationale was to establish a temporary bypass of the proximal aorta to enable surgical treatment of aortic aneurism. This pioneering work also covered experimentation with in-line venous grafts placed in the aorta.

Donovan (1950), as well as providing a useful summary of the main obstacles to successful heart valve surgery over the first half of the century, also reports canine experiments in right-heart surgical bypass. Jugular and femoral vein grafts were used to connect right ventricle to left pulmonary artery, with ligature of the proximal left pulmonary artery, so that right pulmonary arterial flow was supplied through the pulmonic valve, whilst left pulmonary arterial flow was provided through the graft, the proximal end of which was cuffed over a polythene tube.

Donovan and Sarnoff (1954) is often cited as the first account of the successful use, in

animal experiments, of a valved conduit. It was placed between left ventricular apex and descending aorta, with the ascending aorta ligated in order to divert the entire ventricular outflow through the prosthesis. However, Dembitsky and Weldon (1976) cite Jeger, who, as early as 1923 managed to keep animals alive for four days using a valved conduit as an apico-aortic shunt. Hufnagel, widely regarded as the pioneer of contemporary heart valve replacement operations, first reports (1948) unsuccessful experiments in which a graft containing an aortic valve is connected between a canine left ventricle and descending aorta. This was three years prior to his 1951 paper in which he demonstrated that dogs could survive with a prosthetic valve inserted into the descending aorta, thus marking the beginning of heart valve replacement surgery. The tubular form of this valve and its anatomical siting could lead one to argue that this, too, was in effect a valved conduit.

The first clinical use of a valved conduit appears to have been made by Templeton in 1962 (cited by Norman *et al* (1976)), followed closely by Al-Naaman (1963). Both surgeons were operating to create a second left ventricular outflow tract. Wheat *et al* (1964) offer the first report of the replacement of the aortic valve and ascending aorta. The first clinical use of valved grafts in reconstructive surgery is reported by Ross and Somerville (1966), whilst Wright *et al* (1981) appear to be the first to have used a valved extra-cardiac conduit as a mitral valve bypass.

2.2.2 The valved conduit as an apico-aortic shunt

This procedure is typically employed for the relief of severe congenital aortic stenosis, or as an alternative to prosthetic or natural valve replacement where the tissue annulus is very small or damaged by disease. In these cases, it is necessary to establish an artificial outflow tract from the left ventricle to the systemic circulation, bypassing the aortic valve, providing a parallel blood flow path. Thus a hole must be excised from the ventricular wall, typically at the apex, and a conduit inserted, running to either the ascending aorta or the descending thoracic or abdominal aorta. The conduit must be valved to prevent retrograde blood flow from aorta to ventricle during diastole.

Following the work of Templeton and Al Naaman, referred to above, the technique was developed and established in the 1970s. Detmer *et al* (1971) report aortic allograft implantations in calf trials, whilst Brown *et al* (1974) detail canine experiments employing Starr-Edwards caged ball, Bjork-Shiley tilting disc and Hancock porcine xenograft valves in Dacron conduits.

Bernhard *et al* (1975) detail the use of a composite apico-aortic shunt for the relief of congenital aortic stenosis. The shunt consists of flexibly-jointed 316 stainless steel tube sections with an external coating of polyurethane and internal lining of Dacron fibrils embedded in polyurethane elastomer to promote the formation of a haemocompatible surface. (A similar rigid stent design is reported by Chekanov *et al* (1990)). The distal end of the shunt is connected to a short length of Dacron tubing in which a porcine xenograft valve is mounted. Anticoagulation treatment was necessary until the biological tissue lining was fully formed (about one year). This paper was written too soon post-operatively to offer comment on the long-term success of this procedure, which is unfortunate, given the unconventional conduit materials employed.

By the end of the decade, the technique was becoming widely accepted. Much of the reported work was undertaken by Cooley and Norman's team, who favour the Hancock porcine valve in Dacron tubing (Cooley *et al* (1975), Cooley *et al* (1976), Norman *et al* (1976), Cooley (1977) - which offers a clear explanation of the procedure, and Cooley and Norman (1977)).

Cooley and Norman (1977) provide a graphic illustration of the rationale for an apico-aortic shunt operation, where the aortic annulus is of small diameter. Aortic replacement had resulted in a residual gradient of 55 mmHg and haemolytic anaemia, presumably related to high shear stresses in the turbulent flow through the small valve orifice. A shunt operation reduced this to 5 mmHg.

Weldon's discussion of Cooley *et al* (1976) outlines the *pros* and *cons* of the siting of both the distal anastomosis of the apico-aortic shunt and the valve-bearing section of the conduit. Abdominal positions would seem to offer better haemodynamics, avoiding a 180° flow reversal and improved prognosis where re-operation is indicated. Dembitsky and Weldon (1976) would appear to agree, since they report an apico-aortic shunt operation in which the distal anastomosis is to the intra-renal abdominal aorta. However, Narducci *et al* (1984) and McGoon (1976) describe a procedure in which the distal end is anastomosed to the ascending aorta. No reason is given for this location.

Brown *et al* (1977) report on the development of a procedure both to bypass and close the aortic root following the removal of an infected aortic valve prosthesis. This is accomplished by the use of a valved conduit from the left ventricular apex to the descending thoracic aorta. Results of five canine experiments are reported. Of the four surviving animals, a maximum gradient of 15 mmHg was recorded across the prosthesis. However, the authors provide no basis for comparison of this figure. The prosthesis used was a Starr-Edwards valve mounted in a Dacron tube equal in diameter, one supposes, to that of the valvular sewing ring. It seems likely, then, that this represents a very large forward-flow occlusion.

2.2.3 Replacement of the aortic valve and ascending aorta

In patients requiring replacement of the aortic valve who also suffer from disease of the ascending aorta, it is possible to treat both simultaneously by the removal of the valve and either removal or opening of the aorta and replacement with or insertion of a composite graft, in which there is no upstream length of conduit. Instead the valvular prosthesis is sutured into the tissue annulus, the coronary ostia are re-attached downstream of the valve and the distal end of the graft is sutured into the aortic arch.

In the first report of the replacement of the aortic valve and ascending aorta Wheat *et al* (1964) implanted a Starr-Edwards valve and a separate Teflon graft to replace the ascending aorta.

By the late 1970s the accepted technique was to employ a previously prepared composite graft with holes excised for the anastomosis of the coronary ostia after implantation (Kouchoukos *et al* (1977), Kouchoukos *et al* (1980), Mayer *et al* (1978), Piehler and Pluth (1982), Selle *et al* (1981) and Turinetto *et al* (1983)). Bentall, in his discussion of Mayer *et al* (1978), notes that the use of hot wire cautery for the incision of the holes in the Dacron graft produces firm edges and a superior seal.

In a typical procedure to insert the conduit, the ascending aorta is incised longitudinally, the diseased valve excised, the proximal end (which comprises the valve with its sewing ring) is sutured into place, whilst the distal end of the attached tube is sewn into the aorta just above the incision. The slit is then sewn up, so that the graft lies entirely inside the aorta. This contains bleeding through the conduit walls or suture lines. Other procedures avoid wrapping the conduit in the aorta. The arguments for and against are summarised by Kouchoukos (1989), who points out that wrapping provides a watertight seal, but can mask internal bleeding leading to subsequent formation of a pseudoaneurism (estimating that this occurs in between 7 and 25% of cases). The problem of leakage in an unwrapped conduit can be reduced by pre-clotting or, more recently, through the use of fibrin glue (see Section 2.5.2).

Selle *et al* (1981) - a good introduction to the use of conduits in the aortic position - discuss the advantages and disadvantages of various procedures for the replacement of the aortic valve and the ascending aorta with re-establishment of coronary flow. Bedderman *et al* (1980) report a higher survival rate with aortic valve and supracoronary ascending aorta replacement than with the insertion of a composite graft containing an aortic valve prosthesis. Following Mayer *et al*'s paper (1978), there is a discussion in which the question of the treatment of the coronary ostia is addressed. The alternatives are separate valvular and aortic graft implants with the graft profiled to accept the coronary ostia and surrounding tissue (Wheat *et al* (1964)), direct anastomosis to the graft (Kouchoukos *et al* (1980)) or the use of intermediary small grafts of PTFE (Piehler and Pluth (1982)) or

Dacron (Cabrol's discussion of Mayer *et al* (1978)) running between coronary ostia and the aortic graft. Another possibility is the replacement of the aortic root with a homograft conduit, in which the whole valve, sinus and ostia structure is left undisturbed (Stelzer and Elkins (1989)).

2.2.4 Valved conduits in the repair of congenital malformations

Another important application of valved conduits is in the repair of congenital abnormalities of the heart. This usually involves the placing of a conduit to restore adequate continuity between systemic return and the pulmonary arteries, bypassing a severe pulmonary stenosis. Such operations are necessarily frequently undertaken in children. Conduit size is then limited, particularly by the diameter of the vessel at the site of the distal anastomosis. As the child grows, so stroke volume increases, requiring the passage of a greater blood flowrate. This results in increased conduit gradient. This, and the problems of tissue valve degradation and possible conduit stenosis (see Section 2.4), imply reoperation at various intervals.

Early experiments in right-sided shunt operations are reported by Arai *et al* (1965), and the first clinical use of valved grafts in reconstructive surgery is reported by Ross and Somerville (1966) for the treatment of pulmonary atresia. Indeed, their use of allograft conduits opened the door to a variety of hitherto inoperable conditions (Campbell and Clark (1990)).

Rastelli *et al* (1969) detail the correction of transposition of the great arteries with ventricular septal defect and pulmonary stenosis, a condition also tackled by McGoon *et al* (1975), whose team is responsible for some detailed publications on the correction of a range of abnormalities, such as truncus arteriosus (McGoon *et al* (1968)), and the conditions described in McGoon (1976). In this paper four separate procedures involving conduits are reported. The first, for the correction of congenital aortic stenosis, involved the use of a Hancock porcine xenograft valve in a Dacron conduit, as an apico-aortic shunt (see

above). The second operation was for transposition of the great arteries with ventricular inversion, ventricular septal defect and pulmonary stenosis. The septal defect was repaired with a patch, whilst continuity of flow from right ventricle to pulmonary arteries was maintained by means of an extra-cardiac conduit to the right pulmonary artery, with ligation of the pulmonary trunk to prevent the flow of oxygenated blood back to the lungs. The third operation was for the correction of a double-outlet right ventricle with numerous complications. Two conduits were employed, both Hancock porcine xenograft valves in Dacron tubing, one to maintain continuity of flow from anatomical left ventricle to ascending aorta, the other from anatomical right ventricle to the bifurcation at the distal end of the pulmonary trunk. The fourth operation was for the repair of transposition of the great arteries with severe pulmonary stenosis and intact ventricular septum. This had previously been treated by the formation of an atrial septal defect and a Blalock-Taussig anastomosis, which augments low pulmonary flow with blood from the aorta. The condition was treated by providing an enlarged atrial septal defect, performing a Mustard operation (Mustard (1964)) for the reversal of venous return, and providing an extra-cardiac conduit from left ventricle to pulmonary trunk.

Stewart (1976) describes the use of extra-cardiac conduits (again the Hancock porcine xenograft valve variety) in a series of procedures for the reconstruction of various abnormalities, in each case to provide continuity between right ventricle and pulmonary artery. The use of the conduit is much the same as in McGoon, in that the proximal end is cut obliquely and sewn directly to the ventriculotomy without a stent. Another point worth noting is that in cases where the heart was in dextroversion, direct routing of the conduit was not possible because this would have risked conduit compression between sternum and ventricle.

Cartmill *et al* (1974), in their description of nine cases of right ventricular outflow reconstruction, prefer the use of the conduit-mounted Bjork-Shiley tilting disc valve, for reasons of long-term durability (see Section 2.4).

Other interesting references are de Vivie *et al* (1981), who report their experience over seven years with a variety of conduits in operations to repair a number of congenital malformations of the heart, Stelzer and Elkins (1989), who concentrate on the uses of homografts in reconstructive and reparatory surgery, and Castaneda and Norwood (1982), who provide a brief history of the use of valved conduits to provide adequate blood flow to the pulmonary arteries in the correction of congenital heart defects. This is a useful source of references detailing surgical procedures.

2.2.5 The valved conduit as a mitral valve bypass

Wright *et al* (1981) report the use of an extracardiac conduit to bypass an irreplaceable mitral valve. Although the procedure was undertaken on a 64-year old woman, the authors note that the same technique may prove useful in the correction of congenital or acquired mitral obstruction in children, so that the period between correction and re-operation might be extended.

2.3 In vitro tests

Since the inception of the current work, at the start of 1984, much attention has been paid to the issue of standardisation of test methods and performance criteria. Many of these issues are clarified by ISO 5840 (1989) on cardiac valve prostheses. A recent example of an *in vitro* test series attempting to work to these standards is provided by She *et al* (1989). Although ISO- and FDA-recommended mean-value pressure and flow parameters are used in the evaluation, the discussion points out the limitations on the use of the specified mean-based parameters in pulsatile tests. Other questionable areas include the ambiguity in the specification that a pulse duplicator shall "produce pressure and flow waveforms which approximate to those found in healthy adult humans". This immediately raises three questions:

- how is standardisation ensured between different laboratories' pulse duplicators? - a sinusoidal ventricular volume, or some similar easily reproducible waveform can, by contrast, be unambiguously defined

- if the pulse duplicator is pressure-driven, the flow waveform will largely be determined by the characteristics of the valve under test and *mutatis mutandi* for positive-displacement flow-driven pulse duplicators - how do we know in advance what both waveforms will look like?

- is this stipulation appropriate when developing conduits or valves for implantation in small children?

These, and other limitations raise questions about how rigidly this standard should be adhered to in the kind of development work which forms the basis of the research presented in this thesis. The standard is largely intended to specify quality assurance procedures for valves either in or aiming for commercial production, rather than specifying the kind of equipment

that must be used in early laboratory-based prototype development work where research methods are often defined by the availability of existing equipment or the price of options for its replacement. Having said this, it is obviously desirable, in such circumstances, to stick as closely as possible to the recommendations of the relevant sections. This, of course, does not preclude the presentation of additional information, such as the results of steady flow tests, or energy loss figures, neither of which is covered by this standard, but both of which can provide useful insights for development work.

From the point of view of the current work, the relevant parts are Section 2 (definitions) and Section 5.3 (hydrodynamic testing of heart valve substitutes). Where the current work departs from its specifications, this will be made explicit.

2.3.1 Performance criteria

One area which has been a long-running source of confusion to surgeons faced with an array of valvular prostheses to choose from, is the question of how performance should be specified. Obviously, the final test is the longevity and quality of life of the patient, but this does not help determine whether or not a new valve is likely to offer better performance than an established model. *In vitro* testing offers the possibility of some objective comparisons between valves in certain defined respects. The issue then becomes which of the available bases for comparison are of the most clinical significance. This question, again, is not as straightforward as might be imagined at first sight, as the following discussion of the literature on the subject will reveal.

Early researchers tended to use the work of Gorlin and Gorlin (1951), who apply theoretical considerations of steady flow through a circular orifice, with the volumetric flow rate replaced by the stroke volume divided by systolic ejection time. Since this is derived from the flow and pressure drop through an orifice, the formulation involves a "loss coefficient" in the Bernoulli equation:

$$v = c_v\sqrt{(2g\Delta h)} \qquad - 2.1$$

and an area factor to take account of the reduction in orifice diameter associated with the vena contracta:

$$Q = c_c A v \qquad - 2.2$$

When the two results are combined, two uses are apparent for the resulting formula. If it is written as:

$$A = Q/\{c_v c_c\sqrt{(2g)}\sqrt{(\Delta h)}\} \qquad - 2.3$$

typical values of the empirical coefficients may be used with measured flow and head loss data to arrive at an estimate of the real area of a stenosed natural valve. This is the use the authors intend. However, if the equation is re-written as:

$$A' = Q/\{\sqrt{(2g)}\sqrt{(\Delta h)}\} \qquad - 2.4$$

(where $A' = c_v c_c A$ is the effective hydraulic area of a restricted orifice - i.e. the diameter of an orifice which in the absence of loss or vena contracta (an ideal orifice) - has the same pressure-drop *versus* flowrate characteristics), then we can see that we have a basis for comparison of valve replacements. A criticism of this approach derives from the work of Yellin *et al* (1970), who stated that the inertial term in trans-mitral valvular flow is such as to affect the calculation of orifice area from pressure and flow measurements. Gentle (1978) disagrees, demonstrating that mean forward-flow pressure drop *versus* flowrate measurements in pulsatile flow give very similar results to those of steady flow tests.

This controversy is avoided by Viggers *et al* (1967), in a plea from the medical profession for a universally acceptable valve-ranking method, based around pressure-drop. They offer a candidate for such a criterion. The steady flow results of orifice flow are again used as a

starting point. This time, instead of averaging the forward flow and corresponding pressure drop, data are taken from the instant at which forward flow is at a maximum. Since at this point the time derivative of flow is zero, inertial effects do not appear and so Bernoulli's equation may be employed with some justification, to extract meaningful information from an unsteady flow. When the instantaneous pressure drop is plotted against the corresponding instantaneous flow rate on a log-log scale, the gradient is (not surprisingly) equal to 2. From this plot, a value of the parameter K can be made, in the relationship:

$$\Delta p = K.v^2/(2g) \qquad - \ 2.5$$

in which v is the mean velocity through the valve orifice and K represents a figure of merit - the higher its value, the worse the valve. Aware of the argument that some measure of forward flow pressure drop does not adequately characterise valve performance, retrograde flow is also examined. Whilst no comparable figure of merit is offered for the retrograde flow, enabling a range of sizes to be covered by one parameter, some very interesting conclusions concerning valve design are drawn, which are of particular relevance to the present work. These are discussed in Section 2.5.1.

Gentle (1977, 1977a and 1977b) recognises for the first time that there is actually a theoretical limit as to the forward flow pressure drop that may be attained with a heart valve prosthesis. This is that which would be obtained if the valve presented no occlusion whatsoever, i.e. behaved as an ideal orifice. The resulting pressure drop is produced as the emergent jet's kinetic energy is dissipated as viscous eddies. Haemodynamic performance can therefore be quantified as a hydraulic efficiency, expressing pressure drop expected for flow through an orifice of a given diameter separating two large chambers as a fraction of the measured forward flow pressure drop produced by a valve of equal orifice diameter, both non-dimensionalised by dividing by $\rho Q^2/d^4$, where ρ is the fluid density, Q is the volumetric flow rate for valve or orifice respectively, and d is the ideal or valve orifice diameter. The advantages of this approach are precisely that a theoretical design limit is identified and that

the hydraulic efficiency pertains to a valve type, across its range of sizes.

The main criticism of both Viggers' and Gentle's analyses is that comparison is made on the basis of valve orifice diameter. The surgeon, faced with a tissue annulus is more concerned with how to provide the minimum pressure drop with a valve that fits this hole. Based on orifice diameter, for example, the Starr-Edwards caged ball valve is shown to be very efficient, but its large sewing ring diameter scores heavily against it if comparison is made on this basis. This point is echoed by Horstkotte *et al* (1983), and addressed squarely by Gabay *et al* (1978), whose analysis provides a performance index by combining the effective orifice area ideas of Gorlin and Gorlin; the means of avoiding the fluid inertia argument for pulsatile flow adopted by Viggers; and the tissue annulus diameter criterion. Thus, the effective orifice area is derived from the Gorlin formula, but using pressure drop and flow rate as instantaneous values at the point of maximum forward flow. From this, either a discharge coefficient for comparison on the basis of orifice area can be derived by expressing the effective orifice area as a fraction of the real orifice area, or alternatively, a performance index for comparison on the basis of tissue annulus diameter can be calculated by expressing effective orifice diameter as a fraction of external mounting area.

The different physical construction of a conduit-mounted valve would render irrelevant the argument about sewing ring versus orifice diameter as a meaningful basis for comparison, but then such a valve could not be described as separating two large chambers - the emergent jet is not dissipated into a closed constant-pressure cavity but channeled into a tube. This removes the theoretical ideal for comparison in the first place. It also brings into question its applicability to the assessment of aortic and pulmonic valves.

Gentle (1983) raises the issue of a performance criterion for valved conduits. Since the fluid dynamical situation differs from that of a mitral valve, for which Gentle's hydraulic efficiency is derived, efficiencies of greater than 100% are obtained, demonstrating conclusively, as just argued, that the ideal orifice pressure drop does not represent a theoretical limit in conduit design. This consideration, together with the question of how to

present data when the conduit is used as an apico-aortic shunt originally motivated the enquiry into energy loss methods as an alternative criterion for conduit evaluation, resulting in the full discussion of Leefe and Gentle (1987) who conclude that energy loss is particularly applicable as a performance criterion for conduit-mounted valves in pulsatile tests.

The principal arguments in favour of energy loss methods can be summarised as follows.

- Since the heart has to re-pump any regurgitant flow, it has to do additional work. Thus retrograde flow is automatically included in the assessment of energy loss, making this a suitable candidate for overall assessment over the whole cardiac cycle

- Energy loss methods (when rigorously applied) take account of changes in kinetic energy, due for example to the change in velocity profile from inlet to outlet (Langhaar (1942)) or to changes in duct cross-section.

Rushmer (1976) contains the following extract on the subject of wasted energy. "Since the useful work of the heart is the only external evidence of energy dissipation, the large quantities of wasted energy are frequently overlooked, although they have considerable functional importance. The myocardium must release energy equal to both the "wasted energy" and the useful work. Enough oxygen must be delivered to the heart through coronary blood flow to meet this total energy expenditure." Not only does this provide a rationale for estimating the performance of a valvular prosthesis in terms of energy loss, but also highlights the necessity to ensure that the prosthesis does not adversely affect flow to the coronary arteries, a subject addressed by Sabbah *et al* (1977).

Skalak *et al* (1966) offer an early and very thorough application of an energy loss method. Although it is derived for the pulmonary circulation, for which it attempts to derive an "effective resistance" to flow, its method is equally applicable to any region with inlet and

outlet tracts, although, as usual, the choice of sections must be made with a clear understanding of what exactly is being measured and how it might be affected by the choice of measurement site. The approach is to use a time integral, over a cardiac cycle, related to the loss function, such that pulsatile and kinetic energy convective effects are included. The author concludes that the considerable data and computation required render unrealistic the use of his derived effective resistance. In the 1990s, however, one may well beg to differ. Whilst it is difficult to ascribe physical meaning to this parameter, it could conceivably form a useful basis for the characterisation of the "systemic" impedance in sophisticated pulse duplicator rigs in conjunction with a standardised, physiologically realistic, pressure- or flow-induced waveform, such as is now demanded by ISO 5840 (1989).

Olin (1971) uses several measures, including energy loss, which, although somewhat crudely tackling the flow work term does at least account for ejection kinetic energy transport. It is not altogether clear, but it would appear that the energy loss is quantified for the systolic phase only. It is expressed as a percentage of the "externally delivered" energy, although this term is not clearly defined.

Swanson and Clark (1976) argue for clear test procedures and attempt to lay guidelines for what is necessary and how it should be measured. Amongst their recommendations is that energy loss should be reported, pointing out that it occurs both in forward and retrograde flow, although they suggest that figures for each of these situations should be given. As is usually the case, the energy loss computation is far from satisfactory, only considering flow work and stating that $\langle Q\Delta p \rangle$ is the same as $\langle Q \rangle . \langle \Delta p \rangle$ since pressure and flow are in phase. This statement cannot be made of waveforms with differing shapes. Further questionable arguments of a similar nature are used in the derivation of a performance index in terms of the sum of valve energy loss normalised with respect to ideal systolic flow work plus backflow normalised with respect to systolic flow. The higher the index, the worse the valve. (The use of this index is illustrated in Swanson and Clark (1974)). They recommend the use of friction factor *versus* Reynolds number plots for steady flow test data, in an attempt to quantify the degree of turbulence engendered by the valve. Similar arguments, when related

to leakage flows are discussed fully in Chapter 3 and by Leefe *et al* (1986a).

Mohnhaupt *et al* (1976) present valve evaluation in terms of an energy loss, formulated as the average flow work over a cardiac cycle. However, they express the opinion that systolic loss is best regarded in terms of pressure drop, whilst diastolic loss is basically a volume loss (i.e. it reduces cardiac output). This view is shared by Walker *et al* (1980), who argue that the "energy loss [of a mitral valve] ... is a composite measure of both the transvalvular pressure loss during diastole and the reflux loss during systole.

Although many examples of muddled thinking over energy loss methods are to be found in the literature, one in particular may be used as an illustration. Taylor and Whammond (1977) make some good points. For example, they demonstrate that the ranking order of various valves can change significantly, *in vitro* and *in vivo*, as the assessment criterion is changed. (Incidentally, it is worth noting that this is in direct contradiction with the results of Walker *et al* (1980), who found that the same ranking order occurred for different assessment criteria.) They also argue for a power loss per unit flow criterion, recognising the need for some kind of normalisation of an energy loss figure in order to make meaningful comparisons. However, they state that, if flow measurement is not possible, one may use mean pressure drop on the grounds that this is mean (flow work/flowrate). Thus they are, in effect, suggesting that one could use a pressure drop measurement and call it energy loss! In fairness, they do at least consider changes in kinetic energy, because they recommend using the impact pressure (i.e. that measured facing into the flow). The root of the confusion seems to lie in the fact that pressure drop and energy loss are equivalent only if steady flow conditions prevail. In fact, this probably explains the emphasis on pressure drop as the performance criterion at a time when the main thrust of prosthesis design was on the improvement of forward flow efficiency, so that much reported work was on steady flow testing.

In contrast, there have been some very good papers on energy loss. One such, although not on the subject of valvular prostheses, is Clark (1979). The author considers energy loss

arising from pulsatile flow through stenosed natural valves. There is sensible emphasis on the choice of control volume boundary, and thorough discussion of all the terms in the formulation. Of particular significance to the present work is the conclusion that most of the loss occurs in the downstream aorta where post-valvular flow disturbances are dissipated. This underlines the point made by Leefe and Gentle (1987), that "energy losses" associated with a valve are not necessarily apparent in its immediate vicinity.

Walburn *et al* (1985) conclude from their LDA investigation of pulsatile flow in bioprosthetic and stenotic aortic valves, that the turbulent flow Reynolds stresses are inadequately calculated on the basis of uniaxial velocity measurement. This adds further weight to the argument that the commonly used formulation of energy loss using readily measurable parameters is not applicable to cases where flow is grossly disturbed at the control volume boundary (see Leefe and Gentle (1987)).

Gormsen *et al* (1984): attempt to provide a quantitative measure of valvular insufficiency, in terms of "real" and "virtual heart valve insufficiency" indices, which consider the retrograde flow as measured and as corrected for closure volume, respectively. That is to say, reverse flow is recognised as consisting of a component necessary to effect valve closure and a subsequent leakage volume. Whilst a theoretical means of extracting the real heart valve insufficiency index is offered, this depends on the separation of closure and leakage volumes and no suggestion is offered as to how this may be achieved in a clinical measurement. There also appears to be a tacit assumption that insufficiency is purely a result of leakage, since one may express the real heart valve insufficiency in terms of the actual leakage volume as a fraction of the net displaced volume for the valve with zero leakage. This would seem to overlook the fact that valvular stenosis may affect closure volume as well as leakage volume. The authors apparently recognise these limitations because in practice diagnosis proceeds on the basis of the virtual (i.e. measured) heart valve insufficiency index, which is simply the ratio of retrograde to ejected volume.

Wright (1977) makes the point that whilst one usually looks upon leakage or reflux volume

as representing a decrease in cardiac output flow, the actual implication is that to deliver the necessary blood flow, the ventricular systole must result in a higher forward flowrate to compensate. This, in turn results in higher forward flow pressure drop. Obviously, the forward flow energy loss will be similarly affected. This lends weight to the argument that pulsatile flow tests should use mean output flow rather than stroke volume as the independent variable. The ISO evidently disagree.

Heiliger (1986), in a comparative study of bileaflet and tilting disc prostheses, offers two measures of performance. His "performance index" is the ratio of mean to maximum flow area in the open phase. The "efficiency index" (of a mitral valve, it should be noted) is defined as the ratio of net forward flow volume to ventricular volume multiplied by the "performance index". It is not clear what benefits accrue through the use of these measures.

Most contemporary investigators appear to have arrived at the conclusion that there is no suitable single all-purpose criterion for valve performance and accordingly there is a tendency to present several different kinds of test data. Perhaps the best example of thorough and methodical testing, evaluation and presentation is offered by Reul *et al* (1986), who use mean systolic and diastolic pressure drop; energy loss as a fraction of ventricular work per cycle for systole, closure and leakage; and regurgitant volume as a fraction of stroke volume. These data are augmented by well annotated diagrams of flow features, all in a consistent style.

2.3.2 Steady flow testing

There is actually very little published work on conduit testing or development. In contrast, however, there is a vast wealth of test data on steady forward flow *in vitro* testing of prosthetic heart valves, some of which is of direct relevance to the present work. Rather than summarise the principal findings of all *in vitro* valve tests, it is more instructive to concentrate on these papers.

Both Gentle (1977 and 1977a) and Swanson and Clark (1974) find that the Starr-Edwards caged ball valve out-performs other mechanical prostheses when forward-flow pressure drop measurements are compared for different valve types of the same valvular orifice diameter. This result is usually deemed irrelevant from the point of view of the surgeon, replacing a diseased valve, who is interested in minimising the forward flow pressure drop for a given tissue annulus diameter. The Starr-Edwards valve actually ranks quite low when comparison is made on this basis. What this result does indicate, however, is that flow around a spherical occluder offers potential as an efficient valvular device from the point of view of its haemodynamics. If, as in the present study, one has the freedom to dispense altogether with a sewing ring at the site of the occluder seat, one is left with a potentially efficient valved conduit. This notion forms the crux of the prototype development work presented herein.

Taking this idea as a starting point, Gentle (1983) demonstrates that drastic improvements in forward flow pressure drop in valved conduits are possible, by comparing the performance in steady flow of three rough prototypes to that of a Hancock prosthesis, which, as was shown in Section 2.2, was at the time of writing probably the most widely used commercially available device of its kind. Further similar test work is presented by Gentle and Benjamin (1984), although the location of the upstream and downstream pressure tappings, both in tubing of conduit diameter calls into question the validity of the conclusion offered, namely that "pressure drops ... were low in comparison with valves in their normal anatomical site", since the basis of experimental comparison is the Gentle "hydraulic efficiency" which is based on measurements across an orifice between two large chambers, simulating the situation for a mitral valve. This point is examined in more detail in Section 2.3.1, above and Section 2.3.6, below. However, what is clearly shown is that the performance of conduit-mounted mechanical valves may be improved by re-design of the internal profile of the valve-bearing section. This point is borne out by Tindale *et al* (1981) in their critique of steady-flow test experimental apparatus. They find that pressure drop depends on the geometry of the aortic root and of the downstream aortic orifice, suggesting that there may be an optimum aortic area for a given valve type and size.

Of course, in conduit design, these variables are not physiologically imposed, but may be chosen.

2.3.3 Pulsatile flow

The literature shows that the pulse-duplicator was in use to study the natural valve well before its application to prosthetic valve development (Davila (1956), Bellhouse and Talbot (1968)). Pulsatile flow testing of prosthetic heart valves, not surprisingly, appears to have evolved at the same time as heart valve replacement operations were becoming widespread in the late 1960s, when new valve types were being introduced (Smeloff *et al* (1966), Kaster *et al* (1967)).

Many of the earlier pulse duplicators were pneumatically controlled, so that a ventricular pressure waveform was imposed and the flowrate followed from the characteristics of the mock circulatory system and the valves under test (Kaster *et al* (1967), Wieting (1969), Mohnhaupt *et al* (1976)). This is surprising not only because of the required complexity as compared to, say, a simple cam or crank-type positive-displacement drive, but also, and more significantly, for the reasons discussed below.

Pettifor and Mockros (1970) provide the following rationale for flow-driven system (although they actually use a pressure-driven pulse-duplicator). "The heart adapts to different valves by altering both pressure and flow histories, thereby *ensuring a constant mean flow rate* within wide limits of incompetency and stenosis...(The) dynamic response of a valve to an unsteady flow having constant, predetermined mean flow...was investigated." (My italics). The first sentence indicates that either a pressure- or flow-driven duplicator would suffice, neither offering perfect reproduction of cardiac behaviour, but suggests that the latter might be more appropriate. From a practical point of view, it is easier to effect precise control of ventricular volume, for example by using a position-controlled displacement pump, than ventricular pressure. The squeezing action can still be maintained, for example, by forcing an incompressible fluid in a controlled way into a chamber

containing a flexible bag which represents the ventricle. A good example of this approach is found in Sabbah and Stein (1982 and 1984). Swanson and Clark (1976) go as far as to infer that a positive displacement pump ought to be used, since the heart "behaves as a nearly constant volume source with respect to pressure variations in the load..." and because "its volume per stroke also remains nearly constant with changing heart rate". They also suggest that the ratio of systolic to diastolic time should be variable. A further reason for using a positive displacement mechanism for control of ventricular volume, which does not appear to have been widely recognised in the literature, is that the resulting transvalvular pressure gradient waveform is largely independent of the details of the systemic resistance provided a sensible mean systemic pressure is retained to ensure a realistic assessment of the (virtually constant) leakage flowrate during ventricular diastole. This is because during the ejection phase, higher or lower mean systemic pressure will simply result in a correspondingly elevated or depressed ventricular pressure, but for the given imposed flowrate, the pressure drop across the valve will be unaffected.

Early examples of the volume control approach do exist (Viggers *et al* (1967), Duff and Fox (1972), Martin *et al* (1978)), but it is only over the past decade that this approach has become the norm (Walker *et al* (1980), Sano (1982), Mitamura *et al* (1985), Leefe *et al* (1986)). Over the same period, increasing sophistication has been incorporated, such as variable stroke rate and systolic fraction; programmable waveform, servo-controlled drive systems (Mitamura *et al* (1985), Leefe *et al* (1986)); sophisticated mock circulatory systems for better modelling of systemic impedance (Arabia and Akutsu (1984)); compliant aortic arch (Farahifar *et al* (1983) and (1984)); and flexible valve mountings (Gentle (1978) and She *et al* (1989)).

This increasing sophistication is brought into question, rather mischievously, by Gentle (1978), who goes as far as to question the necessity for pulsatile flow testing in all cases. Specifically, identifying forward flow pressure drop as being the factor most in need of improvement for mitral valves, he describes a sophisticated pulse duplicator, including flexible mounting of the valve, then reports a series of tests from which the forward flow

efficiency is determined for Bjork-Shiley and Starr-Edwards valves at different stroke volumes and frequencies. The results show very little difference from those obtained under steady flow conditions. However, it is clear that pulse duplicators are necessary to study *in vitro* other aspects of valve behaviour such as closure time and regurgitant fraction.

There is, to the author's knowledge, no published data on the pulsatile flow testing of valved conduits. Indeed, one aim of the present work is to establish some baseline data with a commercially available device. Since the remainder of the thesis will be concerned with the subsequent development of improved designs based on a ball occluder device, it is worth noting the observations of Chandran *et al* (1984), who show that downstream turbulent stresses obtained with a caged-ball aortic valve prosthesis are lower than with the porcine xenograft. Further, its regurgitant fraction is comparable with that of tissue valves and lower than with a tilting disc valve. Again, this shows promising potential.

2.3.4 Regurgitant flow

Since the principal function of a non-return valve is to prevent retrograde flow in the presence of an adverse pressure gradient, it is perhaps surprising that there has been comparatively little published research into this aspect of prosthetic valve performance. Henze and Fortune (1974) study the effect of leakage gap in disc occluder prostheses on regurgitation and haemolysis. This is accomplished *in vitro* with a pulsatile rig using a small quantity of whole blood pumped to and fro. Not surprisingly, they discover that haemolysis is higher in a totally occluding valve ($\approx$ 0.2% cells per day when extrapolated to *in vivo* conditions) than in the device with an inherent leakage gap ($\approx$ 0.08% per day). Shear stress and mechanical crushing during closure are offered as reasons. Having said that, clearance devices can still produce dangerously large shear stresses in leakage flow. Tillmann *et al* (1984) measure wall shear stress in a pulsatile flow test rig for a number of aortic prostheses and find that the backflow through a Bjork-Shiley valve with a finite leakage gap produces significantly large shear stresses - 500 N/m^2 (5000 dyn/cm^2) max - and it is suggested that this correlates with thrombus formation.

Henze and Fortune's finding that the leakage flow rate is proportional to the sealed pressure differential for the Bjork-Shiley valve is consistent with the data published by Gentle (1982). This paper is concerned with the characterisation of leakage flow domain, on the grounds that turbulent leakage could assist in providing a washing action over the occluder, thus preventing thrombus formation. This work is scrutinised in detail by Leefe *et al* (1986) who conclude that there is not much to be gained from this approach, which also has the disadvantage of increasing the leakage flow rate. They also examine data published by Rief *et al* (1980) and offer an explanation for the form of their non-dimensionalised leakage pressure-flow results. The same approach is adopted by Haggag (1990), who concludes that shear stresses in the leakage gaps of various mechanical valves may be sufficient to cause haemolysis.

Sabbah and Stein (1984) are concerned with the total backflow (closure and leakage flow) of various prostheses and conduct their experiments in a pulse duplicator rig, using an electromagnetic flowmeter. They conclude that the porcine valve offered the lowest backflow. This is not surprising, in view of the feature of leaflet valves that closure starts in systole and is almost complete by the commencement of diastole, as reported by Bellhouse and Talbot (1968), and because occlusion is virtually complete under reverse pressure, which contrasts with the finite annular leakage gap with a Bjork-Shiley valve, for example. However, the authors conclude that "in the presence of normal haemodynamics, the amount of backflow with the three mechanical valves appeared to be well below the level of backflow considered to be clinically significant."

Gentle *et al* (1981) show that regurgitant flow due to closure time in the Starr-Edwards caged ball valve is reduced when used as a mitral prosthesis, since the valve body is effectively flexibly mounted, so that the valve tends to shut more by movement of the valve seat towards the occluder than by fluid forces working against ball inertia to push it towards the seat.

2.3.5 Fatigue testing

Again, this is not directly relevant for the present work, concerned, as it is, with haemodynamic efficiency, rather than with materials and construction. Rather than reviewing results of fatigue tests, it will therefore suffice to make two general comments on the subject. Swanson and Clark (1976) discuss fundamental problems with accelerated fatigue testing. The stroke volume has to be greatly reduced in order to provide realistic pressure loading, because of the dynamic inertia effects. However, nothing can be done to correct the change in phase relation between pressure and flow pulses. They conclude that it must suffice to match the pressure amplitude. Taylor (1986) points out the additional problem that "structural durability may not be equivalent to biological durability", by which one may assume he means that *in vivo* conditions may adversely affect valve life. Having said that, most endurance tests are not merely concerned with structural strength, but make attempts to reproduce the chemical environment into which implantation is anticipated.

2.3.6 Techniques

All hydrodynamic *in vitro* testing of valvular prosthetic devices requires a sensible working fluid (real blood, for a variety of practical reasons - its opaqueness and tendency to clot, for example - is rarely, if ever used); and the measurement of at least two pressures or one differential pressure and a means of assessing flowrate. Obviously, other measurements are desirable such as velocity distribution and shear stress in critical areas. Furthermore, when considering pulsatile testing, it should be recognised that the frequency response both of the transducer and of the measuring system is important. Some techniques or devices are simply not appropriate in these circumstances. There follows a brief discussion of some of the key issues in the specification of an *in vitro* test facility for heart valve prostheses.

Blood analogue solutions

Skalak *et al* (1981) review the whole field of blood flow and in particular the question of blood rheology. It would appear that non-Newtonian behaviour is less marked in large

diameter vessels than in arterioles and capillaries, in which it may scarcely even be regarded as a continuum (see also Trowbridge (1982, 1983 parts 1 and 2 and 1984)). However shear stress is a weak function of strain rate, varying, according to their Fig. 6, from about 5 cP at 20 s^{-1} to about 3 cP at 200 s^{-1}. This justifies the use, by most investigators of (Newtonian) aqueous solutions of glycerol or saline as a blood analogue solution with viscosities in the range 3 to 4.5 cP. Idelson *et al* (1985) offer justification for the use of a Newtonian model of blood rheology in a computational study. The same argument applies to physical modelling using an aqueous glycerol blood analogue solution. Chandran seems to be in some doubt as to whether or not glycerol solution should be employed in the testing of tissue valves, aware of the possibility that there may be some adverse chemical effects which would not arise if the more physiologically realistic saline were used. Chandran and Khalighi (1984) discount this theory, but in Chandran (1986) he appears to have changed his mind.

ISO 5840 (1989) goes some way to clearing up the controversy but, once again, stops tantalisingly short of a clear specification, stating that "The test fluid shall be isotonic saline, blood or a blood-equivalent fluid, the physical properties of which (e.g. specific gravity, viscosity at working temperature) are stated". Surely, it would not be unreasonable to specify a viscosity and density with a sensible tolerance.

Flow measurement

Some early papers avoid the issue of direct flow measurement, (presumably with *in vivo* measurement in mind), and attempt to devise means of deriving arterial or aortic flow from differential pressure measurements (McDonald (1954)). Of this variety, two reasonably sensible publications are Womersley (1955) and Fry (1959). The former borrows from the theory of alternating current in electrical conductors, making intelligent use of Fourier analysis. The latter imagines one-dimensional fluid velocity to be governed by a first order differential equation (thus incorporating inertia effects), with empirical constants and a pressure gradient forcing function. Neither offers much practical significance, but both are offered here for historical interest.

The techniques or devices more commonly used are:

- Hot wire or hot film anemometry

  The current required to maintain a constant temperature in a small film or wire element subjected to convective heat transfer provides a measure of the local fluid velocity or velocity gradient. The technique offers fast response, hence can be used to study turbulence (Seki *et al*). However the larger forces associated with liquids make hot wire techniques unrealistic (the technique is most commonly associated with aerodynamic and wind tunnel testing). The hot film anemometer, however finds more widespread use, since when flush-mounted, it may also be used as a measure of wall shear stress (Bellhouse and Bellhouse (1968), Figliola and Mueller (1981), Tillmann and Schlieper (1979), Tillmann *et al* (1984))

- Electromagnetic flowmeter

  If an electrolyte flows through a region in which there is a transverse magnetic field the positive ions experience a mutually perpendicular force, whilst the negative ions are forced in the other direction, i.e. there is charge separation, hence a transverse voltage is induced. Its magnitude is proportional to the velocity of flow. Modern electromagnetic flowmeters usually employ pulsed waveform excitation, resulting in a cleaner signal after demodulation. To operate effectively, the fluid must be conductive (e.g. blood or saline), and the velocity profile through the device reasonably symmetrical. For this latter reason, it is particularly important that the device is situated sufficiently far downstream from the test valve or other obstructions. Maxted (1982) gives a clear account of the theory of such devices, and also offers a wealth of practical information. Electromagnetic flowmeters are widely used for *in vitro* testing in conjunction with flow visualisation techniques, as an alternative to the (generally expensive) methods, such as laser Doppler anemometry, of providing a detailed quantitative picture of the velocity field. Examples of their use may be found in Wieting (1969), Swanson

and Clark (1976), Mohnhaupt *et al* (1976) and in Sabbah and Stein (1984) who use one to measure leakage flow in a pulsatile rig.

- Turbine flowmeter

  Despite its low cost this device is comparatively rare in the literature. Often constructed to give unipolar output, it is suitable for steady flow testing (Gentle and Benjamin (1984)) as a compact alternative to a rotameter. The same comments as above apply about mean flow measurement and downstream siting.

- Doppler ultrasonics

  Flow velocity in a target control volume of fluid is assessed by detection of the Doppler shift in ultrasound waves reflected from micro-particles in the fluid such as blood cells or small seeding particles. This technique is relatively recent and is gaining wide acceptance as a non-invasive technique for *in vivo* flow visualisation. However, it can equally be used for *in vitro* testing (Farahifar *et al* (1985), Woodcock (1985)).

- Laser Doppler Anemometry (LDA)

  This technique has been developed fairly recently, and relies on the interference fringes set up in the region of intersection of two coherent laser beams. Any particle crossing this control volume will be alternately brightly then dimly illuminated, so that detection of the light reflected from the particle will vary in intensity with a frequency directly proportional to its velocity in a direction perpendicular to the fringe pattern. The technique is particularly useful for the following reasons: the control volume formed by the intersection of the beams is very small, offering extremely good resolution; used of pulsed laser beams offers the opportunity instantaneously to observe fast-varying flows such as are found in turbulent flow (which can be extremely helpful in the evaluation of Reynolds stresses to assess whether haemolysis is likely); being non-invasive, the system

does not disturb the flow which is being investigated. This is essentially a tool for the investigation of local velocity, rather than bulk flowrate, although the cyclical nature of *in vitro* pulse duplicator flows do not preclude its estimation from data obtained from a traverse of a cross-section with data taken at each traverse point at the same point in the cycle. Examples of the use of LDA are to be found in Yoganathan *et al* (1979), Figliola and Mueller (1981), Chandran *et al* (1984 and 1984a), Seki *et al*, Walburn *et al* (1985 and 1985a), Tiederman *et al* (1986) and Wood and Yoganathan (1986).

- Magnetic resonance velocity mapping

  This recent technique (Underwod *et al* (1990)) is emerging as an alternative to ultrasound for *in vivo* measurement, but seems well-suited to assessing the functionality of conduits where transparency causes problems with visual techniques.

Pressure measurement

Shirer (1962) is concerned with *in vivo* blood pressure measurements and reviews the available techniques. However, some of the issues raised pertain equally to *in vitro* measurement, particularly the discussion of the effect of fluid-filled catheter length on the dynamic response of the measurement system. This theme is echoed by Duff (1970) and Duff and Fox (1972), who are concerned with the proper recording and interpretation of dynamic pressure measurement, taking into account Bernoulli and transient effects. Many investigators prefer the use of differential pressure transducers, in a bid to minimise errors (e.g. Swanson and Clark (1976)). However, this necessitates fluid-filled catheters and could compromise dynamic readings. The compromises and problems facing the would-be selector of a pressure transducer for *in vitro* valve testing are discussed by Wright and Brown (1977). In addition to those already mentioned, they add the differences in magnitude between systolic and diastolic gradients, resulting in the requirement for a large dynamic range with good sensitivity throughout, and the avoidance of the transducer's resonant frequency.

Nichols *et al* (1983) present a discussion of catheter tip pressure and velocity probes. Although intended for *in vivo* measurements, the principles of operation of the various devices and the rationale for *in situ* measurement rather than, for example fluid-filled catheter pressure measurement, are all clearly explained, making this a useful reference for information on *in vitro* techniques.

Siting of transducers

Mohnhaupt *et al* (1976) are aware of the practical implications of the location of some of their transducers (since the point is made that the electromagnetic flowmeter should be positioned in the upstream entry section where the flow is axisymmetric). However, their Fig.3 raises questions about the downstream pressure measurement site, since it would appear to be susceptible to Bernoulli effects and orifice losses. Swanson and Clark address this issue and suggest that the downstream pressure measurement should be four diameters distant from the valve and that pipe diameters should be the same upstream and downstream. They also suggest that curved aortic models make pressure readings difficult to interpret because of the secondary flow. In some circumstances, however, this is inevitable. It is hard to imagine, for example, how curvature can be avoided when testing a conduit as a shunt. Tindale *et al* (1981) find that the distance for full pressure recovery downstream of a valve is very variable, and depends on the nature of the flow disturbance. They stress the importance of establishing this distance before conducting comparative test series. Furthermore, although their work is on steady-flow testing, they argue that similar recovery distances will pertain in pulsatile flow, so that when pulse duplicator rigs are designed, transducer siting may be determined from previous steady flow test work.

2.4 In vivo performance

The question of which prosthesis to use for conduit operations is, and will remain, a vexed one, bound up, as it is, with commercial considerations, vested interests and lack of long-term comparative follow-up data in significant numbers of patients.

In the early days of valved conduit operations in the 1960s, aortic homografts were the norm (Ross and Somerville (1966), McGoon *et al* (1968), Rastelli *et al* (1969)). Amongst its advantages are its "natural" haemodynamics - the branches can be harvested with the trunk (Livi *et al* (1974)), and the availability of large sizes. Overall, however, availability is a major problem. Although Moore *et al* (1976), Norwood *et al* (1977) and Di Carlo *et al* (1982) suggest that the durability problems of irradiated deep-frozen homografts may be overcome by the use of antibiotic-sterilised fresh ones, (the latter stating that results are "excellent", but avoiding the issue of availability), by the 1970s, the use of the aortic homograft was in question. This process is illustrated nicely in the discussion following Moore *et al* (1976). Norwood *et al* (1977) report on a superior prognosis for right heart reconstruction with the Hancock conduit than with other biological alternatives. Similarly, Bowman *et al* (1973) note that in addition to procurement, sterilisation and storage difficulties, aortic homografts for use in the repair of congenital defects involving right ventricular-pulmonary artery discontinuity are prone to degenerative change such as wall calcification and stenosis. The authors argue for "the ready availability of a uniform, standardised prosthesis, available in multiple sizes, and which can be tailored at the proximal or distal end to fit the patient's anatomical needs". Since Hancock is one of the co-authors, it is not entirely surprising that the porcine valve in a Dacron graft is presented as the preferred alternative. It is also ironic that the charge of valve degeneration could equally be leveled at the porcine valve.

However, that shortcoming was yet to emerge and in the 1970s, the trend in valved conduits, as in other valvular prostheses, was towards the use of the porcine xenograft (Bernhard *et al* (1975), Cooley *et al* (1975), McGoon *et al* (1975), Cooley *et al* (1976), Dembitsky and

Weldon (1976), Norman *et al* (1976), McGoon (1976), Stewart (1976), Cooley (1977), Norwood *et al* (1977), Cooley and Norman (1977)). This was probably due to the widely held view that the haemodynamics of tissue valves are superior and, more importantly, that long-term anticoagulation therapy is necessary with mechanical valves (although, as argued elsewhere in this thesis, neither assumption is necessarily true). As a result of this trend, most of the follow-up data relates to tissue valves. Such papers written in the late 1970s and early 1980s should therefore be regarded with caution, as long-term durability had not, in many cases, been possible to assess adequately because of the length of the follow-up period. This comment is particularly pertinent in the light of emerging reports of calcification and tissue degeneration in biological valves (both in conduits and as conventional replacements), and of recent indications of a swing back towards mechanical valves for their durability. For example, Rupprath *et al* (1981) report the use of the mechanical Lillehei-Kaster valve in conduit tubing as well as a larger number of Hancock prostheses. Unfortunately, the publication is too soon after the operations reported to enable conclusions to be drawn about the relative behaviour of mechanical and porcine xenograft conduit-mounted valves.

An exception to the mid-seventies trend in the use of tissue valves is to be found in Cartmill *et al* (1974), who, as well as raising concern about the long-term durability of tissue valves, argue (perhaps questionably) that "the use of the Bjork-Shiley .... prosthesis, with its excellent ratio of effective flow orifice to external diameter, central flow characteristics [*sic*] and low inertia disc, appears to offer near perfect haemodynamic function."

In any case, the comparative performance of various valve types is particularly difficult to assess when the prosthesis is conduit-mounted for several reasons. Firstly, the number of implanted conduits is very low compared to the number of valve replacements, so that sample sizes tend to be small. Secondly, it is not always possible to determine whether information on conduit performance is a function of the valve or of the conduit tubing, since only explanted conduits can be examined. Thirdly, results tend to depend strongly on surgical considerations. This combination of factors is summarised in a follow-up study by Bisset *et*

al (1981), who discuss data on 33 surviving children treated for a variety of disorders with external porcine xenograft conduits between a right ventriculotomy and the pulmonary artery. Of the 20 patients catheterised six months to four-and-a-half years postoperatively, all but two showed an increase in peak systolic gradient over that measured intraoperatively, 30% had developed a pressure gradient at the proximal anastomosis and 20% at the distal anastomosis in addition to gradients of varying severity at the site of the valve itself. Where reoperation allowed examination of a replaced conduit, the valve cusps were shortened and thickened. Overall, 30% of the entire study group suffered some form of conduit dysfunction over the six-year follow-up period. The authors conclude that this failure rate is not attributable to any specific cause, but factors such as size selection, determined by distal pulmonary artery diameter and complicated by increasing stroke volume in growing children; angulation of the valve ring and its effect on the haemodynamics; and immunology are all likely to play a significant role. It is suggested that the first of these problems may be overcome by the use of over-sized conduits, where mediastinal capacity will allow. However, they conclude, there remains the problem of tissue-valve durability, which may in any case necessitate a replacement operation.

Due to a variety of reasons, many surgeons were moving back towards the use of the allograft by the late 1980s. The historical development of this type, its uses and advantages is fully charted by Stelzer and Elkins (1989) in a major review article. They show how steps have been taken to overcome the inherent shortcomings. In particular, advances in cryopreservation techniques has led to the establishment of a valve bank in the USA. A similar scheme is also underway in the UK (Almeida *et al* (1989)).

2.4.1 Conduit haemodynamics

There is very little published material on the haemodynamics of valved conduits, either *in vitro* or *in vivo*. However, some features may be deduced from a small number of follow-up studies. For example, Narducci *et al* (1984) report seven operations using bioprosthetic valved conduits from apex to ascending aorta without rigid inlet stents. Whilst the only

pressure information offered is intraoperative, follow-up data ranging from one to five years suggest satisfactory conduit operation. The shunt is claimed to accommodate 40% of the left ventricular outflow. Norman *et al* (1976), on the other hand offer clinical catheterisation and flow quantification data, measured ten to fourteen days postoperatively, on six patients, all of whom had the distal anastomosis of their apico-aortic shunts located in the descending abdominal aorta. Similar flow divisions were recorded, ranging from 29% to 43% (mean 37%) of ventricular outflow taken by the conduit. These figures suggest that the conduit represents more flow resistance than the aortic stenosis it is relieving, yet the reductions in ventricular-ascending aortic gradient are greatly improved, with Norman's post-operative gradients ranging from 0% to 29% (mean 13%) of their preoperative values. Shanebrook and Levine (1979) offer an analysis of these data by constructing a steady-flow mathematical model of the shunt and stenosed valve system. Whilst the results are in fairly close agreement, there is a tendency for predicted values to be higher than measured conduit flow. The possibility of partial occlusion of the conduit entrance in practice is suggested as an explanation. This could be backed up by the observation that the conduit flow was slightly higher in Narducci's patients, whose conduits employed rigid stents at the proximal anastomosis. Obviously there are many possible contributing factors which may explain this apparent anomaly, but flow considerations alone clearly indicate that resistance is greater through the conduit than the stenosed aortic valve. What is of great significance to the present work, however, is that it is theoretically demonstrated by Shanebrook and Levine that the simple replacement of the valve in the conduit with a different valve with a better hydraulic efficiency can reverse the situation so that the conduit accommodates up to 62% of the ventricular outflow. This is without adaptation of the conduit to improve its overall haemodynamics.

2.4.2 Surgical considerations

As mentioned in the introduction to Section 2.4, the issue of which conduit might give the best overall *in vivo* performance is clouded by surgical considerations. For example, Norwood *et al* (1977) report progressive conduit gradient becoming problematic in right heart

reconstruction patients. Late complications arose from sepsis at the anastomoses, distal obstruction and proximal gradient due to the fashioning of the ventricular orifice. These last two points are echoed by Castaneda and Norwood (1982), who comment on the observations of Bisset *et al* (1981) that gradients tend to develop at both proximal and distal anastomoses. These are shown not to be inherently conduit-related, but associated with the construction of the ventricular orifice and with occlusions of the pulmonary artery, respectively. They also note that external conduit compression may be averted by avoiding substernal positioning of the conduit, an observation made by several others, such as Bailey *et al* (1977), most of whose replacement operations were necessary for this reason, Heck *et al* (1978) and McGoon *et al* (1982), who also comment on the alleviation of obstruction at the anastomoses by using as large a diameter as possible at these sites.

An additional danger related to conduit positioning is highlighted in Harris (1981), who reports a post-operative mortality caused by compression of a coronary artery by a rigid portion of a conduit. Just as conduit positioning can impede the proper functioning of its surroundings, so the surroundings can affect the valved conduit. An illustration is offered by Kouchoukos *et al* (1980), who report a series of operations in which a Bjork-Shiley valve replaces the diseased aortic valve and the distally attached tubular graft is enclosed within the ascending aorta. In one case, the aorta was closed too tightly over the conduit, causing impaired valve functioning. There are also suggestions that the ventricular wall can partially occlude conduit entrance at the proximal anastomosis. Some surgeons opt for a semi-rigid stent to overcome this problem (see Section 2.5.3)

The issue of the suitability in very young infants of conduits for reconstructive surgery *versus* the temporary use of palliative procedures is examined in Ebert *et al* (1976) and the ensuing discussion. However, Norwood *et al* (1977) point out that "the use of valved conduits in infants must be considered palliative in the sense that grafts of limited size require replacement to accommodate increased cardiac output with growth". They recommend the use of a large conduit at age 4-5. In Danielson's ensuing discussion, an oversized conduit is recommended, obviating the need for replacement.

2.4.3 Durability

As pointed out above, most of the conduit follow-up data in the literature relates to the porcine valve variety and by the 1980s, the long-term degeneration associated with tissue valves was beginning to become apparent in conduit prostheses. Agarwaal *et al* (1981) in their report on replacement operations highlight problems found in explanted porcine valve conduits - cusp fusion and calcification. This point is further borne out by Hellberg *et al* (1981), who note that calcific failure of glutaraldehyde-preserved porcine xenograft valves tends to be more common in children (they suggest because of their higher calcium turnover). They conclude that "the use of bioprostheses in children and adolescents must ... be questioned since they appear to carry a high prospect of early valve deterioration". The "disquietingly high incidence of relatively early failure of porcine xenograft valves in children" is borne out by Geha *et al* (1979) (whose study includes conduit-mounted valve explants). Again, the only common denominator between this observation and bovine trials involving other types of tissue valve is the high rate of calcium turnover which "establishes a propensity to deposit calcium within the "foreign" valvular tissue". McGoon *et al* (1982) go as far as to question "...whether or not many patients with normal pulmonary vascular development and resistance might have a better long-term result if the conduit contained no valve at all...", although they do not consider the use of mechanical valves as a possible alternative.

The emergence of late tissue valve failure is well illustrated with reference to two papers from the Mayo Clinic. Ciaravella *et al* (1979) present a large long-term follow-up study in which only 3 out of 380 Hancock conduits (0.8%) had required reoperation. However, McGoon *et al* (1982) report that of 352 patients surviving conduit implantation operations, 16% required re-operation, most frequently because of the development of a pressure gradient across the conduit. Examination of the replaced Hancock conduits revealed failure due to valve calcification in 69% of cases, sometimes in conjunction with the development a peel separated from the conduit wall by a thick layer of thrombus. The remainder of failures

were due to this latter effect in isolation. This accords with Agarwaal *et al* (1981), in whose study 2/3 of patients requiring conduit replacement did so for valve-related reasons, whilst the conduit neointima alone was responsible for the remainder of failures. McGoon *et al* (1982) relate the development of this progressively thickening layer of thrombus to the use of non-porous Dacron conduit tubing, since this material inhibits a fibrous anchor for the neointima. They retain its use, however, "for reasons of hemostasis". The effect of graft porosity is also raised in Waldhausen's discussion of Kouchoukos *et al* (1980). Here, a fatality is attributed to the use of low porosity graft material. The entire pseudointima had dislodged, causing a massive stroke.

By the late 1980s, due to the accumulating evidence as typified by the previous citations, the pendulum was swinging against the use of the porcine xenograft. The alternative option favoured by many was the aortic or pulmonary allograft (Campbell and Clark (1990)). Here calcification is less problematic particularly insofar at it affects leaflets, the allografts tending to degrade by wall calcification and insufficiency instead. Stelzer and Elkins (1989) concur with this view, stating that calcification problems are not such as to impair function and that re-operation time is much greater than that to be expected with the porcine xenograft conduit in terms of freedom from complication after a given number of years. Furthermore, sizing problems can be addressed by the use of Dacron extensions if necessary - see also Almeida *et al* (1989).

It should be borne in mind that any assessment of conduit performance can only be as good as the available data will allow. This point is recognised and appropriate action has been taken in the UK in the field of prosthetic valve replacement with the inception of the Multicentre Valve Trial (Black *et al* (1983)). The management of this scheme is described and a discussion of the statistical techniques for evaluation of follow-up data is offered by Drury *et al* (1986). The authors favour actuarial analysis, incorporating patient prognosis information to isolate patient-related factors from valve assessment. It would certainly be a worthwhile exercise to encourage the use of this database over as wide a geography as possible. This may help to provide sufficiently large sample sizes to draw useful conclusions

about the performance of valved conduits from the relatively low number of patients benefiting from them.

Finally, in any discussion of the pros and cons of a particular valvular types or of the niceties of operative technique, it is worth bearing in mind the sentiments of Ciaravella *et al* (1979), that "despite the relatively primitive state of our knowledge....", extracardiac conduits offer help "... now for that large group of patients who otherwise would have no prospect of help".

2.5 Conduit design

Gentle (1983) provides the rationale for much of the work described in this thesis. The main contention is this: the surrounding anatomy places restrictions on haemodynamic improvements for prostheses used as direct replacements for malfunctioning natural valves. Nozzles and diffusers will not be accommodated and the orifice area will be reduced by a sewing ring. (The extent to which these problems present design difficulties is clearly illustrated in Sauvage *et al* (1970).) These limitations do not exist with a conduit. An integral valve, or a different means of connecting a valved section into the conduit, obviate the necessity for a sewing ring at the valve orifice plane, whilst there is ample scope for appropriate shaping of valve inlet and outlet geometries. This does not appear to have been recognised by the manufacturers, who continue to mount conventional valves in cylindrical tubes, accompanied, as one might expect, by unnecessarily high forward flow pressure drops. The paper demonstrates that drastic improvements in this respect are possible, by comparing the performance in steady flow of a prototype based on the trapped ball occluder principle to that of a Hancock prosthesis. The issue of a performance criterion is raised. Since the fluid dynamical situation differs from that of a mitral valve, for which Gentle's hydraulic efficiency is derived, efficiencies of greater than 100% are obtained, demonstrating conclusively, as argued in Section 2.3.1, above, that the ideal orifice pressure drop does not represent a theoretical limit in conduit design. This consideration, together with the question of how to present data when the conduit is used as an apico-aortic shunt originally motivated the enquiry into energy loss methods as an alternative criterion for conduit evaluation, resulting in the full discussion of Leefe and Gentle (1987). A potential problem with ball occluder oscillation is noted at higher flowrates (see also Mueller *et al* (1975), Tindale *et al* (1981) and Uglov *et al* (1984)). This was tackled theoretically as detailed in Tansley *et al* (1986) who showed this to be related to the downstream flow area giving rise to increased pressure, tending to push the occluder back towards the orifice. Using this work as a basis, Tansley (1988) completes the prototype development of the valve-bearing section based on steady-flow considerations. The main purpose of this work is to demonstrate that the promising results of steady flow tests may be realised in pulsatile

tests, and to make suggestions concerning constructional detail of a prototype for *in vivo* studies.

2.5.1 Haemodynamics

Behaviour of blood

It is well documented that low shear flow, such as is found in regions of stasis, results in thrombus formation. For example, Yoganathan and Harrison (1981) use an LDA system to study the post-valvular flow field and offer evidence of the correlation of thrombus formation with low shear and stagnation regions. They also measured high shear stresses at mechanical valve struts and noted the formation of thrombus here. This accords with Galanga and Lloyd (1981), who conclude that thrombus formation correlates with high surface mass transfer coefficients, which occur both at high shear rate locations and at separation sites, (where there is stasis). It is worth noting, at this juncture, that Castaneda and Norwood (1982) found that conduit internal peel originated at the point of maximum compression. This strongly suggests a correlation with flow separation in the conduit.

Mechanisms for both high and low shear rate thrombus formation are postulated in Wells *et al* (1970). At low shear rates, (below about 6 s^{-1}) characteristic of the venous system, red cells become entrapped in fibrin strands, whilst at shear rates in excess of 230 s^{-1}, characteristic of arterial flow, platelet aggregates form and clotting proceeds by cohesion between these aggregates. They state that "anticoagulation with heparin does not appear to inhibit this process." The thrombus, once formed, either remains or is swept away, according to surface shear rate, although unfortunately no threshold value is offered. McIntyre and Hubbell (1986) further elucidate the kinematics of clot formation and explain the observation that it is a worse problem at artificial surfaces. The local shear-rate dependency is explained in terms of micro-vortex formation, caused by the rotation of the larger red cells in the shear field, which increases the transport rate of platelets to the wall, and therefore the rate of aggregate formation there. This aggregation rate is greatly enhanced if the surface does not have the endothelial lining which characterises blood vessels. In view

of Wells' observations, the fact that anticoagulation therapy is effective in reducing thromboembolic episodes in patients with mechanical valves would seem to suggest that its action involves surface effects rather than blood bulk chemistry.

Haemolysis, too is dependent on blood shear rate. Niimi and Sugihara (1985) discuss this issue and present quantitative data relating bulk shear stress necessary to cause haemolysis to exposure time at this stress level. They postulate that in practice haemolysis is a fatigue phenomenon caused by cyclical stresses in red cell membranes. An 'S-N' curve for this mechanism is presented along with a relationship between membrane stress amplitude and local bulk fluid shear stress.

The higher incidence of both haemolysis and thrombus formation with high shear rate implies greater risk of these phenomena with turbulent flow. This is indeed the case.

Flow through prostheses

As far as the haemodynamic design of valvular prostheses is concerned, the implication of the preceding discussion is that regions of unduly high and low shear rate, separation, stagnation and high turbulence intensity should all be avoided. The other principal aim of the designer is to minimise pressure drop and associated losses. In both of these respects it is hard to envisage a better arrangement than that which nature has already designed. The natural valve has a large, non-occluding central orifice which presents almost no restriction to forward flow. In the aortic valve, the sinuses of Valsalva act to assist rapid valve closure by initiating the closure process towards the end of ventricular diastole as the flow decelerates, whilst the axial movement of the valve ring further assists. Completion of the closure process is cushioned by the action of an entrained vortex in the sinuses (Bellhouse and Talbot (1968, Bellhouse (1969)). The closed valve is self-sealing and is effectively a zero-leakage arrangement. The mitral valve operates in a similar manner, with the vortex flow structure encouraged in the ventricle in an analogous manner.

It is not surprising, therefore, that the preferred choice of many surgeons is for natural

valves. However, for reasons discussed elsewhere, these features do not always translate into similar performance when the valve is used as a prosthesis - or if they do, availability and durability may mitigate against their use. For example, when a natural valve degrades by calcification, not only is forward flow pressure drop severely worsened by the resulting reduction in orifice size as the valve fails to open properly, but the hitherto attractive central flow also becomes problematic in that it encourages the formation of a turbulent jet. Moreover, the failure to close efficiently and fully means a greater degree of regurgitation and insufficiency. Where the prosthesis is in the form of a xenograft as opposed to a homograft, the necessary allowance for a sewing ring can offset benefits arising in the original animal from the large central orifice.

This is not to suggest that mechanical valves do not suffer from the same problem - although one certainly need not provide space for a stent. Most mechanical varieties suffer their fluid mechanical disadvantages from the existence of rigid occluders which usually present some form of central obstruction, often associated with an asymmetrical post-valvular flow field with associated viscous dissipative losses (see Figs. 2.1 to 2.3). The cages, hinges and struts also present ample opportunity for sites of turbulence and haemostasis. Closure is often slower than in the natural valves, due to inertia of the occluder, and there is either leakage through a gap necessary to accommodate a tilting or pivoting mechanism, or mechanical damage to cells associated with the crushing action of sudden complete closure of an occluder against a seat.

In the design of valved conduits, however, there is more freedom to work around some of the disadvantages that mechanical types present on their own, and even to turn them to advantage. Most obviously are the possibilities for streamlining and for avoidance of a sewing ring.

The first of these was strikingly illustrated by Viggers (1967), who explicitly made the point emphasised in the introduction to Section 2.5, that the design of the valve profile could result in improved haemodynamic performance. For example, a caged ball design separating two large diameter sections creates a diverging flow around the occluder, resulting in early

separation from the ball and a long downstream region of separated flow. The same valve in a profiled section in which the section immediately downstream of the occluder converges reduces the degree of divergence, deters separation and results in a shorter downstream length of separated flow. (Detailed consideration to this kind of downstream profiling is given by Tansley *et al* (1986) and Tansley (1988)). It was not surprising that in 1967, the relevance of this kind of simple streamlining for conduit design was not spotted. What is surprising is that it apparently still hasn't been commercially exploited. Viggers went as far as to show that a simple orifice with a downstream enlargement (such as a sinus cavity with no valve leaflets) actually produced a worse "figure of merit" in terms of forward flow pressure drop than the same geometry with a caged ball occluder inserted, since this helped streamline the flow and reduce the abruptness of area changes. This paper also provides very useful data on recommended ball occluder travel. Figure of merit and retrograde flow are both plotted *versus* occluder travel (as a fraction of ball diameter). Naturally, the pressure drop increases sharply as travel decreases since the occluder is presenting far more of an obstruction. However, regurgitant flow decreases as travel is reduced, since closure time is reduced. It is shown that pressure drop and regurgitant flow are both acceptably low when occluder travel is limited to around half of its diameter.

A further advantage of the trapped ball occluder variety of valvular prosthesis is noted by Akutsu and Modi (1982), They show that the Starr-Edwards valve, when mounted in an orifice in a cylindrical tube such that sewing ring diameter is 60% of tube diameter, gives a comparatively low turbulence intensity - again because of the smooth, streamlined flow which results. This reduces the risk of dangerously high Reynolds stresses, which, as previously noted, are associated with thrombus formation and haemolysis. Of course, were the caged ball principle to be incorporated in a purpose-built conduit rather than placing an existing valve complete with sewing ring in a tube, one would not only obtain this advantage, but also not suffer the accompanying area reduction.

In valved conduit design there are also considerations relating to the sections upstream and downstream of the valve itself. This forms the main subject matter of two useful additions to

the literature from a Russian team. (There are publications, in Russian). The first of these - Roeva *et al* (1990) - is a literature review on the design of apico-aortic shunt valved conduits. Apart from applying well-known results for losses arising from separation and eddies associated with bends and abrupt enlargements, to the development of a critique of other people's proposed designs, they have striven to identify a more rational approach to fluid mechanical design philosophies, as the following quotes serve to illustrate:

"..a valve implanted a long way from the mouth of the conduit creates a damping chamber, the presence of which in an aortic-left ventricular shunt adversely affects the haemodynamic properties..."

"..when discussing the design of conduits, investigators were mainly concerned with the materials from which the conduit is made, the type of valve prosthesis present in it, and the diameter of the conduit, without attaching due importance to the design features of the flow region of the shunt, ensuring a stable and uninterrupted flow of blood along the conduit."

They argue that hydrodynamic criteria should be established for the choice of surgical tactics. Then ".. a hydrodynamically optimal set of prototype conduits of different sizes for the right and left sides of the heart must be defined and produced, allowing for the patient's age, the type of pathology, and the anatomical features of the heart", perhaps a rather ambitious target for an off-the-shelf item!

The second offering - Chekanov *et al* (1990) - reiterates the importance of the hydrodynamic design of the inlet section. It describes the canine trial of valved conduit developed as a second left ventricular outflow tract for the treatment of a variety of congenital and acquired defects. The stent is described as the "principal component of the whole design as it determines the blood flow pattern in the whole conduit". The rigid inlet consists of a stent running into a curved section made of reinforced silicone. The valve is borne in a distal "siliconised vascular prosthesis". It is either biological xenopericardial tricuspid or mechanical low-profile in nature. (The accompanying illustration looks rather

like a Bjork-Shiley design.) What is perhaps more novel is that the whole conduit is convergently tapered from inlet to outlet. This is consistent with Roeva *et al*'s view that: "Advantages of a convergent channel over cylindrical include reduction of zones of separation and lamination of the flow...". However, whilst much effort has obviously been given to the proximal conduit tubing, the valve-bearing section does not appear to be haemodynamically optimised, hence in this author's opinion, the result that the systolic pressure gradient from ventricle to aorta was less with the tissue valve than for the mechanical.

With shunt designs there is one source of hydrodynamic loss which seems inevitable. This arises from separation at the distal end-to-side anastomosis. It seems that the best one could hope to do would be to make an oblique attachment in an effort to encourage early merging of the flows.

The literature reveals some additional haemodynamic considerations pertinent to the design of composite grafts for the replacement of the aortic valve and the ascending aorta. Firstly, Bellhouse's (1969) study of the relationship between aortic stenosis and sinus pressure reveals a potential problem with the siting of the anastomosis of the coronary ostia with central flow valves such as the porcine xenograft or bovine pericardium prosthesis. Valve stenosis results in the formation of a turbulent jet which affects the pressure distribution in the sinuses of Valsalva. The location of the coronary ostia - sinus centre, where pressure is reduced relative to ventricular pressure; or sinus ridge, where pressure is comparatively unaffected - will determine coronary flow. Composite graft design could take advantage of this finding and help reduce the risk of angina during exercise both by avoiding, as far as is possible, jet formation, and providing a sensible location, downstream of the valve, for the anastomosis of the coronary arteries such that the blood pressure they see is as high as possible over as much of the cardiac cycle as possible. Incidentally, the loss referred to in the previous paragraph at the distal anastomosis of an apico-aortic shunt conduit may also result in a slightly reduced coronary flow for the same ventricular pressure, since aortic pressure will be somewhat lower. Alternatively, in order to deliver the same coronary flow, the ventricular pressure would need to be slightly higher. Either view results in the same

conclusion, namely that this state of affairs is detrimental.

Secondly, aortic curvature is shown by Kang and Tarbell (1983) to result in mean flow impedance 2 to 3 times higher under pulsatile conditions in a rigid tube model than it is for steady flow conditions delivering the same net flow rate. This impedance is strongly amplitude-dependent but relatively insensitive to frequency. Chang and Tarbell (1985) provide a possible basis for order-of-magnitude calculation of the velocity distribution in a curved aorta, hence of the kinetic energy term in a calculation of energy loss in a composite graft. (The same method could equally be applied to a conduit used as a shunt.) Thirdly, as pointed out by Farahifar *et al* (1985) "Compliance plays an important role in the damping of post-valvular disturbances...". Rieu *et al* (1985) confirm this assertion and find, from their study of pulsatile flow in an elastic model of the arterial tree, that most irregularities in downstream velocity profile seem to be damped out by the proximal end of the descending aorta. Thus as well as helping to maintain aortic pressure during ventricular diastole, aortic compliance helps to deliver a symmetric flow profile. The advantage of these effects could be realised if an elastic material were to be employed for the tubular graft.

2.5.2 Materials

Probably the greatest challenge facing bioengineers attempting to design successful cardiac prostheses is the development of materials able to withstand the aggressive chemical environment represented by human blood, that resist the formation of thrombus, which are extremely wear-resistant, and which encourage tissue ingrowth. Dowson (1980) amplifies this view. He sees the development of biocompatible materials and research into the effect of surface characteristics on performance as key areas.

Much has been made in the foregoing Sections of this chapter of the durability problems associated with tissue valves. This is not to say that mechanical valves are immune from these problems. Indeed, Anderson *et al* (1978) provide a catalogue of materials-related failures of mechanical valve components such as wear of silastic balls, chemical interaction

between silastic and blood leading to swelling and cracking, wear of plastic discs, and strut wear caused by differential hardness between occluder and strut materials. However, in view of the many recent advances in the field of ceramic and composite materials, it seems likely to this author that materials with appropriate combinations of properties such as toughness, abrasion resistance, hardness, density and surface finish, which could also support a tissue lining are already available and waiting to be tried. Certainly, more development is warranted where materials have already shown initial promise.

Ebert and Schaldach (1978) posit rutile (titanium dioxide) as a suitable ceramic material for implantation, offering the following reasons. Its semiconducting properties are such as to limit electron transfer to coagulation-specific proteins - i.e. it is haemocompatible. It is wear-resistant, non-toxic and "sufficiently strong" (although half the strength of alumina). It has better surface finish than that typically obtainable with other ceramics commonly used for implants, such as alumina or SnO_2 on an alumina substrate. (A figure of 0.1 mm for R_a is suggested as desirable, and compared with 0.6 mm for alumina.) A dense ceramic can be formed by sintering. These last two features may be rendered less relevant in the light of the findings of two other groups concerning tissue ingrowth.

MacGregor *et al* (1976), as well as providing a very useful and compact overview of materials-related issues and a history of the development of materials in valvular prostheses, also offer the results of canine trials in which occluder cages coated with sintered metal powder of varying porosity were suspended in the atrium for up to six months, whilst a control group had untreated cages similarly inserted. On post-mortem examination, there had been no trace of thrombus in any of the experimental group, whilst ten out of the control group exhibited such evidence. This was attributed to the tissue ingrowth encouraged in the treated cages, but not in the untreated ones.

The theme of the usefulness of surface porosity is taken up by Gentle (1980), who describes the development of an all-ceramic mitral valve, with reference to both haemodynamics and materials, pointing out the desirability, particularly to young sufferers, of a durable valve

which does not require the administration of anticoagulants for its effective functioning. The material chosen to fulfill this requirement is porous alumina, for its excellent wear resistance and for its ability to support a biological lining, potentially sidestepping the haemocompatibility problems usually encountered with mechanical valves. This latter point is discussed more fully in Gentle *et al* (1981) in which the alumina valve body is shown to support a uniform, thin, firmly-attached tissue covering. In this prototype, the occluder flaps were made of Delrin. Pig trials resulted in the formation of a fibrous deposit on the back of the flaps - it is unclear whether this was due to the flap material or the haemodynamics. The problem was addressed by replacing the Delrin flaps and stellite hinges with alumina, the hinge pins having a hard glaze (Gentle (1981b)). Unfortunately, there is no report of any *in vivo* trials with this valve. However, further work was done on the assessment of alumina as a suitable material. Gentle and Juden (1984) describe a wear test procedure and report an evaluation of the useful life of the Abrams-Lucas polypropylene prosthesis, which is in agreement with clinical data. Having thus verified the technique, they go on to estimate the wear rates of porous and dense alumina in different combinations, from which they predict a useful life of eleven years for a porous/dense alumina hinged flap valve, and of forty-five years for one in which the rubbing parts are both of dense alumina. It must be borne in mind that this projection is based on wear and not fatigue considerations. Ritchie and Lubock (1986) offer an authoritative fatigue study, using three different metallurgical criteria, of the Bjork-Shiley valve, concluding that a ninety year life may be expected provided all cracks over 0.5mm long are detected in the factory. This contrasts with Quijano (1980), whose study of explanted Hancock bioprostheses concludes that these valves demonstrate "conservation of intrinsic pre-implant characteristics for up to 100 months [about 8 years] *in vivo*". (It should be noted that "up to" is not a very helpful phrase in this context and also that in both cases authors were employed by the manufacturer concerned, although in fairness the Bjork-Shiley study was published after a series of catastrophic strut weld fatigue failures, well publicised by the Sunday newspapers of July 8 1984, obviously providing incentive for an "in-depth probe"!) The promising properties of alumina are summarised by Gentle (1986), who reviews the use of ceramics in heart valve prostheses. He concludes that it is cheap, hard, wear-resistant, has a reasonable fracture

toughness and, most importantly, will support, either on surface roughness or in porosity, a thin tissue overgrowth (details in Juden *et al* (1983)), rendering it non-thrombogenic. Other materials which may show promise are single crystal alumina, titanium nitride and titanium dioxide.

Tissue ingrowth is also a relevant consideration in the selection of materials for the conduit tubing. Jaroshinsky *et al* (1984) offer experimental evidence of tissue overgrowth and canine biocompatibility of Dacron in a porcine xenograft conduit. However, the degree of porosity is important, as explained by McGoon *et al* (1982), who relate the development of a peel separated from the conduit wall by a progressively thickening layer of thrombus to the use of non-porous Dacron conduit tubing, since this material inhibits a fibrous anchor for the neointima. They retain its use, however, "for reasons of hemostasis". Orszulak *et al* (1982) offer another reason for using low porosity Dacron, pointing out that it reduces bleeding across the graft sufficiently to render wrapping unnecessary in the aortic valve and ascending aorta replacement operation.

A more recent technique is the use of fibrin glue. Early work (e.g. Haverich *et al* (1981) and Borst (1981)) showed that this "mimics the essential steps of physiological fibrin formation", whilst avoiding the leakage sometimes associated with high porosity grafts. Furthermore, "no evidence of a higher thrombogenicity of the inner surface of fibrin glue-sealed grafts" was observed. Its use is now fairly widespread (Kouchoukos (1989)).

Thrombogenicity of the conduit material was also investigated by Deacon *et al* (1985), who compare the haemocompatibility of four Dacron cloth types and conclude that filamentous velour is more thrombogenic than plain-knitted or double-knitted velour and plain-knitted with pyrolytic carbon coating, which appeared to be the most suitable material, in terms of alteration of platelet function.

The possibility of a relationship between prosthetic materials and anastomotic hypoplasia is discussed by LoGerfo (1991). Whilst separated flow results from the end-to-side

anastomoses at both ends of a conduit - which is highly dependent on the angle of anastomosis and the flow split between runoff vessels - there is no demonstration of of a relationship of this phenomenon with anastomotic hypoplasia. Since this is more common at the distal end, it is suggested that this may be the result of some interaction between the blood and Dacron or another prosthetic surface aggravating the hypoplastic response at the downstream site.

2.5.3 Constructional detail

Since homograft conduits are of a given construction (i.e. they are as harvested), the ensuing discussion concerns prosthetic conduits. This comment is made with one exception, concerning a rather ingenious use of an excised aortic valve and ascending aorta to construct an aortic homograft conduit with a distal valve, reported by Hoots and Watson (1989) - see Fig. 2.4. An early source of disagreement over conduit construction concerned the issue of whether the conduit should be rigid or flexible. Rigidity offers the guarantee that valve functioning will not be impaired by conduit compression, but offers little room for manoeuvre with regard to positioning or trimming immediately prior to insertion. The opposite is true of a flexible design. In practice, a combination of rigidity and flexibility is often used. Bernhard *et al* (1975) chose a rigid design with flexible joints. Another ploy is to use a flexible conduit with a semi-rigid stent at the proximal (ventricular) anastomosis. Brown *et al* (1977) for example, state that a semi-rigid apical stent is of paramount importance to prevent collapse of an apico-aortic shunt during ventricular systole and conduit kinking. Other pioneers begged to differ, opting for flexible (Dacron cloth) tubing. Cooley *et al* (1975) justify this on the grounds that such a conduit is less likely to encroach on adjacent organs. McGoon (1976) and Narducci *et al* (1984) evidently share the opinion. Cooley *et al* (1975) think that the continuous suture of the fabric conduit end to the ventricular apex is adequate, but his team evidently changed their view on this point since Norman *et al* (1976) report the use of a rigid inlet stent, which Cooley *et al* (1976) describe further as a pyrolytic carbon stent with a Teflon felt sewing ring. Cooley (1977) also reports the use of a semi-rigid stent. It appears that this conduit is in three distinct sections: proximal and distal conduit tubing and an intermediate valve-bearing section. This

facilitates valve replacement should it become necessary.

It would seem to be a useful idea to develop this approach further, employing a rigid, profiled valve-bearing section, attached to the upstream and downstream lengths of conduit tubing, which, as with the Shiley conduit, could be of reinforced Dacron which offers flexural compliance but incompressible cross-section. The upstream, proximal tubing should be of as large a diameter as possible, to ensure plentiful blood supply and reduce the risk of inlet occlusion and, in children, to delay or prevent the necessity for reoperation.

2.5.4 C.F.D. as a design aid

In recent years, the advent of computational fluid mechanics as a realistic option has resulted in the application of the technique to heart valve design. A representative sample in chronological order, broadly speaking, reveals the developing complexity of analysis and is offered below, for the interested reader.

- Underwood and Mueller (1979): laminar, axisymmetric, Newtonian fluid model using finite difference techniques.

- Engelman *et al* (1980): finite element, steady-flow 2-D study of aortic and mitral valve prostheses with non-Newtonian fluid, formulated in terms of an absolute viscosity as a function of erythrocyte concentration, which varies with distance from the wall.

- Rooz *et al* (1982): 1-D time-dependent finite element study of pulsatile flow in a stenosed artery.

- Mazumdar and Thalassoudis (1983): finite difference, steady, 2-D, laminar flow of a Newtonian fluid.

- Uglov *et al* (1984): crude inviscid axisymmetric finite difference model.

- Thalassoudis and Mazumdar (1984): include turbulent effects.

- Stevenson and Yoganathan (1985): axisymmetric, steady, turbulent finite difference model with body-fitted coordinates.

- Leefe *et al* (1986): finite difference laminar/turbulent 2-D model of leakage flow.

- Tansley *et al* (1988): effectively and thoroughly argues the case for a major future role for CFD in the design of heart valve prostheses, by considering the relative strengths and weaknesses of both CFD and experimental techniques, the data available from CFD studies, and the clinical relevance of this information.

Tansley's (1988) PhD thesis offers a detailed account of the incorporation of an appropriate non-Newtonian blood rheology model into the solution algorithm of a commercial finite difference CFD package and illustrates its use in the design development of the heart valve conduit analysed in this work. It also illustrates the usefulness of CFD as an aid to prototype design, by offering accompanying experimental data and showing how information (such as shear stress and turbulence intensity distributions) not readily available from *in vitro* tests without sophisticated instrumentation, may be extracted for the important forward flow and leakage flow parts of the cycle. The possibilities of future time-dependent, non-Newtonian, turbulent models with distensible boundaries is also touched upon. The relevance of CFD, it is argued, can surely only increase as sophisticated software and hardware become more widespread.

3 Prototype development

As explained in Section 1.2, there were originally two generic valve types contending for inclusion in a conduit for prototype development. These were the ball valve and the twin-flap valve. Both offered promising forward flow characteristics. However, considerations of leakage and consequent manufacturing tolerances mitigated against the twin-flap valve. These considerations are laid out in Section 3.1.

However, there had been some indications of a potential problem with oscillatory instability of the occluder during forward flow through the ball valve, observed during steady flow testing. It was therefore necessary to explore this issue and cure the problem before proceeding with the development of the ball valve concept. The approach to this problem is outlined in Section 3.2

3.1 Leakage considerations

Since both the caged ball and the twin-flap valve offered desirably low forward flow pressure drop, leakage flow is scrutinised in an effort to decide which of these two alternatives to develop. The caged ball design involves a silastic sphere seating on an inlet orifice, over which it forms a seal under reverse pressure conditions. It is a zero clearance, zero leakage device. Consequently attention is focussed on the specification of leakage gap for the twin-flap design.

The choice of leakage gap dimensions in pivoting or tilting occluder mechanical prostheses is essentially a compromise between on the one hand an attempt to prevent clot formation by introducing a washing action over portions of the valve which would otherwise encounter flow stasis when the valve is shut, and on the other hand the prevention of excessive regurgitation and accompanying high shear rates. These conflicting aims are nicely illustrated by Rief *et al* (1980), who were aiming to minimise leakage and by Gentle (1982), who was attempting to characterise the flow regime, postulating that turbulent flow would produce a more effective washing action. Both studies presented experimental data on sealed pressure differential versus leakage rate in the form of a logarithmic plot of friction factor *versus* Reynolds number, with the intention of drawing conclusions concerning the nature of the flow by comparison with the standard curves for fully developed pipe flow. Interestingly enough, however, both data sets represent quite significant departure from the predicted curves. All of the valves studied produced curves with lower gradients than predicted whilst Gentle's twin-flap valve demonstrated a measured friction factor an order of magnitude greater than what might be expected for the turbulent flow claimed to be characteristic of the leakage. The offered explanation of surface roughness, however, does not stand up to closer scrutiny, as becomes apparent when one superimposes the observed data on the Moody chart (Fig. 3.1), where the Reynolds number for the valvular leakage flow is based on mean hydraulic diameter. The ensuing analysis, developed as an attempt to explain the form of the curve for the twin-flap valve, demonstrates that leakage flows of this

nature, in which the leakage path length is on the same order of magnitude as the gap width, can in no way be regarded as fully-developed.

3.1.1 Analysis

The accepted equations describing fully-developed flow in smooth pipes and ducts are:

$$f = 24/Re \qquad \text{and} \qquad f = 0.079/Re^{0.25} \qquad - 3.1$$

for laminar and turbulent flow, respectively, where f is the friction factor defined by:

$$f = \tau_w/(1/2 . \rho u_m^2) \qquad - 3.2$$

and the Reynolds number:

$$Re = 2. u_m . \delta/ \nu \qquad - 3.3$$

is based on mean hydraulic diameter, which for a slit is twice the width, δ. Here, τ_w is the wall shear stress, ρ is the fluid's density, ν its kinematic viscosity and u_m is the mean velocity of flow. In each case the equation arises by considering a fully-developed velocity profile, parabolic for laminar flow and 1/7 power law for turbulent. The problem with using these to characterise leakage through a closed prosthestic heart valve is that the axial path length is so short that the system can only be described as entry flow, rather than as fully-developed flow. The significance of this statement is twofold. Firstly, the velocity profile will be changing throughout the length and secondly it will be flatter, for a given flowrate, giving rise to a greater velocity gradient at the wall and hence to a larger friction factor. A numerical example of the latter effect is presented below.

Let us assume, for simplicity, that the shape of the velocity profile remains constant through the gap, but that it differs from both the fully-developed laminar and turbulent profiles. In

general, it may be approximated by a power law, defined to the gap centreline as:

$$u/u_o = \{y/(\delta/2)\}^{1/n} \qquad - 3.4$$

where u is the velocity at distance y from the wall and u_o is the centreline velocity. The value of n defines the shape of the profile: the larger its value, the flatter the profile. (As n $\rightarrow \infty$ the profile approximates to uniform velocity, plug flow.) Let us further assume that the following relationship holds, derived from turbulent boundary layer theory:

$$u/u^* = K_1 \,.\, \{u^*.y/\nu\}^{1/n} \qquad - 3.5$$

where u^* is the "friction velocity", $\sqrt{(\tau_w/\rho)}$ and K_1 is approximated as a constant over the relevant range of Reynolds number (although it is, in fact, a weak function of Re). Following the scheme of Duncan *et al* (1970), with appropriate modifications for parallel plate theory, one may proceed as follows.

Equation 3.5, evaluated at the centreline yields:

$$u_o/u^* = K_1 \,.\, \{u^*.\delta/2.\nu\}^{1/n} \qquad - 3.6$$

whilst the definition of mean velocity, together with the assumed velocity profile of equation 3.4, give:

$$u_m/u_o = n/(n+1) \qquad - 3.7$$

Equations 3.6 and 3.7 combine to give:

$$u^* = \{2.\nu/\delta\} \,.\, \{(n+1)/(n.K_1)\} \,.\, u_m^{\,n} \qquad - 3.8$$

Substituting the definitions of friction factor and Reynolds number (equations 3.2 and 3.3, respectively), yields the result:

$$f = K \,.\, \{Re\}^{-2/(n+1)} \qquad - 3.9$$

where

$$K = 2^{(n+5)/(n+1)} \,.\, \{(n+1)/(n.K_1)\}^{2n/(n+1)} \qquad - 3.10$$

Thus, the gradient of the curve of log(f) *versus* log(Re) for a flow with this velocity profile, is -2/(n+1), so that a fuller profile (larger n) results in a shallower gradient.

Regressional analysis of Gentle's data yields:

$$f = 0.2998 \,.\, Re^{-0.1131} \qquad - 3.11$$

so that for the twin-flap valve, equations 3.9 and 3.10 give, respectively:

$$n = 16.7 \qquad \text{and} \qquad K_1 = 3.15$$

Combining equations 3.4 and 3.7 gives the velocity profile as:

$$u/u_m = (1+1/n) \,.\, \{y/(\delta/2)\}^{1/n} \qquad - 3.12$$

Fig. 3.2 shows the velocity profile with n = 16.7, as just derived, along with that for n=7 (fully developed turbulent), for the same mean velocity. It is apparent that the profile is flatter and has a steeper wall velocity gradient, hence elevated friction factor. Furthermore, the fact that the flow is not fully developed explains the shallow gradient of Gentle's friction factor *versus* Reynolds number curve.

Another contribution to the high quoted values of friction factor arises from the fact that the twin-flap valve under reverse flow conditions constitutes a converging duct, in which there is a considerable loss of static pressure. The effect can be appreciated by a simplistic order-of-magnitude calculation. If one assumes that the duct is as depicted in Fig. 3.3 with a lossless inlet section with an upstream area, A_o at which the pressure is p_o, leading down to a constant width leakage gap of flow area, A_1, around the walls of which friction acts, and that flow then discharges to atmosphere, then one may calculate the actual pressure, p_1 at the inlet to the leakage gap itself, by applying the Bernoulli equation over the converging inlet section:

$$p_1 = p_o - \rho Q^2/2 \; . \; \{1/A_1^2 - 1/A_o^2\} \qquad - 3.13$$

With $A_o^2 >> A_1^2$ and $A_1 = b.2\delta$ (where b is the leakage slit breadth - here the valve orifice diameter and the factor of 2 is arbitrarily introduced to take account of the fact that the gap width is only equal to δ at the narrowest point, so that over a representative length, the width is nearer 2δ) and a typical leakage flowrate, Q, of 5×10^{-5} m^3s^{-1} (3 litre/min) of blood analogue solution for a physiologically realistic sealed pressure of around 12.5 kPa (94 mmHg), one obtains:

$$p_o - p_1 \approx 5.6 \text{ kPa} \qquad - 3.14$$

for b = 15 mm and δ = 0.5 mm, as per Gentle's data. Since the friction factor used in the non-dimensional plots was derived from a leakage gap wall shear stress calculated from the equation:

$$\tau_w = (\delta/2) \; . \; p_o/s \qquad - 3.15$$

where s is the gap length in the flow direction, instead of the better approximation:

$$\tau_w = (\delta/2) \cdot p_1 / s \qquad - 3.16$$

it can be seen that the wall shear stress, hence friction factor (from equation 3.2) is over-estimated in Gentle's analysis by a factor of about 45%.

3.1.2 Numerical model

In order to test the hypothesis suggested, above, that the leakage flow through Gentle's prototype twin-flap valve had been turbulent, but not fully-developed, a numerical model of the system was set up using the "PHOENICS" finite difference computational fluid dynamics software package. Since quantitative information was only likely to be of significance in the immediate vicinity of the leakage gap itself, a 2-dimensional model was employed. Further simplification was possible by virtue of the symmetry of the problem enabling the analysis of a half plane. An inlet velocity consistent with a leakage flowrate of 2.5 litres/min (corresponding in Gentle's experimental data to a conservative sealed pressure differential of about 9 kPa or 68 mmHg) was specified at the inlet plane, well upstream of the valve. The results, near the valve, are summarised on the streamline plot of Fig. 3.4. Flow over most of the flap surface is laminar, except over the region near its base, forming the leakage gap. The downstream flow, where the jet emerges, is turbulent, whilst the highly elliptic nature of the flow field has produced considerable upstream turbulence. Two stagnation points are apparent on the occluder surface: one on the downstream face, the other at the flow separation point at the flap base. The maximum shear stress in the gap is calculated as 2.2 $kN.mm^{-2}$ (compared with 2.0 kPa, predicted by equation 3.16, with s = 1.13, as suggested by Gentle).

3.1.3 Interpretation of results

The numerical and analytical studies presented above, both suggest that flow through the leakage gap is turbulent but not fully-developed. It is also instructive to consider whether this turbulence has the beneficial effect intended. As stated in Section 3.1.2, above, turbulence occurred over the surface of the occluder, only at the flap base. Consideration of

Fig. 3.5 shows that this is precisely the area which is best washed in forward flow anyway. Indeed, Gentle *et al* (1981) report that the hinges have been deliberately placed so as to ensure adequate washing action between the flaps when open. However, the two locations at which there is stasis when the valve is shut are precisely the same points most likely to result in stagnation during forward flow, namely the base, where the dividing streamline impinges, and the flap tip where separation will occur.

The correlation between the predicted shear stress from the numerical model and that from equation 3.16, justifies the use of this equation in design calculations. Thus, for a sealed pressure differential, of 15 kPa (114 mmHg), one might expect a peak wall shear stress of about 3.7 kPa. Blackshear (1972) suggests that short-duration shear stress over about 4 kPa will cause haemolysis, probably by cell membrane rupture, whilst there is a second mechanism requiring a shear stress level of about 0.1 kPa sustained for several seconds. Opinion is divided as to whether the resulting cell damage is caused by mechanical tearing of long thin processes drawn out when cells anchor to the wall, or by fluid shear stress in the bulk flow. The former mechanism is thought to be the principal mode of haemolysis at prosthetic surfaces and is extremely dependent on the nature of the surface. Even assuming that this mechanism is inhibited by tissue ingrowth into the porous alumina and that duration times are sufficiently short to render the fluid bulk shear mode irrelevant, the proximity of the predicted shear stress to the 4 kPa upper threshold should give sufficient concern to question the suitability of a valve with this leakage gap width for clinical use.

Finally, to produce turbulent leakage, the gap must be wide, producing large backflow. For example, Gentle's quoted backflow for a sealed pressure differential of 15 kPa is 3.4 litres/min ($5.7 \times 10^{-5}\ m^3.s^{-1}$). Assuming a typical mean forward flow rate of 25 litres/min ($4.2 \times 10^{-4}\ m^3.s^{-1}$), over 35% of the cardiac cycle, one would obtain, for negligible backflow, a mean delivery rate of 8.75 litres/min ($1.5 \times 10^{-4}\ m^3.s^{-1}$). In order to sustain this delivery rate with the quoted leakage rate, even assuming no reflux on closure, would require the mean forward forward flow rate to be 40 litres/min (6.7×10^{-4}

$m^3.s^{-1}$), with correspondingly increased forward flow pressure drop. The regurgitant fraction would be 25% of cardiac output (16% of ejected volume). The use of over-sized valves for conduit operations in paediatric surgery would only make the situation worse, because of the smaller ejected volume. This is quite clearly unacceptable.

3.1.4 Required leakage gap width

As just demonstrated in Section 3.1.3, a large gap width can have serious consequences. Since the purpose of the leakage gap is to ensure a washing action, it makes sense to derive an upper limit on leakage flow, since to aim for a very low leakage is to preclude the scouring action which is being sought under conditions of retrograde pressure gradient. The foregoing discussion shows that when shear stress considerations are marginal, the regurgitant fraction is excessive. This suggests the use of an upper limit on gap width based on an acceptable regurgitant fraction. Unfortunately, since the known physiological parameter is the pressure difference across the closed valve, calculation of leakage rate presupposes a knowledge of the relationship between the pressure drop and the flowrate. As noted initially and as is apparent from the results displayed in Fig. 3.1, correlation with the fully-developed flow relationships, whilst very poor for turbulent flow, is adequate for design purposes when the flow is laminar. Thus, for slit flow:

$$\delta = \{12.\mu.s.Q/b.\Delta p\}^{1/3} \qquad - \ 3.17$$

where μ is the dynamic viscosity of blood. Here Q represents the maximum permissible leakage flowrate for a typical sealed differential pressure, Δp. If we limit regurgitant fraction to 1% of cardiac output (again neglecting reflux volume on closure), the permissible leakage rate over 65% of a cardiac cycle whose mean delivery rate is 25 litres/min, is 0.4 litres/min ($6.7 \times 10^{-6}\ m^3.s^{-1}$). With b = 15 mm, μ = 3.5 cP, s = 1 mm, and Δp = 15 kPa (114 mmHg), one obtains:

$\delta = 0.1$ mm - 3.18

Such a small gap would be difficult to engineer in the ceramic materials. It would also be unreasonable to expect a valve initially having this dimension to maintain it over a (deliberately long) lifetime, both because of hinge wear and biological overgrowth. For this reason, and to avoid the use of hinges, which could possibly jam or break, it would seem more prudent to concentrate design effort on the caged ball principle.

3.2 Overcoming occluder instability

Development work published by Benjamin (1986), investigating the effect on forward flow pressure drop of the included angle of the valve's conical inlet section, concluded that 40^{o} resulted in the best performance, in conjunction with a 30^{o} included angle in the conical outlet section. However, there was also a problem with occluder instability at higher flowrates, taking the form of a high frequency axial oscillation of the ball. A similar phenomenon was noted by Viggers *et al* (1977), and Uglov *et al* (1984), who reported occluder instability in caged ball valves in the aortic position. This phenomenon was investigated jointly by Tansley, Edwards, Leefe and Gentle (1986). The contention was that, despite Benjamin's efforts to maintain a constant area as "seen by" the flow, the area immediately downstream of the occluder in the fully open position was in fact sufficiently large for the resulting Bernoulli effect to give rise to a large enough pressure over the distal face to result in a net retrograde force, tending to push the occluder back towards its seat. Here, the impact of the incoming fluid stream forced it back towards the fully open position, and so forth.

This hypothesis was tested by investigating the pressure behind the ball and simultaneously monitoring the level of noise and vibration in the valve-bearing section. This was done as illustrated in Fig. 3.6, for a flow rate of 15 litres.min^{-1} and a mean pressure at the valve section of 100 mmHg. The results are shown in Fig. 3.7. This may be explained as follows. The ball moves some distance upstream as a result of the Bernoulli effect described above. The downstream flow area therefore increases and the pressure behind the ball therefore rises. This results in a greater reverse pressure accelerating the ball further towards the seat. As the ball approaches the seat the flowrate drops (since the valve is closing) so that the Bernoulli effect reduces in strength and static forces start to predominate. (If the valve were to shut fully there would be no flow and the ball would see the upstream pressure whilst the downstream would be isolated, allowing the pressure here to decay.) Thus a forward opening force starts to predominate as the ball gets near to the seat. However, the

rearward momentum carries the ball right back until it collides with the seat. Thus, the noise peaks slightly behind the pressure, as is apparent from the traces.

The solution to this vibration problem was to maintain a constant flow area around the downstream side of the ball. Since "flow area" means that part of a given cross-section "seen by" the flow, or alternatively, the area of that surface which is everywhere normal to the velocity, but whose outer edges define the cross-section, it should be apparent that to keep this quantity constant would require *a priori* knowledge of the velocity field produced downstream of the valve occluder. Since this is not possible, calculation must proceed on conservatively-assumed velocity directions, with the flow area defined as normal to the ball surface (hence conical in form), and the velocity taken as everywhere normal to the flow area. Referring to the nomenclature of Fig. 3.8, (in which the restraining struts are omitted), it can be seen that flow area is given by:

$$A = \int_{Rb}^{Ro} 2.\pi.r.\sin\theta.\, dr \qquad - 3.19$$

In this way, the desired profile of the outlet section, down to its intersection with the cylindrical outflow pipe, was defined by the locus of R_o:

$$R_o = \sqrt{\{A/(\pi.\sin\theta) + R_b^2\}} \qquad - 3.20$$

where A is kept equal to the conduit tubing cross-sectional area. An estimate of the error of this "constant flow area" assumption may be made by taking a revised assessment of flow direction as being everywhere parallel to the bisector of the angle between the tangents to the ball and the outlet section's inner surface (i.e. somewhere between tangential to the ball and tangential to the inner surface of the occluder housing). This shows that the assumed flow area is no worse than 1.2% in error.

A prototype conduit with an outlet section as defined by equation 3.20 was manufactured and tested as part of Tansley's thesis (1988). Whilst the occluder instability was not observed in this prototype, the steady forward flow pressure drop, whilst still an improvement on the commercially available devices tested by Benjamin, (when compared on a non-dimensionalised basis, to eliminate the problem of size difference), was not as impressive as Benjamin's prototype with the conical outlet section of 30^{o} included angle. This was thought to be a result of the abrupt angle formed by the intersection of the curved outlet section with the downstream cylindrical conduit tubing. This problem was investigated both numerically and experimentally by Tansley, as reported in detail in his PhD thesis (1988), to which the reader is referred.

3.3 Summary

- The choice of valve type - twin-flap or caged ball - for conduit development revolved around leakage considerations.

- The ball occluder seats fully, so that virtually zero leakage may be expected. Investigation therefore concentrated on the twin-flap design.

- A large leakage gap was provided in Gentle's early prototype to effect a washing action under leakage flow conditions.

- Leakage flow with the large gap was found to be turbulent but not fully-developed.

- Turbulent flow near surfaces was only found at sites best washed during forward flow anyway.

- Stagnant flow regions during forward flow were found to be stagnant under leakage conditions also.

- The large leakage gap produced shear stresses approaching a level deemed to be critical in terms of haemolysis.

- The large leakage gap produced an unacceptably high regurgitant flow.

- An acceptable regurgitant flow could only be produced by providing a narrow leakage gap, of dimensions difficult to engineer to tolerance in the ceramic material, and difficult to maintain over the (deliberately long) valve life.

- The above findings dictated the choice of the caged ball device.

- Early prototype testing had indicated a problem with occluder instability during forward flow conditions.

- This instability was caused by the large flow area immediately downstream of the occluder in its open position, resulting in a net back pressure due to Bernoulli effects.

- The instability was cured by re-profiling the outlet section to maintain constant flow area.

- The prototype design was refined by Tansley to minimise forward flow pressure drop under steady flow conditions.

4 Evaluation criteria - the energy loss method

4.1 Rationale

When valved conduits were first introduced, the only assessment of their performance which was important was whether or not they were an improvement on the diseased or malformed valves which they replaced or bypassed. However, as different designs became available on the market, a quantitative system of assessment became necessary. The same can be said of valvular prostheses in general, and the relatively small number of conduit operations as opposed to valve replacements, led to a wider variety in their designs and evaluation methods. For example, the simple quoting of a pressure drop across the valve in forward flow was replaced by more sophisticated systems of assessment such as the "effective hydraulic diameter" (widely used and deriving from the work of Gorlin and Gorlin (1951)), the "figure of merit" of Viggers *et al* (1967), the "hydraulic efficiency" of Gentle (1977), or the "performance index" of Gabay *et al* (1978). These are all fully discussed in Section 2.3. Again, the small proportion of conduit operations is reflected in a dearth of literature on the *in vitro* testing of valved conduits. But such work as is available reports the use of steady flow pressure drop and hydraulic efficiency.

Unfortunately, the pressure drop method cannot deal effectively with the regurgitant flow when the valve is closing or with leakage flow when the valve is fully shut. For this reason, many engineers have moved towards the energy loss method as being a more rigorous measure of the overall impedance to flow, where "flow" implies net delivery rate, since a significant contribution to the energy loss can arise through having to work harder to re-pump regurgitant flow. Thus, not only is there a "flow work" contribution to energy loss for both the forward flow and regurgitant flow parts of the cardiac cycle, but the ventricular

ejection volume or heart rate, i.e. forward flow ejection rate, has to be greater to compensate for the backflow. This, too, results in a greater pressure drop and forward flowrate, hence energy loss.

The relative *pros* and *cons* of both pressure drop and energy loss methods, apparent from the literature, are also discussed fully in Section 2.3. However, since there is an almost complete absence of published material on pulsatile testing of valved conduits, there is virtually no discussion of the suitability of their evaluation criteria. An attempt to remedy this situation was made by Leefe and Gentle (1987). Two points mitigate in favour of measuring energy loss for a valved conduit.

1. When the conduit is used (as in the majority of cases) as a second ventricular outflow tract, the bulk of the pressure drop during forward flow originates from the proximal and distal anastomoses as well as at the valve itself. These losses, in turn, depend on the flowrate through the conduit which depends not just on the delivery rate, but also on the ratio of forward flowrate through the conduit to that through the bypassed stenosed valve, and on level of regurgitation through the stenosis. The original, stenosed outflow tract and the conduit must be viewed as part of a system. For this reason, there is no immediately obvious "ideal" pressure drop against which to compare that measured in practice, which is one of the principal attractions of the pressure drop method, since for a valve alone it enables the derivation of a single "hydraulic efficiency" covering a range of sizes and delivery rates (Gentle (1977)). Similarly, the conduit on its own does not lend itself to comparison against an "ideal" pressure drop. It is not inserted as a replacement for an orifice, in that it does not separate two chambers. For this reason, pressure drops less than that obtained

through an open orifice are obtained in practice, resulting in "hydraulic efficiencies" greater than 100%.

2. Whilst pressure drop from ventricle to major artery can and should be measured as well, (since this parameter is clinically important), the main point which renders the use of an energy loss method practically essential, is that when the conduit is employed as a second ventricular outflow tract, the level of regurgitant flow through the bypassed stenosis can be very significant, much more so than for a prosthetic valve used as a valvular replacement. This point may be illustrated by a simple example.

Fig. 4.1 shows highly idealised graphs of the net flowrate (i.e. the sum of the ventricular outflow through the stenosis and the conduit), and of the pressure drop from the ventricle to the artery, downstream of the point of confluence. During systole, occupying a fraction β of the cardiac cycle time, T, the net flowrate is Q_s and the forward flow pressure drop is Δp_s. During diastole, occupying the remaining $(1-\beta)$ of the cycle, the net flowrate is $-Q_d$ and the pressure drop is $-\Delta p_d$. To a first approximation, as will be clarified in Section 4.2, the energy loss over ventricular systole and diastole are, respectively:

$$\Phi_s = \beta.T.Q_s.\Delta p_s \qquad - 4.1$$

and

$$\Phi_d = (1-\beta).T.Q_d.\Delta p_d \qquad - 4.2$$

If the energy loss over the whole cycle is given as:

$$\Phi = \Phi_s + \Phi_d \qquad - 4.3$$

then the fraction of the overall energy loss arising from diastole (i.e. from regurgitation through the stenosis) is given by:

$$\Phi_d/\Phi = 1/\{1 + (Q_s/Q_d) . (\Delta p_s/\Delta p_d) . \beta/(1- \beta) \} \qquad - 4.4$$

Typical values can be inserted into this equation. For example, if systole occupies 35% of the cardiac cycle, the forward flow pressure drop is 15 mmHg and the transvalvular pressure gradient during diastole is 85 mmHg, then equation 4.4 becomes:

$$\Phi_d/\Phi = 1/\{1 + 0.095(Q_s/Q_d) \} \qquad - 4.5$$

Thus, if the regurgitant flowrate is as little as 10% of the systolic flowrate, equation 4.5 shows that the diastolic energy loss is 51% of the net loss. Such a significant contribution must be included in any performance evaluation.

As mentioned above, the pressure drop from ventricle to artery is of clinical importance, and so ought to be included in any discussion of a conduit's performance. This is, of course, also a stipulation of ISO 5840 (1989) and so is included in the current evaluation programme.

4.2 Derivation of the formulations

4.2.1 Overview

The analysis starts with the consideration of a convenient form of the energy equation for a Lagrangian fluid element (i.e. a group of fluid molecules moving with the flow). This includes a term representing energy which can no longer be made available as mechanical work. This is the "dissipation function", (which is what is usually meant when referring to "energy loss" in a fluid system) and is an expression for the irreversible dissipation of heat by fluid friction. The equation is then presented in integral form for a system of fluid molecules moving with the flow. Finally, a transformation is applied to convert the the equation to an integral form over a general, deformable control volume through which the fluid flows.

There are two main reasons for this approach. Firstly, by working from a general to a specific formulation, one is forced to note the simplifying assumptions made *en route*, and preferably to estimate the associated error. Secondly, the consideration of a control volume with distensible boundaries on and by which work may be done, raises the possibility of a thorough analysis of flow in the heart, through the valve, and into the elastic aorta. Whilst this is not attempted here, the method is laid out and may be of use in future research.

4.2.2 Development

Let x, y, z be a set of Cartesian coordinates and consider a Lagrangian fluid element of volume δV and density ρ moving relative to the Cartesian axes with velocity $\mathbf{q}$, whose components are u, v and w. The energy equation for this element may be written as:

$$\rho.\delta V.\ D/Dt\ (e + p/\rho) = \delta V.(\ Dp/Dt + DQ/Dt + \phi'\ ) \qquad - 4.6$$

(see, for example, White (1974), p.76), where e is the specific internal energy, p is the pressure and Q is the heat transferred to the element per unit volume. The quantity ϕ' is the dissipation function, and represents the rate at which the fluid element dissipates energy as irrecoverable heat through fluid friction. In this sense it can be taken as energy loss per unit volume (since this energy is no longer available to do mechanical work) and as such forms the subject matter of this chapter. A full derivation and discussion of its meaning may be found in White (1974) pp.72-76 and Raudkivi and Callander (1975) pp.71-76. When equation 4.6 is rearranged as:

$$\rho.\delta V\ De/Dt = \delta V.(\ p/\rho.D\rho/Dt + DQ/Dt + \phi'\ ) \qquad - 4.7$$

its physical meaning can be explained as follows. The rate of increase of internal energy equals the rate of storage of elastic strain energy plus the rate at which heat is added across the boundary plus the rate of viscous dissipation within the element. The elastic strain is recoverable. Of the heat transferred, the second law of thermodynamics shows that some of this may be irrecoverable. Thus the total rate of conversion of energy to irrecoverable form is the irreversible part of the heat transfer plus the rate of dissipation of fluid friction heat. This point is made for two reasons. Firstly, to emphasise that energy is not really "lost" at all, but converted to other forms, and secondly to point out that, if by "energy loss" we really mean energy converted to irrecoverable forms, then to be rigorous, the irreversible part of the heat transfer across the system boundary should be included.

Invoking the first law of thermodynamics for the fluid element, equation 4.6 may be rewritten as:

$$\phi'.\delta V = - D/Dt(gh + q^2/2).\rho.\delta V - (p/\rho\, D\rho/Dt).\delta V - DW/Dt.\delta V \qquad - 4.8$$

where h is the elevation of the element above some horizontal datum plane. The term $-DW/Dt.\delta V$ is the rate of work done on the element and can be written as:

$$-DW/Dt = \text{div}\begin{bmatrix} (u.\sigma_{xx} + v.\tau_{xy} + w.\tau_{xz}) \\ (u.\tau_{yx} + v.\sigma_{yy} + w.\tau_{yz}) \\ (u.\tau_{zx} + v.\tau_{zy} + w.\sigma_{zz}) \end{bmatrix} \qquad - 4.9$$

where σ_{jj} is the normal stress on the element in direction j and τ_{ij} is the shear stress in direction j on the element face normal to direction i. Also, the density derivative term, $(p/\rho).D\rho/Dt$ may be rewritten as $-p.\text{div}\ \mathbf{q}$, so that equation 4.8 becomes:

$$\phi'.\delta V = -D/Dt(gh+q^2/2).\rho.\delta V + p.\text{div}\ \mathbf{q}.\delta V + \text{div}\begin{bmatrix} (u.\sigma_{xx} + v.\tau_{xy} + w.\tau_{xz}) \\ (u.\tau_{yx} + v.\sigma_{yy} + w.\tau_{yz}) \\ (u.\tau_{zx} + v.\tau_{zy} + w.\sigma_{zz}) \end{bmatrix}.\delta V \qquad - 4.10$$

Equation 4.10 may be integrated over space for a system of particles moving with the fluid. The third integral on the right hand side may then be transformed using Gauss' theorem, to yield:

$$\iiint_{SYS}\phi'.dV = - D/Dt\iiint_{SYS}(gh + q^2/2)\rho.dV + \iiint_{SYS}p.\text{div}\ \mathbf{q}\, dV$$

$$+ \iint_{SYS}\begin{bmatrix} (u.\sigma_{xx} + v.\tau_{xy} + w.\tau_{xz}) \\ (u.\tau_{yx} + v.\sigma_{yy} + w.\tau_{yz}) \\ (u.\tau_{zx} + v.\tau_{zy} + w.\sigma_{zz}) \end{bmatrix}.dA \qquad - 4.11$$

The last term is equivalent to:

$$\iint_{SYS}(\sigma_n + \tau).\mathbf{q}dA$$

where σ_n is the outward normal direct stress and τ is the in-plane shear stress acting over the surface element dA of the system boundary.

It is necessary to develop the energy equation in this form because, in order for the first law of thermodynamics to apply, there must be no mass transfer across the boundary of the system under consideration, i.e. one must consider a group of molecules moving with the fluid. However, in studying the flow of fluid through a region in space, a control volume formulation is of more use. This can be derived by defining the "system" as occupying the same region of space as the "control volume" at a given instant (so that integral limits over the "system" may be replaced by those over the "control volume") and applying a transformation to relate the time derivative of the "system" integral to that of the "control volume", namely:

$$D/Dt \iiint_{SYS} f\rho.dV \int d/dt \iiint_{C.V.} f\rho.dV + \iint_{C.V.} f\rho\mathbf{q}_r.d\mathbf{A} \qquad - 4.12$$

where f is any specific fluid property (i.e. per unit mass) and $\mathbf{q}_r$ is the velocity of the fluid relative to that of the surface element, d**A**, of the control volume boundary where the fluid crosses it. A full discussion of this transformations may be found in Hansen (1965). Thus treated, equation 4.11 becomes:

$$\iiint_{C.V.}\phi'.\delta V = -\frac{d}{dt}\iiint_{C.V.}(gh + q^2/2)\rho.dV - \iint_{C.V.}(gh + q^2/2)\rho\mathbf{q}_r.d\mathbf{A}$$
$$+ \iiint_{C.V.}p.\text{div}\,\mathbf{q}\,dV + \iint_{C.V.}(\sigma_n + \tau).\mathbf{q}d\mathbf{A} \quad - 4.13$$

This equation is completely general. It describes the rate at which the mechanical energy in an arbitrary time-varying deformable control volume is degraded into irrecoverable heat. Only in the absence of heat transfer across the boundaries of this control volume, or if heat transfer is reversible can this be taken as the "energy loss" rate.

4.2.3 Assumptions

Equation 4.13 is hardly a convenient form from which to evaluate losses from measured data. It is therefore necessary to make some simplifying assumptions. These are enumerated below, and justification offered as appropriate.

1. **Negligible heat transfer between control volume and surroundings**
   Laboratory testing of valved conduits at room temperature involves no heat transfer processes. In view of the concluding sentence of the previous sub-section, equation 4.13 may be taken to represent the instantaneous rate of "energy loss" (in the sense already defined) within the control volume.

2. **Blood analogue fluid is incompressible**
   This means that ρ is a constant and div $\mathbf{q} = 0$, so that the third integral on the right hand side of equation 4.13 vanishes.

The boundary of the control volume may be sub-divided into the inlet and outlet sections across which there is fluid flow, and walls, across which there is not, hence for which

$\mathbf{q}_r = 0$. It may also be noted that the surface stress integrals over the walls represent the rate of work done, W'_{walls} , on the fluid by the solid boundary. If the integral on the left hand side of equation 4.13 is denoted by Φ', the rate of energy dissipation inside the control volume, the energy equation may be written as:

$$\Phi' = - d/dt\iiint_{C.V.}(gh + q^2/2)\rho.dV - \iint_{in/out}(gh + q^2/2)\rho\mathbf{q}_r.d\mathbf{A} + \iint_{in/out}(\sigma_n + \tau).\mathbf{q}dA + W'_{walls} \quad - 4.14$$

3. Work done by fluctuations over inlet and outlet sections in turbulent flow is negligible

It is first necessary to determine whether turbulent flow is likely. The Reynolds number for a typical forward flow of 20 litre/min of a blood analogue of viscosity 3.5×10^{-3} Pa.s flowing through a conduit of diameter 22 mm is 5519, i.e. characteristic of turbulent flow. An estimate of the Reynolds stresses may be made by considering steady, one-dimensional pipe flow with a fully-developed turbulent velocity profile. The Reynolds stresses are terms such as:

$$(\tau_R)_{xy} = - \rho < u'.v' > = \mu_t \partial u/\partial y \quad - 4.15$$

where primed quantities represent fluctuating components and u here represents the quasi-steady velocity. Time average is represented by the parentheses < >, thus, and μ_t is the apparent turbulent viscosity. (See, for example, Markatos (1986)). Prandtl's mixing length model enables this last term to be approximated by:

$$\mu_t = \rho l^2 . \left| \partial u / \partial y \right| \qquad - 4.16$$

where l is the characteristic length scale of turbulence. This may be estimated, following Von Karman's reasoning, by:

$$l = \kappa . \left| (\partial u / \partial y) / (\partial^2 u / \partial y^2) \right| \qquad - 4.17$$

where κ is Von Karman's constant, which may be taken as 0.4 (Markatos (1986)). The derivatives may be evaluated for fully-developed turbulent pipe flow in which:

$$u / u_o \approx (y/r_o)^{1/7} \qquad - 4.18$$

where u_o is the centreline velocity, r_o is the pipe radius and y is the distance from the wall. Substitution of equations 4.16, 4.17 and 4.18 into equation 4.15 yields:

$$\left| < u'v' > \right| \approx (\kappa^2/36) . u_o^2 . (y/r_o)^{2/7} \qquad - 4.19$$

For the purposes of order-of-magnitude calculation, and assuming isotropic turbulence, $\left| < u'v' > \right|$ may be approximated by $<u'>^2$. Thus:

$$< u' > / u_o \approx (\kappa/6) . (y/r_o)^{1/7} \qquad - 4.20$$

Dividing equation 4.20 by equation 4.18 yields the final result:

$$< u' >/u \approx \kappa/6 \approx 0.067 \qquad - 4.21$$

That is, the fluctuating components of velocity are around 6% of the quasi-steady flow velocity. This means that the contribution to the work by terms such as the product of the quasi-steady pressure and the normal velocity fluctuation will be on the order of 6% of that provided by the mean velocity and mean pressure. The contribution from the product of Reynolds stress terms and quasi-steady velocity may also be estimated, in comparison to that from the quasi-steady pressure and velocity, by comparing the Reynolds stresses to the pressure gradient. This ratio may be approximated by $\rho.< u' >^2/ \Delta p$, which, by the preceding result is approximately $0.067^2.\rho.u^2/ \Delta p$. For the typical conditions described at the start of this discussion, the mean velocity is 0.88 $m.s^{-1}$. Experience with prototype testing shows that the corresponding pressure gradient may be expected to be around 10 to 15 mmHg, that is around 1500 Pa. This gives a ratio of Reynolds stress to pressure gradient of 0.002, i.e. 0.2%, which is clearly negligible.

4. Blood analogue solution is Newtonian

This means that the direct stress (ignoring any Reynolds stresses, in line with assumptions 2 and 3, above), can be written as:

$$\sigma_n = - p + 2.\mu \, \partial q/\partial n \qquad - 4.22$$

where μ is the dynamic viscosity and the derivative is taken along the normal direction.

5. Convective accelerations are not excessive

If $\partial q/\partial n << p/2\mu$, then the normal stress can be replaced by the pressure. A typical path length for acceleration through a valve may be 5 mm, pressure will be on the order of 10^4 Pa, whilst the viscosity will be around 4 Pa.s. Approximating the derivative by $\delta q/s$ (the change in velocity over the path length, s), the requisite inequality becomes:

$$\delta q << p.s/2\mu \qquad - 4.23$$

or, using the numbers suggested above, $\delta q << 6250\ m.s^{-1}$, which will obviously be satisfied in all conceivable cases. Thus σ_n may be replaced by -p. Furthermore, since $\sigma_n\, dA$ is identical with $\sigma_n\, \mathbf{dA}$, we can replace $\sigma_n.\mathbf{q}dA$ with $\sigma_n\mathbf{q}.\mathbf{dA}$ and hence with $-\mathbf{pq}.\mathbf{dA}$.

Incorporating the above assumptions into equation 4.14 and integrating with respect to time over one cardiac cycle:

$$\Phi = -\left[\iiint_{C.V.}(gh + q^2/2)\rho.dV\right]_0^T - \int_0^T\left\{\iint_{in/out}(gh + q^2/2)\rho\mathbf{q}_r.\mathbf{dA} + \iint_{in/out}\mathbf{pq}.\mathbf{dA} - \iint_{in/out}\boldsymbol{\tau}.\mathbf{q}dA\right\} dt + W_{walls} \qquad - 4.24$$

where Φ is the total energy dissipated per cycle inside the region of interest, W_{walls} is the work done per cycle by the walls, and T is the cycle time.

6. **The pulse duplicator gives steady cyclic conditions**

The quantity of kinetic and potential energy inside the control volume is therefore the same at the beginning of the cycle as at the end, so that the first integral on the right hand side of equation 4.24 vanishes.

7. **Inlet and outlet sections are fixed in space**

This assumption permits the replacement of $\mathbf{q}_r$ by $\mathbf{q}$ in the second integral on the right hand side of equation 4.24 and therefore its combination with the following term.

Writing p* for the piezometric pressure, the energy equation now becomes:

$$\Phi = -\int_0^T \left\{ \iint_{in/out} (p^* + \rho q^2/2)\mathbf{q}.d\mathbf{A} - \iint_{in/out} \tau.\mathbf{q}\,dA \right\} dt + W_{walls} \qquad - 4.25$$

8. **The walls are rigid or perfectly elastic**

Since conduits are being tested in a pulse duplicator, in which conduits are retained within firm plastic tubing (see Chapter 5), and in the case of the shunt configuration the second outflow tract is also of firm tubing, this assumption is justified. As a consequence, the term W_{walls} vanishes.

In order to simplify the equation further into a usable form, careful attention must be paid to the choice of control volume boundary such that the following assumptions are satisfied to a reasonable approximation.

9. **All energy loss associated with the valve takes place within the control volume**

If, for example, a biased velocity distribution is produced downstream of the valve, as in the Bjork-Shiley valve, or if the valve sets up large-scale eddies, clearly there will be some viscous dissipation further downstream as the velocity distribution rearranges itself to the fully-developed profile. Ideally, therefore, the control volume should be chosen such that its boundary does not intersect significant flow disturbances caused by the valve itself. Similarly, if upstream inlet sections are such that disturbances are convected into the control volume, losses may be inferred which are not the consequence of the valve. In practice, of course, if the upstream and downstream lengths are too great, losses will also be inferred due to viscous dissipation arising from wall friction. As will be demonstrated under assumption 12, below, provided the velocity profile is reasonably symmetrical, mean velocity does not vary and velocity does not anywhere greatly exceed the mean value, changes in velocity distribution need not cause great concern. However, in many conceivable experimental circumstances, these conditions may not be met, and so this point should be borne in mind.

10. **The control volume boundary is chosen such that flow across it is everywhere normal**

In view of assumption 7, this may not in practice be possible over the entire cycle, since flow directions may change throughout the cycle. It does suggest the choice of a control volume inlet section outside the ventricle, for example just inside the conduit entry section. The outlet section is not such a problem, since it will be in a length of tubing. It does, however, caution against the use of the point of confluence for the double outflow case. A short length downstream would seem more appropriate.

Incidentally, this assumption was implied in the order-of-magnitude calculation undertaken in justification for assumption 3.

The effect of assumption 10 on the energy equation is to eliminate the vector dot product of in-plane shear stress and velocity over inlet and outlet sections. Since velocity has been assumed everywhere normal to **dA** over inlet and outlet sections, the vector notation may be dropped. Since **dA** defines the direction of the outward facing normal, **q.dA** may be replaced by -qdA over inlet sections and by +qdA over outlet sections, provided velocity is considered positive when directed into the inlet sections and out of the outlet sections. Application of assumptions 8, and 10 thus results in the following modification to equation 4.25:

$$\Phi = \int_0^T \left\{ \iint_{in} (p^* + \rho q^2/2)\, \mathbf{q}.d\mathbf{A} - \iint_{out} (p^* + \rho q^2/2)\, \mathbf{q}.d\mathbf{A} \right\} dt \qquad - 4.26$$

11. Piezometric pressure may be regarded as constant over an inlet or outlet section

The worst case would be for a horizontally mounted conduit, where piezometric pressure would vary by plus or minus ρgd/2 from the mean value from top to bottom of the section (where d is the conduit diameter). This represents a variation of about 1% of typical pressures, but if pressure measurement is taken on the vertical mean of the (circular) section, it can be shown to have no effect at all on the flow work term, provided the velocity distribution is axisymmetric. This assumption results in:

$$\Phi = \int_0^T \left\{ \Sigma_{in} p^*.Q - \Sigma_{out} p^*.Q \right\} dt + \int_0^T \left\{ \iint_{in} \rho q^3/2.d\mathbf{A} - \iint_{out} \rho q^3/2.d\mathbf{A} \right\} dt \qquad - 4.27$$

(recognising that the integral of the velocity over the area, $\int\int q.dA$ is, by definition, the volumetric flowrate, Q, at any section, where the sign convention for Q is the same as for q).

For general experimental purposes, with both valves and conduits, this formulation is probably the most useful, in that it is usable in terms of parameters which may be measured without too much difficulty, but does not overlook the effect that, for example, changes in area from inlet to outlet can have on the kinetic energy term. Where the mean value of velocity changes significantly from inlet to outlet, as for example, from ventricle (where zero velocity might be assumed) to major artery, the change in convected K.E. from inlet to outlet can be appreciable, perhaps as much as half of the overall pressure drop.

At this point, it is convenient to consider separately the two applications, or test methods. In the first case, the conduit is tested on its own, as a replacement for the ventricular outflow valve and artery. In the second case, it is considered as part of a flow system in which the ventricle has one outflow through an orifice representing a stenosed valve and a second outflow through the conduit. These cases are illustrated in Figs. 4.2 and 4.3, respectively.

Single outflow formulation

Here, the conduit diameter is assumed to be the same upstream as it is downstream of the valved section and the control volume is chosen to cross the upstream and downstream ends of the conduit. This means that the volumetric flowrate and hence mean velocity is the same at inlet and outlet. A further assumption may be applied:

12. The difference between kinetic energy convected in and that convected out is negligible

If we assume that the downstream outlet section is sufficiently far removed from the valve for the velocity distribution to be a good approximation to its fully-developed form, the worst case against which this assumption may be tested, is for flat profile entry conditions at the inlet section. Recognising that the significant contributions to energy loss occur during forward flow when, as demonstrated above, flow is turbulent, one needs to compare the kinetic energy convected in by turbulent flow with a uniform velocity distribution with that convected out where the velocity distribution is that of fully-developed turbulent pipe flow. For a mean flowrate, Q, this difference is:

$$\iint_{in} \rho q^3/\ 2.dA \ - \iint_{out} \rho q^3/\ 2.dA \ = \ 8.\rho Q^3/\ (\pi^2.d^4) \ - \ 8.467.\rho.Q^3/\ (\pi^2.d^4)$$

$$= \ - \ 0.467\{\rho Q^3/\ (\pi^2.d^4)\ \} \qquad - \ 4.28$$

Using typical values of a 20 litre/min forward flowrate of blood analogue with a density of 1000 $kg.m^{-3}$ through a 22 mm diameter conduit this formula gives around 7.5×10^{-3} W. This compares with a typical contribution of 0.5 W from the flow work terms (i.e. it represents about 1.5 % of the significant terms).

Assumption 12 justifies the removal of the second time integral from the right hand side of equation 4.27. For a conduit providing the ventricle with a single outflow, there is only one inlet and one outlet to the control volume, and continuity dictates that the volumetric flowrate of the incompressible fluid is the same across both sections. If we denote the drop in piezometric pressure from inlet to outlet by Δp^*, then equation 4.27 reduces to the familiar

formulation:

$$\Phi = \int_0^T Q\Delta p^*.\, dt \qquad - 4.29$$

Should a correction for the K.E. term be deemed necessary, (which it will not in the current work, as justified above) then incorporation of equation 4.28 suggests the use of the following formula:

$$\Phi = \int_0^T \{Q.\Delta p^* - K.Q^3\}\, dt$$

where

$$K = 0.467\rho. / (\pi^2.d^4) \qquad - 4.30$$

Double outflow formulation

From the experimental viewpoint, it is desirable that assumption 12, made for the case in which the ventricle has one outflow (the conduit), should also apply to the case of double ventricular outflow. This obviates the necessity for assessing the velocity profiles at three sections, involving the use of laser Doppler anemometry, or some similar technique. Again, an estimate may be made of the effect of the convected kinetic energy terms. This time, however, the analysis is complicated by the fact that the areas of the inlet and outlet sections may vary from one site to another, and that the flow is split between the ventricular outfow tracts in an unknown proportion which will depend on conduit performance and the degree of stenosis.

The control volume is depicted in Fig. 4.3, with inlet section 1 at the end of the conduit, inlet

section 2 just upstream of the stenosed valve, deliberately placed a short distance along a ventricular outlet section which enables assumption 10 to apply, and outlet section 3 a suitable distance downstream of the confluence. Since, in practice, the split of the flow will be primarily determined by conduit performance and degree of stenosis, the choice of diameter at section 2 is to all intents quite arbitrary, within the limits of physiological similarity. Therefore, it would seem reasonable to make the following stipulation:

13. **The two inlet sections are of the same diameter, namely that of the conduit, d_c**

For the purposes of evaluating the order of magnitude of the K.E. terms on the loss formulation, it will suffice to consider the forward flow, ventricular ejection phase of the cardiac cycle, during which most of the loss occurs, and to approximate reality by steady flow conditions at typical flowrates. The task, then is to estimate the term:

$$\iint_{in} \rho q^3/\,2.dA - \iint_{out} \rho q^3/\,2.dA$$

which will be denoted by $\phi'_{K.E.}$, and compare it with the flow work term. For this control volume, we have:

$$\phi'_{K.E.} = \iint_1 \rho q^3/\,2.dA + \iint_2 \rho q^3/\,2.dA - \iint_3 \rho q^3/\,2.dA \qquad - 4.31$$

Volumetric flowrate at section i is denoted by Q_i so that the continuity relation is:

$$Q_3 = Q_1 + Q_2 \qquad - 4.32$$

If the fraction of the forward flow taken by the conduit is α, i.e.

$$Q_1 = \alpha Q_3 \qquad - 4.33$$

then equation 4.32 dictates that:

$$Q_2 = (1 - \alpha).Q_3 \qquad - 4.34$$

With assumption 13 and with d_3 representing the tube diameter at section 3, it can be shown that:

$$\phi'_{K.E.} = \rho.Q_3^3/\pi^2 \{A/d_c^4 - B/d_3^4\} \qquad - 4.35$$

where, for plug flow conditions at inlet,

$$A = 8.(1 - 3.\alpha + 3.\alpha^2) \qquad - 4.36$$

and B depends on the velocity profile at outlet section 3. Two pertinent cases are:

$$B = 8 \quad \text{(plug flow)}$$
$$= 8.467 \quad \text{(fully-developed turbulent flow)} \qquad - 4.37$$

If we define the ratio:

$$r = d_3/d_c \qquad - 4.38$$

then equation 4.35 may be rewritten as:

$$\phi'_{K.E.} = 8.\rho.Q_3^3/ (\pi^2.d_c^4) \; . \; \{1 - 3.\alpha + 3.\alpha^2 - C/r^4\}$$

where

$C = 1$ (plug flow)

$= 1.058$ (fully-developed turbulent flow) - 4.39

In order to avoid evaluating the proportion of flow down each ventricular outflow tract at each instant and estimation of the errors thus arising, and to enable the use of just one flowmeter, the following assumption will be made:

14. Flow is lossless from a stagnant point in the ventricle to each of the control volume inlet boundaries

If the stagnant point in the ventricle is denoted by the subscript v, then, using Bernoulli's equation the piezometric pressures at sections 1 and 2 may be written as:

$$p^*_1 = p^*_v - 8\rho Q_1^2/ \pi^2 d_c^4$$

and

$$p^*_2 = p^*_v - 8\rho Q_2^2/ p^2 d_c^4 \qquad - 4.40$$

since equation 4.27 may be written as:

$$\Phi = \int_0^T \{ p^*_1 Q_1 + p^*_2 Q_2 - p^*_3 Q_3 + \phi'_{K.E.} \} dt \qquad - 4.41$$

substitution of equations 4.40 and 4.39 results in:

$$\Phi = \int_0^T \{ p^*_v.(Q_1 + Q_2) - p^*_3 Q_3 - 8\rho/\pi^2 d_c^4 (Q_{13}+Q_{23}) + 8.\rho.Q_3^3/(\pi^2.d_c^4) . \{1 - 3.\alpha + 3.\alpha^2 - C/r^4\} \} dt \quad - 4.42$$

Using equations 4.32, 4.33 and 4.34, this becomes:

$$\Phi = \int_0^T \{ (p^*_v - p^*_3).Q_3 - \{8\rho Q_3^3/(\pi^2 d_c^4)\}\{1 - 3.\alpha + 3.\alpha^2\} + 8.\rho.Q_3^3/(\pi^2.d_c^4) . \{1 - 3.\alpha + 3.\alpha^2 - C/r^4\} \} dt \quad - 4.43$$

which simplifies to:

$$\Phi = \int_0^T \{ (p^*_v - p^*_3).Q_3 - 8\rho Q_3^3 C/(\pi^2 r^4 d_c^4) \} dt$$

where

$C = 1$ (plug flow)

$= 1.058$ (fully-developed turbulent flow) - 4.44

It can be seen from equation 4.44 that the assumption concerning the profile of the outlet flow alters the K.E. correction term by some 6%. However, the K.E. correction term itself is only around 10% to 20% of the integrand during forward flow (and significantly less during retrograde flow where the smaller flowrate has a less marked effect, due to the cubic power. Thus, one may, for simplicity, assume plug

flow outlet conditions and noting equation 4.38, finally, write:

$$\Phi = \int_0^T \left\{ \left(\Delta p^* . Q_3 - 8\rho Q_3^3 / (\pi^2 d_3^4) \right) \right\} dt \qquad 4.45$$

where Δp^* is understood as the difference in piezometric pressure between a stagnant point in the ventricle and the control volume outlet section.

This formula (equation 4.45), whilst reasonably accurate, justifies an extremely simple experimental set-up, involving the use of just one flowmeter (although another may be employed in order to learn something about the proportion of flow taken by the conduit) and one height-corrected differential pressure transducer. This is the formulation that is employed in this work.

4.3 Summary

- The energy loss method is recommended as a suitable parameter for the comparative performance evaluation of valved conduits, because:

  1. Conduits are most commonly used as a second ventricular outflow tract in parallel with a blood flow path through a stenosed valve or small orifice, this system giving rise to a significant contribution to losses in terms of the regurgitant flow through the stenosis during ventricular diastole, these losses being overlooked by the pressure drop criterion.

  2. One of the principal benefits of any pressure drop criterion is the ability to compare the drop with that expected through an ideal orifice, to derive an efficiency, covering a range of sizes and flowrates. The absence of an orifice in a conduit can and does give rise to "efficiencies" of greater than 100%, demonstrating the inapplicability of the use of ideal orifice flow as the basis for an efficiency figure. No alternative ideal on which to base an efficiency is readily apparent.

- Forward flow pressure drop is, nevertheless, a useful indicator of performance, carrying clinical significance. Both evaluation methods are used.

- A rigorous energy equation is derived and presented as equation 4.13

- Assumptions are systematically applied and justified, for the experimental conditions pertaining to the practical test work presented elsewhere in this thesis. This

culminates in the recommendation that equation 4.29 be used for the evaluation of the conduit alone and equation 4.45 be used for the evaluation of the conduit as a second ventricular outflow.

5 Experimental method

5.1 Description of apparatus

5.1.1 Fluid circuit

The heart may be viewed in essence as a positive displacement pump. Changes in ventricular volume determine the flow of blood into or out of the chamber, whilst pressures throughout the system are a function of the load impedance. This standpoint underpins the pulse duplicator design (see Fig. 5.1). Blood analogue fluid is pumped in a controlled way by a reciprocating piston into and out of a cylindrical enclosure representing the ventricle. This enclosure is sealed from above by a plate containing inlet and outlet ducts. The inlet duct contains a Bjork-Shiley mitral valve prosthesis, whilst the outlet tract accommodates either the conduit under test or a small diameter orifice representing a stenosed valve. There is a further outlet tract at the base of the model ventricle, which is either plugged or accommodates the test conduit in shunt mode. Systemic compliance is provided by a pressure chamber containing a fixed mass of air, which may be varied as required. Systemic resistance is provided by a simple cross-clamp downstream of the compliance. Adjustment of this cross-clamp enables the determination of peak systolic pressure. Atrial and ventricular pressure during ventricular diastole may be varied by altering the height of the discharge reservoir which feeds the venous return. End systolic pressure is set by the elevation of the outlet pipe (downstream of the mock systemic impedance and draining into the venous return reservoir). With the exception of the conduit under test, all fluid circuit pipework is in clear plastic tubing of 22mm bore.

The Trent Polytechnic prototype conduit (see Fig. 5.2) is a rigid model with its body fabricated in acrylic. As such, this makes its incorporation into the test circuit somewhat simple. However, the flexibility and porosity of the commercial product tested, together with the nature of the ending of the conduit tubing pose several problems. The approach adopted is to seal the conduit into a length of straight 25 mm bore plastic tubing, by sewing

O-rings to the o.d. of the conduit near each end, and locating these in shallow grooves in the tubing. The end of the tubing is then pushed over a tapered stub pipe terminating in a flanged connector (see Fig. 5.3). This approach enables continuity of bore diameter, ease of connection into the circuit and retention of any conduit leakage. It also allows the conduit to be held straight for comparison with the Trent Polytechnic prototype.

The blood analogue is a 40/60 solution (by weight) of glycerol in distilled water, with a small quantity of common salt added to ensure electrical conductivity, hence compatibility with the electromagnetic flowmeter. The density is 1250 $kg.m^{-3}$ and the kinematic viscosity is 3.0 cSt.

5.1.2 Drive system

A simple but flexible system was employed to drive the reciprocating piston, whose velocity is proportional to the flowrate out of the ventricle during systole and flowrate into the ventricle during diastole. This consists of a crank mechanism with a variable throw, which controls stroke volume driven by an infinitely variable speed electric motor. However, the line along which the piston reciprocates may be offset from the crank axis by a variable amount. This has the effect of altering the ratio of systolic to diastolic time. The pulse rate is determined by the speed of rotation of the crank.

Relationships for the stroke volume and the systolic time as a fraction of the cycle time may be deduced quite simply from geometric considerations (see Fig. 5.4). Considering the piston at its furthest displacement, x_1, following the nomenclature of Fig.5.5, one may write down:

$$x_1 = \sqrt{\{(s+r)^2 - h^2\}} \qquad - 5.1$$

and

$$\omega t_1 = \sin^{-1}\{h/(s+r)\} \qquad - 5.2$$

where ω is the angular velocity of the crank. Similarly, at the opposite end of the piston

stroke, one may write down:

$$x_2 = \sqrt{\{(s-r)^2 - h^2\}} \qquad - 5,3$$

and

$$\omega t_2 = \pi + \sin^{-1}\{h/(s-r)\} \qquad - 5.4$$

If the piston area is A, then the stroke volume is given by:

$$S.V. = A.(x_1 - x_2) \qquad - 5.5$$

which, using equations 5.1 and 5.3, becomes:

$$S.V. = 2.r.A\psi$$

where

$$\psi = [\sqrt{\{(1 + r/s)^2 - (h/s)^2\}} - \sqrt{\{(1 - r/s)^2 - (h/s)^2\}}] \;/\; (2.r/s) \qquad -5.6$$

The fraction, β, of systolic to cycle time is given by:

$$\beta = 1 - (t_2 - t_1)/T \qquad - 5.7$$

where T is the cycle time. Since the cycle time is related to the angular velocity by:

$$T = 2.\pi/\omega \qquad - 5.8$$

one may substitute equations 5.2, 5.4 and 5.8 into equation 5.7 to obtain:

$$\beta = (1/2).\alpha$$

where

$$\alpha = 1 - [\sin^{-1}\{(h/s)/(1 - r/s)\} - \sin^{-1}\{(h/s)/(1 + r/s)\}]/\pi \qquad - 5.9$$

Written in this form, equations 5.6 and 5.9 show that when h = 0, the stroke volume reduces to 2.A.r and the systolic time reduces to 1/2 of the cycle time, as one might expect. They also show that the stroke volume and systolic fraction are functions of two independent variables, so that these parameters may be selected independently.

5.2 Instrumentation

As explained in Chapter 4, all the necessary information to infer conduit performance, both on its own and as a shunt, may be inferred from readings taken using one flowmeter (Gould SP2202 Electromagnetic Blood Flowmeter with 22mm dia. lumen probe) and one differential pressure transducer (SE 1150/D594 25WG connected to transducer converter type SE 90S). Output from these instruments is fed to a Gould 1602 digital storage oscilloscope, which may also be used for signal processing. The set-up for both modes is explained below.

5.2.1 Conduit alone

The control volume for testing in this configuration is as shown in Fig. 4.2. This dictates the physical arrangement shown in Fig. 5.6 with the upstream pressure tapping located a short distance inside the ventricular outflow tract.

The static head due to the difference in elevation is calibrated out of the differential pressure measurement as follows. With the motor switched off, the reservoir is raised until the free surface is just above the discharge pipe so that the static head is just sufficient to open the conduit valve. Since for this condition, flow is negligible, p_1 is greater than p_2 by an amount due to the elevation of point 2 above point 1. The offset is removed from the oscilloscope trace, so that subsequent traces are actually measuring differential piezometric pressure, as required.

The flowmeter calibration is checked *in situ* as follows. With the motor switched on, flowmeter output for one cycle is recorded on the oscilloscope and the signal processed to determine the area under the curve for the forward flow phase. Since this must correspond to the stroke volume, which is known from the piston stroke length and cylinder bore, a calibration factor to convert screen units to flowrate may readily be calculated.

5.2.2 Conduit as a shunt

As explained at the end of Section 4.2, experimental simplification can be obtained by using an upstream pressure tapping inside the ventricle. The set-up is illustrated in Fig. 5.7. The stenosed ventricular outflow valve is modelled by a profiled constriction presenting an 80% occlusion. As before, the instruments required are a differential pressure transducer (measuring the difference in piezometric pressure between the ventricle and the combined outflow section) and electromagnetic flowmeter (measuring the combined outlet flowrate). The portion of systolic outflow taken by the conduit may be inferred by comparison of the net outflow with that measured with no shunt in place (i.e. only the stenosed valve).

The flowmeter calibration is checked in the way described in the preceding section, whilst the height adjustment is made to the differential pressure trace by simply zeroing the trace with the motor switched off, there being no necessity to open a valve in this configuration, since the ventricle is connected to the combined outlet section via the "stenosis".

5.3 Test programme

Whilst ISO 5840 does not cover the laboratory testing of conduit-mounted valves, the programme laid out below duplicates the features of test procedures and measurements defined for valvular prostheses which are relevant to the current investigation. Definitions of parameters are as in Section 2 of that document, but are re-defined, below, for ease of reference. In addition to these parameters, the energy loss derived in Chapter 4 is recorded, along with related parameters.

The tests described below are carried out on a typical commercially available conduit of mechanical type (Bjork-Shiley plano-convex 22-GVPC) and on the Trent Polytechnic prototype.

Along with the first test series (described in Section 5.3.1, below), identical tests are conducted with no conduit in place, but with a restriction in the ventricular outflow tract representing an 80% occlusion - that is a local restriction in flow area of only 20% of the upstream and downstream cross-sectional area. The purpose of this is to model the functioning (or rather malfunctioning!) of a stenosed valve. This is done both to gain insight into the problems that such a condition causes under pulsatile flow, and to provide data for pressure-drop versus flowrate to assist in the estimation of the ratio of flowrates taken by the conduit and passing through the valve when the conduit is employed as a second ventricular outflow tract. The occlusion is formed as an annulus with a profiled bore (see Fig. 5.8).

5.3.1 Conduit alone

For each of three stroke volumes and for pulse rates covering realistic physiological ranges and with the systole occupying 39% (±1%) of the cardiac cycle, with arterial pressure of 120/80 mmHg and using the set-up described in Section 5.2.1 the following data are measured (see Fig. 5.9 to assist with definitions):

- cardiac output (= f. $\int_0^T Q.dt$ where f is frequency)

- mean forward flow differential piezometric pressure (= $\{1/\beta T\}.\int_0^{\beta T} \Delta p^*.dt$)

- mean systolic forward flowrate (= $\{1/\beta T\}.\int_0^{\beta T} Q.dt$ = SV. f/β)

- regurgitant volume (= $-\int_{\beta T}^{T} Q.dt$)

- regurgitant fraction (= {Regurgitant Vol.} / SV)

- energy loss per cycle ($\Phi = \int_0^T Q\Delta p^*.dt$)

- mean energy loss rate (Φ' = Φ.f)

- mean energy loss rate per unit cardiac output (= Φ'/cardiac output)

5.3.2 Conduit as a shunt

Before testing the conduit in shunt configuration, the readings described above are taken with no conduit in place, all outflow being directed through the stenosis. This enables comparison of the relieved condition with that which it replaces. This procedure also enables the estimation of the fraction of flow taken by the conduit. This is achieved by obtaining a functional relationship between mean forward flowrate through and mean piezometric pressure drop across the stenosis, thus:

$$<Q_{stenosis}>_{systole} = f\{<\Delta p^*_{stenosis}>_{systole}\} \qquad - 5.10$$

When the shunt is introduced, the forward flow fraction, α, through the conduit may be inferred from the measured total systolic output and the relationship in equation 5.10, since:

$$\alpha = (1/SV).\int_0^{\beta T} Q_{conduit}.dt$$

$$= (1/SV).\int_0^{\beta T} \{Q_{combined} - Q_{stenosis}\}.dt$$

$$\approx (1/SV.) \; . \; \{SV - \beta T.\langle Q_{stenosis}\rangle_{systole}\}$$

$$\text{i.e. } \alpha \approx 1 - (\beta T/SV) \; . \; f\{\langle \Delta p^{*}_{stenosis}\rangle_{systole}\} \qquad - \; 5.11$$

It must be stressed that the use of this relationship only enables an <u>estimate</u> of the flow through the stenosis with the shunt present also, since the details of the waveforms will be affected to some extent by inclusion of the shunt. Accurate measurement would require the use of a second flowmeter. However, a check may be made to ensure that this estimate is within reasonable bounds. This entails using the functional relationship between the mean systolic pressure drop across the conduit and the mean systolic flowrate through it, thus:

$$\langle Q_{conduit}\rangle_{systole} = g\{\langle \Delta p^{*}_{conduit}\rangle_{systole}\} \qquad - \; 5.12$$

Using an argument similar to that laid out, above, it can readily be demonstrated that the fraction, α, can also be given by:

$$\alpha \approx (\beta T/SV) \; . \; g\{\langle \Delta p^{*}_{conduit}\rangle_{systole}\} \qquad - \; 5.13$$

In both cases (equations 5.11 and 5.13 the piezometric pressure drop is not that which is measured, since for the shunt test series the upstream measurement site is in the ventricle,

whilst the downstream site is a short distance beyond the confluence of the two outflow tracts. However, a reasonable estimate may be made by employing the formulation:

$$\langle \Delta p^{*}_{measured} \rangle_{systole} = \langle \Delta p^{*}_{stenosis} \rangle_{systole} + 8.\rho.\langle Q_{stenosis} \rangle^{2}_{systole} / \pi^{2}.d^{4} \quad - 5.14$$

or

$$\langle \Delta p^{*}_{measured} \rangle_{systole} = \langle \Delta p^{*}_{conduit} \rangle_{systole} + 8.\rho.\langle Q_{conduit} \rangle^{2}_{systole} / \pi^{2}.d^{4} \quad - 5.15$$

as justified by the argument advanced in Section 4.2 (ignoring the loss associated with the confluence itself).

For the same range of stroke volumes and pulse rates and for the same systolic fraction and arterial pressure as for the arrangement considered in Section 5.3.1, above, the following parameters are assessed (see Fig. 5.10 to assist with definitions):

- cardiac output (= $f. \int_0^T Q.dt$)
- mean forward flow differential piezometric pressure (= $\{1/\beta T\}.\int_0^{\beta T} \Delta p^{*}.dt$)
- mean systolic forward flowrate (= $SV. f/\beta$)
- regurgitant volume (= $-\int_{\beta T}^{T} Q.dt$)
- regurgitant fraction (= {Regurgitant Vol.} / SV)
- fraction of net forward flowrate accommodated by the conduit

(equations 5.11, 5.13, 5.14 and 5.15)

- energy loss per cycle ($\Phi = \int_o^T \{Q.\Delta p^* - 8.\rho Q^3/ (\pi^2.d_3^4)\}.dt$)

- mean energy loss rate ($\Phi' = \Phi.f$)

- mean energy loss rate per unit mean cardiac output (= Φ'/cardiac output)

6 Test results and discussion

As explained in Section 5.3, a series of tests was first undertaken with no ventricular outlet valve or conduit in place, but rather with an 80% occlusion in the ventricular outflow tract, representing the severely stenosed condition that the conduit is intended to alleviate. The results of this series is summarised in Table 6.1. Since the tests performed in this arrangement are identical with those for the conduit as an aortic replacement, these results will be discussed in the following section. They are also drawn upon in Section 6.2 in the estimation of the split of flow between the two ventricular outflow tracts when the conduit is employed as a shunt.

6.1 Conduit as aortic replacement

The results of this test series, undertaken on the Bjork-Shiley conduit and the ball valve prototype are summarised in Tables 6.2 and 6.3, respectively.

6.1.1 Forward flow pressure drop

Two comparisons are of interest here. The first is the mean systolic piezometric pressure drop as a function of mean forward flowrate. This is of interest both as a comparison of pulsatile flow results with their steady flow analogues, and, for the case of the stenosis alone, to assist in the estimation of the flow split in the shunt test series. These results are of fundamental importance to the engineer, since they are the best indicator of inherent haemodynamic performance. The second, which is of more clinical significance, is the comparison of mean systolic piezometric pressure drop as a function of cardiac output. This figure is of interest because it gives an idea of the pressure drops to be expected as the heart delivers a net flowrate. Thus the effect of regurgitation will result in additional pressure drop as the heart has to deliver a higher forward flowrate both because systole occupies a certain fraction of the cycle time and to compensate for the reflux. The former effect can be calculated, but it is the latter effect which is particularly overlooked by steady flow testing.

Fig. 6.1 shows the results of the correlation with mean systolic flowrate. This shows that whilst both conduits, not surprisingly, offer a substantial reduction in pressure drop over the uncorrected stenosis, the degree of improvement is not particularly spectacular. It also shows the performance of the ball-valve prototype to be superior to that of the Bjork-Shiley conduit. There is no significant degree of scatter despite the fact that these forward flowrates were achieved by varying both the stroke volume and the pulse-rate. As expected, the data follow a parabolic form. This square-law dependence is emphasised in Figs. 6.2 - 6.4, in which pressure drop is plotted against the square of the forward flowrate. It is quite apparent that the resultant data may be fitted well by a straight line through the origin. The slope of this line enables quantification of a coefficient, K, relating mean systolic pressure

drop to mean systolic flowrate, in a relationship of the form:

$$<\Delta p^{*}>_{systole} = K \cdot \{<Q>_{systole}\}^{2} \qquad - 6.1$$

These results are shown on Figs. 6.2 - 6.4. They show that the prototype conduit results in a reduction of the forward flow pressure drop produced by the uncorrected stenosis by a factor of 2.0 for a given mean systolic flowrate, and that the pressure drop produced by the Bjork-Shiley conduit is 25% greater than that produced by the ball valve prototype.

Fig. 6.5 shows the mean systolic pressure drop *versus* mean systolic flowrate data in non-dimensionalised form for the ball valve prototype conduit, plotted on the same axes as results derived from steady flow evaluation of the same conduit, taken from Fig. 7.17 of Tansley (1988). It is necessary to show this comparison in non-dimensional form since Tansley's test fluid was water. Despite the considerable scatter of the pulsatile flow data and the higher Reynolds numbers of the steady flow data, it can be seen that the Euler numbers produced are of comparable value in both cases - the best fit curves differing by no more than 30% where Reynolds number ranges overlap. If the steady flow data were to be extrapolated to lower Reynolds numbers, it appears likely that data here would be largely coincident between the two cases. It is not clear whether this is a phenomenon related to stroke volume (points to the left of the pulsatile flow data were produced at lower stroke volume) or simply to flowrate, with better correlation at lower values.

The point made, above, concerning the effect of systolic fraction and regurgitant flow on the pressure drop associated with a given cardiac output, as opposed to that for a given forward flowrate, is amply illustrated by comparison of Fig. 6.6 with Fig. 6.1. The extent of this phenomenon is particularly apparent when the graphs of pressure drop *versus* the square of the cardiac output (Figs. 6.8 - 6.10) are contrasted with Figs. 6.2 - 6.4. For example, Figs. 6.8 and Fig. 6.2 show that for the uncorrected stenosis, the pulsatile nature of the flow results in a pressure drop twenty times greater for a given flowrate. That is to say, if it

were possible for the same cardiac output to be achieved through steady flow, one would obtain only a very small fraction of the pressure drop which is actually entailed by having to pump a pulsatile flow at much higher instantaneous flowrates over systole and to compensate for the reflux.

For the tests presented herein, the systolic fraction of cycle time was 39%. An ideal, zero leakage instantaneously closing valve would produce a pressure drop 6.6 times (i.e. $1/0.39^2$) greater under these pulsatile conditions than it would to deliver the same cardiac output as a steady flowrate. By way of comparison, the Bjork-Shiley conduit produces 8.8 times the pressure drop, whilst the ball valve prototype conduit produces 7.1 times the pressure drop. The difference between these figures and the ideal are primarily the result of regurgitation - having to pump a higher forward flowrate (resulting in increased pressure drop) to compensate for the reflux.

Another point to note is that this effect results in an amplification of the difference in haemodynamic performance between prostheses, should one have worse regurgitation than another. Again this is a feature which is missed by steady flow testing. By way of illustration, as noted above, the Bjork-Shiley conduit produced pressure drops some 25% higher than the ball valve prototype conduit for a given mean systolic flowrate. However, to produce the same cardiac output, its pressure drop is 56% greater.

Finally, it may be seen by comparison of Fig. 6.6 with Fig. 6.1 that the effect of stroke volume becomes noticeable when pressure drop is plotted against cardiac output. This is marked by a greater degree of scatter on the linear plots (Figs. 6.8 - 6.10). To clarify this effect, separate point graphs for each stroke volume are produced in Fig.6.7. This shows that the two conduits and the uncorrected stenosis behave in different ways to stroke volume and pulse-rate. The stenosis would appear to produce separate and distinct curves for different stroke volumes, with slightly higher pressure drops associated with larger stroke volumes. For the Bjork-Shiley conduit, the opposite would appear to be true, namely that the data

appear to lie on the same curve, with deviations caused not by differing stroke volumes, but rather by differing pulse-rates, particularly at high pulse-rates. The ball-valve prototype, on the other hand, appears to form little differentiation between these two means by which cardiac output is increased.

6.1.2 Energy loss

Fig. 6.11 shows the energy loss per cycle as a function of cardiac output for the uncorrected stenosis and for the two conduits. This shows that the prototype conduit reduces the energy loss associated with the stenosis by a whole order of magnitude and halves the energy loss produced by the Bjork-Shiley conduit under the same conditions. To establish whether the energy loss follows a power law, the data are plotted on a logarithmic scale in Fig. 6.12. As well as emphasising the conclusions just drawn above about the relative magnitude of the energy loss, it shows that, to a reasonable approximation (and for the ball valve prototype conduit, to a good approximation) the energy loss follows a square law with cardiac output.

Again, to examine the effect of stroke volume on the results, these are plotted as separate curves for different stroke volumes in Fig. 6.13, so that each curve represents the change in energy loss with changing pulse-rate. This shows that, whilst there may be a slight tendency to produce higher energy loss with larger stroke volumes, the effect is of minor significance for both conduits. The effect is further reduced if the mean power loss (energy loss per pulse times pulse-rate) is plotted against cardiac output (Figs. 6.14 and 6.15). This parameter is a measure of the average additional rate of work required from the heart to overcome conduit-related losses for a given cardiac output and is the most clinically useful of the energy loss-related parameters.

A further parameter sometimes quoted is the specific power loss, or power loss per unit cardiac output. This is plotted in Fig. 6.16 and shows that the specific power loss of the ball valve prototype is between 1/2 and 1/3 of that produced by the Bjork-Shiley conduit, with the improvement more pronounced at higher flowrates.

6.1.3 Regurgitation

Regurgitation is plotted in Fig. 6.17 as a function of cardiac output, with stroke volume as parameter. This shows not only that the total reflux produced by the Bjork-Shiley conduit is surprisingly large and that this is very significantly reduced by the ball valve prototype conduit, but also that varying the pulse-rate affects the regurgitation produced by the conduits in a different manner from that in which it affects the uncorrected stenosis. With the conduits, for a given stroke volume, an increasing pulse-rate produces an increasing regurgitant fraction, more so for the Bjork-Shiley conduit, whereas it produces a decreasing regurgitant fraction for the uncorrected stenosis. The reason for this observation is unclear, but may well be a function of the conditions employed to model systemic impedance. At constant pulse-rate, the regurgitant fraction is sensibly independent of stroke volume. This dependency on pulse-rate rather than stroke volume is emphasised when regurgitant fraction is plotted against pulse-rate, as in Fig. 6.18. This shows that the data thus plotted for each conduit and for the uncorrected stenosis tend to fall on a smooth curve. The regurgitation produced by the prototype conduit at a standard pulse-rate of 70 bpm is only 1/3 of that produced by the Bjork-Shiley conduit, whilst this fraction falls to 1/4 at 120 bpm. This improved regurgitation performance is a function both of the better closure time of the ball valve prototype, which employs a neutral buoyancy occluder and was engineered to produce a low net streamwise force in forward flow conditions (see Section 3.2, Tansley *et al* (1986) and Tansley (1988)), and of the zero leakage gap associated with the ball occluder, as opposed to the finite leakage gap of the Bjork-Shiley device (see Section 3.1). Even at the highest pulse-rates, the regurgitant fraction of the prototype conduit does not exceed 5%, whereas the Bjork-Shiley's reaches some 24%. This is every bit as significant for the overall performance improvement in terms of pressure drop for a given cardiac output, as is the inherent improvement in forward flow haemodynamics.

6.2 Conduit as a shunt

The results of this test series, undertaken on the Bjork-Shiley conduit and the ball valve prototype are summarised in Tables 6.4 and 6.5, respectively. Comparing with Tables 6.2 and 6.3 one cannot help but be struck by the significantly lower cardiac outputs for similar pulse-rates and stroke volumes. This emphasises the degree to which the large reflux volumes caused by the non-closing stenosis are detrimental to overall performance of the system - the heart has to work harder to deliver the same net flowrate. This difference is more apparent with regard to the energy loss results than to the pressure drop results. This is because the provision of a second outflow tract causes a large increase in the forward flow area, hence a very low overall pressure drop, which offsets the higher pressure drops associated with the larger systolic flowrates required to compensate for the large regurgitation. Thus, whilst ventricular pressure may be drastically reduced by the provision of a second outflow, the heart muscles have to work harder to provide adequate net flowrate.

6.2.1 Forward flow pressure drop

Referring to Fig. 6.19, one can see that the introduction of either conduit as a second ventricular outflow results in a very marked reduction in forward flow pressure drop for a given mean systolic flowrate - so much so that the difference in performance between the two conduits becomes almost insignificant. Comparison with Fig. 6.1 reveals the extent of the improvement, which is a further threefold reduction in mean systolic pressure drop beyond that to be attained with the conduit as a replacement for the stenosed valve. Fig 6.20 shows the same data as Fig. 6.19 but permits a clearer distinction between the forward flow pressure drop performance of the two conduits. It can be seen that use of the ball valve prototype results in a pressure drop of about 3/4 of that obtained with the Bjork-Shiley conduit. The linear regression plots of Figs. 6.21 and 6.22 show that again the mean systolic pressure drop is proportional to the square of the mean systolic flowrate and confirm that

the performance of the ball valve prototype is better than that of the Bjork Shiley by a factor of 0.77.

However, as pointed out in the preceding section, the result of more clinical significance is the pressure drop for a given cardiac output. Here, the detrimental effect of the stenosis becomes apparent, since it is responsible for a very large regurgitant volume. The apparent threefold improvement in pressure drop performance suggested above is seen in Fig. 6.23 to be eliminated. Comparison of this graph with Fig. 6.6 shows that very similar pressure drops are obtained for a given cardiac output when the conduit is used as a shunt for an 80% stenosis as when it is used as a valvular replacement. The quadratic dependence of mean systolic pressure drop on cardiac output is again confirmed by Figs. 6.24 and 6.25, which also enable a numerical comparison of the two conduits with each other as shunts and of either conduit as a shunt for an 80% stenosis with that as a valvular replacement. Thus we can see that the pressure drop for a given cardiac output obtained when the ball valve prototype is used as a shunt is less than that produced by the Bjork-Shiley conduit in the same situation by a factor of 0.72. Since the inherent retrograde flow performance of the ball valve conduit is superior to that of the Bjork-Shiley conduit (see Section 6.1), the effect of the reflux through the stenosis has a more marked effect on the pressure drop *versus* cardiac output results. Thus, whilst the pressure drop for a given cardiac output is 6% greater when the Bjork-Shiley conduit is used as a shunt than when it is used as a valvular replacement, the pressure drop obtained with the ball valve prototype as a shunt is 20% greater than when it is used as a replacement.

Fig. 6.26 shows the pressure drop data as separate curves for each stroke volume. This indicates a dependence of pressure drop on stroke volume, such that for a given cardiac output a greater pressure drop may be expected from a large stroke volume and low pulse-rate than from a small stroke volume and high pulse-rate. Comparison with Fig. 6.7 shows this effect to be far more significant when the conduit is used as a shunt than when it is used as a valvular replacement. This is again due to the strong influence of the stenosis on the overall behaviour of the system. This assertion is borne out by Fig. 6.27 in which pressure

drop is plotted against pulse-rate with stroke volume as parameter. It can be seen that the overriding dependence is on stroke volume and not on the choice of conduit, similar results appearing for either conduit in the same situation.

6.2.2 Energy loss

Comparison of Fig. 6.28 with Fig. 6.11 shows that the energy loss for a given cardiac output is greater when either conduit is used as a shunt for an 80% stenosis than when it is used as a valvular replacement. When the data are plotted on a logarithmic scale (Fig. 6.29), quantitative comparison is facilitated. The energy loss is around 2 to 3 times greater in shunt usage at low flowrates, dropping to around 1.6 to 1.9 times greater at a moderate flowrate of 8 litre/min. The greater energy loss figures for shunt mode are because of the energy wasted having to re-pump the large reflux volume caused by the ever-open orifice of the stenosis, whilst the variation in performance with cardiac output is due to the weaker dependence on cardiac output in shunt mode than in valvular replacement mode. (In replacement mode a power law with an index of 2 - i.e. a square law - provided a reasonable approximation, whereas in shunt mode power law approximations require indices in the range 1.3 to 1.5.) Again, the performance of the ball-valve prototype is superior, providing energy losses of around half those produced with the Bjork-Shiley conduit over a realistic range of flowrates.

Fig. 6.30 shows energy loss data for each stroke volume plotted on a separate curve. Comparison with Fig. 6.13 reveals a stronger dependence of energy loss per pulse on stroke volume when the conduit is used as a shunt than when it constitutes a valvular replacement. For a given cardiac output, the energy loss is lower for a larger stroke volume (hence a lower pulse-rate).

Fig. 6.31 shows the results of power loss *versus* cardiac output, and again demonstrates a reduction in the differentiation between the two conduits in shunt mode as compared to their

performance as valvular replacements, whilst the average power loss is higher in shunt mode.

Comparison of the data for specific power loss *versus* cardiac output for the two modes of usage (Figs. 6.32 and 6.16, respectively) emphasises both the reduction in performance in shunt mode. More strikingly, the shunt results are far more dependent on stroke volume, with substantially lower specific power loss at a given flowrate if that flowrate is provided by a larger stroke volume.

6.2.3 Regurgitation

Figs. 6.33 and 6.34 show, respectively, regurgitant fraction as a function of cardiac output with stroke volume as parameter, and as a function of pulse-rate. Whilst it may be observed again that the ball valve prototype offers reductions in net regurgitation over the Bjork-Shiley conduit, the immediately striking feature of both graphs is the shear magnitude of the reflux volume - the introduction of the conduit has very little effect in reducing it. This is particularly apparent in Fig. 6.33, in which it is quite difficult to distinguish between the curves representing regurgitation for the stenosis alone and for the system created by the use of either conduit as a shunt, particularly at lower stroke volumes.

Fig. 6.34 shows that regurgitation decreases with increased pulse-rate more or less independently of stroke volume, emphasising the predominance of the stenosis in determining the overall behaviour of the system. Clearly, as the extent of the stenotic occlusion increases, one might expect the behaviour of the system to approximate more closely to that of the conduit as a valvular replacement. What is perhaps surprising is that even with 80% occlusion, the regurgitation is almost unaffected, with corresponding detriment to overall system performance.

6.2.4 Ratio of flowrates

As mentioned in Section 5.3, an estimate of the fraction of the total systolic flow accommodated by the conduit may be made by two means. (It must be emphasised that this refers to the flowrates during ventricular systole, not over the whole cycle.) The results of this estimation for each of the conduits are shown in Figs. 6.35 and 6.36. The error bands arising from the two different estimates are shown on these graphs. Whilst the ball valve prototype accommodates a fractionally higher percentage of the total forward flow, both conduits show very little variation with stroke volume, and both accommodate about 55% of the flow. This is in line with the findings of Shanebrook and Levine (1979) - see Section 2.4.1 - who predicted from theoretical steady-flow considerations that insertion of a Bjork-Shiley valved conduit as a shunt for an 80% stenosis would result in the conduit accommodating 52% of the forward flow.

6.3 Summary

The principal experimental findings may be summarised as follows:

- Mean systolic pressure drop varies as the square of mean systolic flowrate in both modes of usage.

- Forward flow pressure drop results from pulsatile tests on the ball valve prototype are broadly in line with those indicated by steady flow tests.

- Forward flow pressure drop for the conduits alone show that the Bjork-Shiley conduit produces 25% greater pressure drop than the ball valve prototype for the same forward flowrate.

- The difference in pressure drop results is amplified when considering pressure drop for a given cardiac output, due to having to re-pump different regurgitant volumes - the Bjork-Shiley conduit produces 56% higher pressure drop.

- The energy loss per pulse (and the mean power loss) of the ball valve prototype is half that of the Bjork-Shiley conduit for a given cardiac output.

- Zero-leakage and reduced closure time of the ball-valve prototype result in regurgitant fractions of between 1/4 and 1/3 of those obtained with the Bjork-Shiley conduit in valvular replacement tests, with better improvements at higher pulse-rates.

- The regurgitant fraction of the ball valve prototype was within 5% in all valvular replacement tests.

- Neither conduit used as a shunt for an 80% stenosis has a significant effect on the regurgitant fraction, this being almost entirely a function of the stenosis.

- The provision of a second, parallel valved ventricular outflow tract produces marked reductions in the overall pressure drop - similar to those for produced by the conduit as a valvular replacement.

- Pressure drop is more strongly dependent upon stroke volume when the conduit is used as a shunt, with higher values at higher stroke volume.

- Energy loss is more strongly dependent upon stroke volume when the conduit is used as a shunt, with higher values at lower stroke volume.

- The previous two observations provide conflicting imperatives with regard to paediatric cardiac surgery.

- Since better overall pressure drop and energy loss performance is obtained with the conduit as the sole outflow tract, it may be more sensible to ligate the artery immediately downstream of the stenosed valve when bypassing it with a conduit.

- This would have the additional benefit of eliminating the dichotomy referred to above, since when the conduit is the sole ventricular outflow, the energy loss and pressure drop are far less dependent on stroke volume.

- Energy loss figures are significantly greater when the conduit is used in shunt mode than when it is used as a valvular replacement.

- Energy loss is a weaker function of cardiac output when the conduit is used as a shunt.

- Whilst the performance of the ball valve prototype conduit is superior to that of the Bjork-Shiley conduit, the difference in performance is less marked in shunt mode, since the behaviour of the stenosis predominates.

- The fraction of systolic flow accommodated by either conduit is about 55% when used as a shunt for an 80% stenosis - in line with theoretical predictions.

7 Conclusions

7.1 Prototype development

The choice of valve type - twin-flap or caged ball - for conduit development revolved around leakage considerations. Since the ball occluder seated fully, so that virtually zero leakage was expected, theoretical investigation concentrated on the twin-flap design. Retrograde flow through a large leakage gap was found to be turbulent but not fully-developed. Where turbulent flow occurred near prosthetic surfaces, these locations were best washed during forward flow anyway, thus undermining the proposed benefit of an engineered leakage gap, namely to provide a scouring action to help prevent thrombus formation. Furthermore, stagnant flow regions during forward flow were also found to be stagnant under leakage conditions. Since a large leakage gap produced shear stresses approaching a level deemed to be critical in terms of haemolysis as well as producing an unacceptably high regurgitant flow, a narrower leakage gap would be required. However, to provide an adequately narrow gap, the dimensions would be difficult to engineer to an appropriate tolerance in the ceramic material, and difficult to maintain over the (deliberately long) valve life. Therefore, the ball valve concept was developed instead.

Steady flow testing of an early ball valve prototype conduit had indicated a problem with an oscillatory instability of the occluder. This instability was found to be caused by the large flow area immediately downstream of the occluder in its open position, resulting in a net back pressure due to Bernoulli effects. The instability was cured by re-profiling the outlet section to maintain constant flow area.

7.2 Performance evaluation methods

The energy loss method is recommended as a suitable parameter for the comparative performance evaluation of valved conduits for the following reasons:

- Conduits are most commonly used as a second ventricular outflow tract in parallel with a blood flow path through a stenosed valve or small orifice. This system gives rise to a significant contribution to losses arising from the regurgitant flow. This has two effects. The first is to necessitate a higher forward flowrate to compensate for the reflux, resulting in higher systolic loss, whilst the second is to cause direct flow work losses during ventricular diastole. This second loss is overlooked by the pressure drop criterion.

- One of the principal benefits of any pressure drop criterion is the ability to compare the drop with that expected through an ideal orifice, to derive an efficiency, covering a range of sizes and flowrates. The absence of an orifice in a conduit can and does give rise to "efficiencies" of greater than 100%, demonstrating the inapplicability of the use of ideal orifice flow as the basis for an efficiency figure. No alternative ideal on which to base an efficiency is readily apparent, particularly when the conduit is being used as a shunt.

Forward flow pressure drop is, nevertheless, a useful indicator of performance, carrying clinical significance, so that both evaluation methods should be used.

A rigorously derived expression for energy loss may be considerably simplified for the conduit either as a valvular replacement or as a shunt when justifiable assumptions are made about the experimental conditions or, putting it conversely, when experimental conditions are chosen such that these assumptions may be made. These relate primarily to a considered choice of appropriate control volume hence of transducer location and of flow areas.

7.3 Conduit performance

7.3.1 Conduit as a valvular replacement

The mean systolic pressure drop was shown to be proportional to the square of both mean systolic flowrate and of cardiac output for both conduits, with forward flow pressure drop results for the ball valve prototype in broad agreement with results indicated by previous steady flow testing.

Despite the Bjork-Shiley conduit only producing a 25% greater forward flow pressure drop than the ball valve prototype for a given mean systolic flowrate, its inferior regurgitation performance and the consequent necessity to deliver higher forward flowrates resulted in its pressure drop being 56% higher for a given cardiac output. This observation demonstrates that steady flow testing alone is insufficient, since differences in regurgitation and leakage performance show up as pressure drop (and hence also as energy loss) for a given cardiac output.

Despite the ball valve prototype producing only half the forward flow pressure drop produced by the uncorrected 80% occluding stenosis for a given mean forward flowrate, a 6-fold reduction in pressure drop was produced for a given cardiac output. Again, this is due to the large reflux of the uncorrected stenosis.

Energy loss (or power loss) was also found to be approximately proportional to the square of cardiac output, with power loss per unit cardiac output lower by a factor of 2 - 3 for the ball valve prototype than for Bjork-Shiley conduit.

The regurgitation of the ball valve conduit was shown to be significantly better than that of the Bjork-Shiley device, with regurgitation depending on pulse-rate rather than stroke volume. The regurgitation through the Bjork-Shiley was some 3 - 4 times greater than that through the ball valve prototype conduit with better comparative performance at higher

pulse-rates. Regurgitant fraction was within 5% in all test conditions with the ball valve prototype.

In summary, the ball valve prototype was shown to be superior to the Bjork-Shiley conduit in every measured respect.

7.3.2 Conduit as a shunt

The overwhelming conclusion of this test series is that when used as a shunt for a stenosis representing as much as an 80% occlusion, the shunt does almost nothing to reduce overall regurgitation - it simply reduces the power loss on forward flow and reduces systolic pressure drop. The heart still has to work hard to re-pump the large regurgitant fraction (typically around 55%, but almost as much as 70% at low stroke volume and pulse-rate). The implication of this is that (perhaps ironically) the more severe is the occlusion represented by the stenosis, the more effective is the shunt. That is, better performance may be obtained from the system comprising shunt and stenosis if the stenosis is more severe. One may even go as far as to suggest, that the best performance would be obtained if the artery immediately downstream of the stenosed valve were to be ligated altogether.

Again, the ball valve prototype performs better than the Bjork-Shiley on every criterion, but the consequence of the overwhelming influence of regurgitation

through the open stenosis is that the comparative performance of the different conduits is less marked than when they are being used as valvular replacements.

Whilst the pressure drop for a given cardiac output produced with either conduit in place is really very similar to that produced with the conduit as a valvular replacement (although slightly higher), the energy loss is typically around twice that previously obtained (more at low outputs, less at high outputs). This emphasises the point made in Section 7.2 that pressure drop methods neglect the losses associated directly with diastolic regurgitation.

In shunt mode the effect of stroke volume on both pressure drop and power loss is more marked. However, pressure drop increases whilst power loss is reduced as stroke volume increases.

8 Recommendations

In view of the results presented in this thesis, it is clear that the ball valve concept, when engineered into a purpose-designed extracardiac conduit, can offer significant improvements over other commercially available devices. It is therefore recommended that the development programme be continued. In the first instance, this might take the form of extending the test regime reported herein, with the same rigid acrylic prototype to cover its use as a shunt for different degrees of stenotic occlusion, augmenting these studies with a direct measurement (rather than a theoretical estimate) of the fraction of systolic flow accommodated by the conduit, and with flow visualisation studies.

Since one of the principal attractions of the cardiac valve prosthesis development work at Nottingham Polytechnic is the prospect of combining the inherent longevity of a mechanical device with the haemocompatibility of a biological lining, by virtue of the use of appropriate ceramics, it is recommended that a valve body be cast in porous alumina with integral restraining struts and small sewing holes to facilitate the attachment or replacement of proximal and distal conduit tubing, or of the valve-bearing section (see Fig. 8.1). Several devices in a range of sizes should be manufactured. The tubing should be of a reinforced bellows-type construction, offering flexural compliance and cross-sectional strength but with some radial elasticity to mimic the natural compliance of the ascending aorta, which helps maintain aortic pressure during ventricular diastole.

This set of prototypes should then be submitted to an *in vitro* test programme, sticking as closely as is permitted by physical limitations to the ISO standard, prior to animal trials. In addition to the ISO test specifications, the conduit should be tested in shunt mode against a range of stenotic occlusions, and at cardiac outputs such as may be found in infants, since many conduits are used for the early correction of congenital cardiac malformations. In this mode, as explained previously, the power loss should be quoted as well as the pressure drop.

TABLES

UNCORRECTED STENOSIS

Stroke volume litre	Pulse-rate beats per mir	Mean systolic piezometric pressure drop mmHg	Cardiac output litres/min	Mean systolic flowrate litres/min	Regurgitant fraction %	Regurgitant volume litres	Energy loss per cycle J	Mean power loss W	Power loss per unit cardiac output W/(litre/min)
0.059	72.5	4.0	1.41	11.0	67.0	0.040	0.085	0.103	0.073
0.059	104.5	8.0	3.21	15.8	47.9	0.028	0.198	0.345	0.107
0.059	122.0	11.2	3.96	18.5	45.0	0.027	0.318	0.647	0.163
0.108	71.1	12.4	3.38	19.7	56.0	0.060	0.408	0.483	0.143
0.108	105.6	28.2	6.16	29.2	46.0	0.050	1.280	2.253	0.366
0.108	123.4	38.5	7.73	34.2	42.0	0.045	1.990	4.093	0.529
0.192	69.8	38.9	6.03	34.4	55.0	0.106	2.210	2.571	0.426
0.192	103.3	85.2	10.71	50.9	46.0	0.088	6.700	11.535	1.077
0.192	120.6	116.0	13.66	59.4	41.0	0.079	10.340	20.783	1.521

Table 6.1 Test results for uncorrected stenosis (80% occlusion)

BJORK-SHILEY CONDUIT

Stroke volume	Pulse-rate	Mean systolic piezometric pressure drop	Cardiac output	Mean systolic flowrate	Regurgitant fraction	Regurgitant volume	Energy loss per cycle	Mean power loss	Power loss per unit cardiac output
litre	beats per min	mmHg	litres/min	litres/min	%	litres	J	W	W/(litre/min)
0.059	61.0	1.9	3.24	9.2	10.0	0.006	0.167	0.170	0.052
0.059	73.2	2.9	3.80	11.1	12.0	0.007	0.180	0.220	0.058
0.059	100.0	6.1	5.00	15.1	15.3	0.009	0.204	0.340	0.068
0.059	125.4	7.5	5.95	19.0	19.6	0.012	0.215	0.449	0.076
0.059	149.5	10.1	6.70	22.6	24.0	0.014	0.225	0.561	0.084
0.108	79.2	9.1	7.60	21.9	11.1	0.012	0.417	0.550	0.072
0.108	94.7	13.5	8.90	26.2	13.0	0.014	0.526	0.830	0.093
0.108	113.0	22.9	10.00	31.3	18.1	0.020	0.595	1.121	0.112
0.192	68.6	25.2	11.80	33.8	10.4	0.020	1.360	1.555	0.132
0.192	78.1	30.3	13.20	38.4	12.0	0.023	1.880	2.447	0.185
0.192	83.0	35.8	13.50	40.9	15.3	0.029	2.420	3.348	0.248

Table 6.2 Test results for Bjork-Shiley conduit as a valvular replacement

BALL VALVE CONDUIT

Stroke volume	Pulse-rate	Mean systolic piezometric pressure drop	Cardiac output	Mean systolic flowrate	Regurgitant fraction	Regurgitant volume	Energy loss per cycle	Mean power loss	Power loss per unit cardiac output
litre	beats per min	mmHg	litres/min	litres/min	%	litres	J	W	W/(litre/min)
0.059	60.0	1.5	3.43	9.1	3.10	0.0018	0.070	0.070	0.020
0.059	89.0	3.3	5.08	13.5	3.20	0.0019	0.090	0.134	0.026
0.059	104.5	4.3	5.89	15.8	4.50	0.0027	0.130	0.226	0.038
0.059	123.0	6.1	6.85	18.6	5.60	0.0033	0.150	0.308	0.045
0.110	61.0	4.9	6.57	17.2	2.05	0.0023	0.220	0.224	0.034
0.110	92.0	12.5	9.92	25.9	1.95	0.0021	0.340	0.521	0.053
0.110	108.0	15.1	11.24	30.5	5.40	0.0059	0.410	0.738	0.066
0.192	53.5	12.1	10.04	26.3	2.24	0.0043	0.499	0.445	0.044
0.192	60.0	15.0	11.26	29.5	2.30	0.0044	0.539	0.539	0.048
0.192	72.0	19.1	13.51	35.4	2.25	0.0043	0.965	1.158	0.086
0.192	81.0	27.7	14.58	39.9	6.22	0.0119	1.480	1.998	0.137

Table 6.3 Test results for ball valve conduit as a valvular replacement

BJORK-SHILEY SHUNT

Stroke volume	Pulse-rate	Mean systolic piezometric pressure drop	Cardiac output	Mean systolic flowrate	Regurgitant fraction	Regurgitant volume	Energy loss per cycle	Mean power loss	Power loss per unit cardiac output
litre	beats per min	mmHg	litres/min	litres/min	%	litres	J	W	W/(litre/min)
0.059	60.0	0.9	1.13	9.1	68.1	0.040	0.05	0.055	0.049
0.059	74.3	1.3	1.53	11.2	65.1	0.038	0.14	0.176	0.115
0.059	102.0	2.1	3.13	15.4	48.0	0.028	0.24	0.415	0.132
0.059	126.1	2.9	4.54	19.1	39.0	0.023	0.41	0.872	0.192
0.107	79.8	3.5	3.93	21.9	54.0	0.058	0.30	0.402	0.102
0.107	95.1	5.2	5.50	26.1	45.9	0.049	0.47	0.739	0.134
0.107	114.0	8.8	7.20	31.3	41.0	0.044	1.01	1.915	0.266
0.191	69.2	9.7	5.95	33.9	55.0	0.105	0.47	0.547	0.092
0.191	79.1	11.5	7.40	38.7	51.0	0.097	0.70	0.929	0.125
0.191	82.0	13.7	8.14	40.2	48.0	0.092	0.95	1.294	0.159

Table 6.4 Test results for Bjork-Shiley conduit as a shunt for an 80% occlusion

BALL VALVE SHUNT

Stroke volume	Pulserate	Mean systolic piezometric pressure drop	Cardiac output	Mean systolic flowrate	Regurgitant fraction	Regurgitant volume	Energy loss per cycle	Mean power loss	Power loss per unit cardiac output
litre	beats per min	mmHg	litres/min	litres/min	%	litres	J	W	W/(litre/min)
0.059	58.2	0.6	1.13	8.8	67.1	0.040	0.04	0.034	0.030
0.059	73.4	0.9	1.56	11.1	64.0	0.038	0.05	0.064	0.041
0.059	99.2	1.5	3.16	15.0	46.0	0.027	0.09	0.157	0.050
0.059	125.4	2.1	4.59	19.0	38.0	0.022	0.28	0.575	0.125
0.11	78.8	2.9	4.16	22.2	52.0	0.057	0.10	0.129	0.031
0.11	93.8	4.8	5.68	26.5	45.0	0.049	0.34	0.533	0.094
0.11	113.0	6.0	7.33	31.9	41.0	0.045	0.75	1.412	0.193
0.192	69.0	6.8	6.23	34.0	53.0	0.102	0.21	0.242	0.039
0.192	78.9	9.5	7.69	38.8	49.2	0.095	0.62	0.814	0.106
0.192	84.0	11.5	8.87	41.4	45.0	0.086	0.82	1.148	0.129

Table 6.5 Test results for ball valve conduit as a shunt for an 80% occlusion

FIGURES

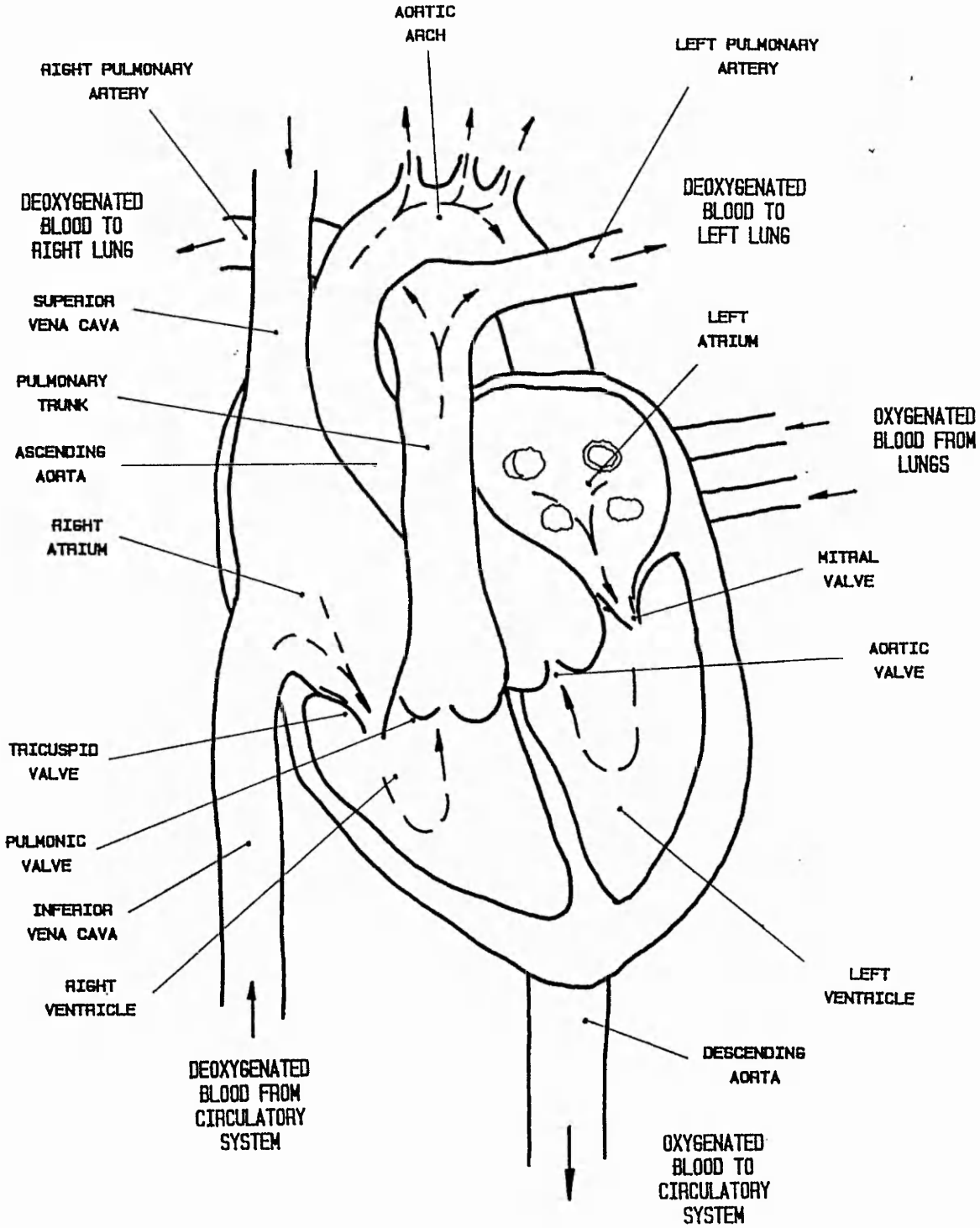


Fig. 1.1 The human heart

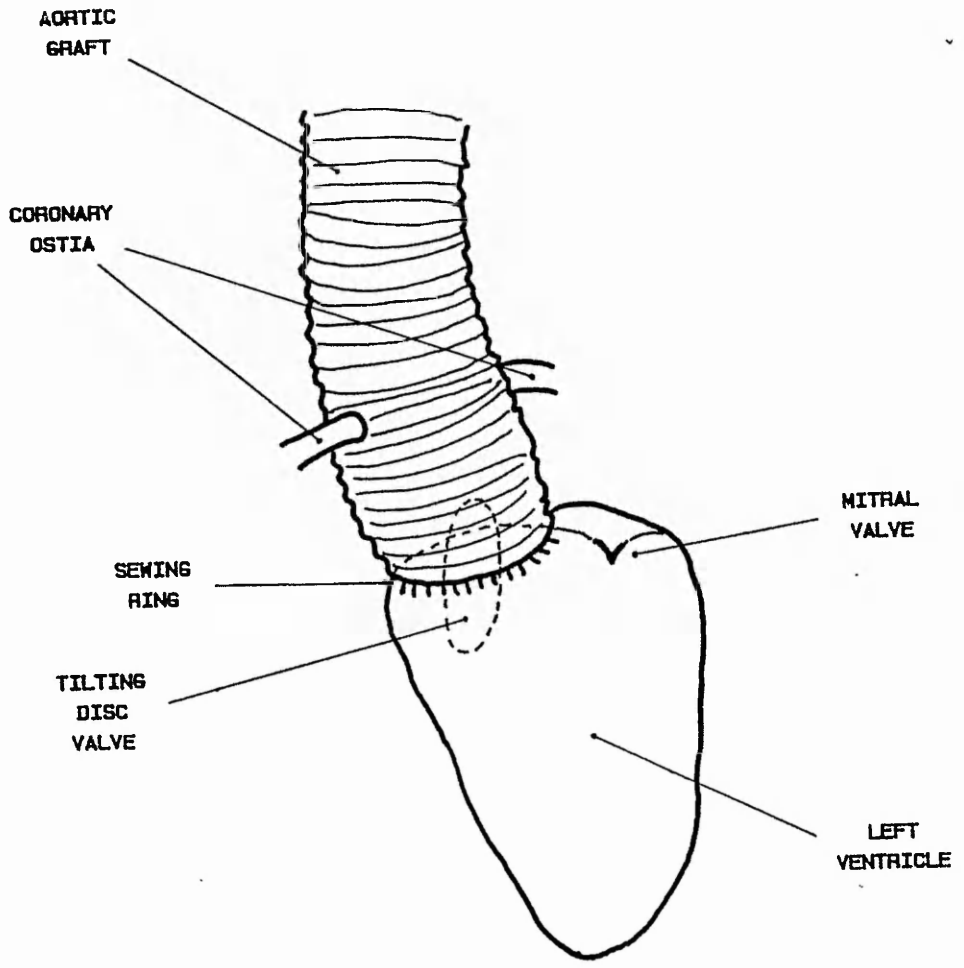


Fig. 1.2 Replacement of aortic valve and ascending aorta with composite graft (conduit)

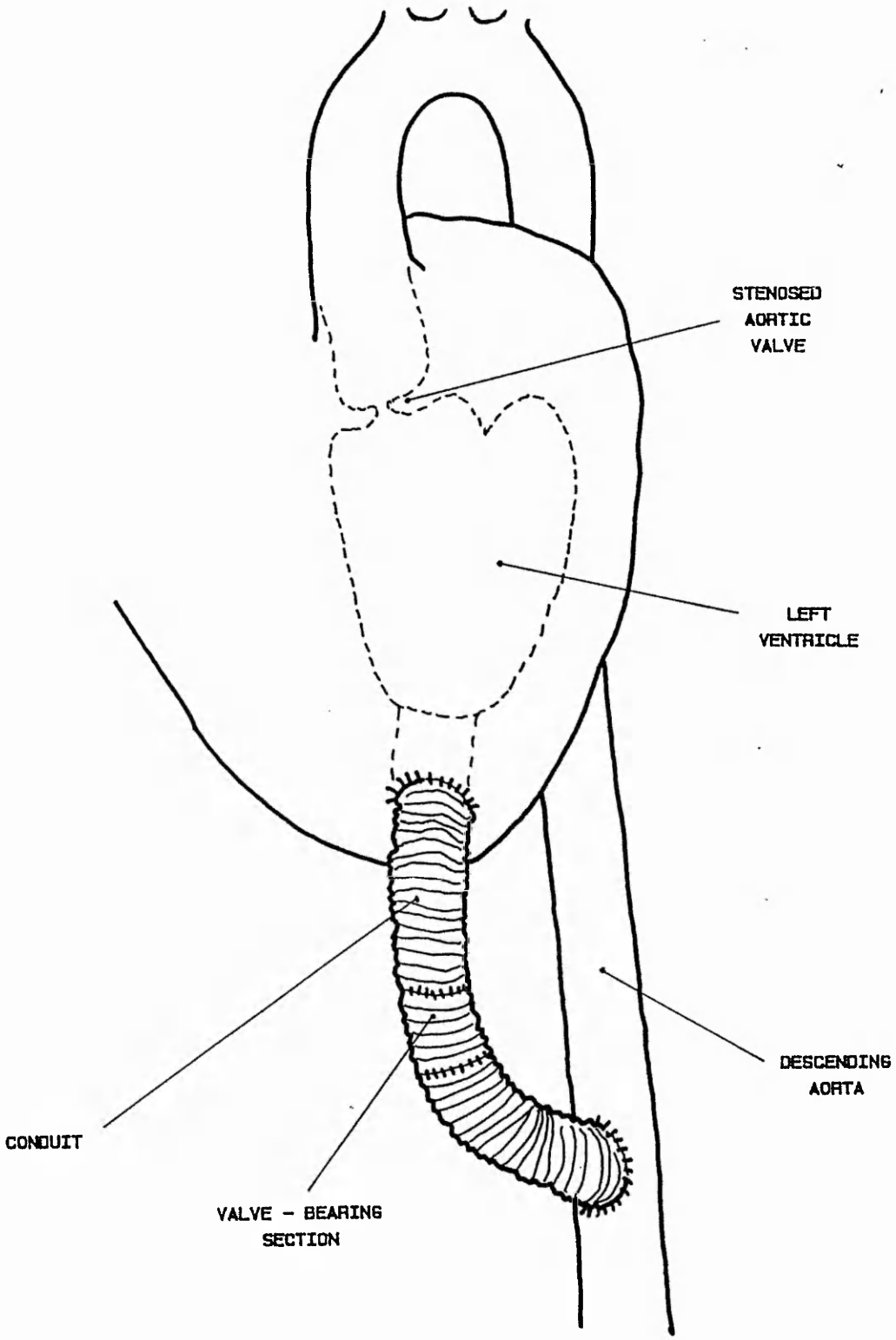


Fig. 1.3 Valved conduit as an apico-aortic shunt

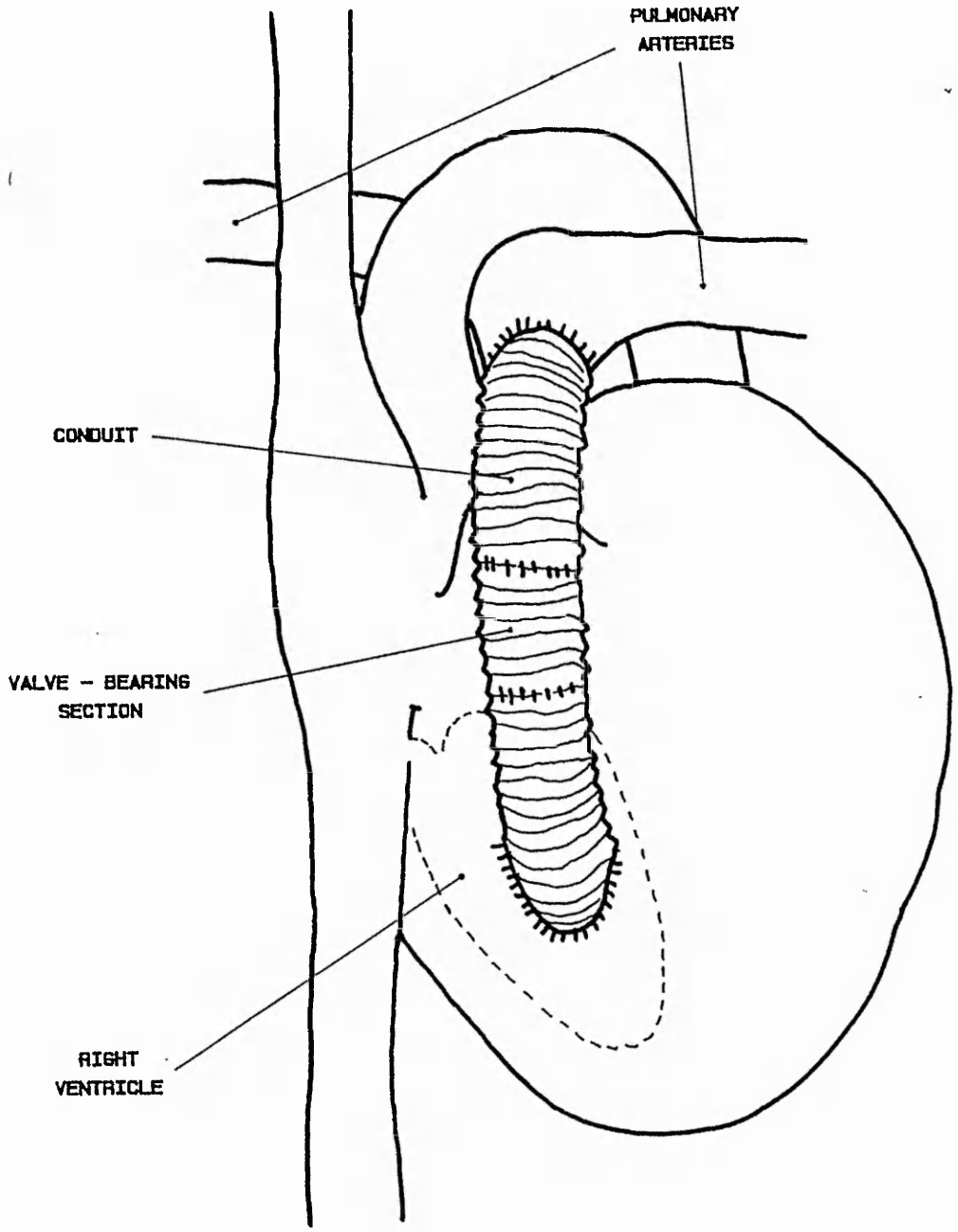


Fig. 1.4 Valved conduit used to establish flow from right ventricle to pulmonary arteries

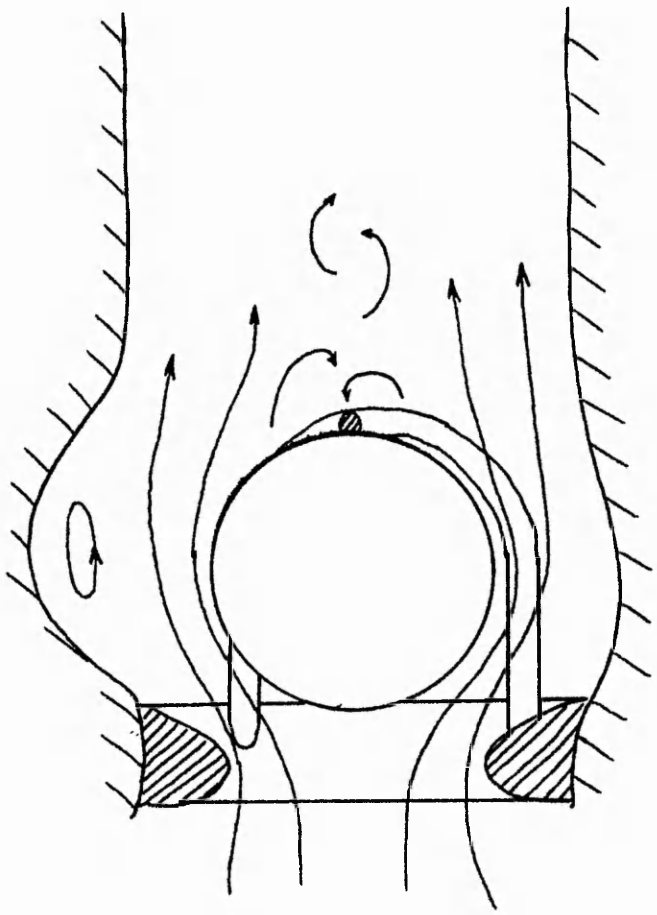


Fig. 2.1 Flow through a ball valve

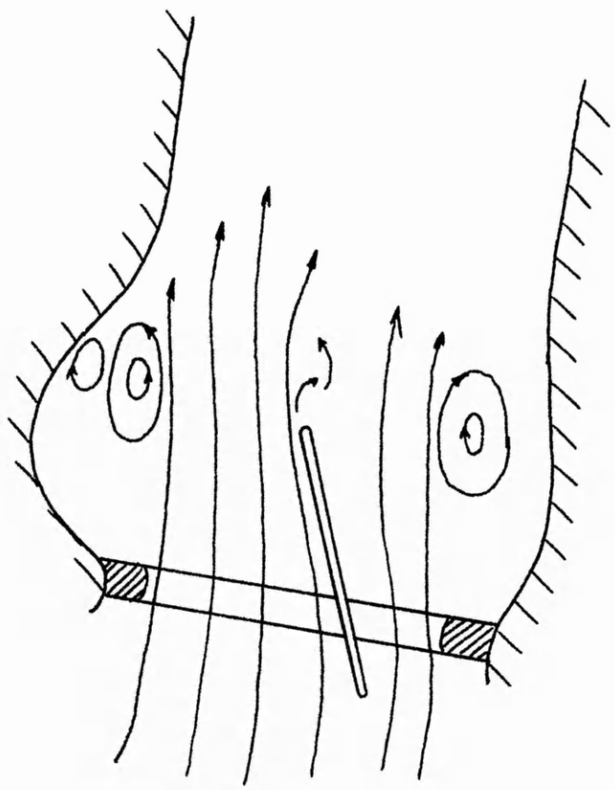


Fig. 2.2 Flow through a tilting disc valve

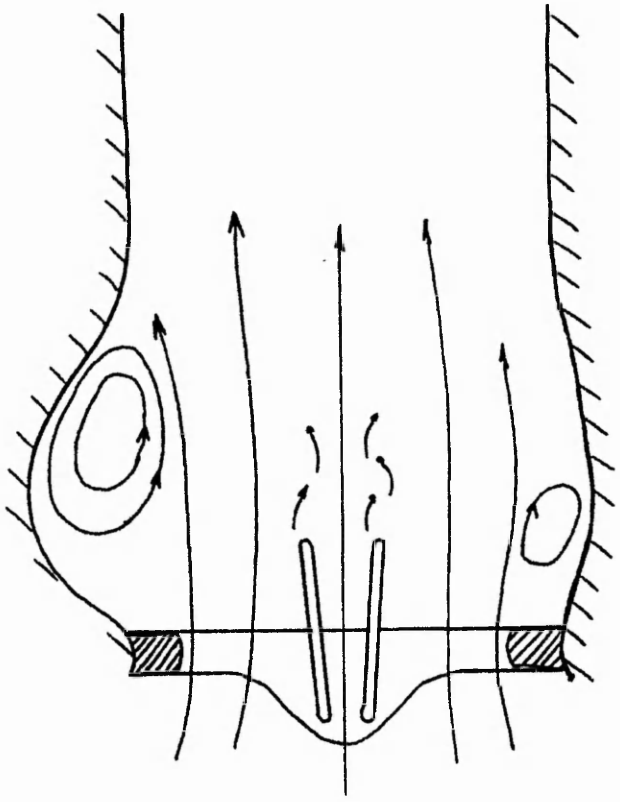


Fig. 2.3 Flow through a twin-flap valve

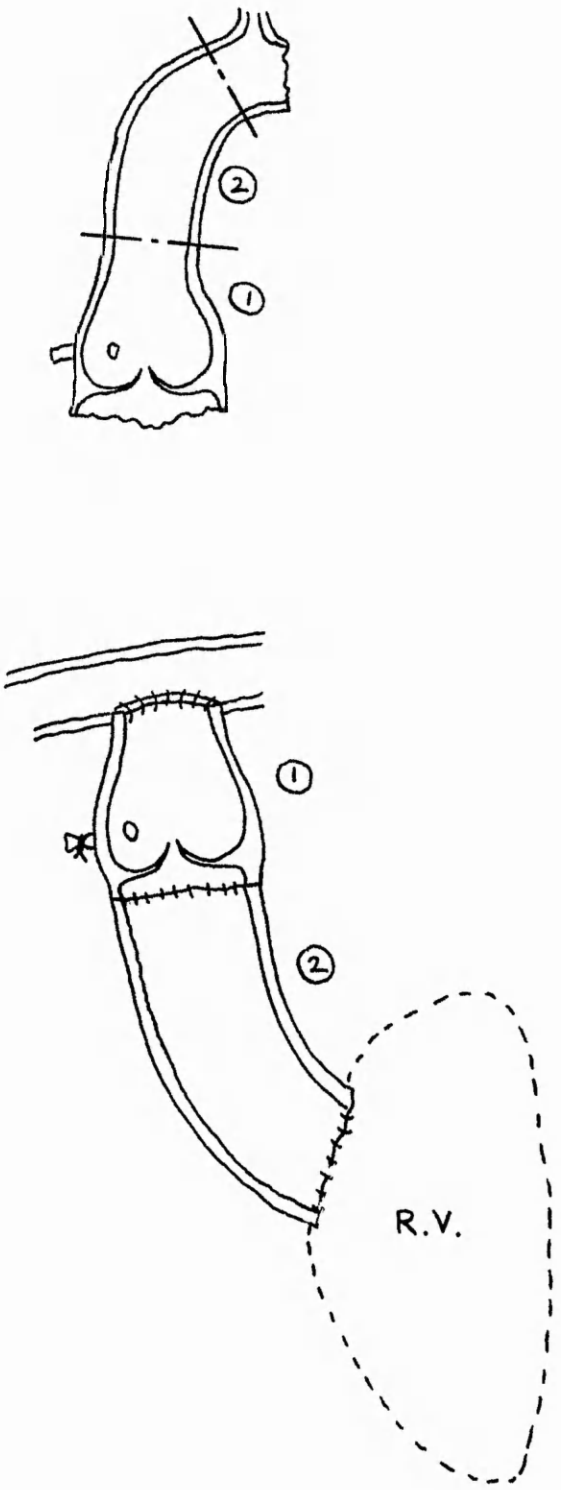


Fig. 2.4 Construction of an aortic homograft conduit with distal valve from an excised aortic valve with ascending aorta

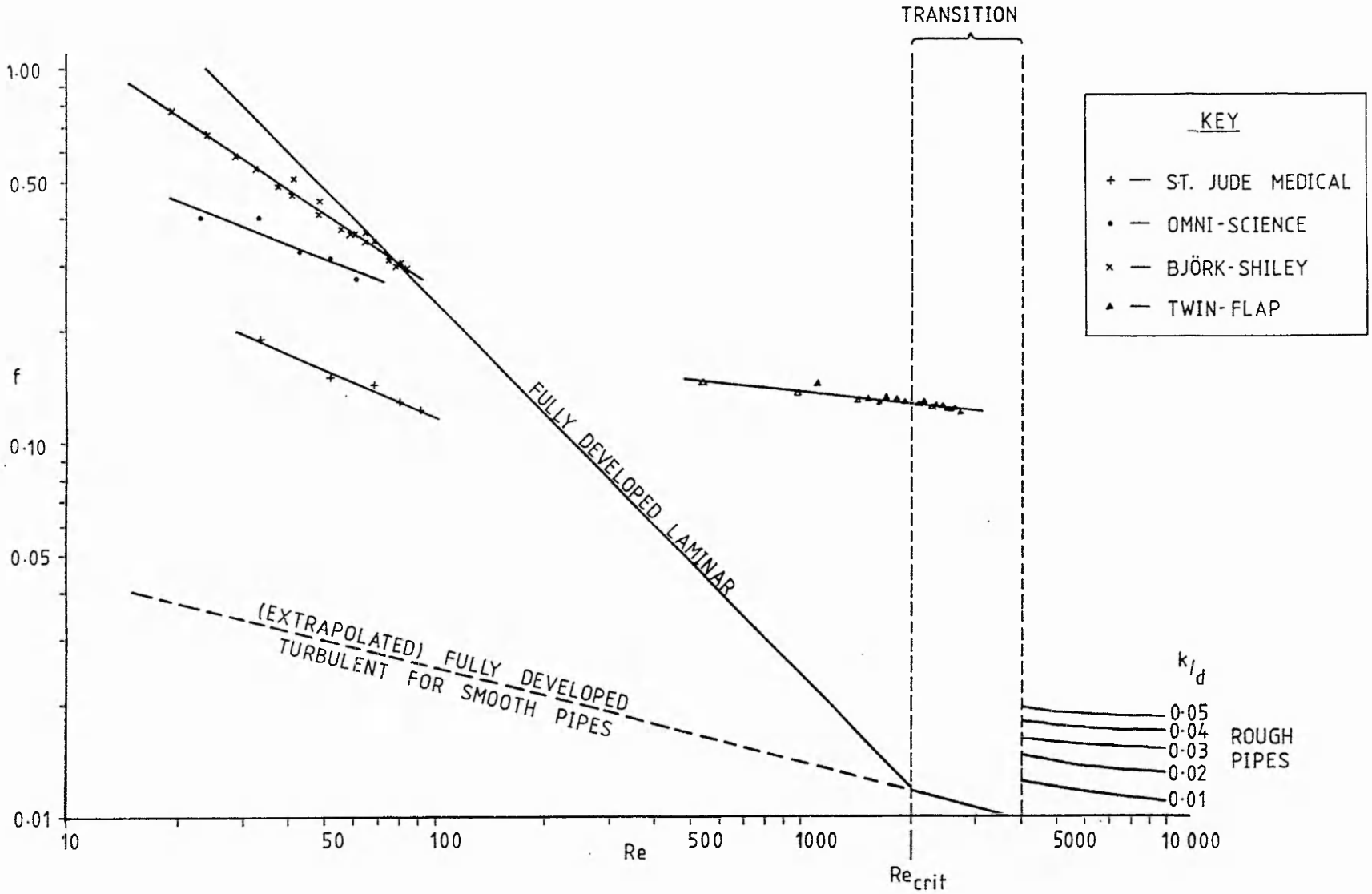


Fig. 3.1 Experimental leakage flow data superimposed on Moody chart (relationship between friction factor, f, and Reynolds number, Re) for fully-developed pipe flow

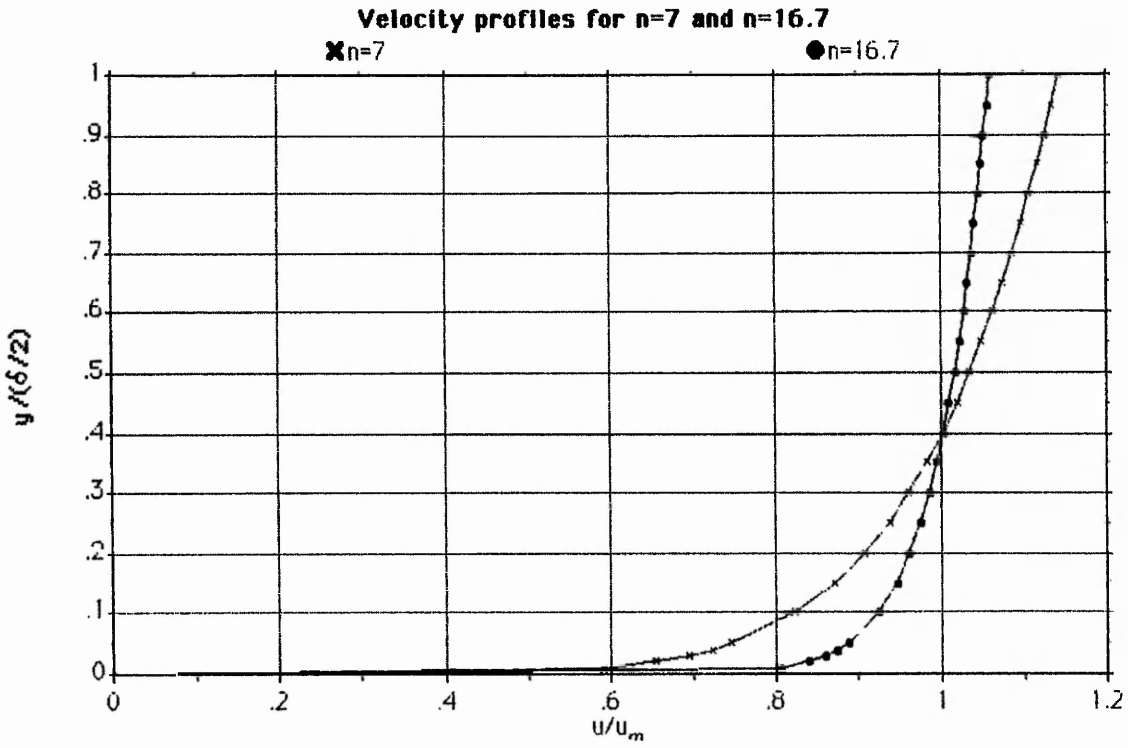
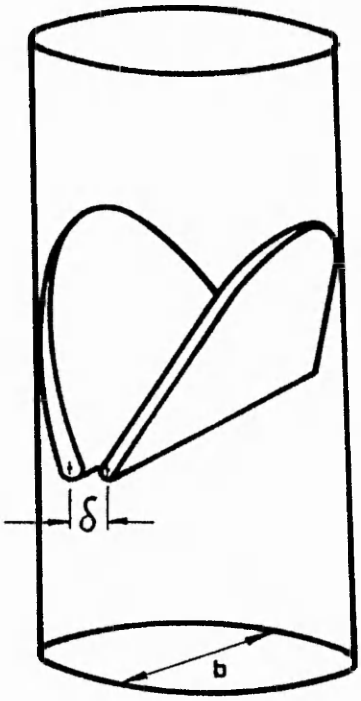


Fig. 3.2 Velocity profiles for n=7 and n=16.7



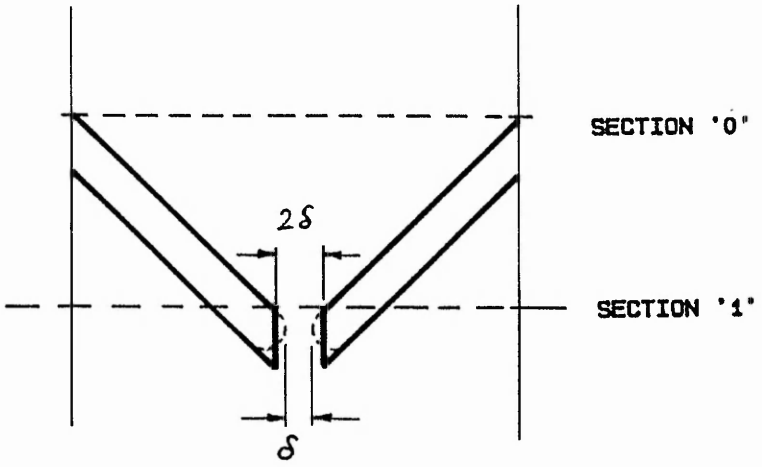


Fig. 3.3 Idealised representation of geometry for leakage analysis of twin-flap valve

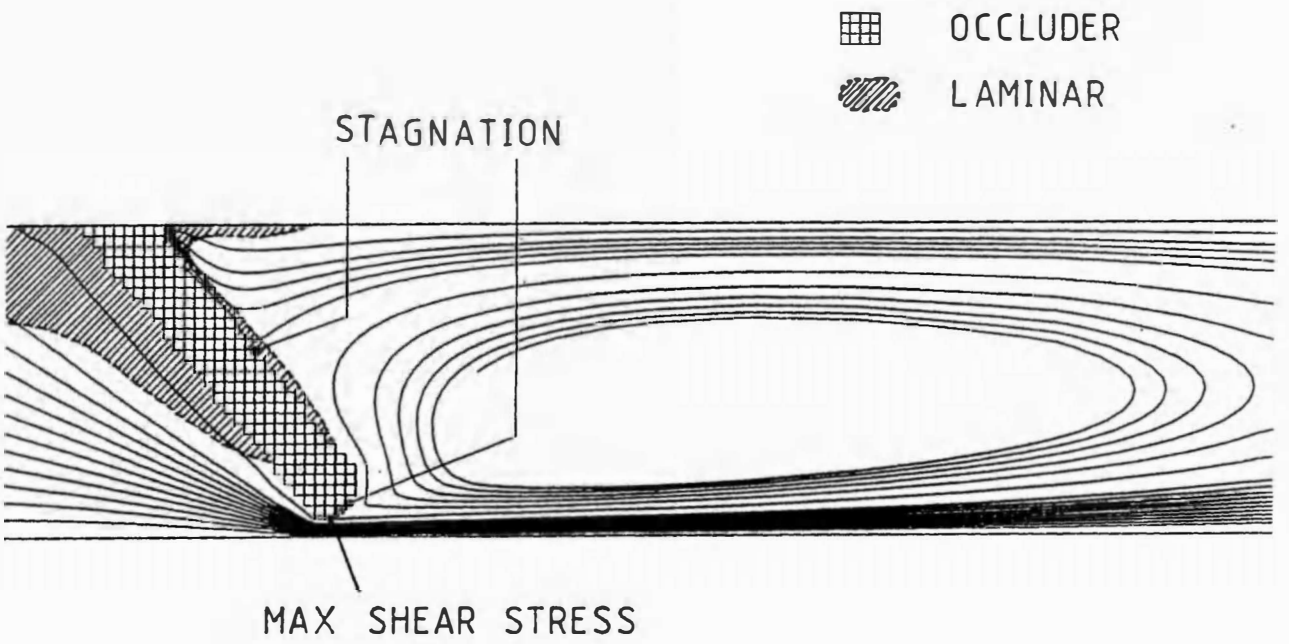


Fig. 3.4 Summary of numerical model results for the twin-flap valve

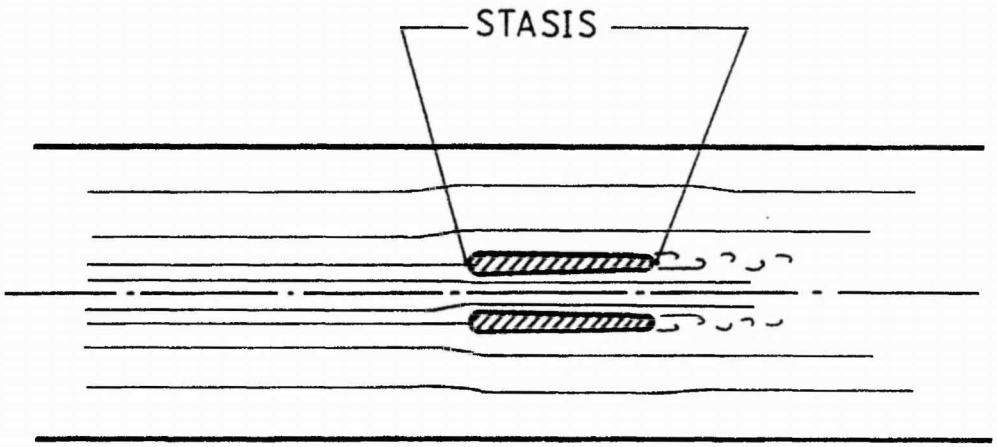


Fig. 3.5 Forward flow through the twin-flap valve

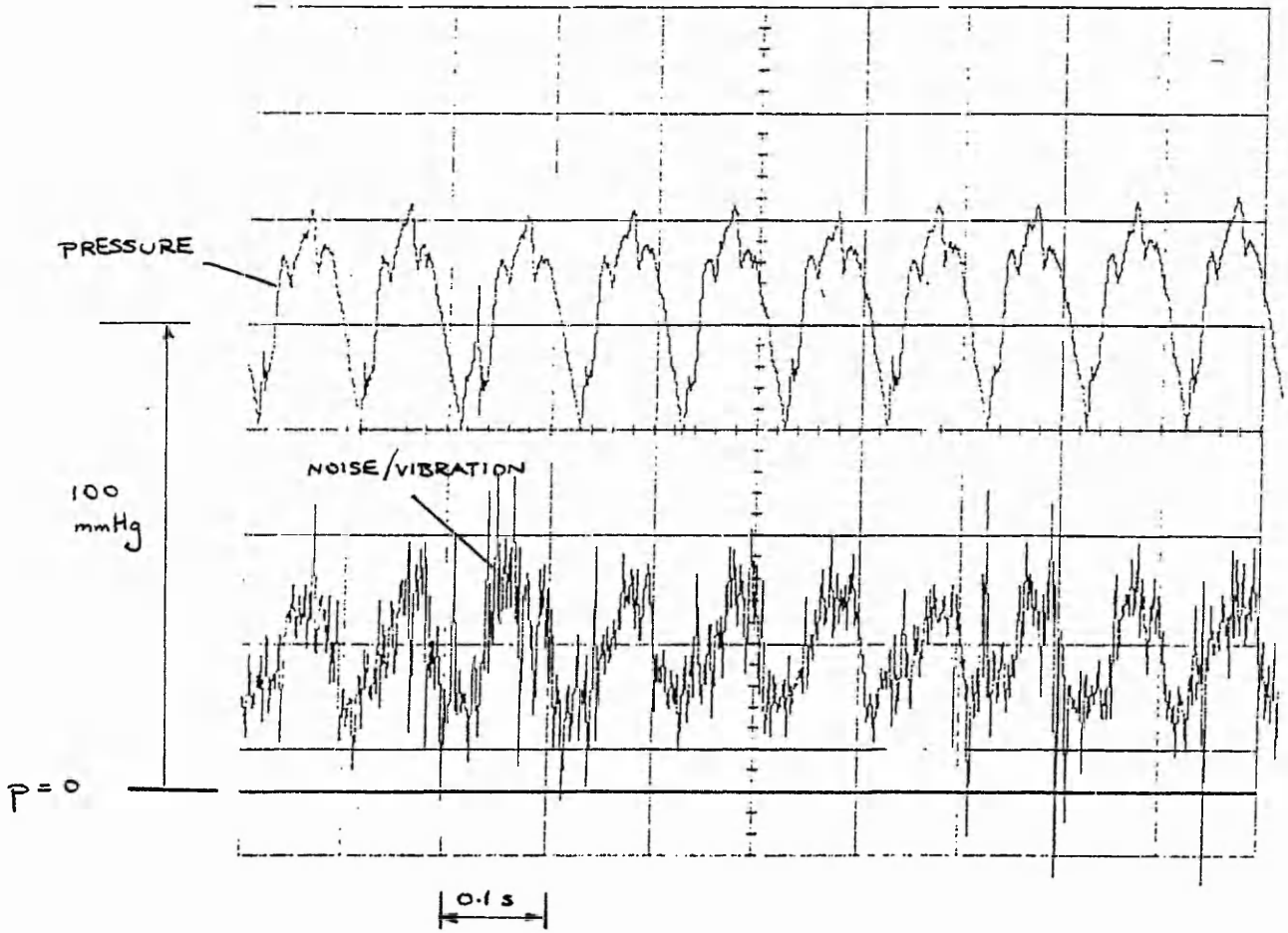


Fig. 3.6 Investigation of ball vibration - experimental set-up

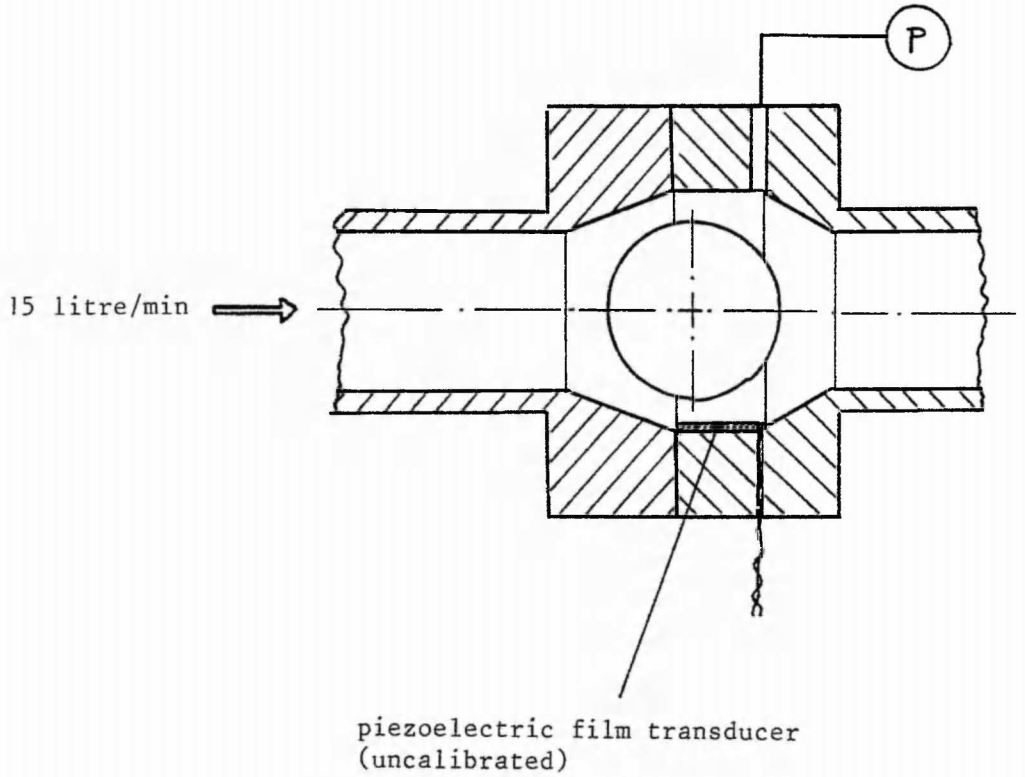


Fig. 3.7 Investigation of ball vibration - experimental results

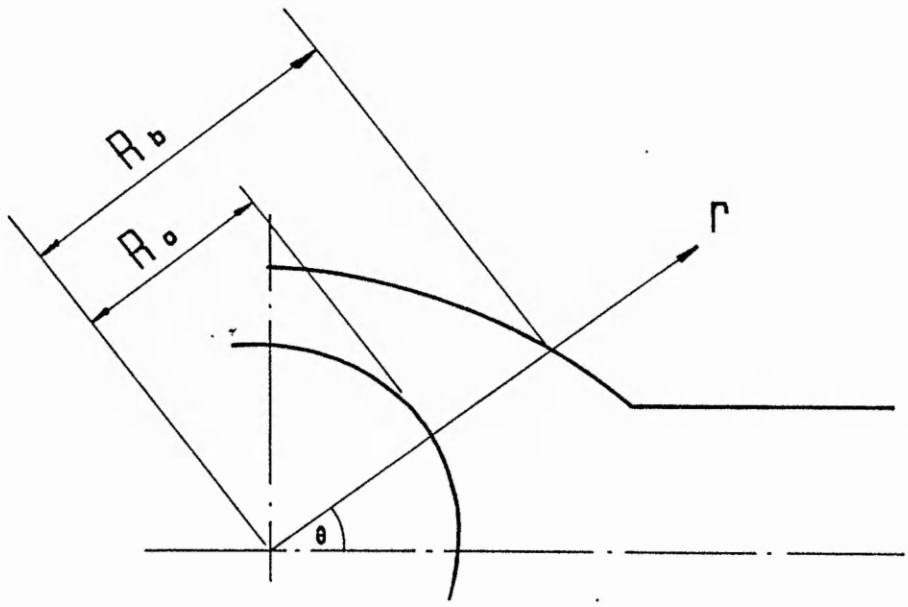


Fig. 3.8 Stability design of valve outlet section of prototype conduit

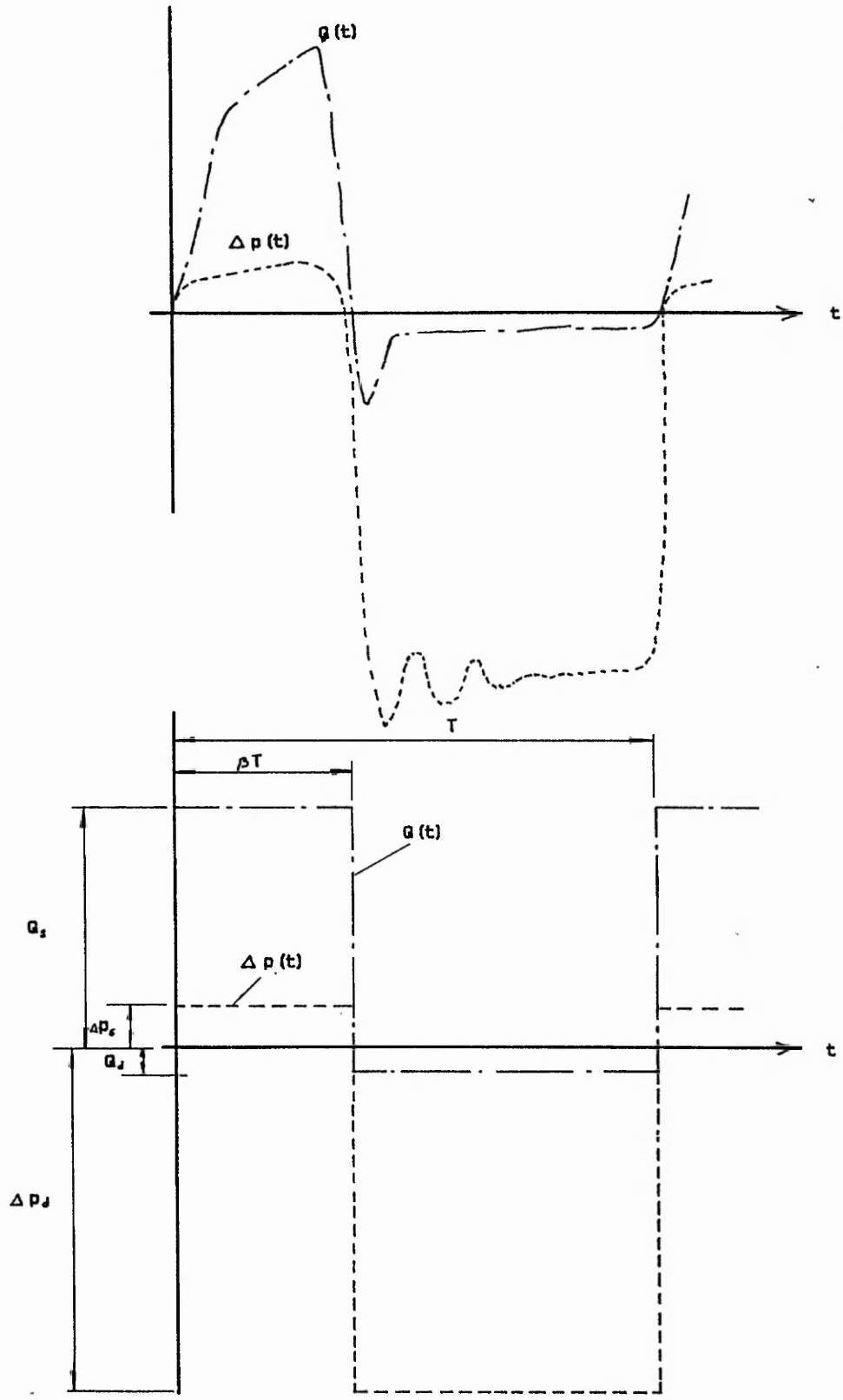


Fig. 4.1 Idealised representation of pressure drop and net flowrate over a cardiac cycle

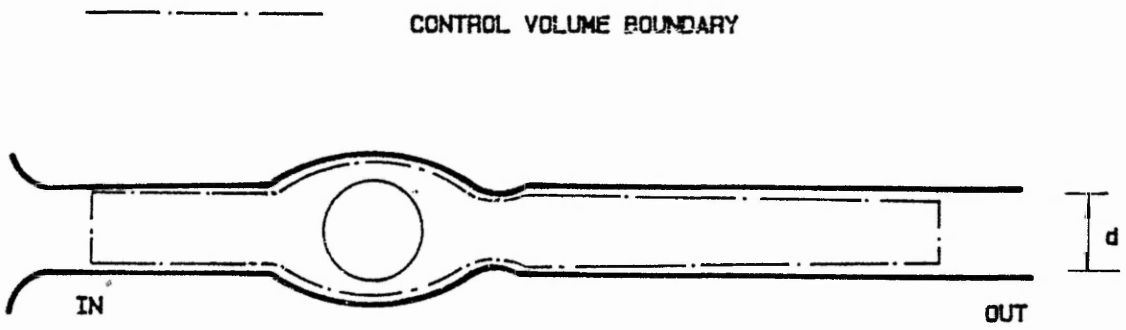


Fig. 4.2 Control volume for testing of conduit on its own

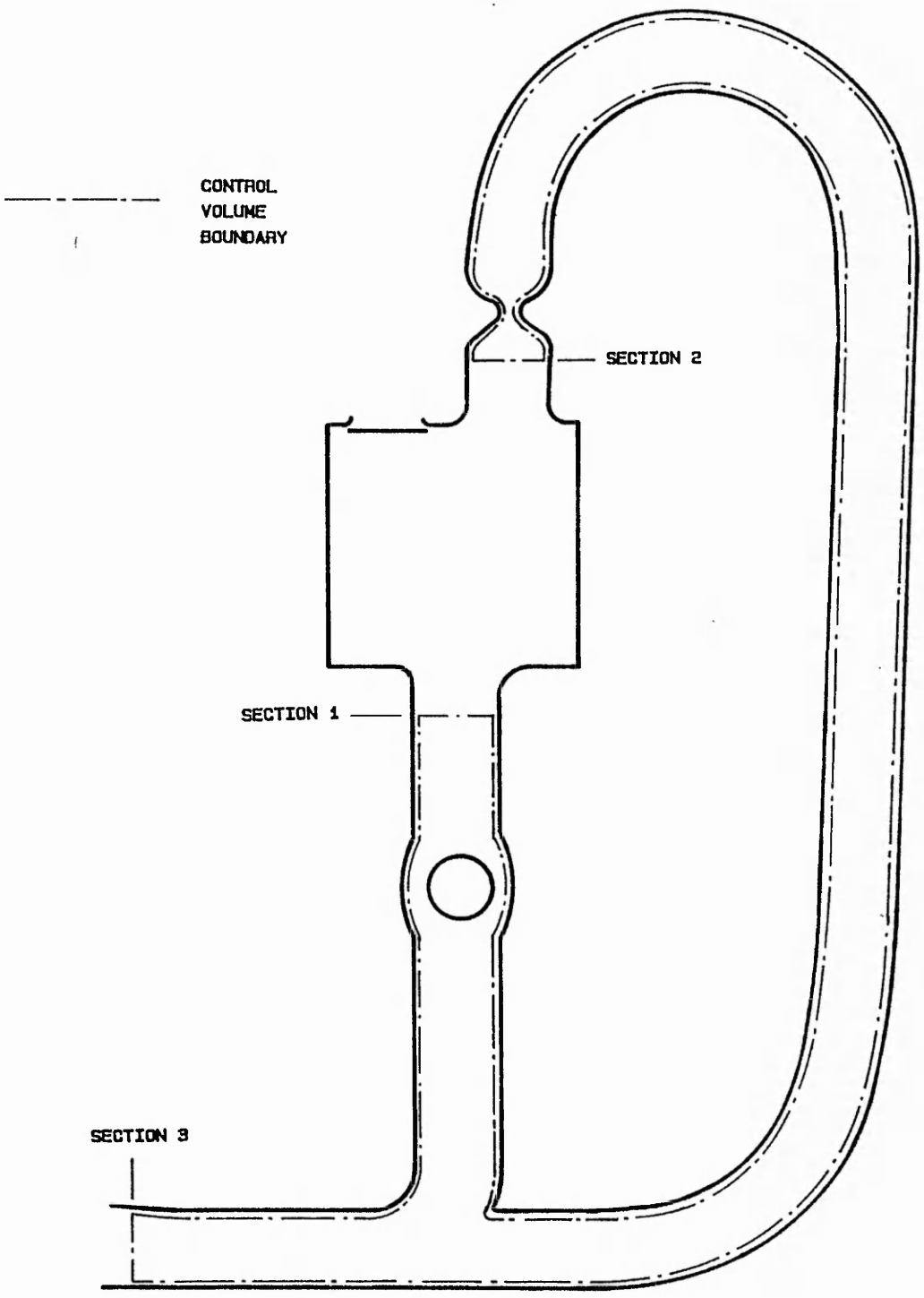


Fig. 4.3 Control volume for testing of conduit as a second ventricular outflow

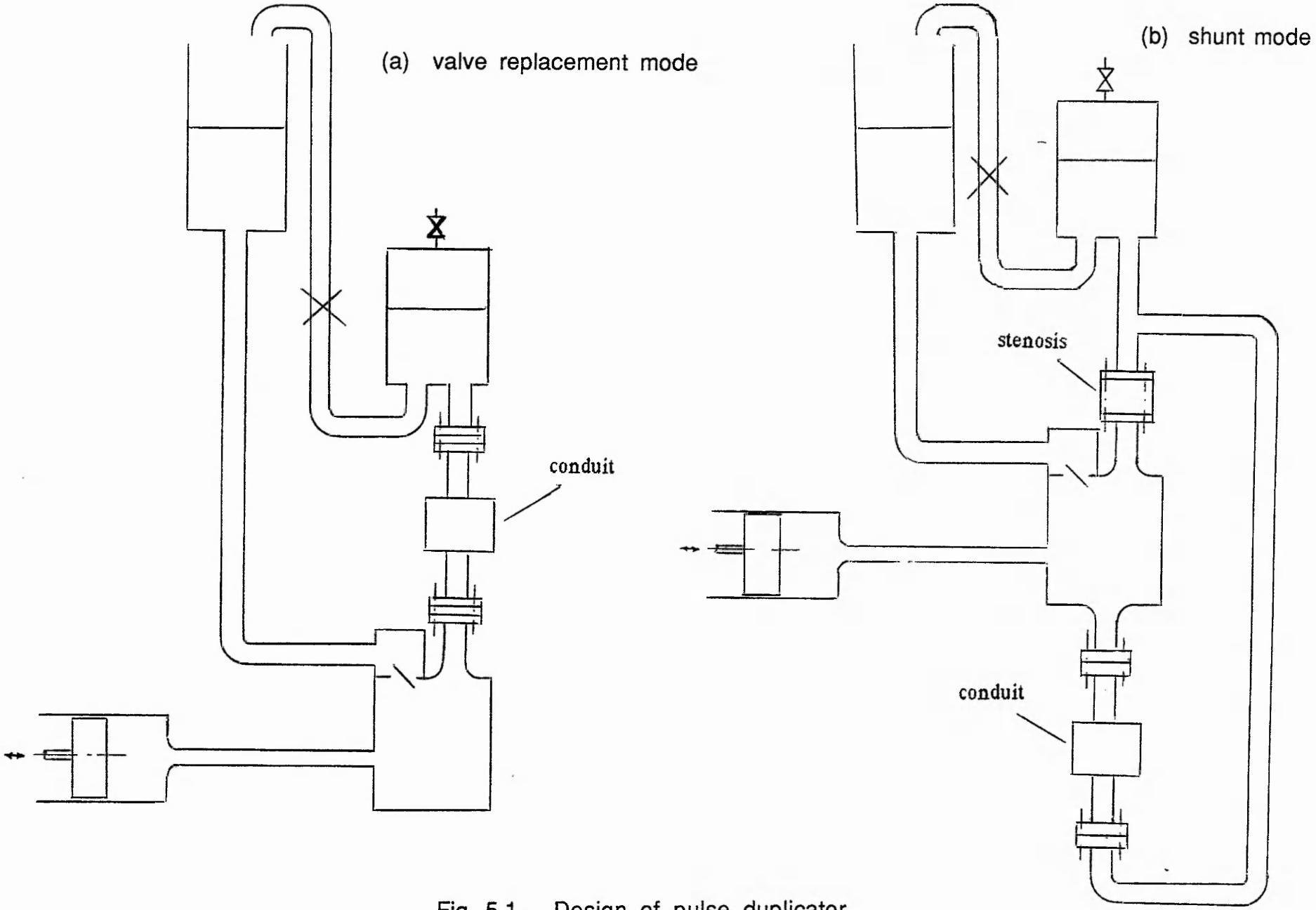
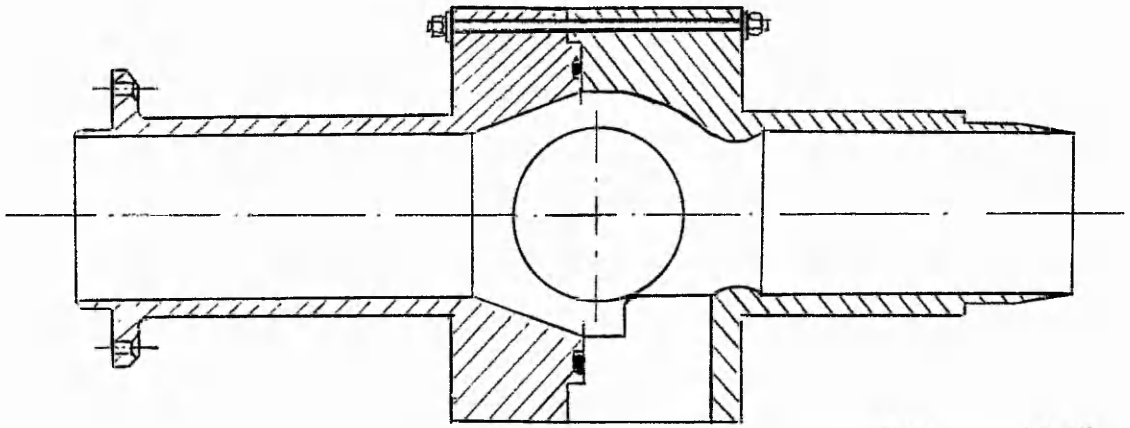


Fig. 5.1 Design of pulse duplicator

Rigid version in acrylic for in vitro testing

(Restraining strut shown at lower section only)



As envisaged for surgical application

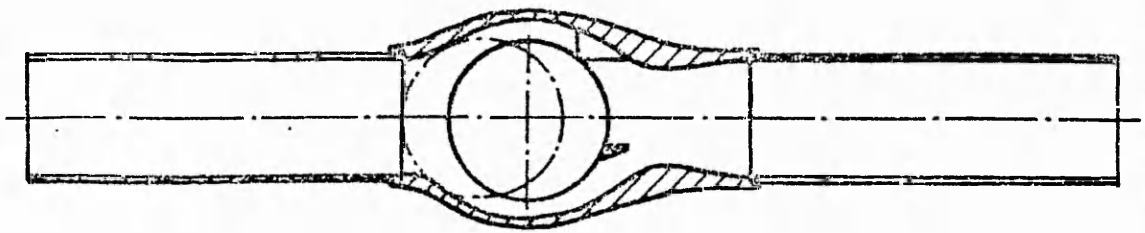


Fig. 5.2 Ball valve prototype conduit

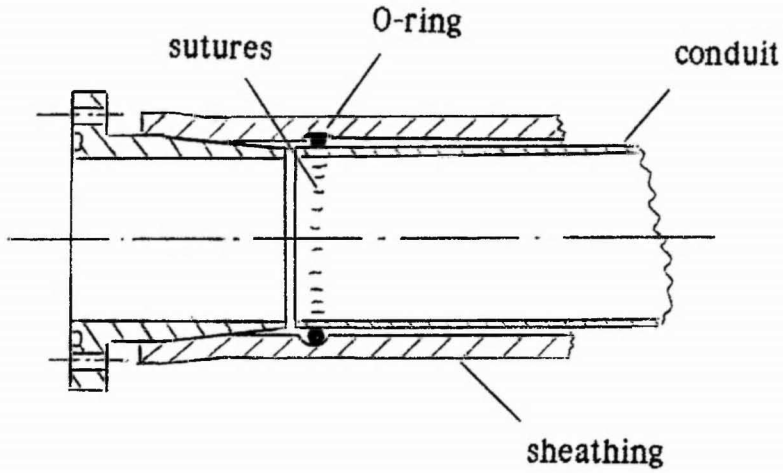


Fig. 5.3 Attachment of conduit into circuit

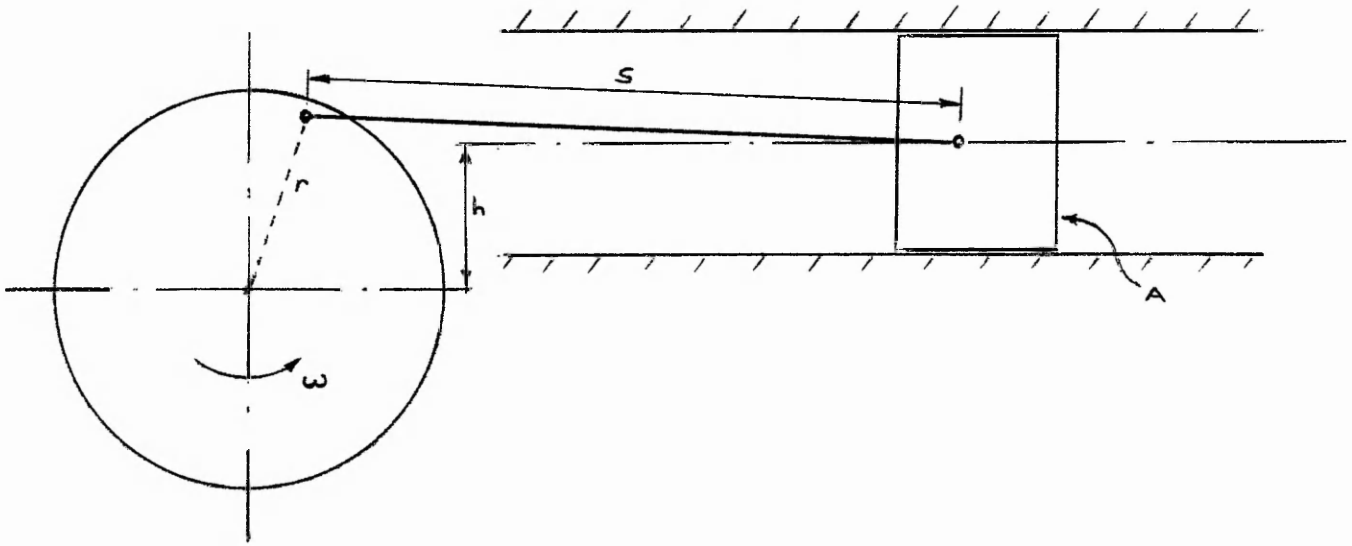


Fig. 5.4 Drive mechanism

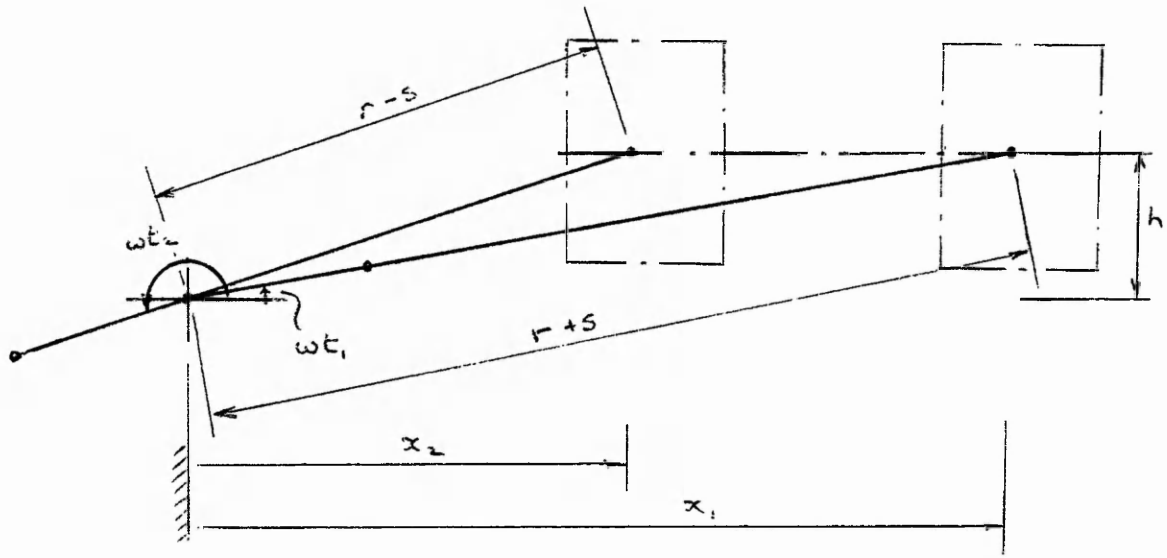


Fig. 5.5 Schematic of drive mechanism at full extents

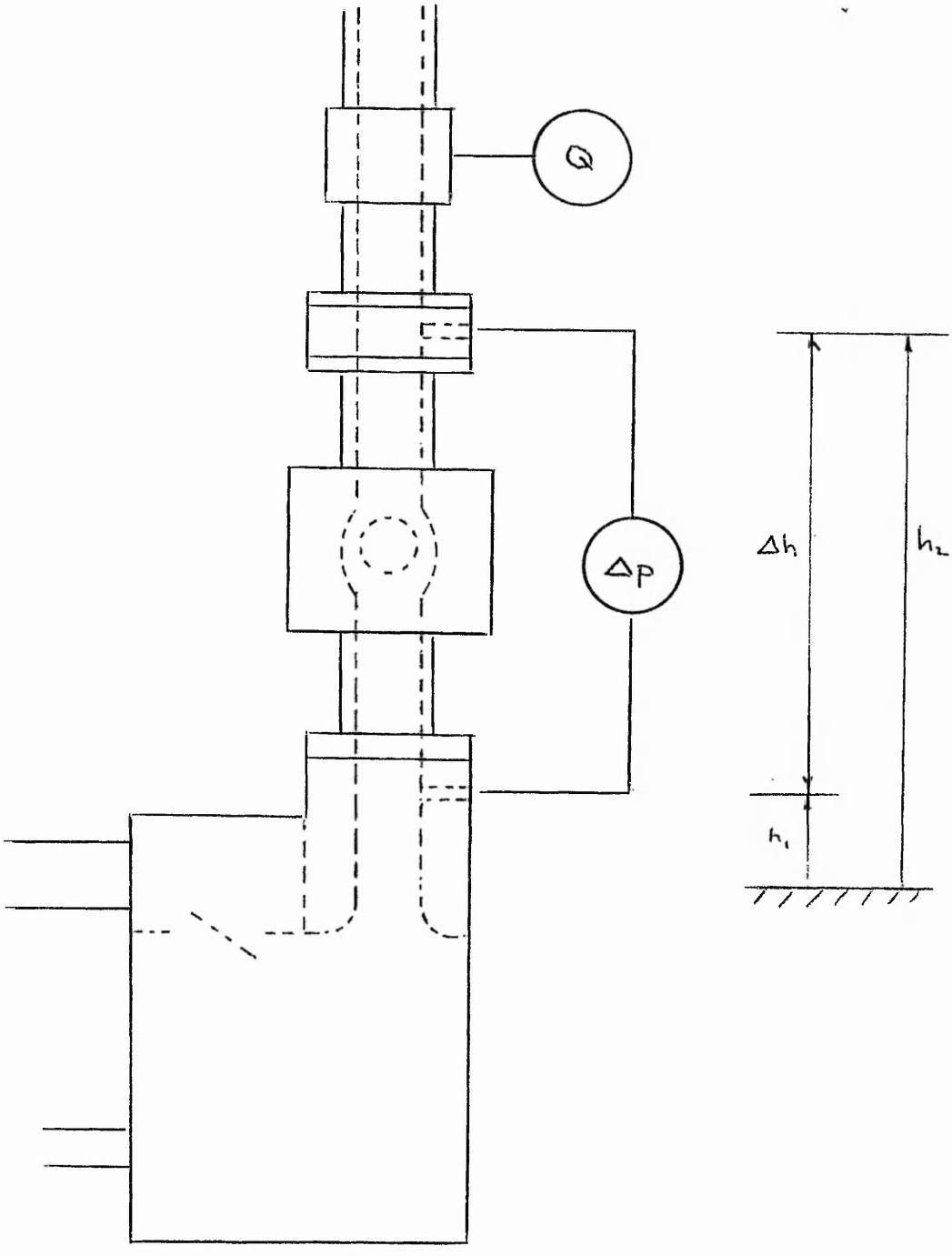


Fig. 5.6 Instrumentation set-up for conduit as valve replacement

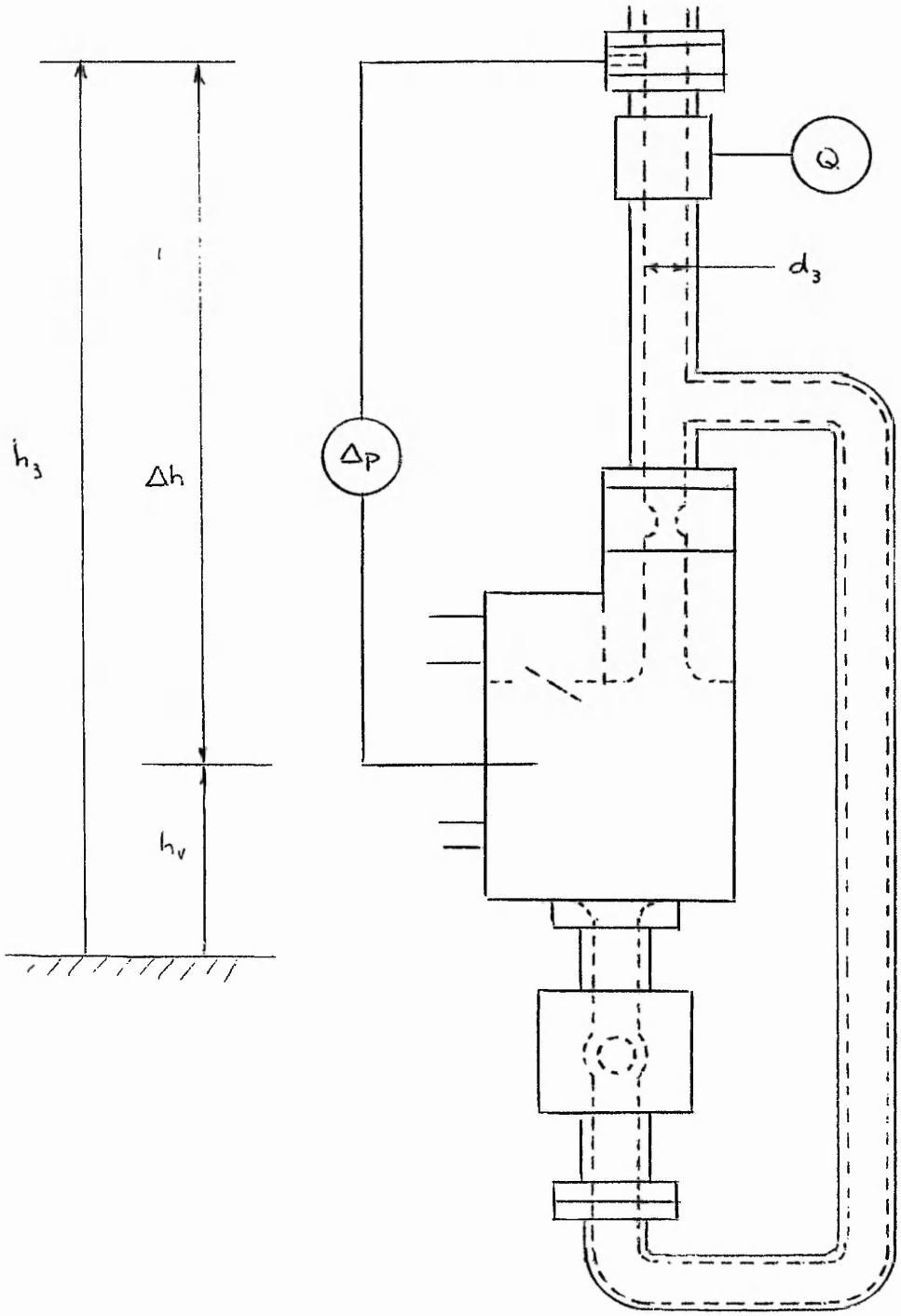


Fig. 5.7 Instrumentation set-up for conduit as a shunt

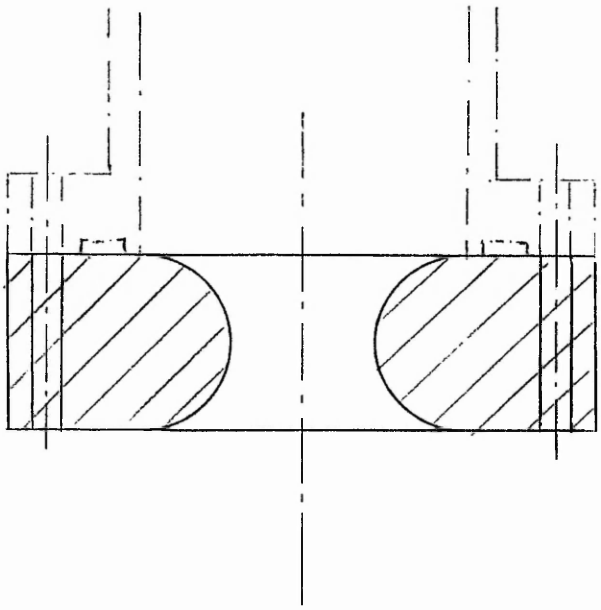


Fig. 5.8 Model of stenosis

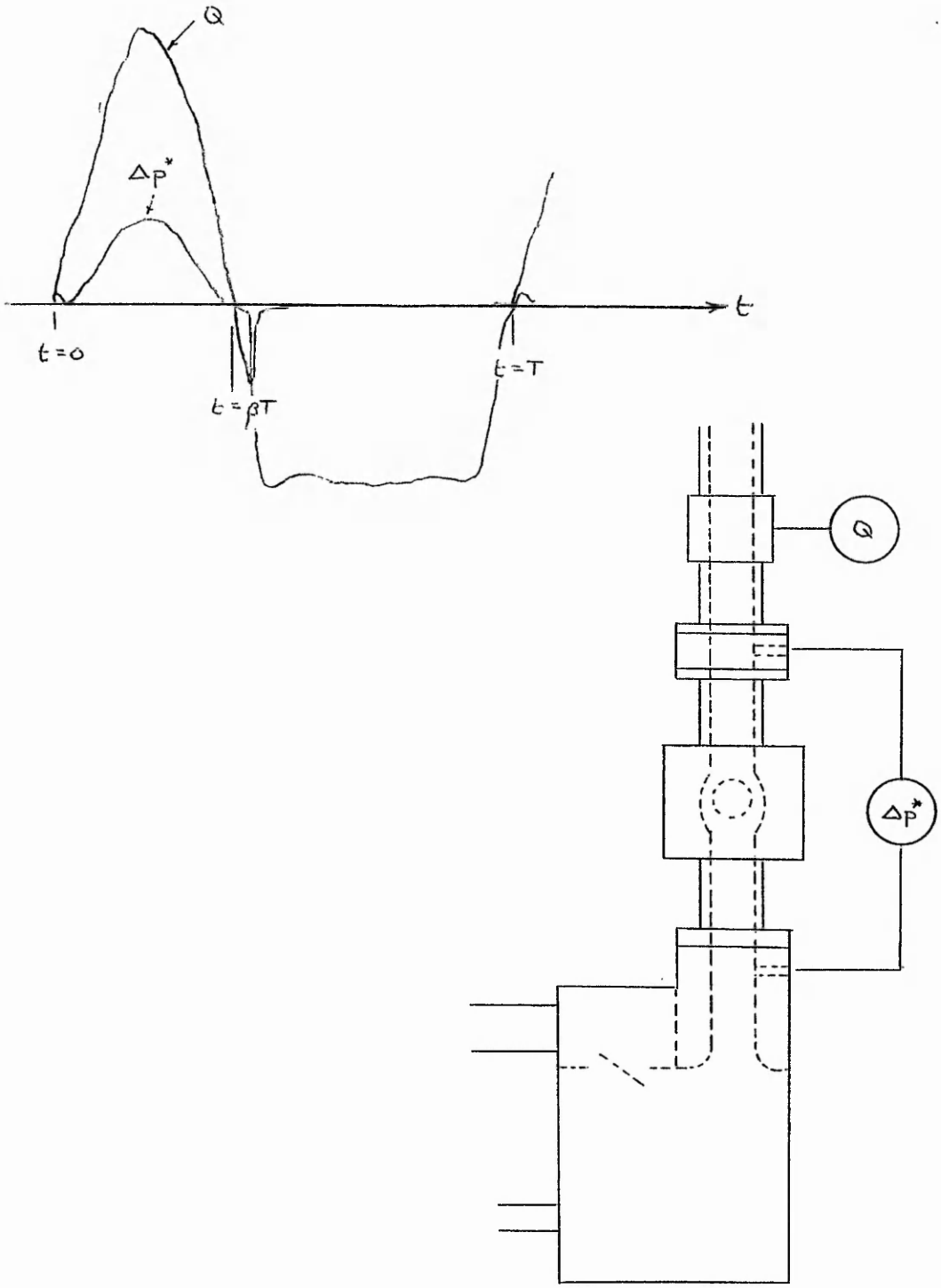
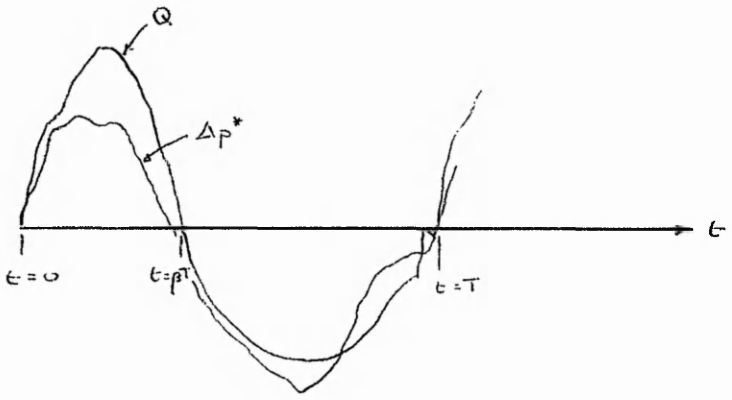


Fig. 5.9 Nomenclature for test measurements with conduit as valve replacement



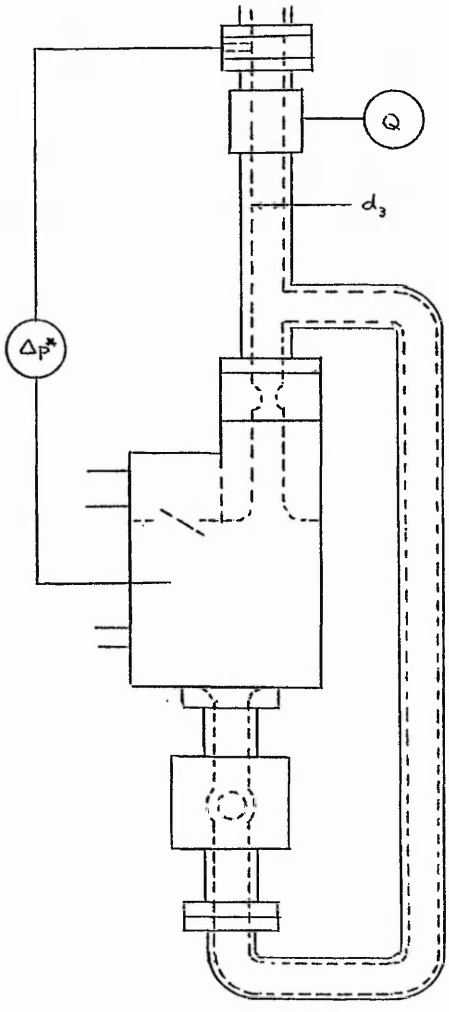


Fig. 5.10 Nomenclature for test measurements with conduit as a shunt

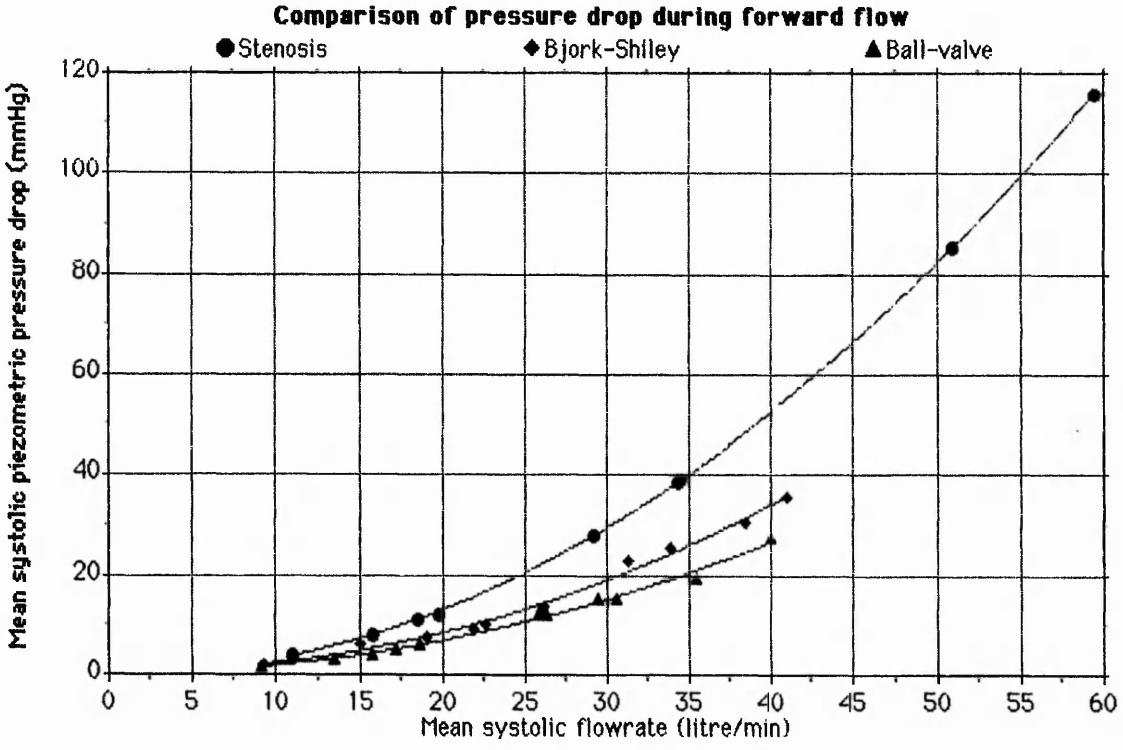


Fig. 6.1 Mean systolic pressure drop versus mean systolic flowrate for uncorrected stenosis and conduits as valve replacements

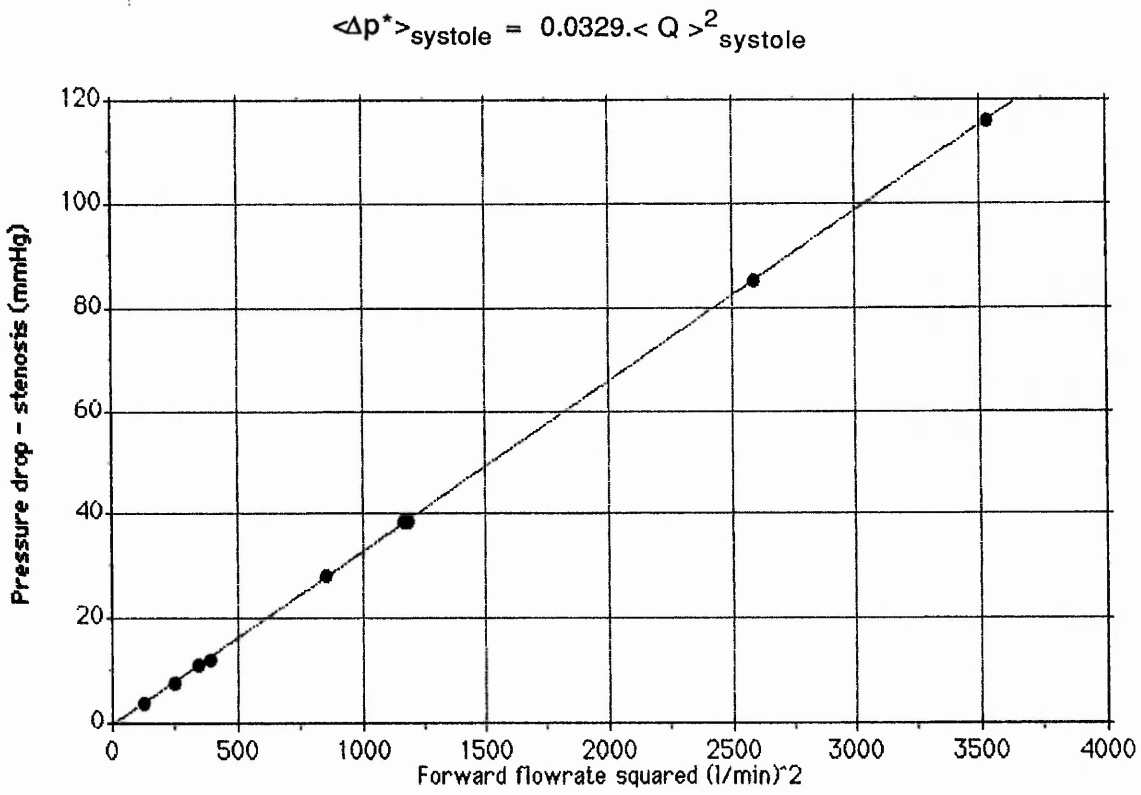


Fig. 6.2 Mean systolic pressure drop versus square of mean systolic flowrate for uncorrected stenosis

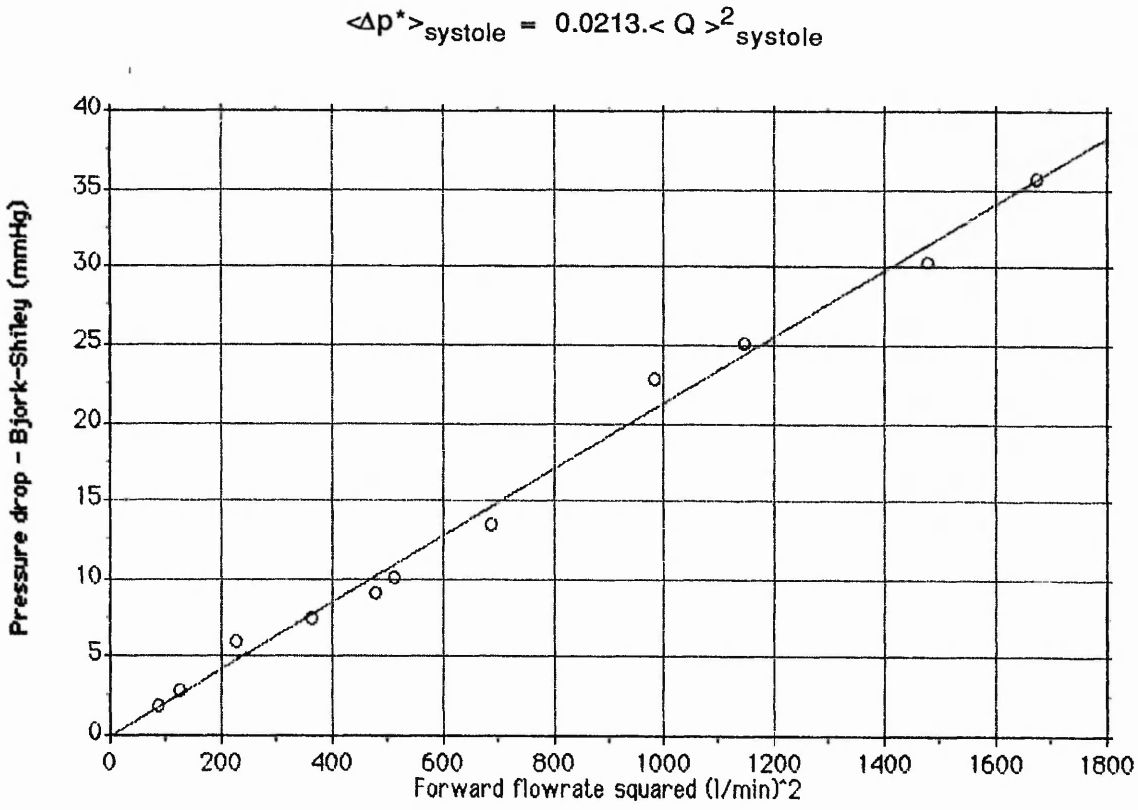


Fig. 6.3 Mean systolic pressure drop versus square of mean systolic flowrate for Bjork-Shiley conduit as valve replacement

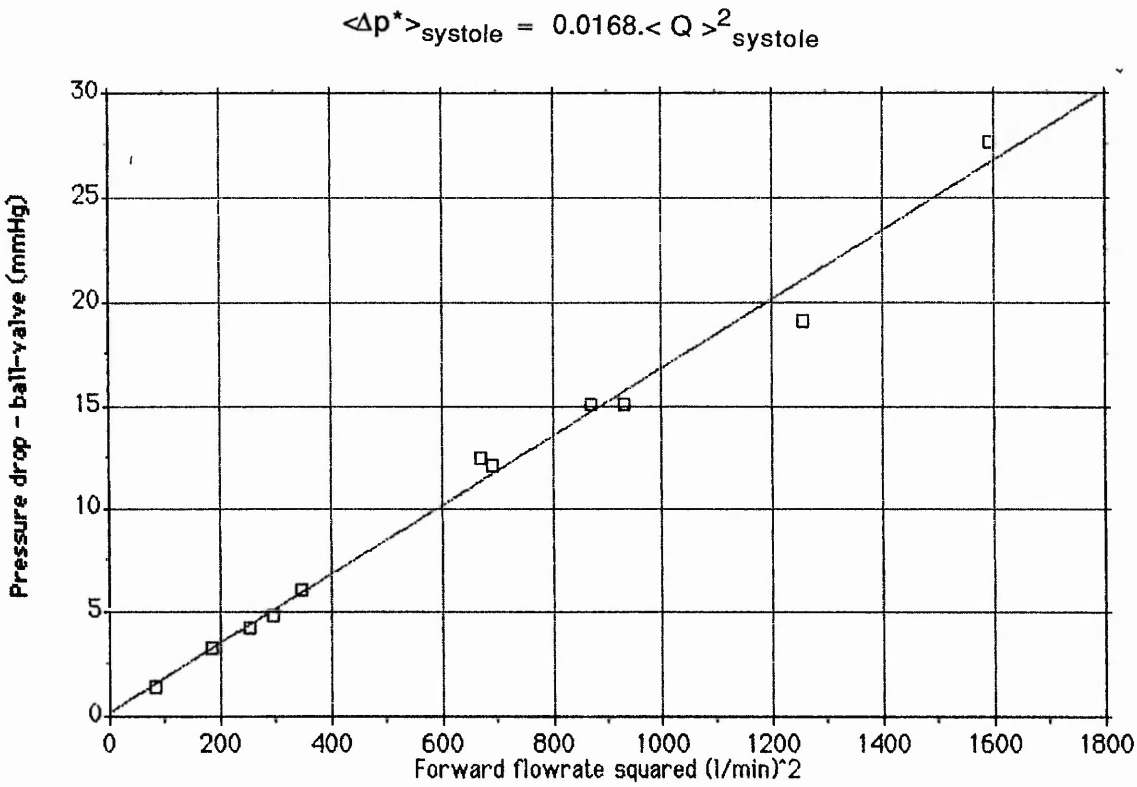


Fig. 6.4 Mean systolic pressure drop versus square of mean systolic flowrate for ball valve conduit as valve replacement

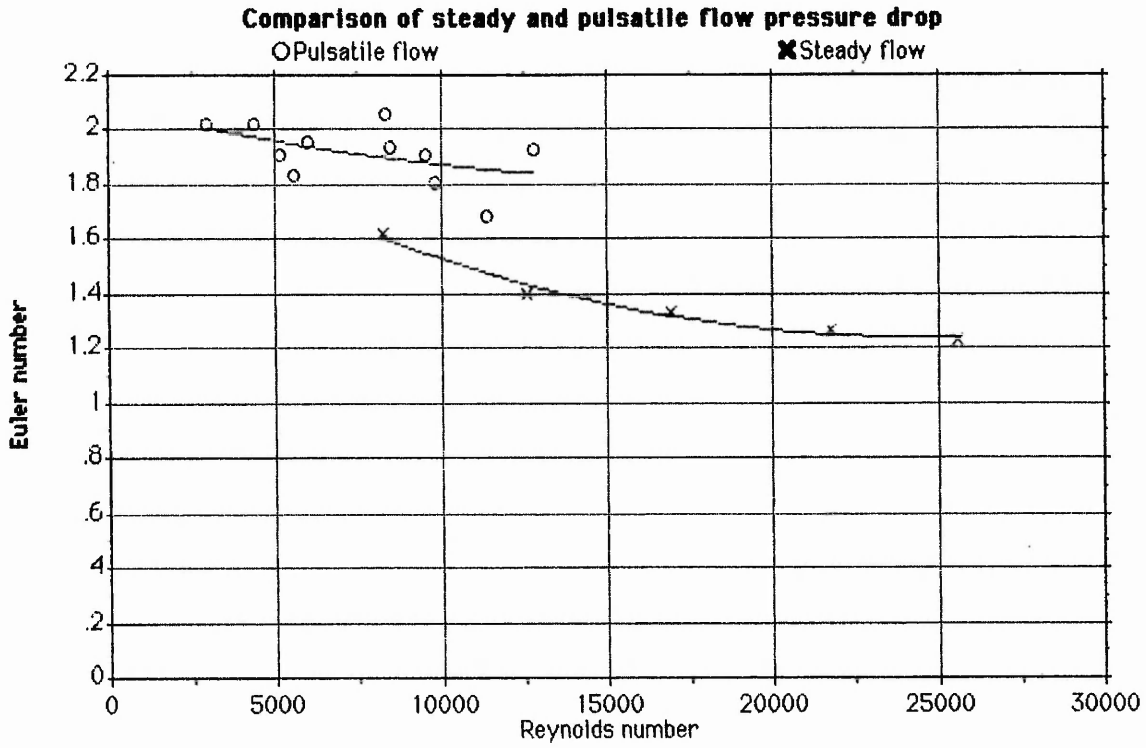


Fig. 6.5 Comparison of forward flow pressure drop for pulsatile and steady flow through ball valve prototype

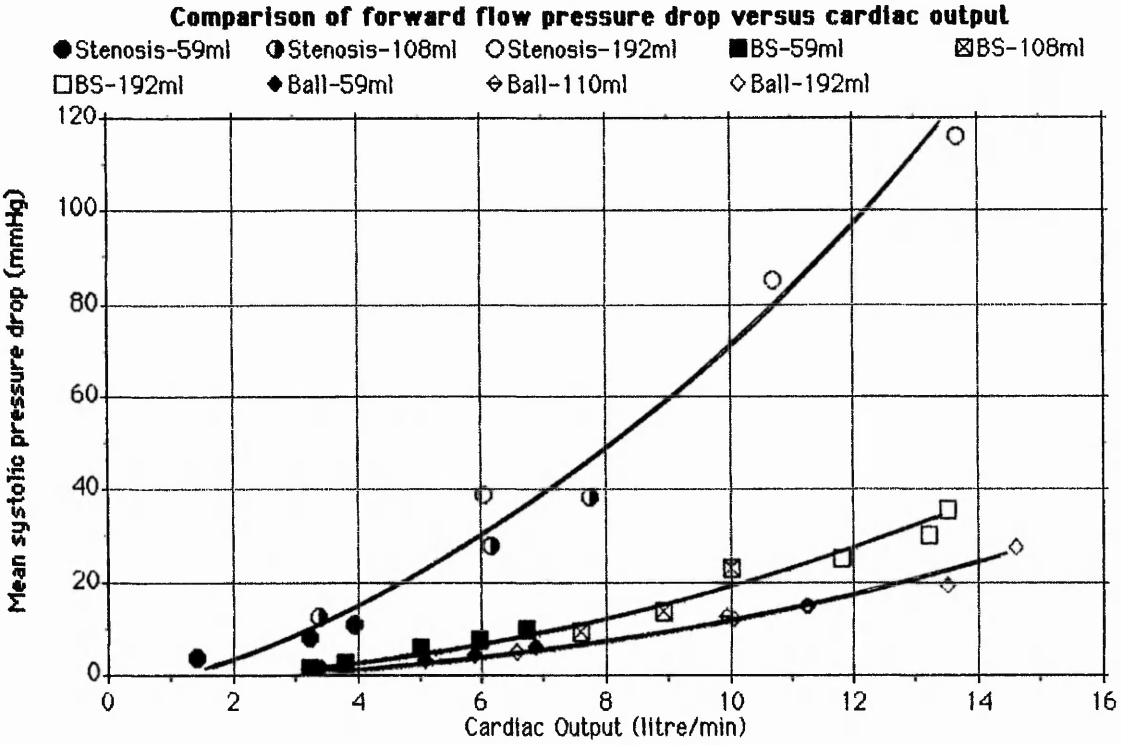


Fig. 6.6 Mean systolic pressure drop versus cardiac output for uncorrected stenosis and conduits as valve replacements

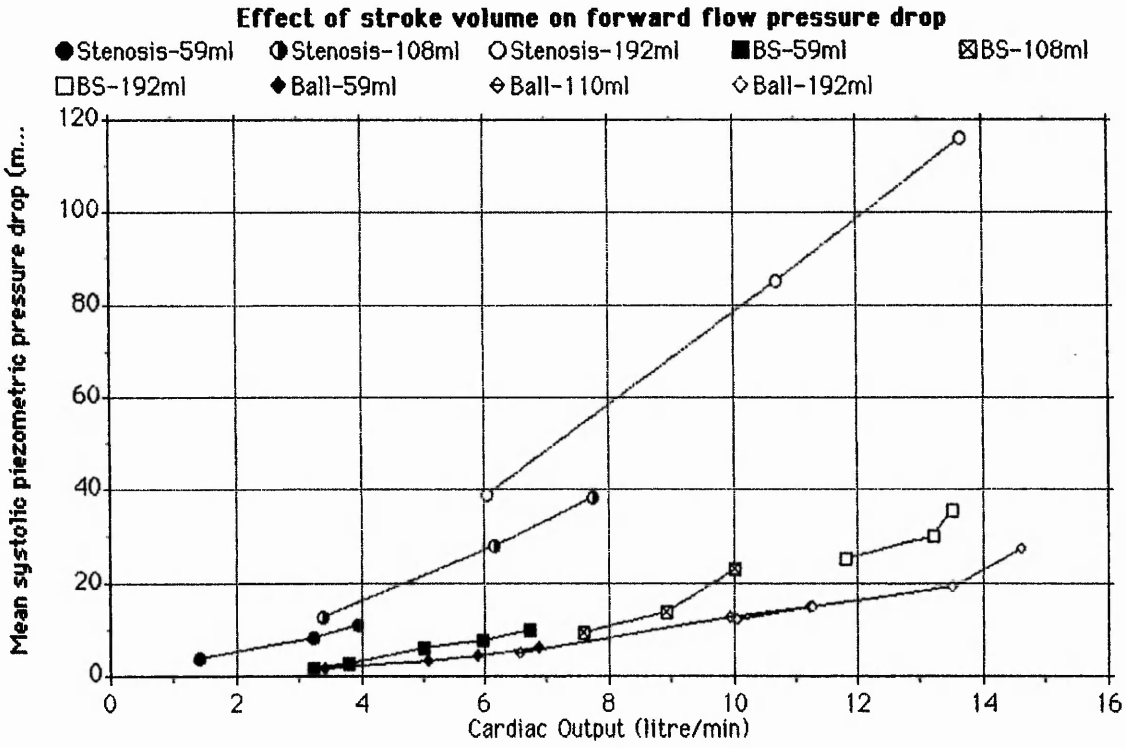


Fig. 6.7 Effect of stroke volume on forward flow pressure drop for uncorrected stenosis and conduits as valve replacements

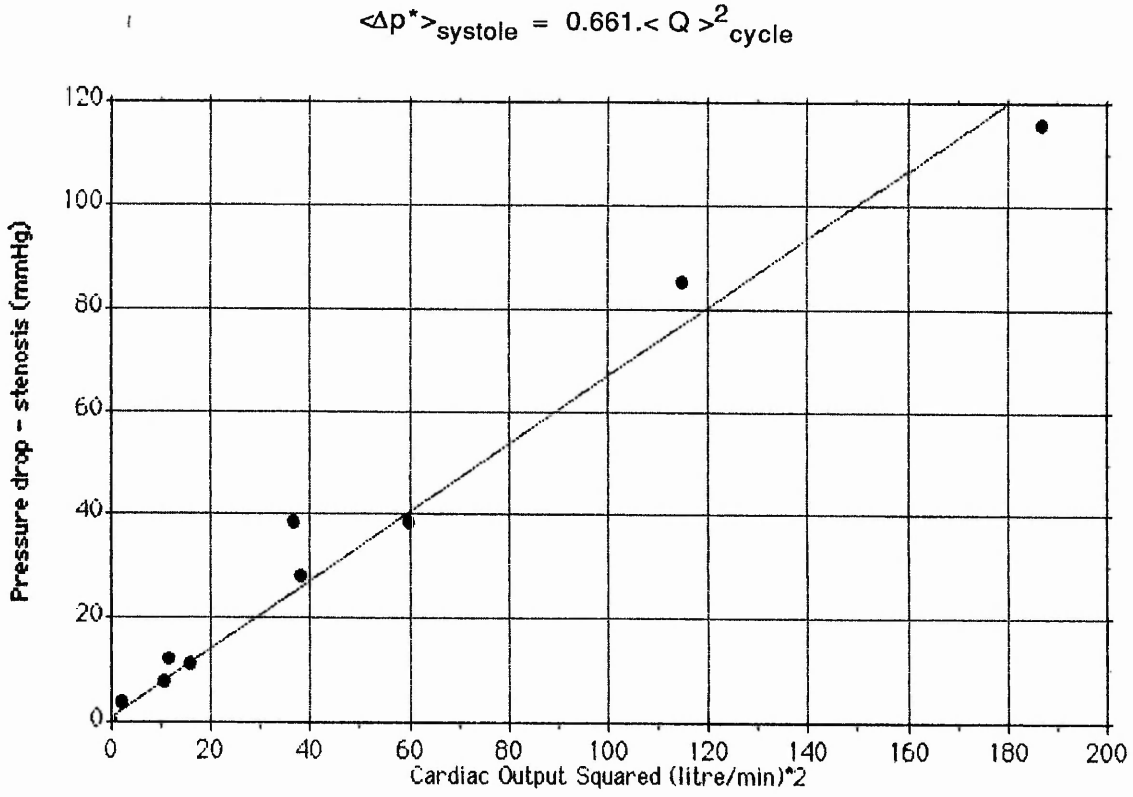


Fig. 6.8 Mean systolic pressure drop versus square of cardiac output for uncorrected stenosis

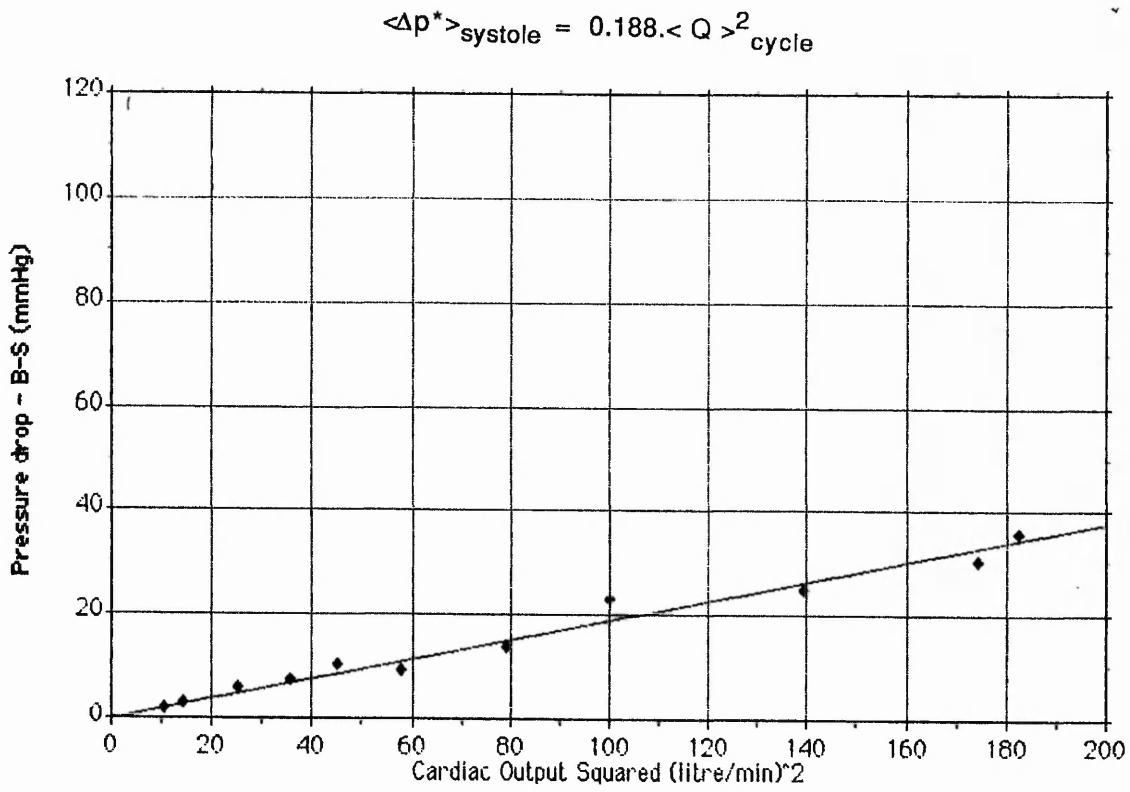


Fig. 6.9 Mean systolic pressure drop versus square of cardiac output for Bjork-Shiley conduit as valve replacement

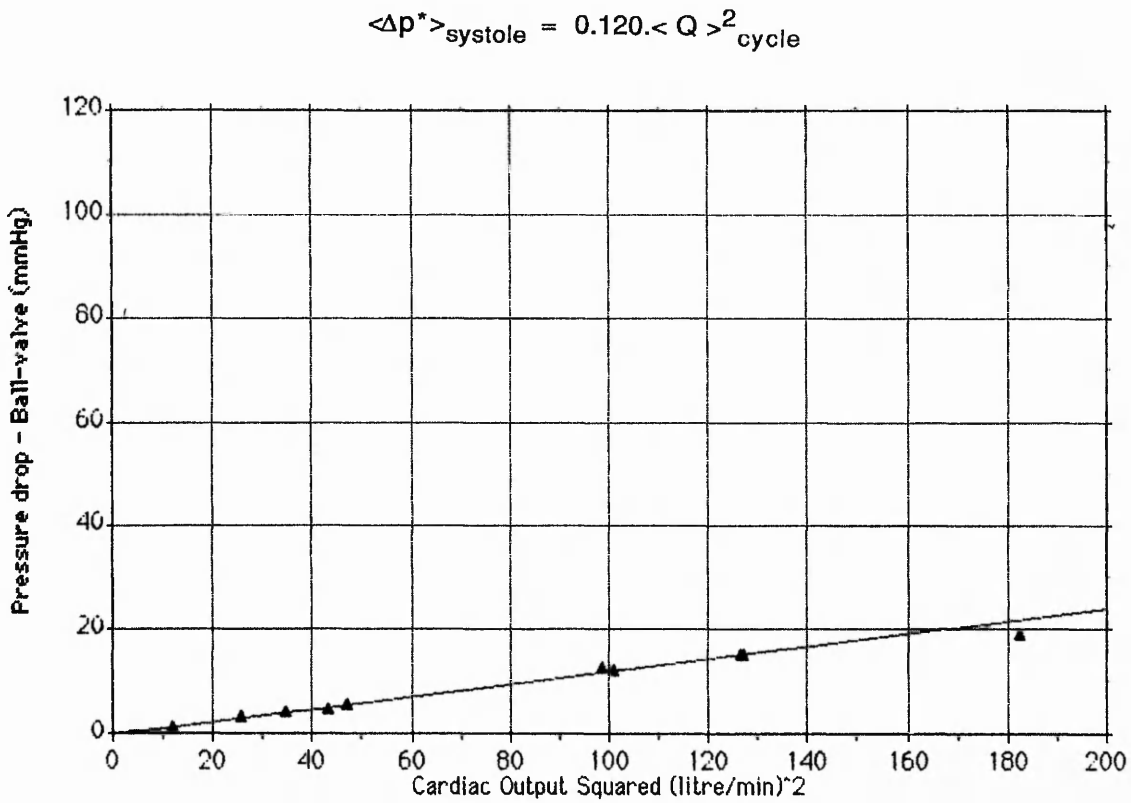


Fig. 6.10 Mean systolic pressure drop versus square of cardiac output for ball valve conduit as valve replacement

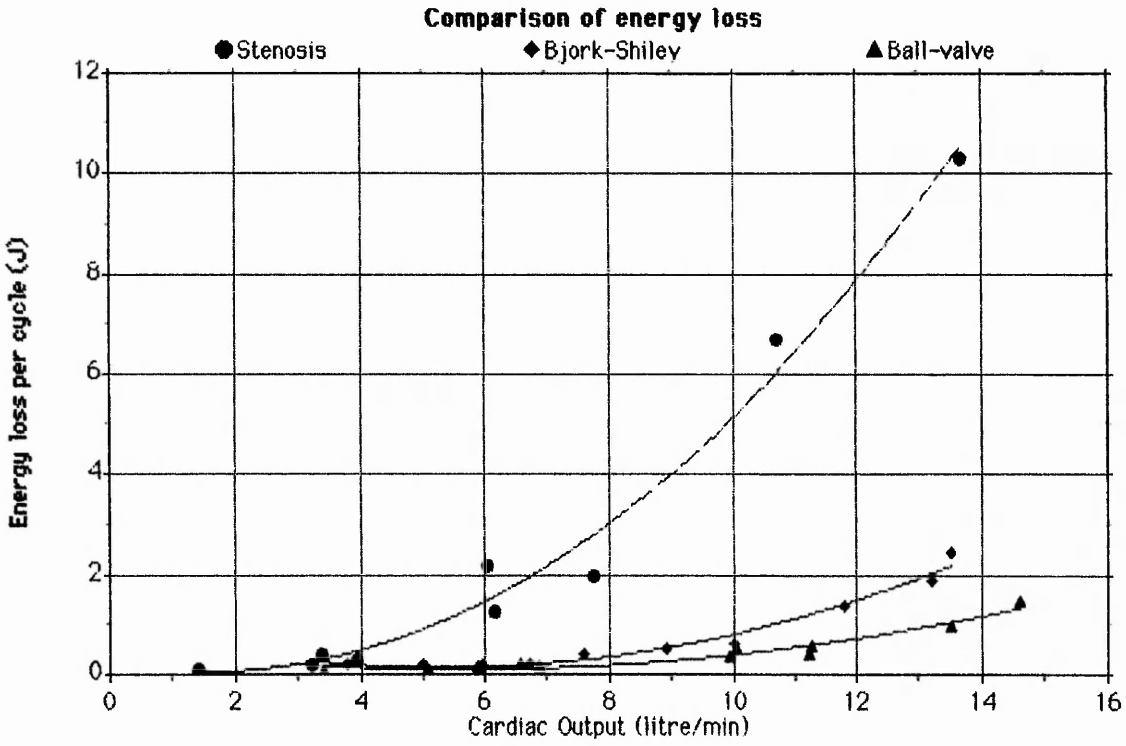


Fig. 6.11 Energy loss per pulse versus cardiac output for uncorrected stenosis and conduits as valve replacements

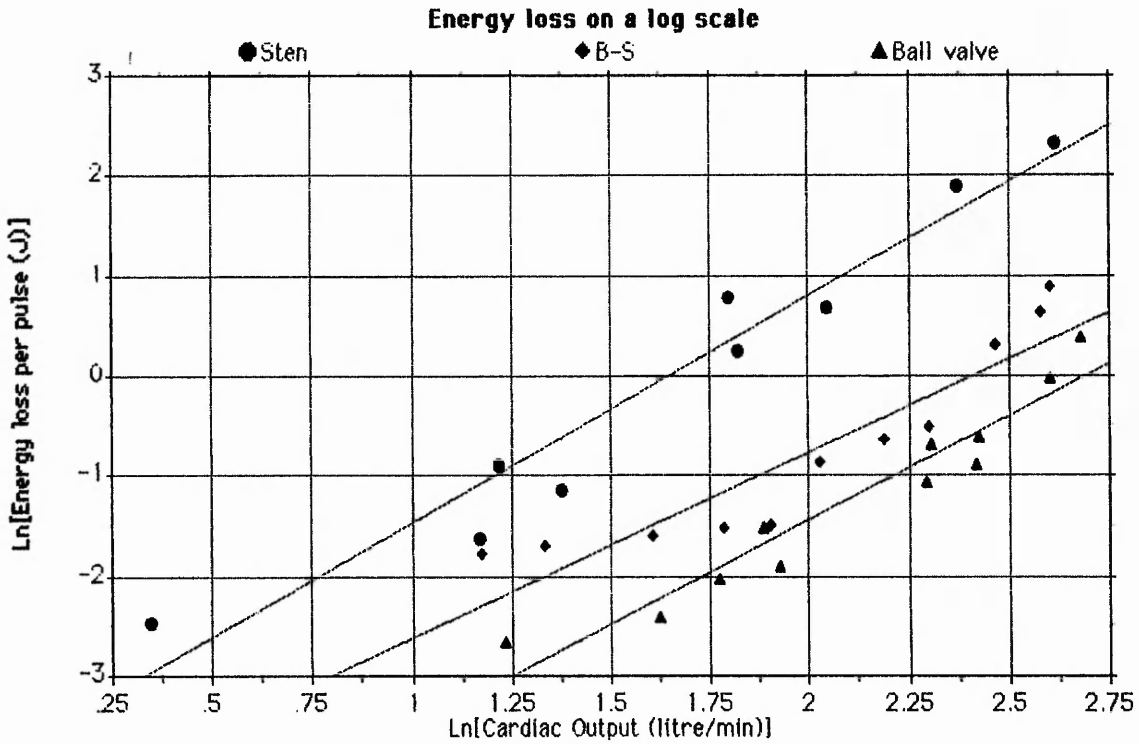


Fig. 6.12 Energy loss per pulse versus cardiac output for uncorrected stenosis and conduits as valve replacements, plotted on a logarithmic scale

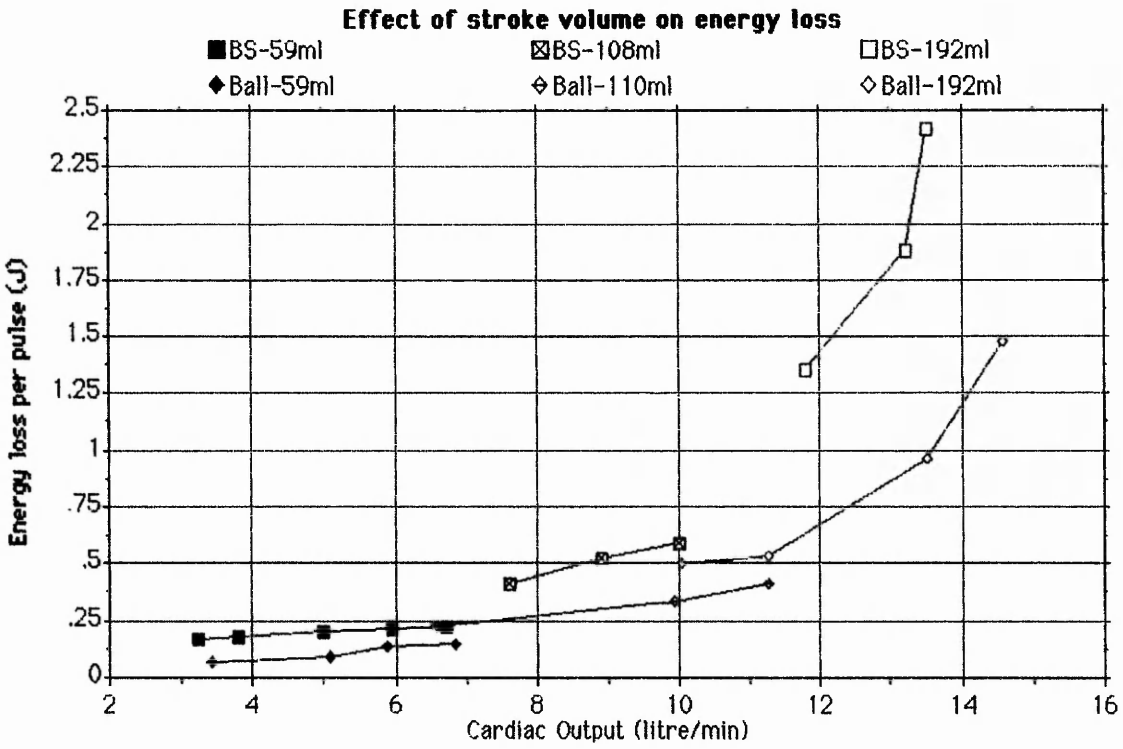


Fig. 6.13 Effect of stroke volume on energy loss per pulse for conduits as valve replacements

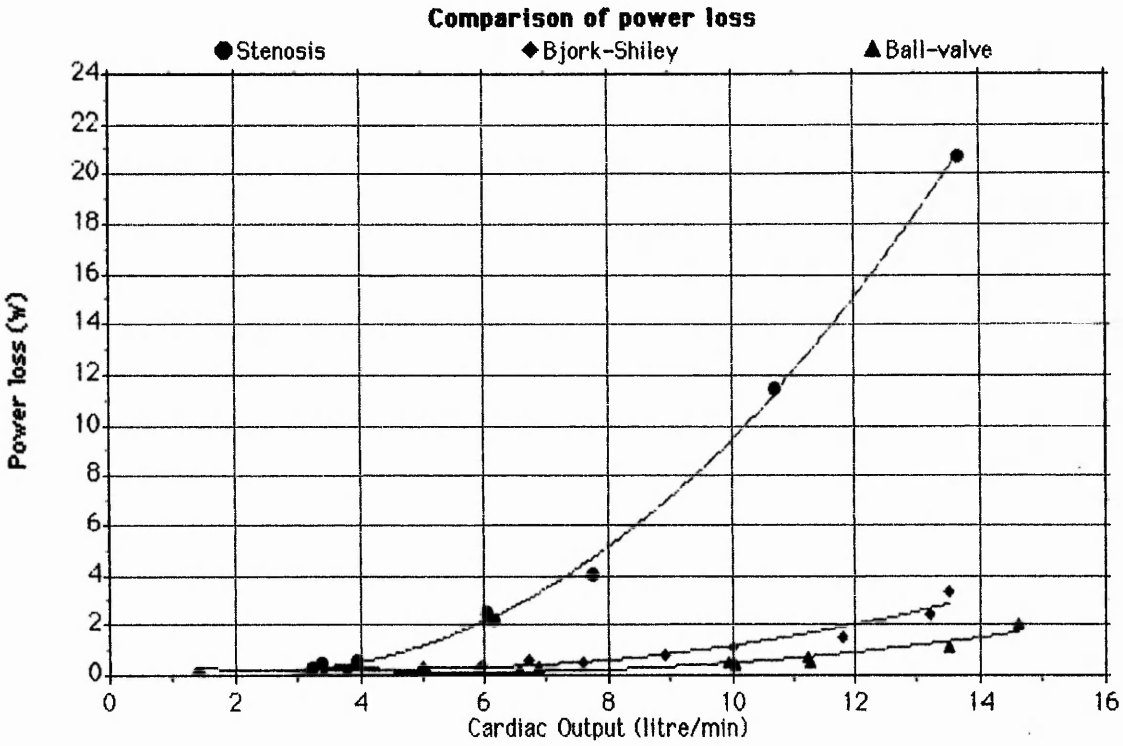


Fig. 6.14 Mean power loss versus cardiac output for uncorrected stenosis and conduits as valve replacements

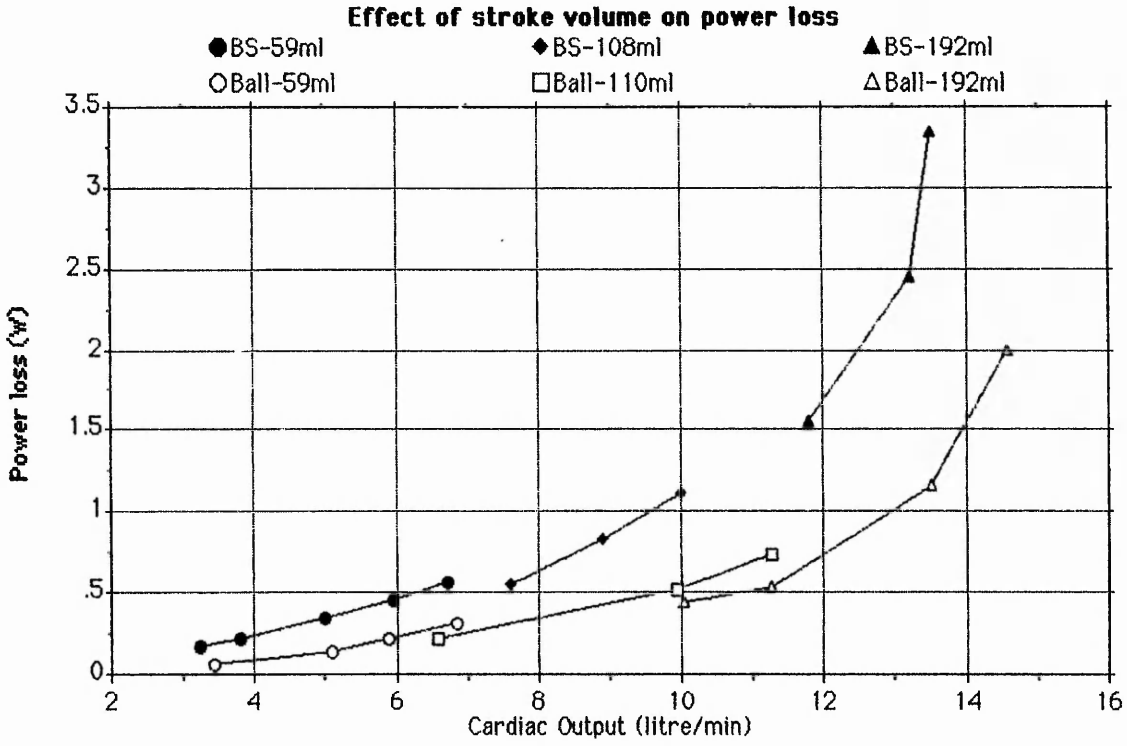


Fig. 6.15 Effect of stroke volume on power loss per pulse for conduits as valve replacements

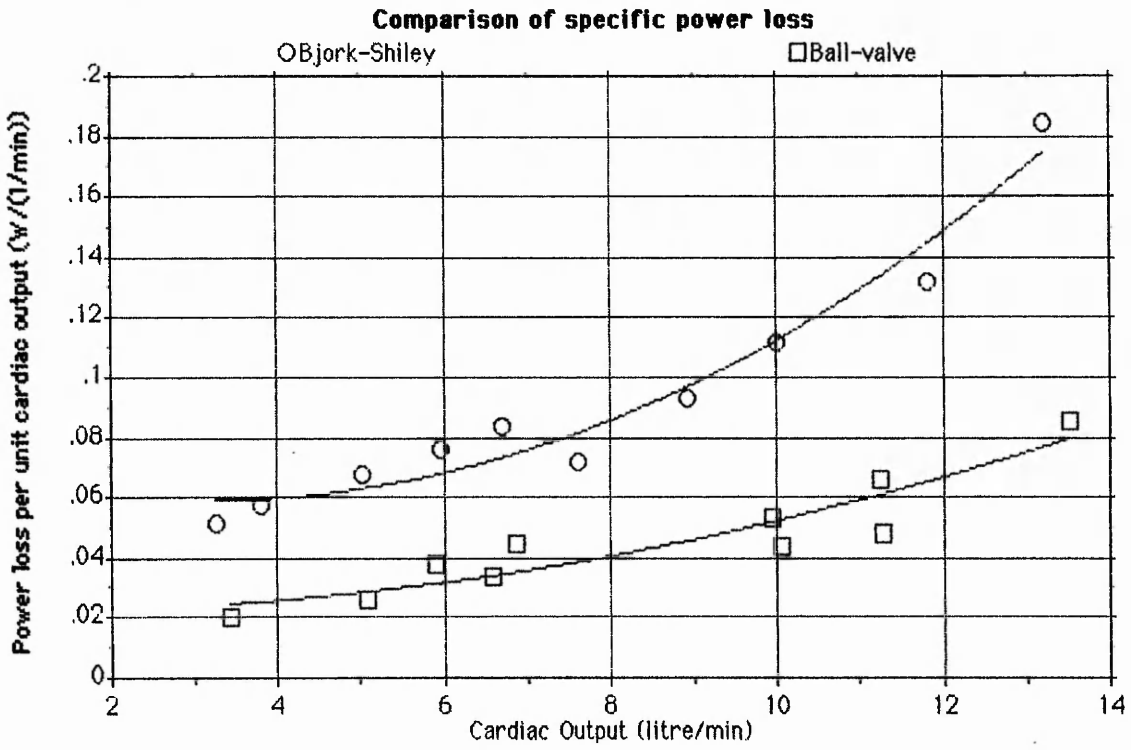


Fig. 6.16 Power loss per unit cardiac output versus cardiac output for conduits as valve replacements

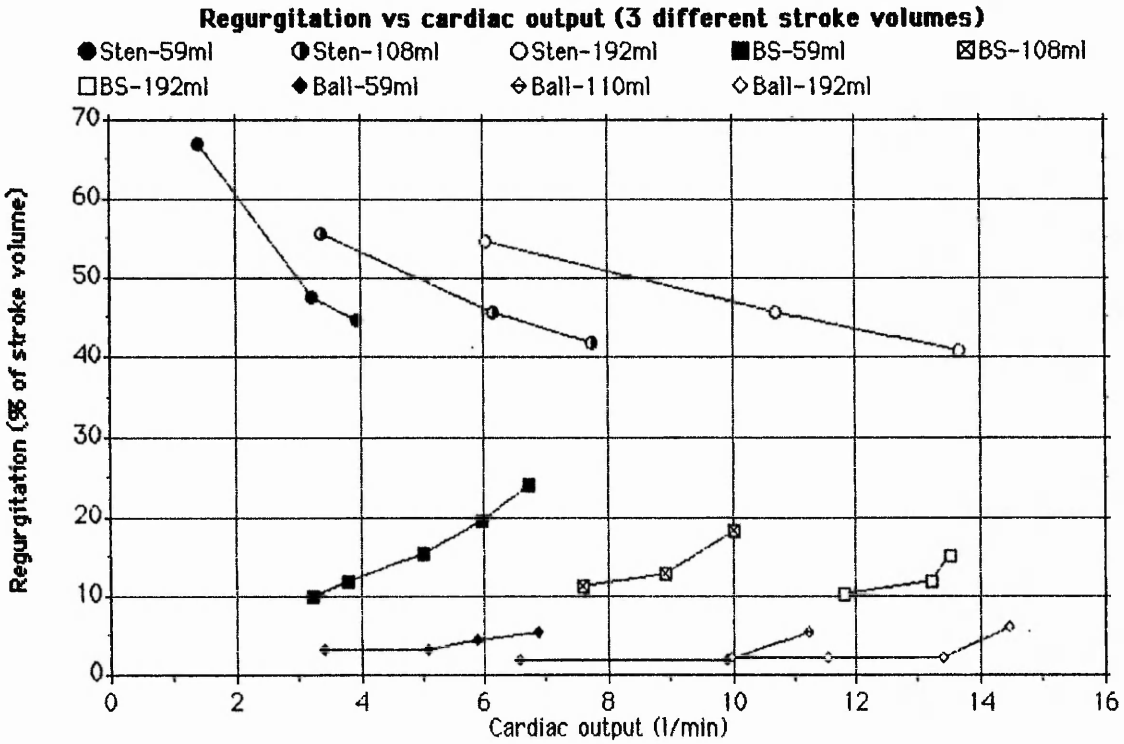


Fig. 6.17 Regurgitant fraction versus cardiac output with stroke volume as parameter for uncorrected stenosis and conduits as valve replacements

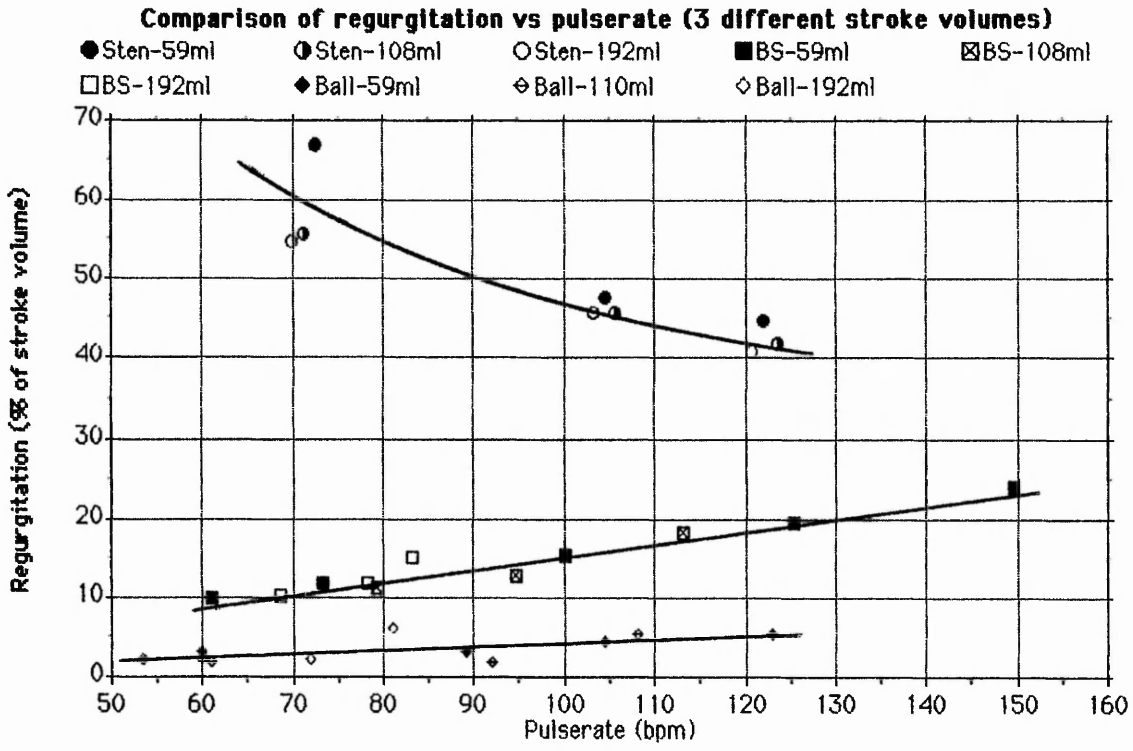


Fig. 6.18 Regurgitant fraction versus pulse-rate for uncorrected stenosis and conduits as valve replacements

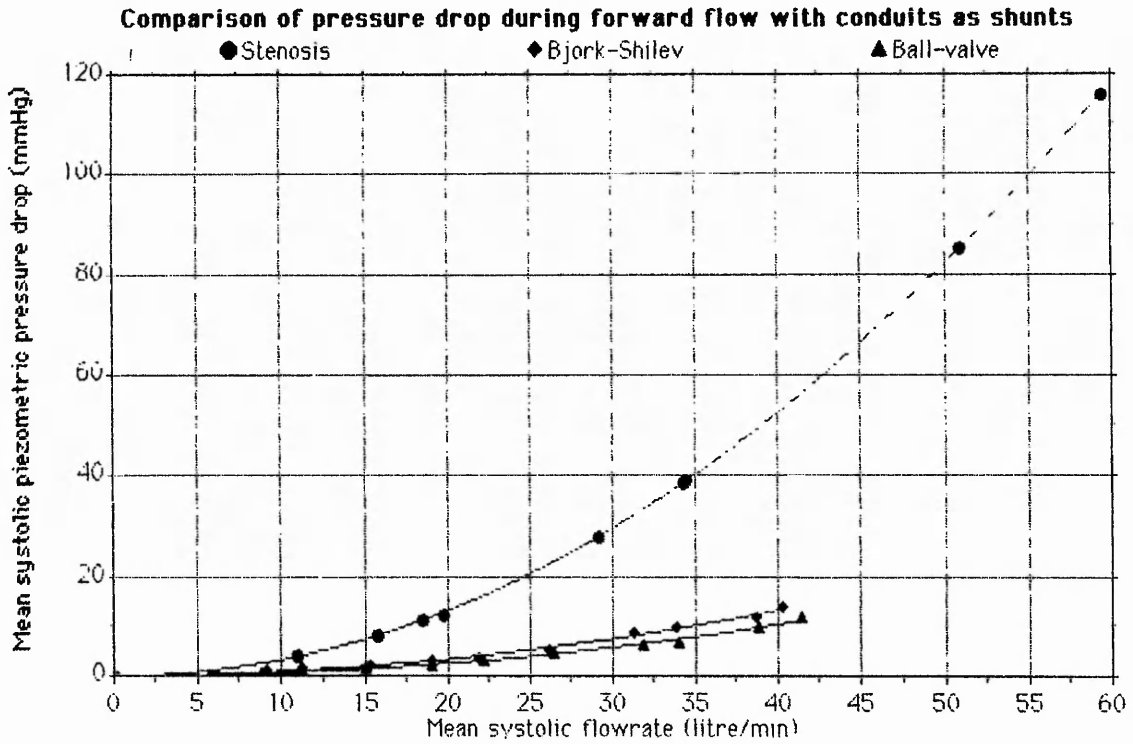


Fig. 6.19 Mean systolic pressure drop versus mean systolic flowrate for uncorrected stenosis and conduits as shunts

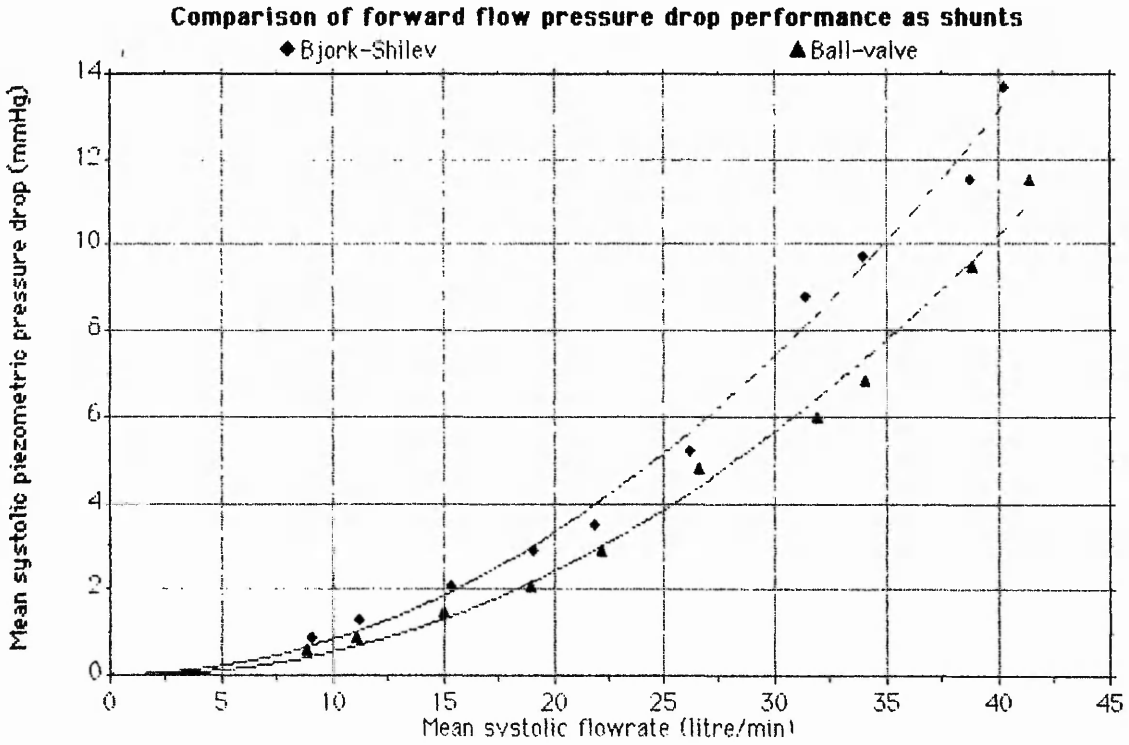


Fig. 6.20 Mean systolic pressure drop versus mean systolic flowrate for conduits as shunts

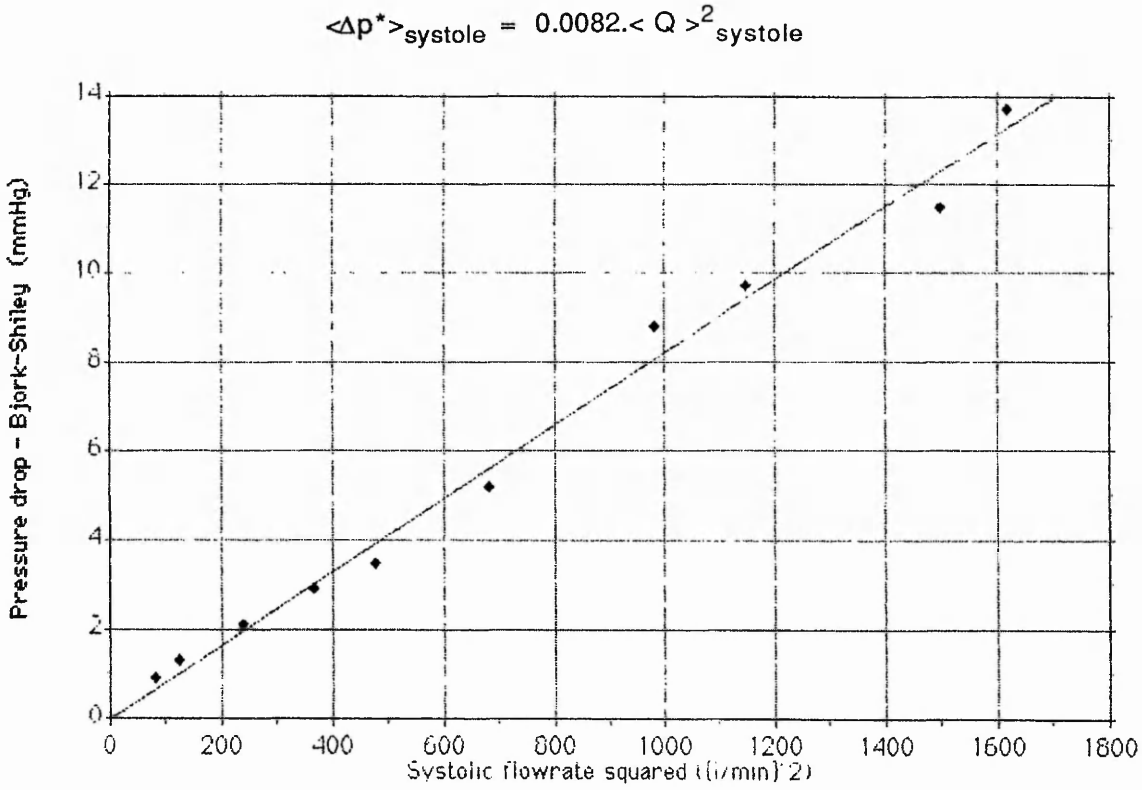


Fig. 6.21 Mean systolic pressure drop versus square of mean systolic flowrate for Bjork-Shiley conduit as shunt

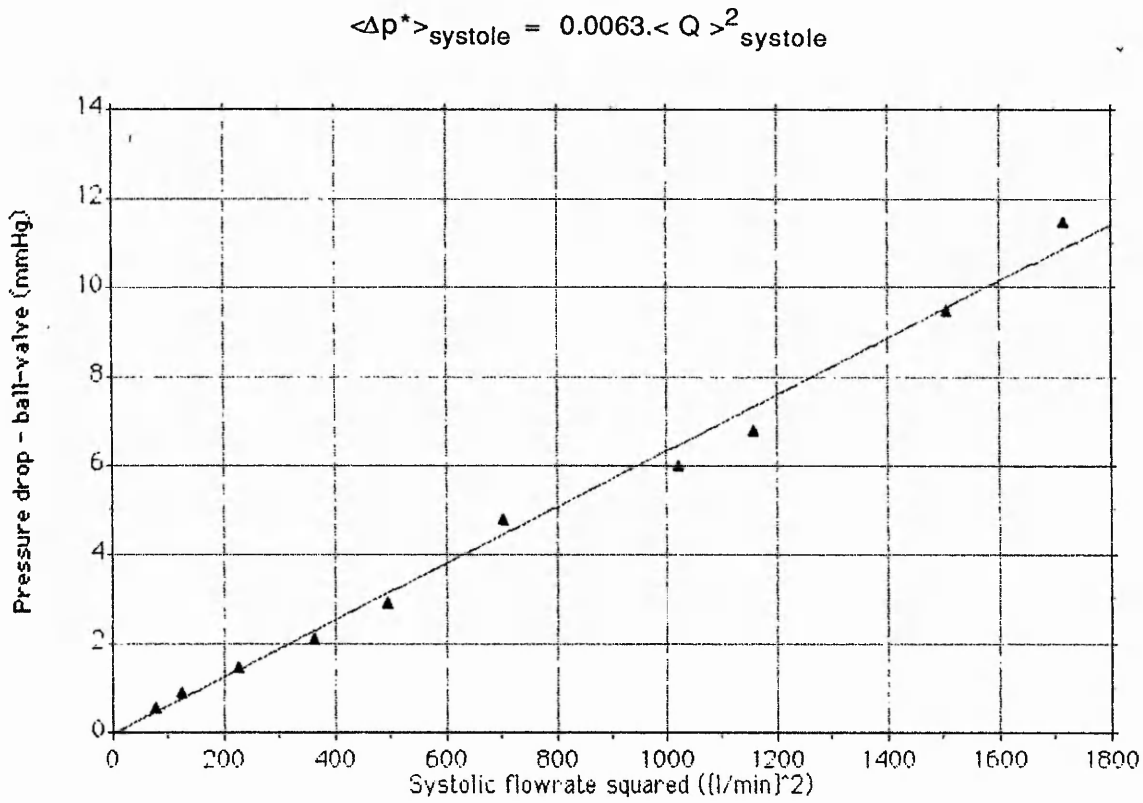


Fig. 6.22 Mean systolic pressure drop versus square of mean systolic flowrate for ball valve conduit as shunt

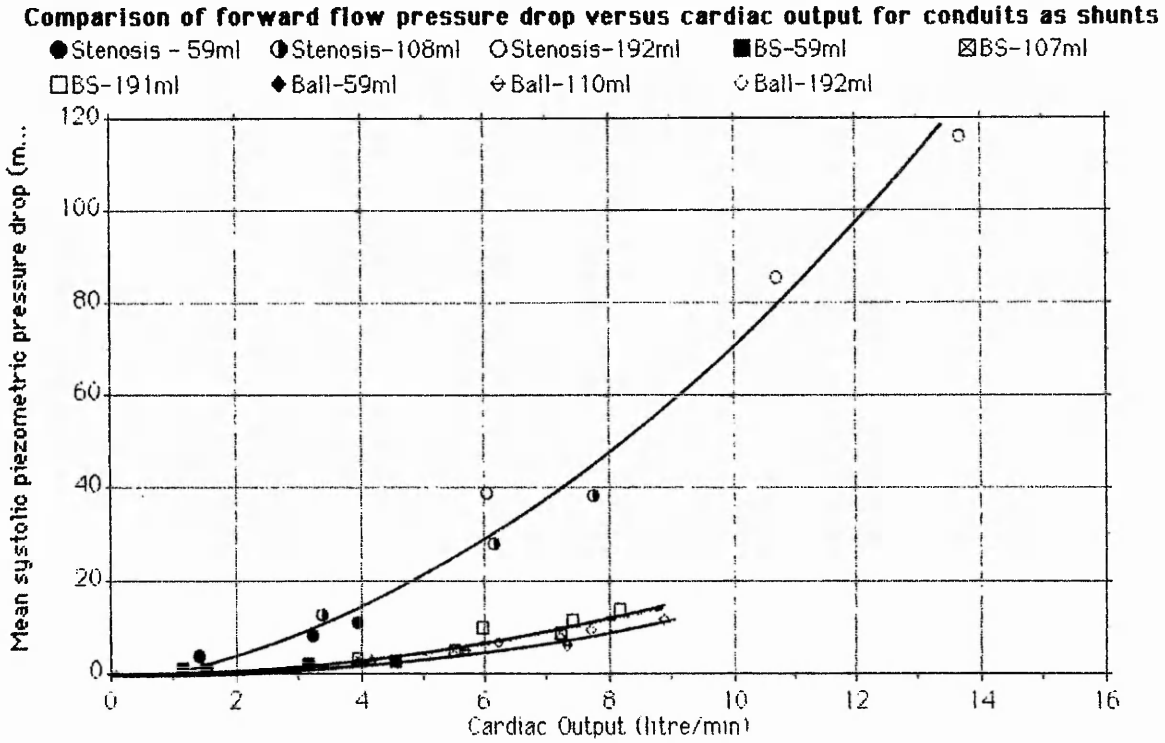


Fig. 6.23 Mean systolic pressure drop versus cardiac output for uncorrected stenosis and conduits as shunts

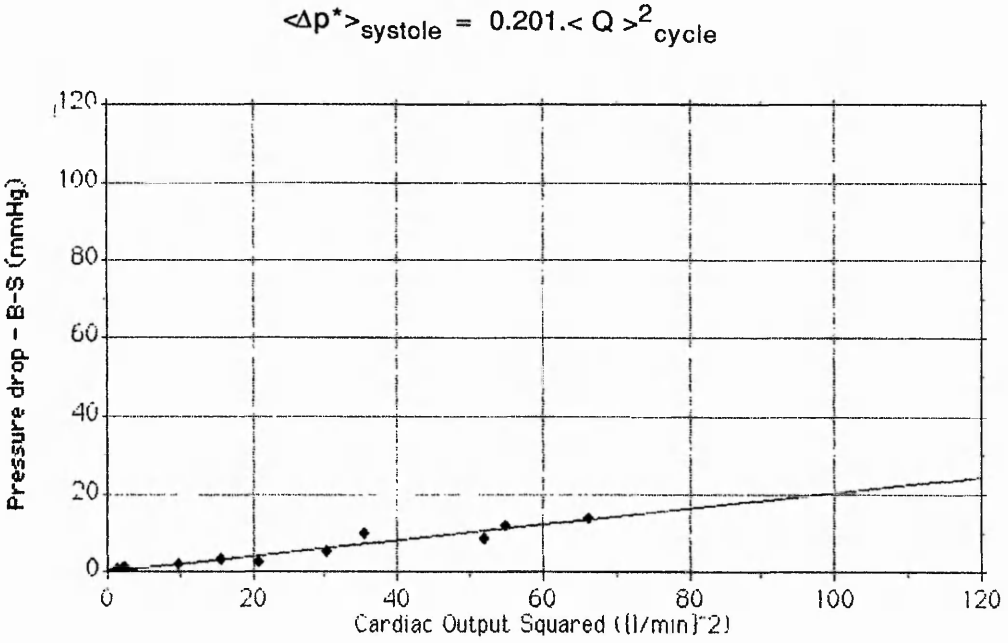


Fig. 6.24 Mean systolic pressure drop versus square of cardiac output for Bjork-Shiley conduit as shunt

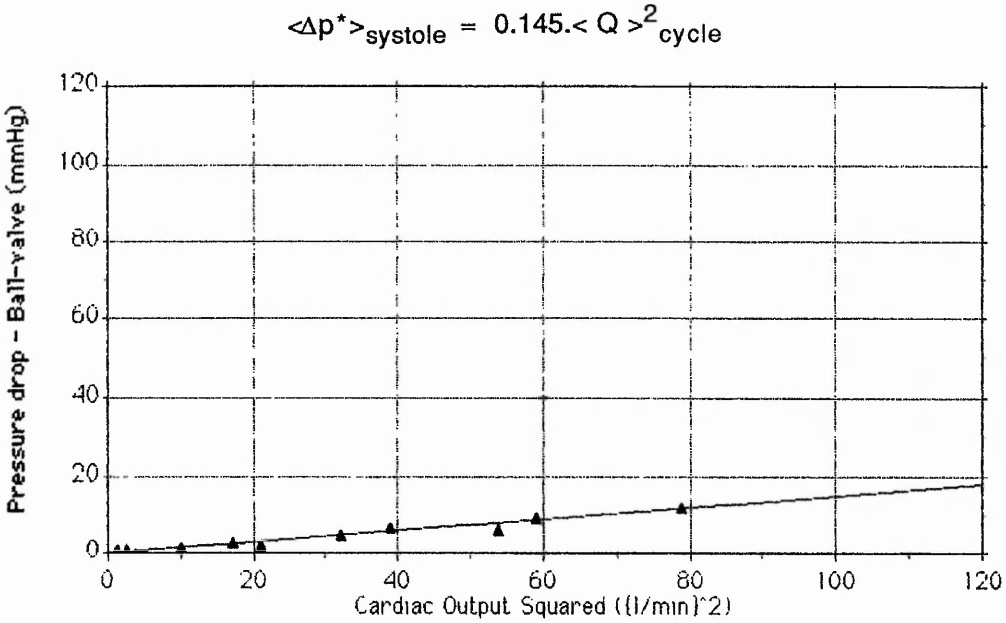


Fig. 6.25 Mean systolic pressure drop versus square of cardiac output for ball valve conduit as shunt

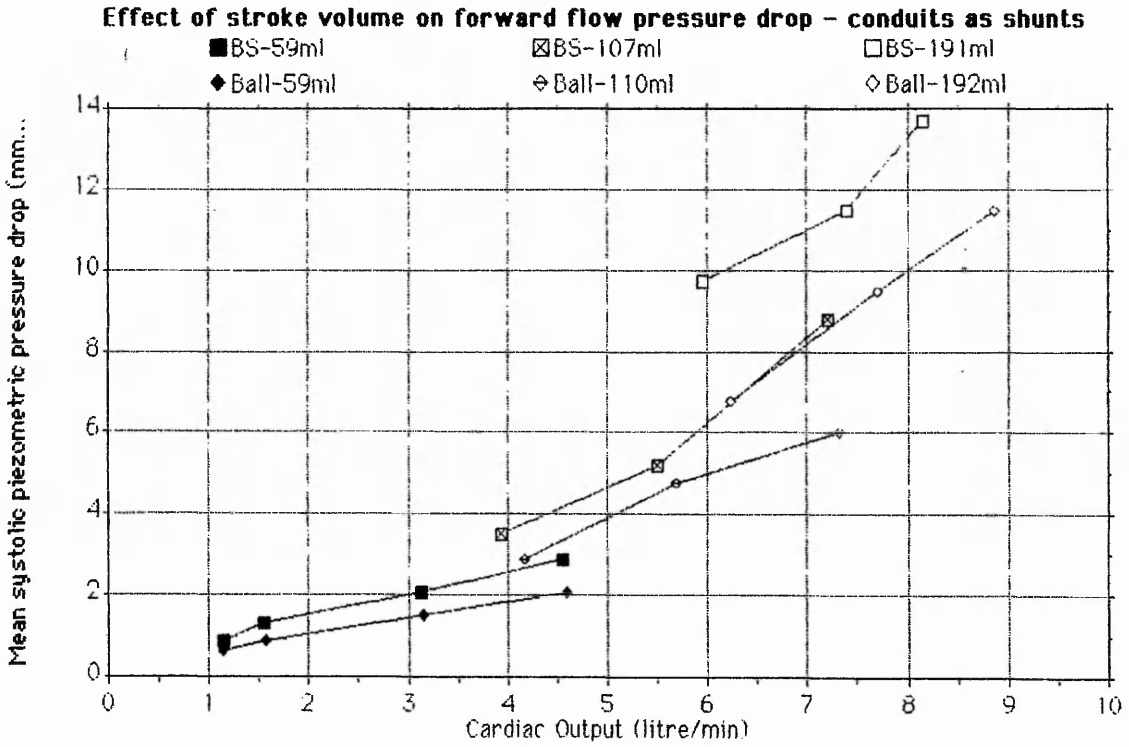


Fig. 6.26 Effect of stroke volume on forward flow pressure drop for conduits as shunts

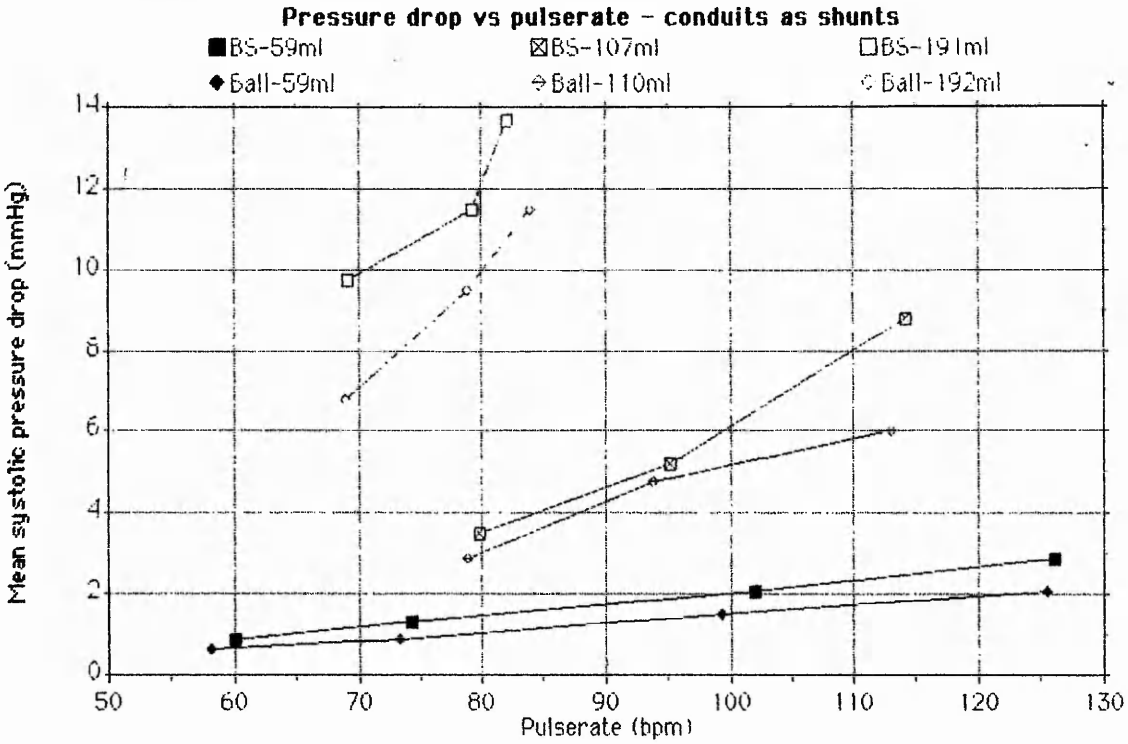


Fig. 6.27 Mean systolic pressure drop versus pulse-rate with stroke volume as parameter for conduits as shunts

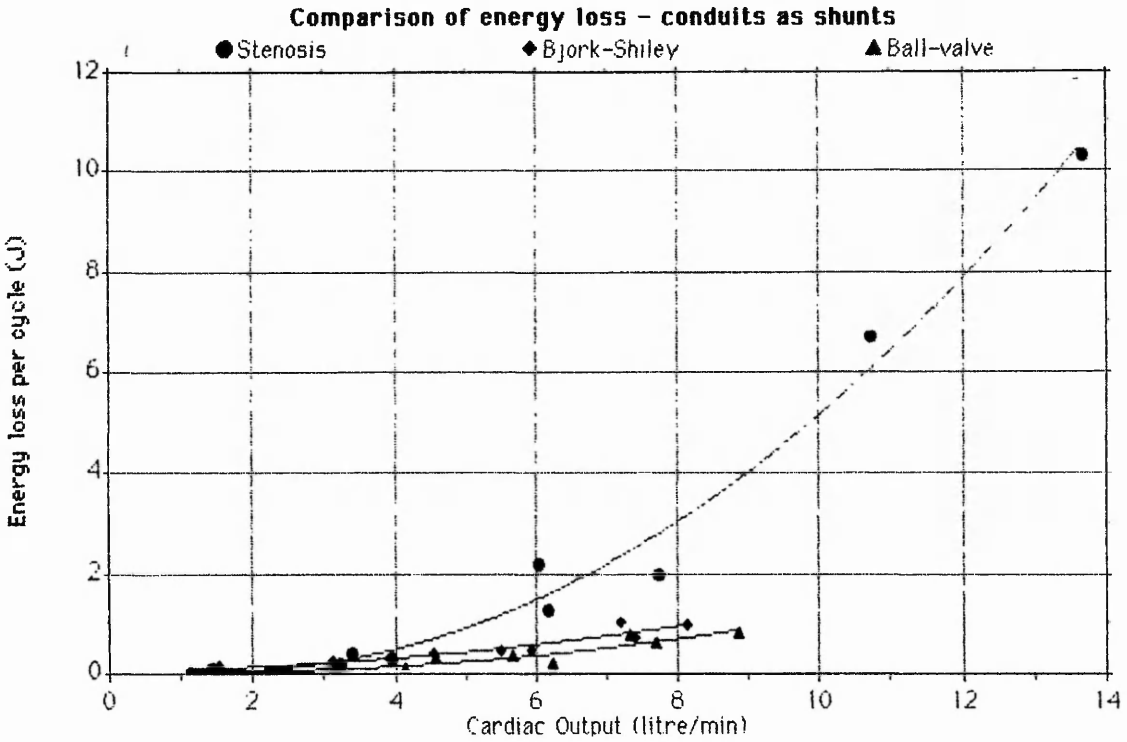


Fig. 6.28 Energy loss per pulse versus cardiac output for uncorrected stenosis and conduits as shunts

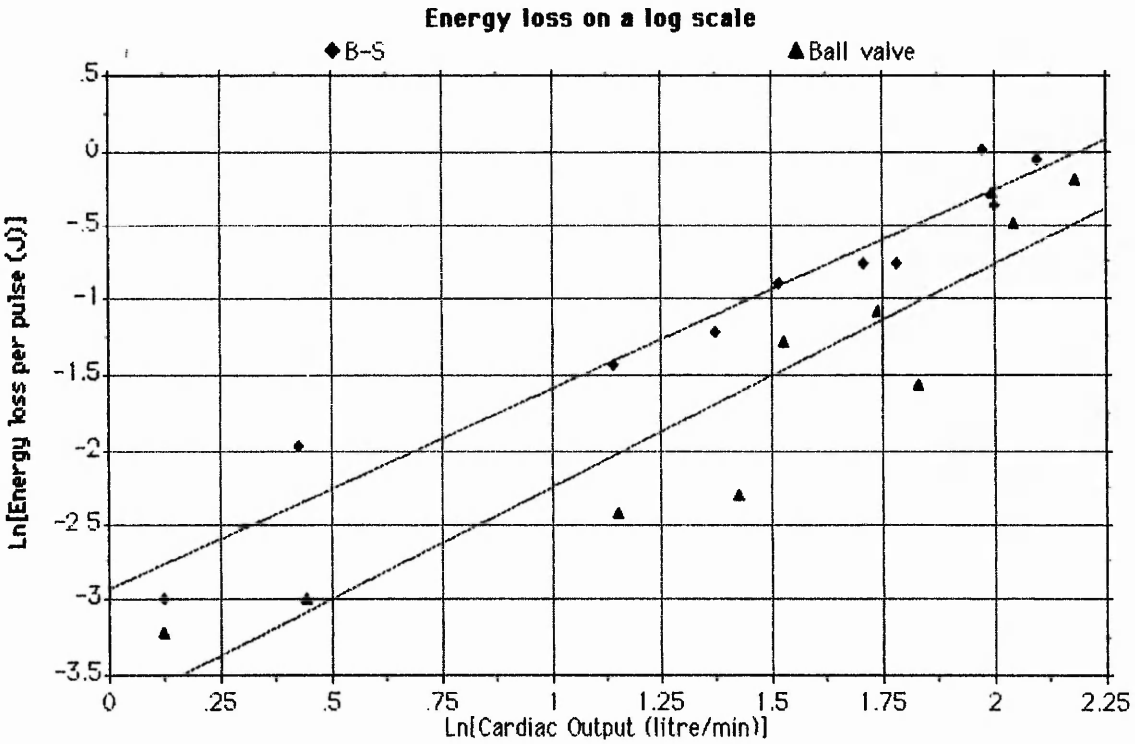


Fig. 6.29 Energy loss per pulse versus cardiac output for conduits as valve replacements, plotted on a logarithmic scale

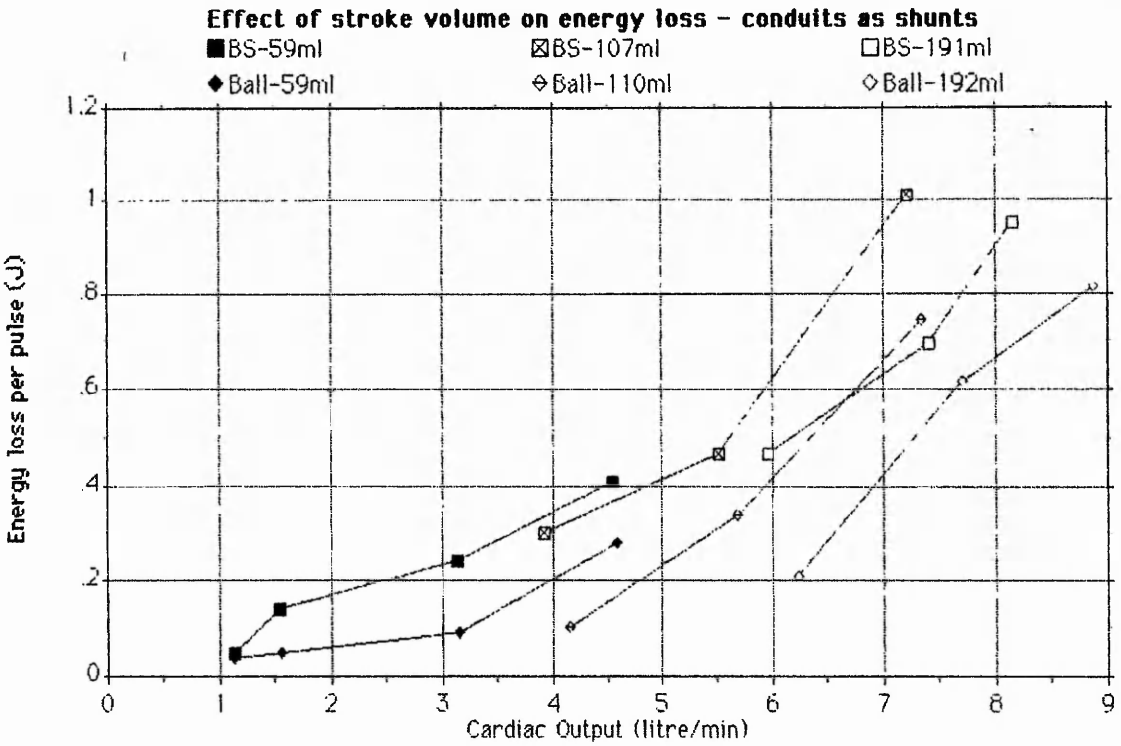


Fig. 6.30 Effect of stroke volume on energy loss per pulse for conduits as shunts

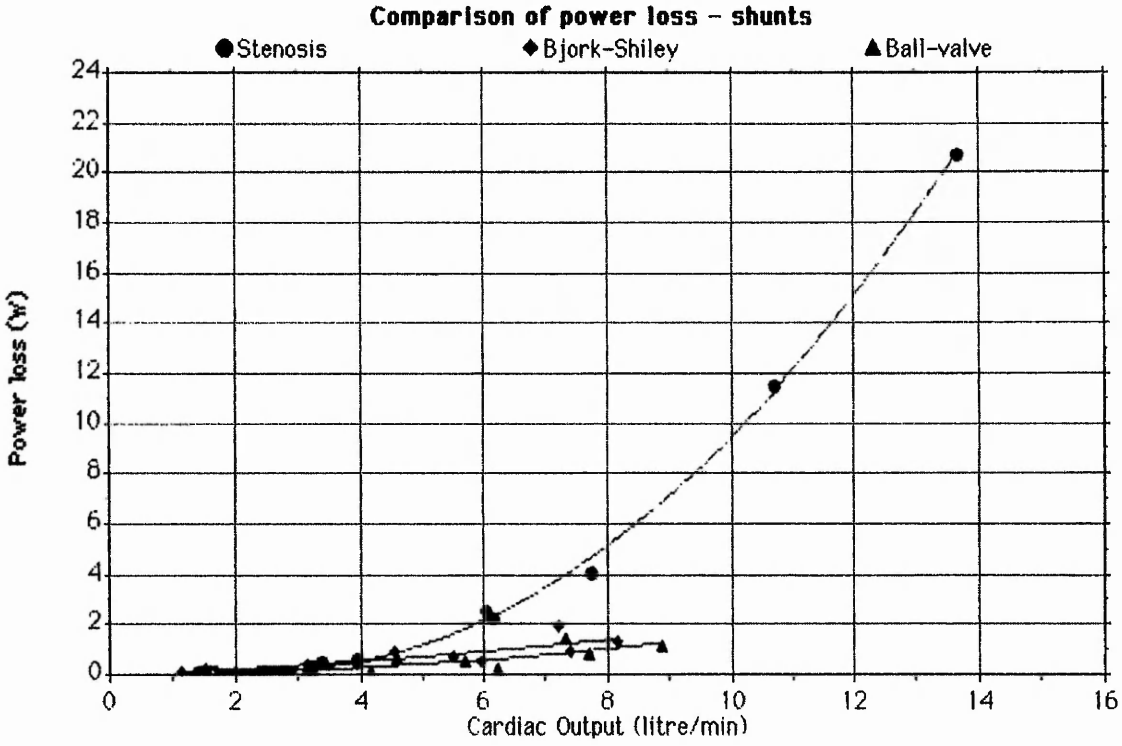


Fig. 6.31 Mean power loss versus cardiac output for uncorrected stenosis and conduits as shunts

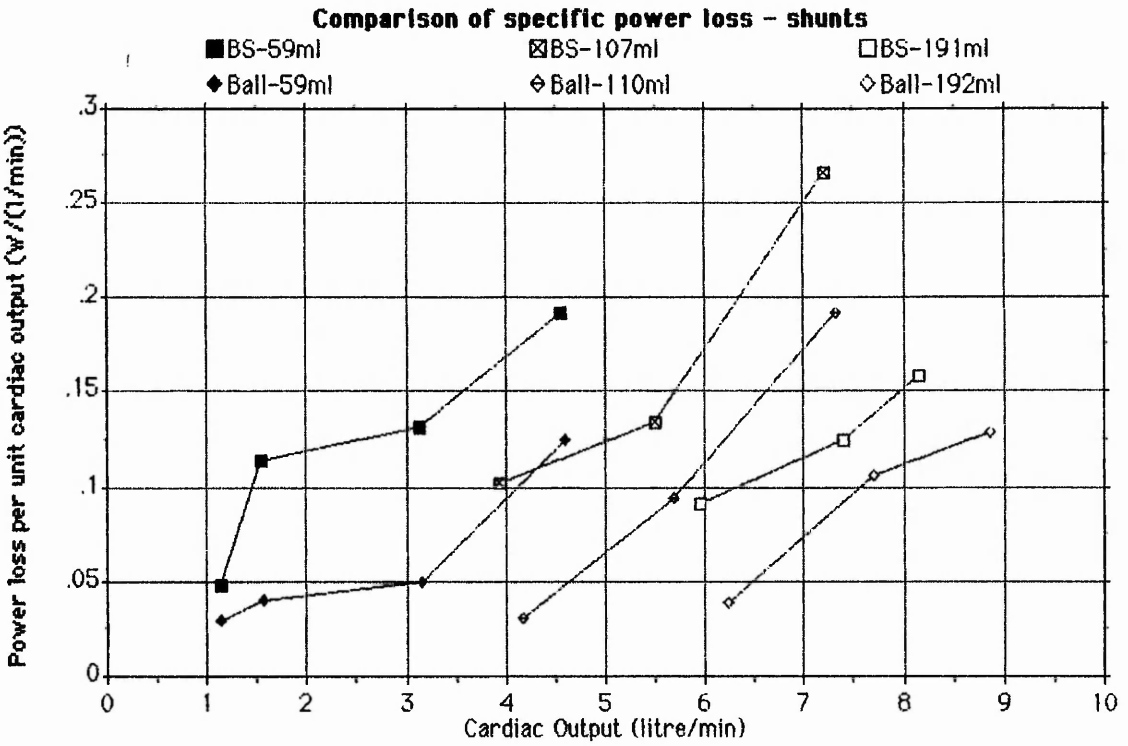


Fig. 6.32 Power loss per unit cardiac output versus cardiac output with stroke volume as parameter for conduits as shunts

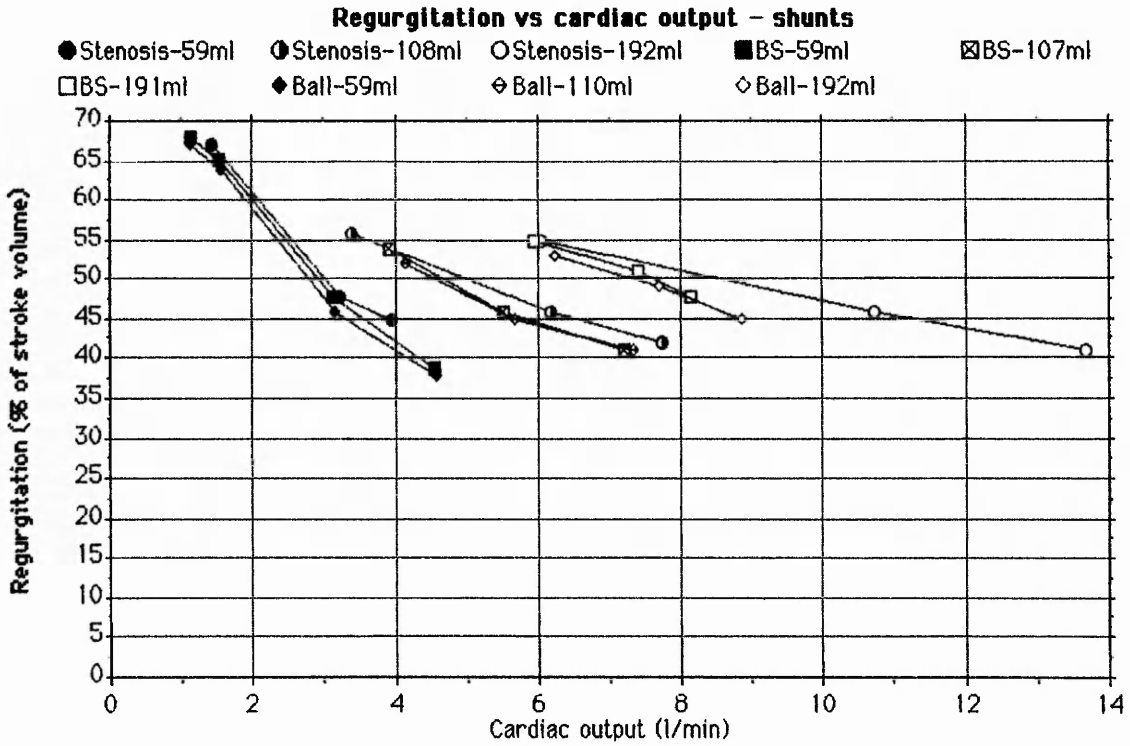


Fig. 6.33 Regurgitant fraction versus cardiac output with stroke volume as parameter for uncorrected stenosis and conduits as shunts

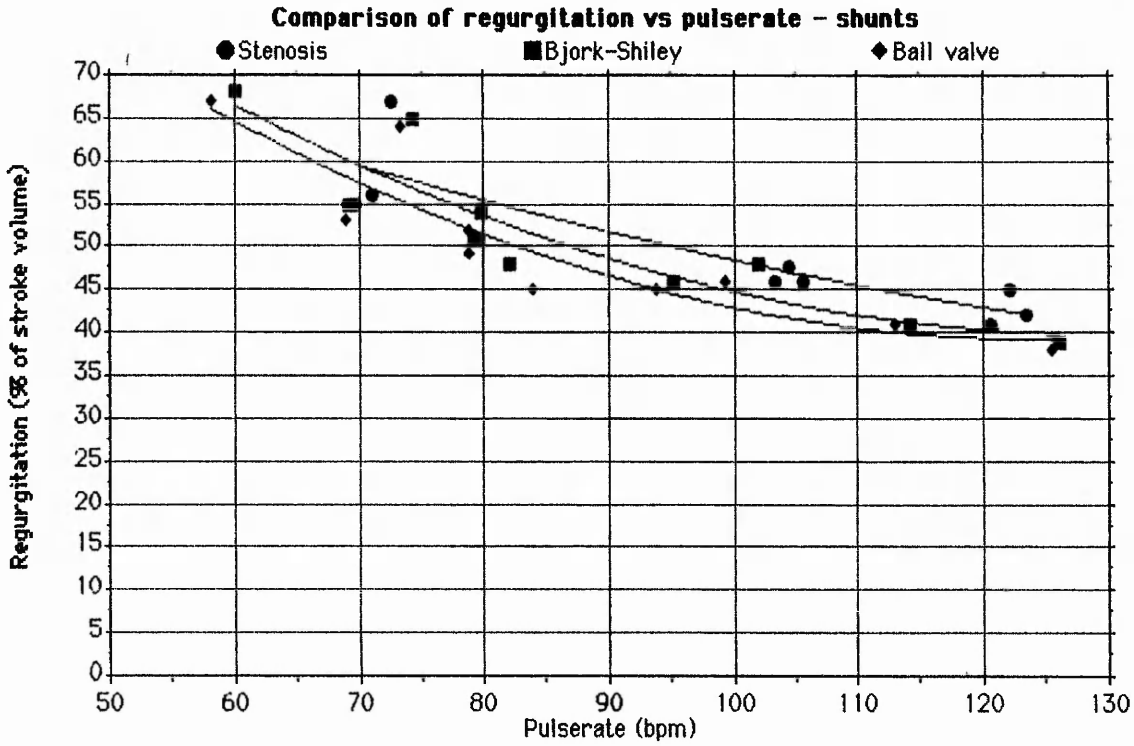


Fig. 6.34 Regurgitant fraction versus pulse-rate for uncorrected stenosis and conduits as shunts

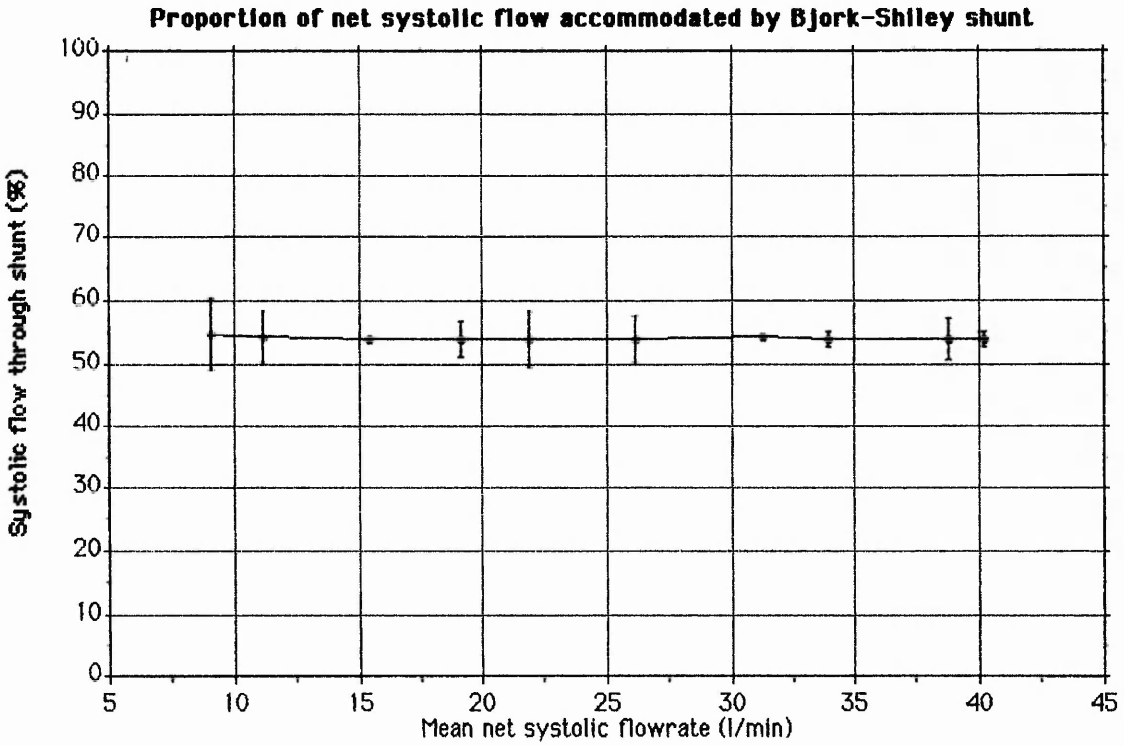


Fig. 6.35 Estimated proportion of net systolic flowrate accommodated by Bjork-Shiley conduit as a shunt

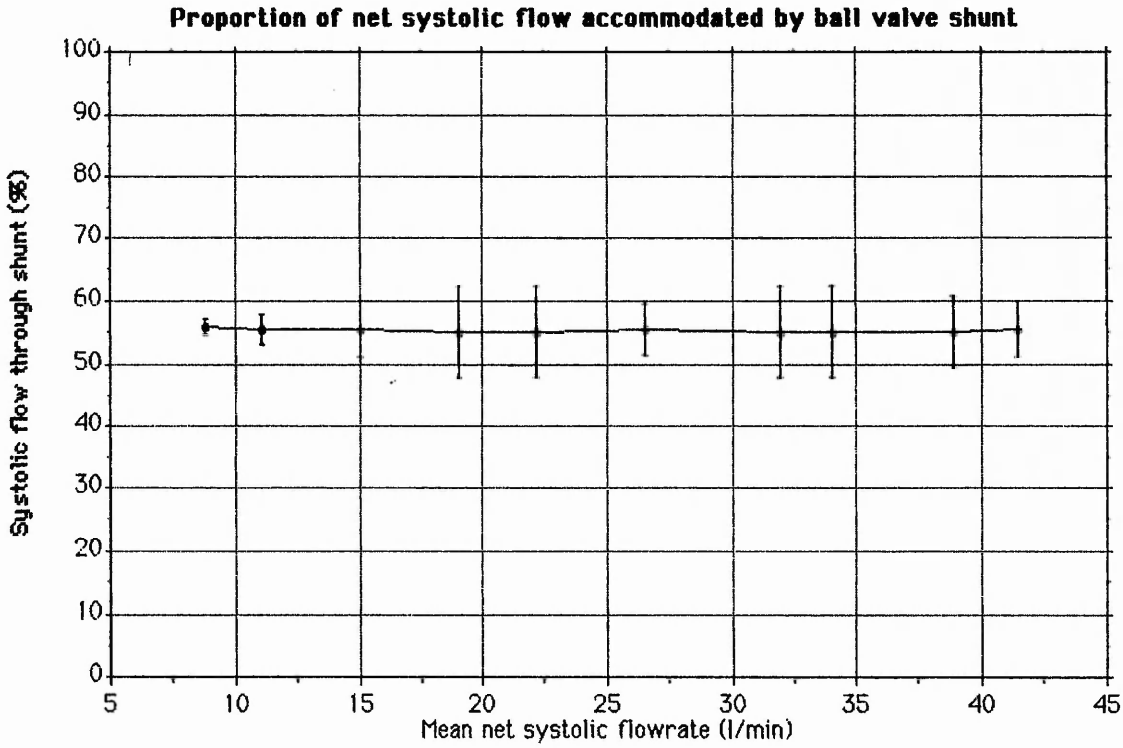


Fig. 6.36 Estimated proportion of net systolic flowrate accommodated by ball valve conduit as a shunt

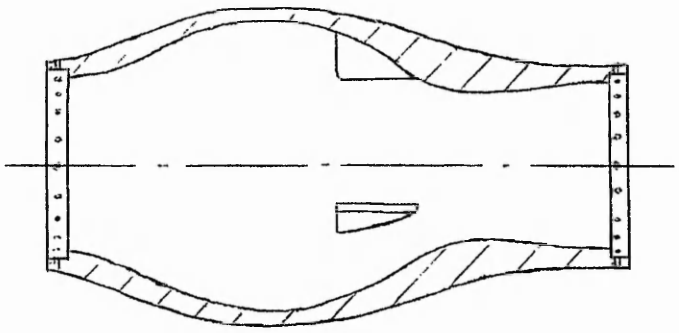


Fig. 8.1 Recommended constructional detail of valve-bearing section

REFERENCES

References

Agarwal, K.C., Edwards, W.D., Feldt, R.H., Danielson, G.K., Puga, F.J. and McGoon, D.C. 1981. Clinicopathological correlates of right-sided porcine-valved extracardiac conduits. *Journal of Thoracic and Cardiovascular Surgery*, **81**, 4, 591-601

Akutsu, T. and Modi, V.J. 1982. Fluid dynamics of several mechanical prosthetic heart valves using laser Doppler anemometry. *Proceedings of the 9th Annual Meeting of the European Society for Artificial Organs*, 107-110

Almeida, R.S., Wyse, R.K., deLeval, M.R., Elliott, M.J. and Stark, J. 1989. Long-term results of homograft valves in extracardiac conduits. *European Journal of Cardiothoracic Surgery*, **3**, 6, 488-93

Al-Naaman, Y.D. 1963. Bypass procedure for aortic stenoses simplified. *Medical Tribune*, **4**, 27 *et seq*

Anderson, J.M., Barenberg, S. and Hiltner, A. Prosthetic valve wear products - embolization and systemic presentation. *National Bureau of Standards Special Publication* **601**, 283-298

Arabia, M. and Akutsu, T. 1984. A new test circulatory system for research in cardiovascular engineering. *Annals of Biomedical Engineering*, **12**, 29-48

Arai, R. *et al.* 1965. Experimental study on bypass between the right ventricle and pulmonary artery, left ventricle and aorta by means of a homograft with valve. *Bulletin of the Heart Institute of Japan*, **9**, 49 *et seq*

Bailey, W.W., Kirklin, J.W., Bargeron, L.M., Pacifico, A.D. and Kouchoukos, N.T. 1977. Late results with synthetic valved external conduits from venous ventricle to pulmonary arteries. *Cardiovascular Surgery, Supplement 2, Circulation*, **56**, 3, 73-79

Bedderman, C., Norman, J.C. and Cooley, D.A. 1980. Combined replacement of the ascending aorta and the aortic valve in 42 consecutive patients: a comparison of composite graft techniques and conventional techniques over one year. *Bulletin of the Texas Heart Institute*, **7**, 2, 214-229

Bellhouse, B.J. and Bellhouse, F.H. 1968. Thin-film gauges for the measurement of velocity or skin-friction in air, water or blood. *Journal of Physics, E, Journal of Scientific Instruments*, **2**, 1, 1211-1213

Bellhouse, B.J. and Talbot, L. 1968. The fluid mechanics of the aortic valve. *Journal of Fluid Mechanics*, **3 5**, 4, 721-735

Bellhouse, B.J. 1969. Velocity and pressure distributions in the aortic valve. *Journal of Fluid Mechanics*, **3 7**, 3, 587-600

Benjamin, E.L. 1986. Steady flow investigations of valved heart conduits. *MPhil thesis, Trent Polytechnic, Nottingham.*

Bernhard, W.F., Poirier, V. and LaFarge, C.G. 1975. Relief of congenital obstruction to left ventricular outflow with a ventricular-aortic prosthesis. *Journal of Thoracic and Cardiovascular Surgery*, **6 9**, 2, 223-229

Bisset, G.S., Schwartz, D.C., Benzing, G., Helmsworth, J., Schreiber, J. and Kaplan, S. 1981. *Annals of Thoracic Surgery*, **3 1**, 5, 437-443

Black, M.M., Drury, P.J. and Smith, G.H. 1983. Long term clinical assessment of heart valve substitutes. *Life Support Systems*, **1**, Supplement 1, 301-304

Black, M.M. 1986. Artificial heart valves - past, present and future. *Heart Valve Engineering, Seminar at the Institution of Mechanical Engineers 4-5 December 1986*

Borst, H.G. 1981. Replacement of ascending aorta and aortic valve. *Annals of Thoracic Surgery*, **3 2**, 6, 613-614

Bowman, F.O., Hancock, W.D. and Malm, J.R. 1973. A valve-containing Dacron prosthesis. *Archives of Surgery*, **1 0 7**, 724-728

Brown, J.W., Myerowitz, P.D., Cann,M.S., McIntosh, C.L. and Morrow, A.G. 1974. Apical-aortic anastomosis: a method for relief of diffuse left ventricular outflow obstruction. *Surgical Forum*, **2 5**, 147-149

Brown, J.W., Salles, C.A. and Kirsh, M.M., 1977. Extraanatomical bypass of the aortic root: an experimental technique. *Annals of Thoracic Surgery*, **2 4**, 5, 433-438

Campbell, D.N., Clark, D.R. 1990. Use of the allograft aortic valved conduit. *Annals of Thoracic Surgery*, **5 0**, 2, 320-2

Carrel, A. 1910. On the experimental surgery of the thoracic aorta and the heart. *Annals of Surgery*, **5 2**, 83-95

Cartmill, T.B., Celermajer, J.M., Stuckey, D.S., Bowdler, J.D., Johnson, D.C. and Hawker, R.E. 1974. Use of Bjork-Shiley tilting disc prosthesis in valved conduits for right ventricular outflow reconstruction. *British Heart Journal*, **3 6**, 1106-1108

Castaneda, A.R. and Norwood, W.I. 1982. Valved conduits: a panacea for complex congenital heart defects? *Cardiac Bioprostheses. Proceedings of the 2nd International Symposium.* (Ed. Cohn, L.H. and Gallucci, V., Yorke Medical Books, N.Y.), 205-216

Chandran, K.B., Cabell, G.N., Khalighi, B. and Chen, C.J. 1984. Pulsatile flow past aortic valve bioprostheses in a model human aorta. *Journal of Biomechanics*, **1 7**, 8, 609-619

Chandran, K.B. and Khalighi, B. 1984. A note on the blood analog for in-vitro testing of heart valve bioprostheses. *Transactions of the A.S.M.E., Journal of Biomechanical Engineering*, **1 0 6**, 112-114

Chandran, K.B., Khalighi, B. and Chen, C.J. 1985. Experimental study of physiological pulsatile flow past valve prostheses in a model of human aorta - I. Caged ball valves. *Journal of Biomechanics*, **1 8**, 10, 763-772

Chandran, K.B. 1986. Prosthetic heart valves - 1. Flow dynamics. *Mechanical Engineering*, 1986, **1**, 53

Chang, L.J. and Tarbell, J.M. 1985. Numerical simulation of fully-developed sinusoidal and pulsatile (physiological) flow in curved tubes. *Journal of Fluid Mechanics*, **1 6 1**, 175-198

Chekanov, V.S., Shatalov, K.V. and Roeva, L.A. 1990. Left ventricular-to-aortic conduit as a variant of surgical treatment of left ventricular outflow tract obstructions. An experimental study. *Cor et Vasa* **3 2**, 2, 134-44

Ciaravella, J.M., McGoon, D.C., Danielson, G.K., Wallace, R.B. and Mair, D.D. 1979. Experience with the extracardiac conduit. *Journal of Thoracic and Cardiovascular Surgery*, **7 8**, 6, 920-930

Clark, C. 1979. Energy losses in flow through stenosed valves. *Journal of Biomechanics*, **1 2**, 737-746

Cooley, D.A, Norman, J.C., Mullins, C.E. and Grace, R.R. 1975. Left ventricle to abdominal aorta conduit for relief of aortic stenosis. *Bulletin of the Texas Heart Institute*, **2**, 3, 376-383

Cooley, D.A, Norman, J.C., Reul, G.J., Kidd, J.N. and Nihill, M.R. 1976. Surgical treatment of left ventricular outflow tract obstruction with apicoaortic valved conduit. *Surgery*, **8 0**, 6, 674-680

Cooley, D.A. 1977. A double outlet to relieve left ventricular hypertension. *Hospital Practice*, June 1977, 60-67

Cooley, D.A. and Norman, J.C. 1977. Severe intravascular hemolysis after aortic valve replacement. *Journal of Thoracic and Cardiovascular Surgery*, **7**, 2, 322-324

Davila, J.C., Trout, R.G., Sunner, J.E. and Glover, R.P. 1956. A simple mechanical pulse duplicator for cinematography of cardiac valves in action. *Annals of Surgery*, **1 4 3**, 4, 544-551

Deacon, P., Crow, M.J., Rajah, S.M. and Kester, R.C. 1985. Comparison of the thrombogenicity of four types of knitted Dacron arterial graft in an artificial circulation. *Biomaterials*, **6**, 64-67

Dembitsky, W.P. and Weldon, C.S. 1976. Clinical experience with the use of a valve-bearing conduit to construct a second left-ventricular outflow tract in cases of unresectable intra-ventricular obstruction. *Annals of Surgery*, **1 8 4**, 3, 317-323

Detmer, D.E., Johnson, E.H. and Braunwald, N.S. 1971. Left ventricular to apex-to-thoracic aorta shunts using aortic valve allografts in calves. *Annals of Thoracic Surgery*, **1 1**, 5, 417-422

Di Carlo, D., Stark, J., Revignas, A. and De Leval, M.R. 1982. Conduits containing antibiotic preserved homografts in the treatment of complex congenital heart defects. *Cardiac Bioprostheses. Proceedings of the 2nd International Symposium.* (Ed. Cohn, L.H. and Gallucci, V., Yorke Medical Books, N.Y.), 259-265

Donovan, T.J. 1950. The experimental use of homologous vein grafts to circumvent the pulmonic valves. *Surgery, Gynecology and Obstetrics*, **9 0**, 204-214

Donovan, T.J. and Sarnoff, S.J. 1954. Apical-aortic anastomosis for the relief of aortic stenosis. *Circulation Research*, **Vol II**, 1954, 381-382

Dowson, D. 1980. Priorities for research on internal prostheses. *Engineering in Medicine*, **9**, 3, 165-166

Drury, P.J., Kay, R., Black, M.M. and Lawford, P.V. 1986. Long-term clinical assessment of heart valve substitutes. *Heart Valve Engineering, Seminar at the Institution of Mechanical Engineers 4-5 December 1986*

Duff, W.R. 1970. Fluid dynamics of prosthetic heart valves. PhD thesis, Purdue University, Lafayette, Indiana, USA

Duff, W.R. and Fox, R.W. 1972. Prosthetic cardiac valves - an in vitro study. *Journal of Thoracic and Cardiovascular Surgery*, **6 3**, 1, 131-142

Duncan, W.J., Thom, A.S. and Young, A.D. 1970. Mechanics of Fluids. (Edward Arnold, London). 311-313

Ebert, P.A., Robinson, S.J., Stanger, P. and Engle, M.A. 1976. Pulmonary artery conduits in infants younger than six months of age. *Journal of Thoracic and Cardiovascular Surgery*, **7 2**, 3, 351-356

Ebert, R. and Schaldach, M. 1978. Rutile ceramics as an alternative in cardiovascular design. *Proceedings of the European Society for Artificial Organs*, **5**, 42-45

Engelman, M.S., Moskowitz, S.E. and Borman, J.B. 1980. Computer simulation: A diagnostic method in comparative studies of valve prostheses. *Journal of Thoracic and Cardiovascular Surgery*, **7 9**, 3, 402-412

Farahifar, D., Cassot, F., Bodard, H., Pelissier, R. and Siouffi, M. 1983. A cardiovascular pulse duplicator for the in vitro assessment of artificial heart valves under physiological conditions. *Life Support Systems*, **1**, Supplement 1, 332-335

Farahifar, D., Pelissier, R., Cassot, F. and Bodard, H. 1984. An experimental investigation of post-valvular flow field. *Life Support Systems*, **2**, Supplement 1, 73-74

Farahifar, D., Cassot, F. and Bodard, H. 1984. Velocity profiles in the wake of two prosthetic heart valves using a new cardiovascular simulator. *Journal of Biomechanics*, **18**, 10, 789-802

Figliola, R.S. and Mueller, T.J. 1981. On the hemolytic and thrombogenic potential of occluder prosthetic heart valves from in-vitro measurements. *Transactions of the A.S.M.E. Journal of Biomechanical Engineering*, **103**, 83-90

Fry, D.L. 1959. The measurement of pulsatile blood flow by the computed pressure gradient technique. *I.R.E. Transactions on Medical Electronics*, **Dec 1959**, 259-264

Gabay, S., McQueen, D., Yellin, E.L. and Frater, R.W.M. 1978. In vitro hydrodynamic comparison of mitral valve bioprostheses. *Cardiovascular Surgery, Supplement 1, Circulation*, **60**, 2, 62-70

Galanga, F.L. and Lloyd, J.R. 1981. An experimental study of the flow-induced mass transfer distribution in the vicinity of prosthetic heart valves. *Transactions of the A.S.M.E. Journal of Biomechanical Engineering*, **103**, 1-10

Geha, A.S., Laks, H., Stansel, H.C., Cornhill, J.F., Kilman, J.W., Buckley, J. and Roberts, W.C. 1979. Late failure of porcine valve heterografts in children. *Journal of Thoracic and Cardiovascular Surgery*, **78**, 3, 351-363

Gentle, C.R. 1977. A limit to hydraulic design of heart valve prostheses. *Engineering in Medicine*, **6**, 1, 17-21

Gentle, C.R. 1977a. A theoretical limit to flow design in heart valve prostheses. *Journal of Medical Engineering and Technology*, **1**, 3, 161-163

Gentle, C.R. 1977b. Assessment of the performance of heart valves. *Journal of Medical Engineering and Technology*, **1**, 4, 229

Gentle, C.R., Swales, P.D. and Dowson, D. 1978. In vitro evaluation of prosthetic heart valves. Chapter 1 in "*Heart Valve Research in the UK*" (Eds. Gentle, C.R. and Swales, P.D. Leeds University Press)

Gentle, C.R. 1978. The role of simulation studies in cardiac valve prosthesis design. *Engineering in Medicine*, **7**, 2, 101-106

Gentle, C.R. 1980. A porous, ceramic mitral valve prosthesis. *Mechanical Properties of Biomaterials. (Ed. Hastings, G.W. and Williams, D.F., John Wiley and Sons Ltd.)* 557-563

Gentle, C.R., Juden, H. and Swales, P.D. 1981. Testing of an all-ceramic mitral valve. *Proceedings of the European Society for Artificial Organs*, **8**, 352-355

Gentle, C.R., Arundel, P.A., Hamilton, D.I. and Swales, P.D. 1981a. Development of a twin-flap, porous ceramic mitral valve prosthesis. *Journal of Biomedical Engineering*, **3**, 3 - 8

Gentle, C.R. 1982. Minimisation of pressure drop across heart valve conduits: a preliminary study. *Life Support Systems*, **1**, 263-270

Gentle, C.R. 1982a. Characterisation of leakage flow in cardiac valve prostheses. *Proceedings of the European Society for Artificial Organs*, **9**, 102-106

Gentle, C.R. and Benjamin, E.L. 1984. Measurement of steady flow characteristics of valved conduit prostheses. *Proceedings of the 10th Annual Meeting of the European Society for Artificial Organs*, 324-327

Gentle, C.R. and Juden, H. 1984. Wear tests on alumina for application in heart valve prostheses. *Engineering in Medicine*, **1 3**, 2, 79-82

Gentle, C.R. 1986. The use of ceramics in prosthetic heart valves. *Heart Valve Engineering, Seminar at the Institution of Mechanical Engineers 4-5 December 1986*

Gorlin, R. and Gorlin, S.G. 1951. Hydraulic formula for the calculation of the area of the stenotic mitral valve, other cardiac valves, and central circulatory shunts. *American Heart Journal*, **4 1**, 1, 1-29

Gormsen, J., Mathar, R., Hasenkam, J.M. and Paulsen, P.K. 1983. Quantitative determination of heart valve insufficiency. *Life Support Systems*, **1**, Supplement 1, 328-331

Haggag, Y.A.M. 1990. A comparative study of the shear stress induced in the leakage backflow produced by four types of heart valve prostheses. *Proceedings of the Institution of Mechanical Engineers - Part H, Journal of Engineering in Medicine*, **204**, H2, 111-114

Harken, D.E., Soroff, H.S., Taylor, W.J., Lefemine, A.A., Gupta, S.K. and Lunzer, S. 1960. Partial and complete prostheses in aortic insufficiency. *Journal of Thoracic and Cardiovascular Surgery*, **40**, 744-762

Harken, D.E. 1977. 'Perfection may be the enemy of good'. *Medical Instrumentation*, **11**, 2, 70-71

Harris, J.P., Stewart, S., Anderson, V., Mitchell, M.L. and Manning, J.A. 1981. Coronary artery injury by a valved external conduit. *Annals of Thoracic Surgery*, **31**,3, 271-273

Haverich,A., Walterbusch, G. and Borst, H.G. 1981. The use of fibrin glue for sealing vascular prostheses of high porosity. *Thoracic Cardiovascular Surgeon*, **29**, 252-254

Heck, H.A., Schieken, R.M., Lauer, R.M. and Doty, D.B. 1978. Conduit repair for complex congenital heart disease. Late follow-up. *Journal of Thoracic and Cardiovascular Surgery*, **75**, 6, 806-814

Heiliger, R. 1986. Hydrodynamic advantages and disadvantages of mechanical bileaflet valves and tilting disc valves No. 29. *Heart Valve Engineering, Seminar at the Institution of Mechanical Engineers 4-5 December 1986*

Hellberg, K., Ruschewski, W. and De Vivie, E.R. 1981. Early stenosis and calcification of gluteraldehyde-preserved porcine xenografts in children. *Thoracic Cardiovascular Surgeon*, **29**, 361-374

Henze, A. and Fortune, R.L. 1974. Regurgitation and Haemolysis in artificial heart valves. *Scandinavian Journal of Thoracic Cardiovascular Surgery*, **8**, 167-175

Hoots, A.V., Watson, D.C. Jr. 1989. Construction of an aortic homograft conduit for right ventricle to pulmonary artery continuity. *Annals of Thoracic Surgery*, **48**, 5, 731-2

Horstkotte, D., Schulte, H.D., Loogen, F., Bircks, W. and Budde, T. 1984. Natural history versus heart valve replacement in severe mitral, aortic and double valve legions. *Proceedings of the 11th Annual Conference of the European Society for Artificial Organs*, 136-139

Horstkotte, D., Schulte, H.D., Loogen, F., Bircks, W. and Budde, T. 1984. Rest and exercise hemodynamics after mitral valve replacement using different allo- and xenografts. *Proceedings of the 11th Annual Conference of the European Society for Artificial Organs*, 128-131

Hufnagel, C.A. 1948. Resecting and grafting of the thoracic aorta with minimal interruption of the circulation. *Clinical Congress of the American College of Surgeons -Los Angeles, 1948.*

Hufnagel, C.A. 1951. Aortic plastic valvular prosthesis. *Bulletin of the Georgetown University Medical Center*, **4**, 60-61

Hufnagel, C.A. and Harvey, W.P. 1953. The surgical correction of aortic regurgitation. *Bulletin of the Georgetown University Medical Center*, **6**, 128-130

Idelson, S.R., Costa, L.E. and Ponso, R. 1985. A comparative computational study of blood flow through prosthetic heart valves using the finite element method. *Journal of Biomechanics*, **1 8**, 2, 97-115

ISO 5840, 1989

Jaroshinsky, J.N., Tchekanov, V.S. and Krassikov, L.I. 1984. Morphology and morphogenesis of implanted valve-containing prostheses of the pulmonary trunk for surgical correction of complex congenital heart diseases. *Biomaterials*, **5**, 180-182

Jeger 1923. Cited by Kuttner, H., *Chirurgische Operationslehre*, Ed. 5, Leipzig, Barth, Vol. 2

Juden, H., Gentle, C.R. and Dowson, D. 1983. Scanning electron microscopy examination of a porous alumina prosthetic heart valve. *Biomaterials*, **4**, 139-141

Kang, S.G. and Tarbell, J.M. 1983. The impedance of curved artery models. *Transactions of the A.S.M.E., Journal of Biomechanical Engineering*, **1 0 5**, 275-282

Kaster, R.L., Mantini, E., Tanaka, S. and Lillehei, C.W. 1967. Analysis of dynamic characteristics of heart valves in a pulse duplication - analog computer system. *Proceedings of the 5th National Biomedical Scientific Instrument Symposium*, **Vol. 4**, 112-118

Kouchoukos, N.T., Karp, R.B. and Lell, W.A. 1977. Replacement of the ascending aorta and aortic valve with a composite graft: results in 25 patients. *Annals of Thoracic Surgery*, **2 4**, 2, 140-148

Kouchoukos, N.T., Karp, R.B., Blackstone, E.H., Kirklin, J.W., Pacifico, A.D. and Zorn, G.L. 1980. Replacement of the ascending aorta and aortic valve with a composite graft: results in 86 patients. *Annals of Surgery*, 192, 3, 403-413

Kouchoukos, N.T. 1989. Aortic graft - valve (composite) replacement at 20 years: Wrap or no wrap? Shunt or no shunt? *Annals of Thoracic Surgery*, **4 8**, 5, 615 - 6

Langhaar, H.L. 1942. Steady flow in the transition length of a straight tube. *Journal of Applied Mechanics*, **6 4**, 55-58

Lawford, P.V., Roberts, K.V., Black, M.M., Drury, P.J. and Bilton, G. 1986. The in vivo durability of bioprosthetic heart valves - modes of failure observed in explanted valves. *Heart Valve Engineering, Seminar at the Institution of Mechanical Engineers 4-5 December 1986*

Leefe, S.E., Edwards, R.E., Tansley, G.D. and Gentle, C.R. 1986. Investigation into leakage design in prosthetic heart valves. *Proceedings of the 26th Annual Meeting of the Biological Engineering Society, Glasgow, 1986*

Leefe, S.E., Tansley, G.D. and Gentle, C.R. 1986a. Pulsatile flow testing of prosthetic heart valve conduits. *Heart Valve Engineering, Seminar at the Institution of Mechanical Engineers 4-5 December 1986*

Leefe, S.E. and Gentle, C.R. 1987. Theoretical evaluation of energy loss methods in the analysis of prosthetic heart valves. *Journal of Biomedical Engineering*, **9**, 121-127

Livi, U., Kay, P. and Ross, D. 1974. The pulmonary homograft: an improved conduit for RVOT reconstruction. *Circulation,* **7 4**, (Supplement 2), 250

LoGerfo, F.W. 1991. The blood-materials interaction and downstream effects in prosthetic conduits. *Journal of Vascular Surgery*, **1 3**, 5, 740-2

MacGregor, D.C., Pilliar, R.M., Wilson, G.J., Goldstein, W., Rubis, L.J., Blackwell, R., Lixfield, W., Scully, H.E. and Silver, M.D. 1976. Porous metal surfaces: a radical new concept in heart valve design. *Transactions of the American Society for Artificial Internal Organs*, **2 2**, 646-653

Markatos, N.C. 1986. The mathematical modelling of turbulent flows. *Applied Mathematical Modelling*, **1 0**, 190-220

Martin, T.R.P., Palmer, J.A. and Black, M.M. 1978. A new apparatus for the in vitro study of aortic valve mechanics. *Engineering in Medicine*, **7** , 4, 229-230

Maxted, K.J. 1982. Electromagnetic flowmetry. Chapter 7 of "*Blood Flow Measurement in Man*" (Ed. Mathie, R., Castle House, Tunbridge Wells)

Mayer, J.E., Lindsay, W.G., Wang, Y., Jorgensen, C.R. and Nicoloff, D.M. 1978. Composite replacement of the aortic valve and ascending aorta. *Journal of Thoracic and Cardiovascular Surgery*, **7 6**, 6, 816-823

Mazumdar, J. and Thalassoudis, K. 1983. A mathematical model for the study of flow through disc-type prosthetic heart valves. *Medical and Biological Engineering and Computing*, **2 1**, 400-409

McDonald, D.A. 1955. The relation of pulsatile pressure to flow in arteries. *Journal of Physiology*, **1 2 7**, 533-552

McGoon, D.C., Rastelli, G.C. and Ongley, P.A. 1968. An operation for the correction of truncus arteriosus. *Journal of the American Medical Association*, **2 0 5**, 2, 69-73

McGoon, D.C., Wallace, R.B. and Danielson, G.K. 1975. Transposition with ventricular septal defect and pulmonary stenosis: the Rastelli operation. *Israeli Journal of Medical Science*, **1 1**, 2-3, 82-88

McGoon, D.C. 1976. Left ventricular and biventricular extracardiac conduits. *Journal of Thoracic and Cardiovascular Surgery*, **7 2**, 1, 7-14

McGoon, D.C., Danielson, G.K., Schaff, H.V., Puga, F.J., Ritter, D.G., Mair, D.D., Ilstrup, D.M. and Edwards, W.D. 1982. Factors influencing late results of extracardiac conduit repair for congenital cardiac defects. *Cardiac Bioprostheses. Proceedings of the 2nd International Symposium* (Ed. Cohn, L.H.,and Galucci, V., Yorke Medical Books, New York), 217-230

Mcintyre, L.V. and Hubbell, J.A. 1986. Visualising clots on artificial surfaces. *Mechanical Engineering*, **January 1986**, 32-35

Mitamura, Y., Mikami, T., Suzuki, Y., Yamamoto, H., Okamoto, E. and Shimooka, T. 1985. Design of a motor driven artificial heart. *Design and Synthesis*, **1 9 8 5**, 701-705

Mohnhaupt, A., Affeld, K., Mohnhaupt, R. and Bücherl, E.S. 1975. A comparative performance analysis of heart valve prostheses. *Transactions of the European Society for Artificial Organs*, **2**, 39-45

Moore, C.H., Martelli, V. and Ross, D.N. 1976. Reconstruction of right ventricular outflow tract with a valved conduit in 75 cases of congenital heart disease. *Journal of Thoracic and Cardiovascular Surgery*, **7 1**, 1, 11-17

Mueller, T.J., Lloyd, J.R., Chetta, G.E. and Galanga, F.L. 1975. Effect of test section geometry on the occluder motion of caged ball prosthetic heart valves. *A.S.M.E. Symposium on Biomechanics*, 55-56

Mustard, W.T. 1964. Successful two-stage correction of transposition of the great vessels. *Surgery*, **5 5**, 469-472

Narducci, C., Russo, L. and Chidichimo, G. 1982. Hemodynamic properties of valved apico aortic conduit: noninvasive evaluation of bioprosthetic valves and of left ventricle dynamics. *Life Support Systems. Proceedings of the 9th Annual Meeting of the European Society for Artificial Organs*, 120

Nichols, W.W., Pepine, C.J. and Conti, C.R. 1983. Catheter tip manometer system: pressure and velocity measurement. *Advances in Cardiovascular Physics*, **Vol 5**, (part 2) 144-176

Nichols, W.W. 1983. Instantaneous ascending aortic blood flow derived from the pressure-gradient. *Advances in Cardiovascular Physics*, **Vol 5**, (part 2) 1-15

Niimi, H. and Sugihara, M. 1985. Cyclic loading on the red cell membrane in a shear flow: a possible cause of hemolysis. *Transactions of the A.S.M.E., Journal of Biomechanical Engineering*, **1 0 7**, 91-95

Norman, J.C., Nihill, M.R. and Cooley, D.A. 1976. Creation of double outlet left ventricles for left ventricular outflow obstructions: initial clinical results in six patients. *Transactions of the American Society for Artificial Internal Organs*, **2 2**, 332-339

Norwood, W.I., Freed, M.D., Rocchini, A.P., Bernhard, W.F. and Castaneda, A.R. 1977. Experience with valved conduits for repair of congenital cardiac legions. *Annals of Thoracic Surgery*, **2 4**, 3, 223-232

Olin, C. 1971. Pulsatile flow studies of prosthetic aortic valves. *Scandinavian Journal of Thoracic Cardiovascular Surgery*, **5**, 1-12

Orszulak, T.A., Clements, I.P. and Tinker, J.H. 1982. Bjork-Shiley leaflet impairment in an ascending aortic conduit due to extrinsic compression by a false aneurism. *Annals of Thoracic Surgery*, **3 4**, 6, 706-709

Parsons, R.W.T. 1981. Leakage flow testing of artificial heart valves. *Undergraduate project thesis,* Trent Polytechnic, Nottingham.

Pettifor, A.H. and Mockros, L.F. 1970. Hydrodynamic characteristics of prosthetic aortic valves. *Annals of Thoracic Surgery*, **9**, 2, 122-135

Piehler, J.M. and Pluth, J.R. 1982. Replacement of the ascending aorta and aortic valve with a composite graft in patients with nondisplaced coronary ostia. *Annals of Thoracic Surgery*, **3 3**, 4, 406-409

Quijano, R.C. 1980. Study of 208 explanted bioprostheses: distribution of dysfunction patterns and assessment of durability at 10 years of use. *National Bureau of Standards Special Publication 601.* Conference on Implant Retrieval, Materials and Biological Analysis, May 1-3 1980, Gaithersburg, Maryland, 363-393

Rastelli, G.C., Wallace, R.B. and Ongley, P.A. 1969. Complete repair of transposition of the great arteries with pulmonary stenosis. *Circulation*, **3 9**, 83-95

Reul, H., Giersiepen, M. and Knott, E. 1986. Laboratory testing of prosthetic heart valves. *Heart Valve Engineering, Seminar at the Institution of Mechanical Engineers 4-5 December 1986*

Rief, T.H., Cocolin, G. and Huffstutler, M.C. 1980. A note on the leakage of the new Omniscience/tm pivoting disc prosthetic heart valve. *Transactions of the A.S.M.E., Journal of Biomechanical Engineering*, **1 0 2**, 342-344

Rieu, R., Friggi, A. and Pelissier, R. 1985. Velocity distribution along an elastic model of human arterial tree. *Journal of Biomechanics*, **1 8**, 9, 703-715

Ritchie, R.O. and Lubock, P. 1986. Fatigue life estimation procedures for the endurance of a cardiac valve prosthesis. *Transactions of the A.S.M.E., Journal of Biomechanical Engineering*, **1 0 8**, 153-160

Roeva, L.A., Shatalov, K.V., Zorina, A.P. and Chekanov, V.S. 1990. Design of left ventricular-aortic conduits (review of the literature). *Biomedical Engineering* **2 3**, 6, 229-235

Rooz, E., Young, D.F. and Rogge, T.R. 1982. A finite element simulation of pulsatile flow in flexible obstructed tubes. *Transactions of the A.S.M.E., Journal of Biomechanical Engineering*, **1 0 4**, 119-124

Ross, D.N. and Somerville, J. 1966. Correction of pulmonary atresia with a homograft aortic valve. *The Lancet*, **1 9 6 6**, Vol. 2, 1446-1447

Rupprath, G., Vogt, J., DeVivie, E.R. and Beuren, A.J. 1981. conduit repair for complex congenital heart disease wit pulmonary atresia or right ventricular outflow tract obstruction. Part 2: Early and late hemodynamic and echocardiographic findings. *Thoracic Cardiovascular Surgeon*, **2 9**, 337-344

Rushmer, R.F. 1976. *"Cardiovascular Dynamics"*, 4th Edn., W.B. Saunders, Philadelphia, 1976.

Sabbah, H.N., Blick, E.F. and Stein, P.D. 1977. Effects of structural configuration of prosthetic aortic valves upon coronary blood flow. *British Journal of Surgery*, **64**, 561-566

Sabbah, H.N. and Stein, P.D. 1982. Mechanism of early systolic closure of the aortic valve in discrete membranous subaortic stenosis. *Circulation*, **65**, 2, 399-402

Sabbah, H.N. and Stein, P.D. 1984. Comparative study of the amount of backflow produced by four types of aortic valve prostheses. *Transactions of the A.S.M.E., Journal of Biomechanical Engineering*, **106**, 66-71

Sano, M., Nagahiso, T., Sasakawa, E., Yokoi, K. and Iwa, T. 1982. Hydraulic evaluation of heart prosthetic valves (in case of mechanical valve). *University of Kanazawa, Memoranda of the Faculty of Technology*, **15**, 2, 95-102 (In Japanese with English abstract)

Sauvage, L.R., Viggers, R.F., Berger, K., Wood, S.J., Rao, A.M., Dedomenico, M., Ross, M.A. and Robel, S.B. 1970. Aortic ball valve design based upon healing and hydraulic considerations. *Surgery*, **67**, 1, 151-167

Seki, J., Hayashi, K., Nakamura, T. and Umeza, M. 1985. Laser-Doppler and hot-film anemometers for measurement of unsteady turbulent flow behind a prosthetic heart valve. *Fluid control and Measurement. The Society of Instrument and Control Engineers, Japan.* **Vol. 1**, (Ed. Harada, M., Pergamon Press), 269-274

Selle, J.G., Robicsek, F., Daugherty, H.K., Cook, J.W. and Hess, P.J. 1981. Technical options in repairing the diseased ascending aorta with aortic valve involvement. *Annals of Thoracic Surgery*, **32**, 6, 578-583

Shanebrook, J.R. and Levine, M.L. 1979. Hemodynamics of left ventricular apex-aortic valved conduits. *Cardiovascular Diseases, Bulletin of the Texas Heart Institute*, **6**, 4, 425-438

She, J., Li, M., Wang, K. and Yu, Y. 1989. Waveform comparison and average value disposition for prosthetic heart valves in pulsatile flow in vitro. *Department of Anatomy, Shanghai Medical University.*

Shirer, H.W. 1962. Blood pressure measuring methods. *I.R.E. Transactions on Biomedical Electronics*, **9**, 116-125

Skalak, R., Wiener, F., Morkin, E. and Fishman, A.P. 1966. The energy distribution in the pulmonary circulation, I: Theory. *Physics in Medicine and Biology*, **1 1**, 2, 287-294

Skalak, R., Keller, S.R. and Secomb, T.W. 1981. Mechanics of blood flow. *Transactions of the A.S.M.E., Journal of Biomechanical Engineering*, **1 0 3**, 102-115

Smeloff, E.A., Huntley, A.C., Davey, T.B., Kaufmann, B. and Gerbode, F. 1966. Comparative study of prosthetic heart valves. *Journal of Thoracic and Cardiovascular Surgery*, **5 2**, 6, 841-848

Stelzer, P., Elkins, R.C. 1989. Homograft valves and conduits: applications in cardiac surgery. *Current Problems in Surgery*, **2 6**, 6, 381-452

Stevenson, D.M. and Yoganathan, A.P. 1985. Numerical simulation of steady turbulent flow through trileaflet aortic heart valves - 1. Computational scheme and methodology. *Journal of Biomechanics*, **1 8**, 12, 899-907

Stewart, S. 1976. Reconstruction of right ventricular-pulmonary artery continuity with a valved external conduit. Unusual technical considerations. *Journal of Thoracic and Cardiovascular Surgery*, **7 2**, 1, 39-47

Swales, P.D., Holden, M.P., Dowson, D. and Ionescu, M.I. 1973. Some experiments on tissue heart valve prostheses. *Engineering in Medicine*, **2**, 2, 27-31

Swanson, W.M. and Clark, R.E. 1974. Comparative prosthetic valve performance based on power measurements. *Proceedings of the 27th Annual Conference on Engineering in Biology and Medicine, Philadelphia, Pennsylvania*. 117

Swanson, W.M. and Clark, R.E. 1976. Testing of prosthetic heart valves. *A.S.M.E. Preprint No. 76-WA/Bio 3*

Tansley, G.D., Edwards, R.J., Leefe, S.E., Gentle, C.R. 1986. Ball occluder instability during forward flow through prosthetic heart valve conduits. *Proceedings of the 13th Annual General Meeting of the European Society for Artificial Organs. Life Support Systems*, **4**, Supplement 2, 169-191

Tansley, G.D. 1988. Computational investigation of turbulent, non-Newtonian flow in heart valve conduits. PhD Thesis, Trent Polytechnic, Nottingham.

Tansley, G.D., Edwards, R.E. and Gentle, C.R. 1988. The role of computational fluid mechanics in the analysis of prosthetic heart valve flow. *Medical and Biological Engineering and Computing*, **2 6**, 175-185

Taylor, D.E.M. and Whammond, J.S. 1977. The assessment of haemodynamic function of diseased and prosthetic heart valves in patients. *Journal of Medical Engineering and Technology*, 1 2, 81-85

Taylor, K.M. 1986. The clinical aspects of heart valve replacement: an overview. *Heart Valve Engineering, Seminar at the Institution of Mechanical Engineers 4-5 December 1986*

Thalassoudis, K. and Mazumdar, J. 1984. Mathematical model for turbulent blood flow through a disk-type prosthetic heart valve. *Medical and Biological Engineering and Computing*, **2 2**, 529-536

Tiederman, W.G., Steinle, M.J. and Phillips, W.M. 1986. Two-component laser velocimeter measurements downstream of heart valve prostheses in pulsatile flow. *Transactions of the A.S.M.E. Journal of Biomechanical Engineering*, **1 0 8**, 59-64

Tillmann, W. and Schlieper, H. 1979. Modification of miniature flush surface wall shear probes for biomedical use. *Journal of Physics, E: Scientific Instrumentation*, **1 2**, 371-372

Tillmann, W. and Schlieper, H. 1979. A device for the calibration of hot-film wall shear probes in liquids. *Journal of Physics, E: Scientific Instrumentation*, **1 2**, 373-380

Tillmann, W., Reul,H., Herold, M., Bruss, K.-H. and VanGilse, J. 1984. In vitro wall shear measurements at aortic valve prostheses. *Journal of Biomechanics*, **1 7**, 4, 263-279.

Tindale, W.B., Martin, T.R.P., Van Noort, R. and Black, M.M. 1981. Problems of measurement in aortic valve in vitro evaluation. *Proceedings of the 8th Annual Meeting of the European Society for Artificial Organs*, 356-360

Trowbridge, E.A. 1982. The fluid mechanics of blood: equilibrium and sedimentation. *Clinical Physics and Physiological Measurement*, **3**, 4, 249-265

Trowbridge, E.A. 1983. The physics of arteriole blood flow. I. General theory. *Clinical Physics and Physiological Measurement*, **4**, 2, 151-175

Trowbridge, E.A. and Meadowcroft, P.M. 1983. The physics of arteriole blood flow. II. Comparison with experiment. *Clinical Physics and Physiological Measurement*, **4**, 2, 177-196

Trowbridge, E.A. 1984. Mathematical concepts of blood flow and blood rheology. *Life Support Systems*, **2**, 25-38

Tuna, I.C. and Danielson, G.K. 1989. Use of conduits in surgical repair of congenital heart disease. *Annals of Thoracic Surgery*, **48**, 1, 143-4

Turinetto, B., DiBartolomeo, R., Marinelli, G., Galli, R. and Pierangeli, A. 1983. Surgical replacement of the aortic valve and of the ascending aorta with a composite graft. *Life Support Systems*, **1**, Supplement 1, 316-319

Uglov, F.G., Orlovskii, P.I., Mursalova, F.A., Evdokimov, S.V., Bushmarin, O.N., Yakovenko, V.V. and Belov, E.F. 1984. Investigation of self-induced vibrations of the ball closing an artificial aortic valve. *Biomedical Engineering*, **18**, 2, 48-50

Underwood, F.N. and Mueller, T.J. 1979. Numerical study of the steady axisymmetric flow through a disk-type prosthetic heart valve in an aortic-shaped chamber. *Transactions of the A.S.M.E., Journal of Biomechanical Engineering*, **101**, 198-204

Underwood, S.R., Firmin, D.N., Rees, R.S. and Longmore, D.B. 1990. Magnetic resonance velocity mapping. *Clinical Physics and Physiological Measurement*, **11** Supplement A, 37-43

Viggers, R.F., Robel, S.B. and Sauvage, L.R. 1967. A hydraulic figure-of-merit for heart valve prostheses. *Journal of Biomedical Materials Research*, **1**, 103-112

DeVivie, E.R., Hellberg, K., Kutzner, D.D., Rahlf, G., Rupprath, G. and Beuren, A.J. 1981. Conduit repair for complex congenital heart disease with pulmonary atresia or right ventricular outflow tract obstruction. Part 1: Surgical results. *Thoracic Cardiovascular Surgeon*, **29**, 329-336

Walburn, F.J., Sabbah, H.N. and Stein, P.D. 1985. Turbulent stresses in the region of a Hancock porcine bioprosthetic aortic valve. *Transactions of the A.S.M.E., Journal of Biomechanical Engineering*, **1 0 7**, 200-205

Walker, D.K., Scotten, L.N., Modi, V.J. and Brownlee, R.T. 1980. In vitro assessment of mitral valve prostheses. *Journal of Thoracic and Cardiovascular Surgery*, 79, 5, 680-688

Wells, R., Goldstone, J., Hutchins, P. and Schmid-Schonbein, H. 1970. Analysis and direct visualisation of effects of shearing forces upon thrombus formation. *Proceedings of the 23rd Annual Conference of engineering in Medicine and Biology, Washington, D.C.*, 97

Wheat, M.W., Wilson, J.R. and Bartley, T.D. 1964. Successful replacement of the entire ascending aorta and aortic valve. *Journal of the American Medical Association*, **1 8 8**, 8, 717-719

Wieting, D.W. 1969. "Dynamic flow characteristics of heart valves". PhD dissertation, University of Texas at Austin.

Womersley, J.R. 1955. Method for the calculation of velocity, rate of flow and viscous drag in arteries when the pressure gradient is known. *Journal of Physiology*, **1 2 7**, 553-563

Woo, Y.-R. and Yoganathan, A.P. 1986. In vitro pulsatile flow velocity and shear stress measurements in the vicinity of mechanical mitral heart valve prostheses. *Journal of Biomechanics*, **1 9**, 1, 39-51

Woodcock, J.P. 1985. Noninvasive blood flow measurement. *C.R.C. Critical Reviews in Biomedical Engineering*, **1 2**, 3, 201-235

Wright, J.T.M. 1977. An in-vitro assessment of the hydraulic characteristics of the Mark II Abrams-Lucas mitral valve prosthesis. *Thorax*, **3 2**, 296-302

Wright, J.T.M. and Brown, M.C. 1977. A method for measuring the mean pressure gradient across prosthetic heart valves under in vitro pulsatile flow conditions. *Medical Instrumentation*, **1 1**, 2, 110-113

Wright, J.S., Thomson, D.S. and Warner, G. 1981. Mitral valve bypass by valved conduit. *Annals of Thoracic Surgery*, **3 2**, 3, 294-296

Yellin, E.L., Silverstein, M., Frater, R.W.F. and Peskin, C. 1970. Pulsatile flow dynamics across the natural and prosthetic mitral valve. *Proceedings of the 23rd Annual Conference of Engineering in Medicine and Biology, Washington, D.C.*, 96

Yoganathan, A.P., Reamer, H.H., Harrison, E.C. and Corcoran, W.H. 1979. Laser-Doppler anemometer to study velocity fields in the vicinity of prosthetic heart valves. *Medical and Biological Engineering and Computing*, **1 7**, 38-44

APPENDIX

PUBLISHED PAPERS

Leefe, S.E., Edwards, R.E., Tansley, G.D. and Gentle, C.R. 1986

Investigation into leakage design in prosthetic heart valves.

Proceedings of the 26th Annual Meeting of the Biological Engineering Society, Glasgow, 1986

TITLE: INVESTIGATION INTO LEAKAGE DESIGN IN PROSTHETIC HEART VALVES

ABBREVIATED TITLE: Valve leakage flow

AUTHORS: S.E. LEEFE, R.J. EDWARDS, G.D. TANSLEY, C.R. GENTLE

ABSTRACT

The design of pivoting or tilting occluder prosthetic heart valves deliberately incorporates a degree of leakage in the "shut" position in order to effect a washing action, thus preventing clot formation. It has been suggested that experimentally determined relationships between friction factor and Reynolds number for leakage flow demonstrate turbulence and that this could represent a means of improving the washing action.

This paper presents a physical argument augmented by numerical simulation using the "Phoenics" finite difference code to offer an alternative explanation for the form of the friction factor versus Reynolds number curve. Some shortcomings of designing for a turbulent leakage flow are highlighted and guidelines are recommended on the suitable choice of leakage gap dimensions.

INTRODUCTION

The design of pivoting or tilting occluder prosthetic heart valves has, over recent years, deliberately incorporated a degree of leakage flow in the "closed" position. This is intended to inhibit thrombus formation both by preventing the blood from becoming stagnant and, principally, by forcing a high pressure jet through the leakage gap, thus effecting a scouring action.

Leakage flow has been studied by Rief et al (1980) and Gentle (1982), with a view, in each case, to demonstrating the superior characteristics of one valve over another, although the criteria for comparison differ. Rief et al were apparently seeking to minimise leakage, whilst Gentle was concerned with characterising the flow regime on the grounds that turbulent leakage flow would produce a more effective washing action than laminar. Both studies presented experimental data in terms of pressure drop versus flow rate and both used the gradient of a logarithmic plot of friction factor versus Reynolds number to draw conclusions as to the nature of the flow, by comparison with the expected values for fully-developed pipe flow. It is apparent, however, that there was poor quantitative agreement between such predicted curves and the experimental data. Rief et al seemed aware of this point and offered a plausible explanation in terms of the irregular pattern of leakage flow. Indeed, Parsons (1982) presented a similar explanation for his findings on the Bjork-Shiley valve's leakage behaviour, which could go some way towards accounting for Gentle's results with this valve.

All of the valves studied produced curves whose gradients were less than predicted and one had a measured friction factor an order of magnitude larger than expected. The explanation offered, namely that the high friction factor was caused by surface roughness, is questionable when considering the curves for fully turbulent flow in rough pipes. These ambiguities are clearly apparent when experimental data are superimposed on the Moody chart, figure 1.

Since the data for Gentle's twin-flap valve shows the furthest departure from theoretical predictions, and since no other plausible explanation for this discrepancy has been offered elsewhere, this paper will concern itself only with this valve. Its purpose is to offer an alternative explanation for the form of the non-dimensional plots of experimental data. This explanation, in turn, calls into question the idea of designing for turbulent leakage and suggests a basis for the rational selection of leakage gap dimensions. The work also forms part of the development programme for an all-ceramic twin-flap mitral valve (Gentle, 1980, Gentle, Arundel et al, 1981 and Gentle, Juden et al, 1981).

ANALYSIS

The accepted equations describing fully-developed laminar or turbulent flow in smooth pipes are:

$$f = \frac{2.4}{Re} \quad \text{and} \quad f = \frac{0.079}{Re^{0.25}} \qquad - (1)$$

respectively, where f is the friction factor, defined by:

$$f = \frac{\tau_w}{\frac{1}{2}\rho\bar{u}^2} \qquad - (2)$$

and the Reynolds number:

$$Re = \frac{2\bar{u}\delta}{\nu} \qquad - (3)$$

is based on the hydraulic diameter, which, for a slit, is 2δ, where δ is the slit width. Here, τ_w is the wall shear stress, ρ is the fluid density, ν is its kinematic viscosity and $\bar{u}$ is the mean velocity. In each case the equation arises by considering a fully-developed velocity profile, parabolic for laminar and $^1/_7$th power law for turbulent. The problem with using these to characterise leakage flow through a closed prosthetic heart valve is that the axial path length is so short that the conditions could only be described as entry flow, and therefore it is unreasonable to assume that the velocity profile through the gap is the same as it would be for fully-developed conditions. This will have twofold significance. Firstly, the profile will be changing throughout the length and secondly, it will be flatter than the fully-developed profile for the same flowrate and will, therefore, give rise to a greater wall velocity gradient, hence friction factor.

For simplicity, we will concentrate on the effect of this "fuller" profile and assume that its shape lies somewhere between the totally flat and the fully-developed and remains unchanged throughout the

length of the leakage gap. Three further assumptions are made, as follows:-

1) Since the leakage gap is a thin slit whose breadth is some 30 times its width, a "parallel plate" analysis is more appropriate than a "circular pipe" analysis. Plate separation is taken as δ.

2) Velocity profile is defined, to the centreline, as:

$$\frac{u}{u_m} = \left\{\frac{y}{(\delta/2)}\right\}^{\frac{1}{n}} \qquad - (4)$$

where u_m is the maximum (centreline) velocity and y is the distance from the wall. Here, the value of n defines the shape of the velocity profile: the larger its value the "fuller" the profile. For fully developed turbulent flow, n=7.

3) The non-dimensional relationship:

$$\frac{u}{u^*} = K_1 \left\{\frac{u^* y}{\nu}\right\}^{\frac{1}{n}} \qquad - (5)$$

holds, where u* is the "friction velocity", $\sqrt{\tau_w/\rho}$. K_1 is taken as a constant over the relevant range of Reynolds number (although it is, in fact, a weak function of Re).

We now follow the scheme of Duncan et al (1970) with appropriate modifications for parallel plate theory. Equation (5) evaluated at the centreline yields:

$$\frac{u_m}{u^*} = K_1 \left\{ \frac{u^* \delta}{2\nu} \right\}^{\frac{1}{n}} \qquad - (6)$$

whilst the definition of mean velocity, $\bar{u}$, together with the assumed velocity profile of equation (4), gives:

$$\frac{\bar{u}}{u_m} = \frac{n}{(n+1)} \qquad - (7)$$

Equations (6) and (7) combine to give:

$$u^* = \left(\frac{2\nu}{\delta}\right)\left(\frac{n+1}{n K_1}\right)^n \bar{u}^n \qquad - (8)$$

and substituting the definitions of friction factor and Reynolds number (equations (2) and (3)) yields the result:

$$f = K\, Re^{-\frac{2}{(n+1)}} \qquad \text{where} \qquad K = 2^{\frac{(n+5)}{(n+1)}} \left(\frac{n+1}{n K_1}\right)^{\frac{2n}{(n+1)}} \qquad - (9)$$

Thus, the gradient of the log(f) vs log(Re) curve is $-2/(n+1)$, so that a "fuller" profile (larger n) results in a shallower gradient.

Regressional analysis of Gentle's data yields:

$$f = 0{\cdot}2998\, Re^{-0{\cdot}1131} \qquad - (10)$$

so that, for the twin-flap valve we have:

$n = 16{\cdot}7$ and $K_1 = 3{\cdot}15$

Combining equations (4) and (7) yields:

$$\frac{u}{\bar{u}} = \left(1 + \frac{1}{n}\right)\left\{\frac{y}{(\delta/2)}\right\}^{\frac{1}{n}} \qquad - (11)$$

The velocity profile defined by equation (11) with $n = 16{\cdot}7$ is shown together with that for n=7 (fully developed turbulent) for the same mean velocity, in figure 2. It is apparent that this profile is significantly flatter, and hence has a steeper wall velocity gradient resulting in the elevated friction factor observed in practice. Further, the flat profile resulting from the fact that the flow is not fully-developed, explains the shallow gradient of Gentle's curve.

The high friction factor can also be explained by considering that the closed valve constitutes a converging duct, for which the momentum theorem yields:

$$\tau_w = -\frac{\delta}{2}\frac{d}{dz}\left(p + \tfrac{1}{2}\rho\bar{u}^2 + \rho g h\right) \qquad - (12)$$

where p is the static pressure, h the elevation and z the flow direction. Thus, to assume f is proportional to Δp is to neglect the velocity and elevation terms. Order-of-magnitude calculation shows the velocity term is comparable with the pressure term. Thus

for the analysis to be valid, $\bar{u}$ must be assumed constant throughout the slit, i.e. that the slit is of constant width. This constrains us to a short length around the flap base where $\bar{u}$ is already large and so the pressure is significantly lower than its assumed inlet value. Thus, the quoted pressure drop, hence friction factor, is too large.

NUMERICAL MODELLING

Leakage flow was modelled using the "Phoenics" finite difference package. Since quantitative information was required only for flow through the leakage gap itself, a 2-dimensional model was employed. Since flow is symmetrical, only a half-plane was modelled. The flowrate was set at 2.5 litres/min (4.2×10^{-5} m^3/s). A converged solution was obtained whose results are summarised on the streamline plot of figure 3. There is turbulence as the jet emerges. Whilst the highly elliptic nature of the flow field has produced considerable upstream turbulence, flow over most of the flap surface, except for a small region at its base, is laminar. Two stagnation points are apparent on the occluder surface: one on the downstream face and the other just distal to the flow separation point at the base. This, significantly, coincides with a small local laminar region. The maximum shear stress in the gap is calculated as 2.2 kN/m^2.

DISCUSSION

As demonstrated above, flow through the gap itself, albeit not fully-developed, does appear to be turbulent, with the accompanying advantages, but consideration of where on the occluder surface this turbulence lies reveals that it is primarily in regions which are best washed in forward flow anyway (see figure 4). Indeed, Gentle, Juden et al (1981) report that the hinges have been deliberately placed so as to ensure adequate washing action between the flaps when open. However, the two places at which there is stasis when the valve is shut are precisely the same points most likely to provide stagnation during forward flow, namely the base where the dividing streamline impinges and the flap tip where separation will occur.

As reported above, the maximum shear stress was 2.2 kN/m². The pressure drop for this flowrate was approximately 9 kN/m², since this was central to the reported test range. Rough calculation shows that a more typical physiological pressure differential of 17 kN/m² would yield a wall shear stress of 4.2 kN/m² for the twin-flap valve, as compared with 0.62kN/m² for the Bjork-Shiley valve (for the same pressure drop) estimated from the equation:

$$\tau_w = \frac{\delta}{2} \frac{\Delta p}{l} \qquad - (13)$$

where l is the leakage gap length in the flow direction. Blackshear (1972) states that short-duration shear stress over 4 kN/m² will cause haemolysis, probably by cell membrane rupture, whilst there is

a second mechanism requiring a shear stress level of about 0.1 kN/m^2 sustained for several seconds. Opinion is divided as to whether the resulting cell damage is caused by mechanical tearing of long thin processes drawn out when cells anchor to the wall, or by laminar shear stress in the bulk flow. The former mechanism is thought to be the principal mode of haemolysis at prosthetic surfaces and is extremely dependent on surface treatment. Clearly then, despite the choice of porous alumina to encourage tissue growth to reduce the chance of haemolysis at the surface, the values of shear stress produced merely by the choice of leakage gap are unacceptably high for clinical use.

Finally, to produce turbulent leakage, the gap must be wide, producing large backflow. For example, quoted flow for a back pressure of 20 kN/m^2 is 3.95 litres/min (6.6×10^{-5} m^3/s). Assuming a typical mean forward flow of 25 litres/min (4.2×10^{-4} m^3/s), a regurgitant fraction well over 15% is readily produced.

LEAKAGE GAP DIMENSIONS

Evidently, a wide leakage gap can have serious consequences, so design criteria are required. Obviously, it is of prime importance to ensure that any planned washing action scours the surface regions where thrombus formation is most likely. A lower limit on leakage flow might be suggested by that necessary to prevent cell adhesion to the occluder surface, but, as previously mentioned, the nature of the surface is the significant factor here. However, since it is

agreed that the washing action of leakage flow is desirable, it seems sensible not to limit this to a minimum, but rather to fix gap width around an upper limit set by consideration of haemolysis or regurgitation. Such a criterion might be the 4 kN/m² shear stress for haemolysis by cell membrane rupture. This, however, is likely to produce a large gap and hence excessive regurgitation. Also, Blackshear's comments concerning the mechanisms of haemolysis at prosthetic surfaces indicate that the 4 kN/m² shear stress criterion may be too high in these circumstances. Since the 0.1 kN/m² shear stress condition applies to situations in which this level is sustained for several seconds, it does not appear relevant for pulsatile cardiac flow, and anyhow, would lead to leakage gaps only a few microns wide.

This leaves the acceptable regurgitant fraction as the criterion for an upper limit. Unfortunately, since the known physiological parameter is the pressure difference across the closed valve, calculation of regurgitant flow presupposes a knowledge of the Q- p relationship. We have seen that although correlation with fully-developed predictions is poor, it is better for Reynolds numbers well into the laminar regime -- possibly good enough for a rough design calculation. Thus, for slit flow:

$$\delta = \left(\frac{12\mu l Q}{w \Delta p}\right)^{\frac{1}{3}} \qquad -(14)$$

where μ is the dynamic viscosity, Q is the volumetric flowrate and w is the leakage gap breadth, dictated by valve geometry. Equation (14) could yield an estimate of δ required to produce an acceptable

leakage flow for a given pressure drop. We may also check whether the Reynolds number:

$$Re = \frac{2\rho Q}{\mu w} \qquad - (15)$$

is sufficiently low. Equation (13) could then produce a rough check on the shear stress level.

CONCLUSION

In order to obtain a turbulent washing action, one must risk excessive regurgitation, high shear stress and consequent mechanical haemolysis. Even then the turbulence is not necessarily produced where washing is most needed. It seems more sensible, therefore, to design for laminar flow through a well-placed leakage gap in a valve made from a suitable material. This has been seen to provide adequate protection against thrombus formation and haemolysis in clinical use.

REFERENCES

Blackshear, P.L. (1972). Mechanical hemolysis in flowing blood, in: Y.C. Fung, N. Perrone and M. Anliker (eds.), Biomechanics -- Its Foundations and Objectives, Prentice-Hall, Englewood Cliffs, N.J.

Duncan, W.J., Thom, A.S. and Young, A.D. (1970). Mechanics of Fluids, Edward Arnold, London, pp.311-313.

Gentle, C.R. (1980). A porous ceramic mitral valve prosthesis, in: G.W. Hastings and D.F. Williams (eds.), Mechanical Properties of Biomaterials, John Wiley & Sons, New York.

Gentle, C.R. (1982). Characterisation of leakage flow in cardiac valve prostheses. Proceedings of ESAO, vol IX, 102-106.

Gentle, C.R., Arundel, P.A., Hamilton, D.I. and Swales, P.D. (1981). Development of a twin-flap porous ceramic mitral valve prosthesis. J. Biomed. Eng., 3, 3-8.

Gentle, C.R., Juden, H. and Swales, P.D. (1981). Testing of an all-ceramic mitral valve prosthesis. Proceedings of ESAO, vol VIII, 352-355.

Parsons, R.W.T. (1981). Leakage flow testing of artificial heart valves. Undergraduate project thesis, Trent Polytechnic, Nottingham.

Rief, T.H., Cocolin, G. and Huffstutler, M.C. (1980). A note on the leakage of the new Omniscience/tm pivoting disk prosthetic heart valve. J. Biomech. Eng. Trans. ASME, 102, 342-344.

FIGURE CAPTIONS

Figure 1. Experimental leakage flow data superimposed on Moody chart for fully-developed pipe flow.

Figure 2. Predicted and estimated velocity profiles in the leakage gap.

Figure 3. Summary of numerical model results for the twin-flap valve.

Figure 4. Forward flow through the twin-flap valve.

AUTHORS' AFFILIATIONS

S.E. LEEFE BA (Oxon)

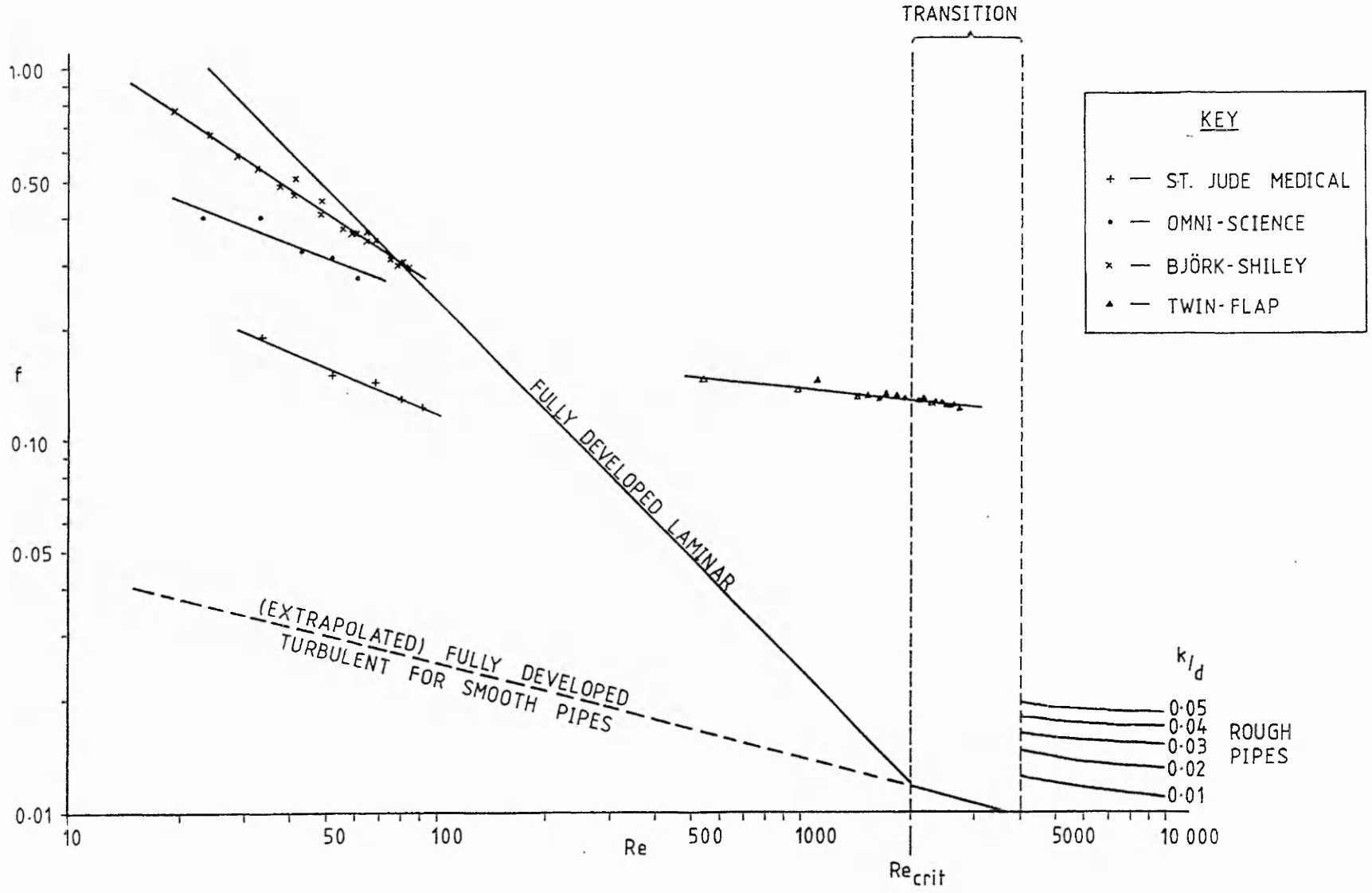
R.J. EDWARDS BSc (Hons)

G.D. TANSLEY BSc (Hons)

C.R. GENTLE PhD, BSc, DIC, ARCS, MBES, MESAO

Department of Mechanical Engineering, Trent Polytechnic,
Burton St., Nottingham NG1 4BU, UK.

Queries to: Dr. C.R. Gentle


TRANSITION
KEY
+ — ST. JUDE MEDICAL
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× — BJÖRK-SHILEY
▲ — TWIN-FLAP
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FULLY DEVELOPED LAMINAR
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Fig 1

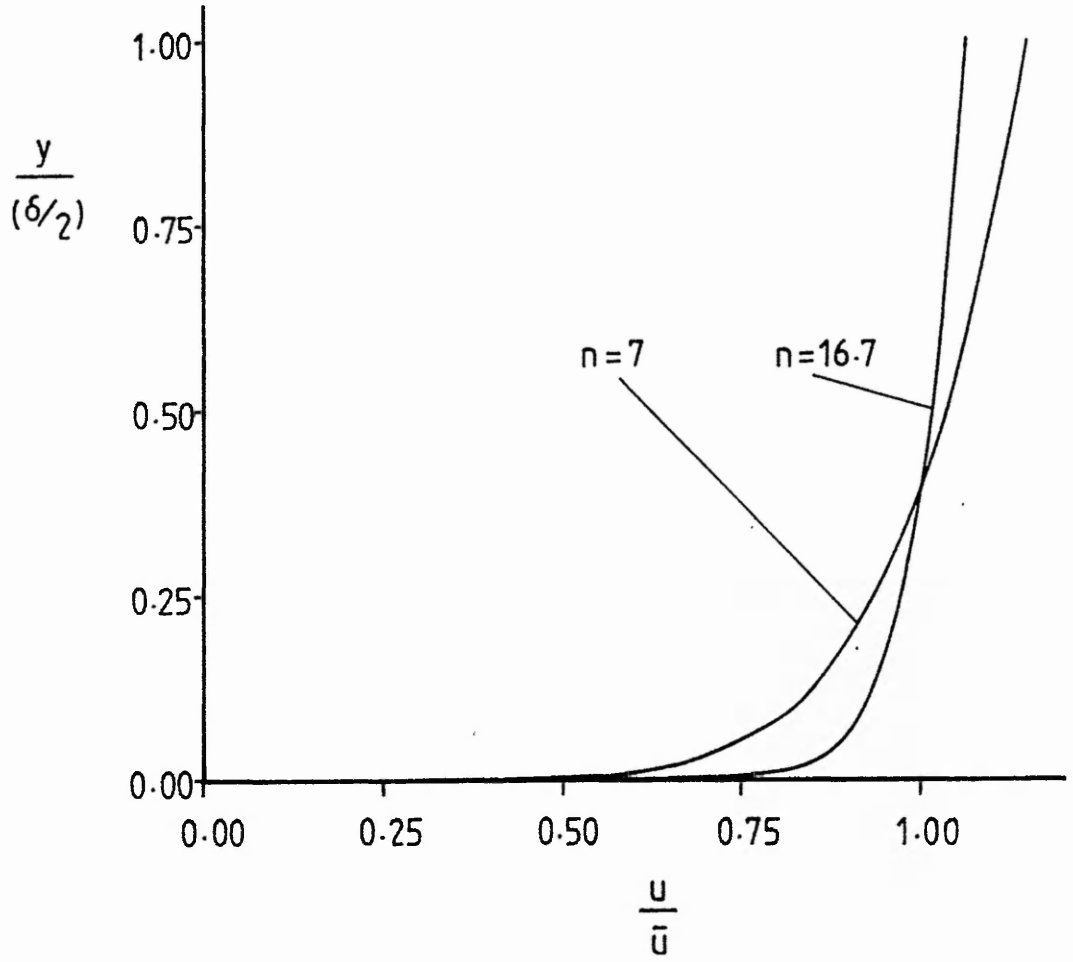

Fig 2

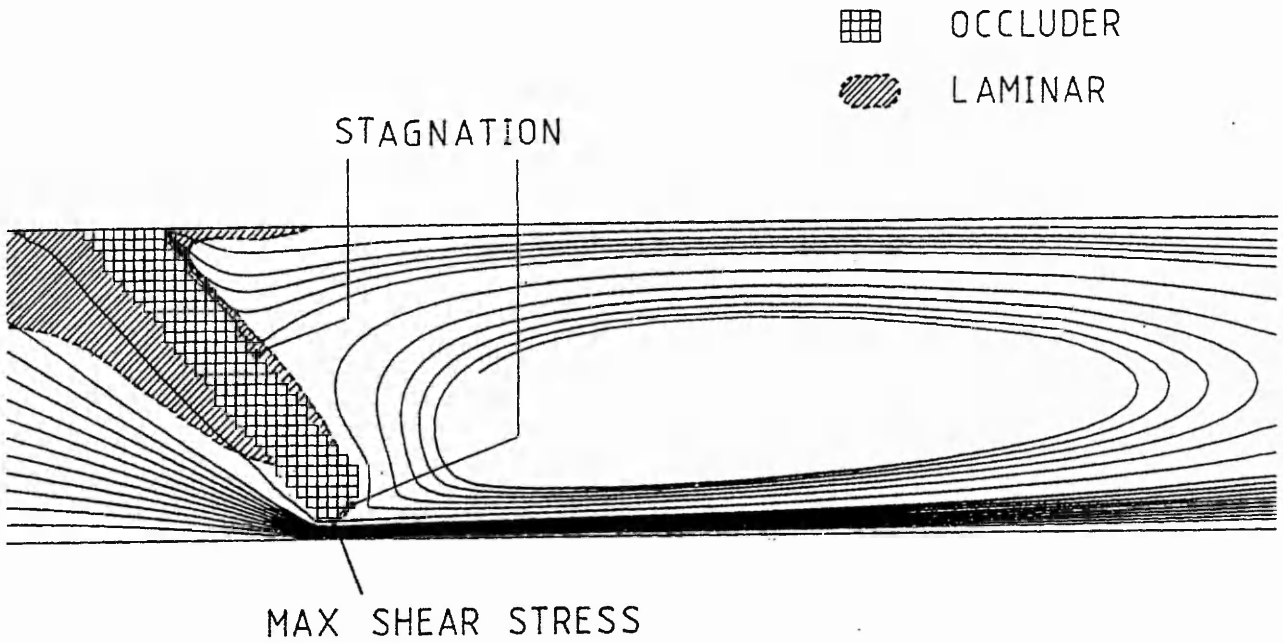

Fig 3

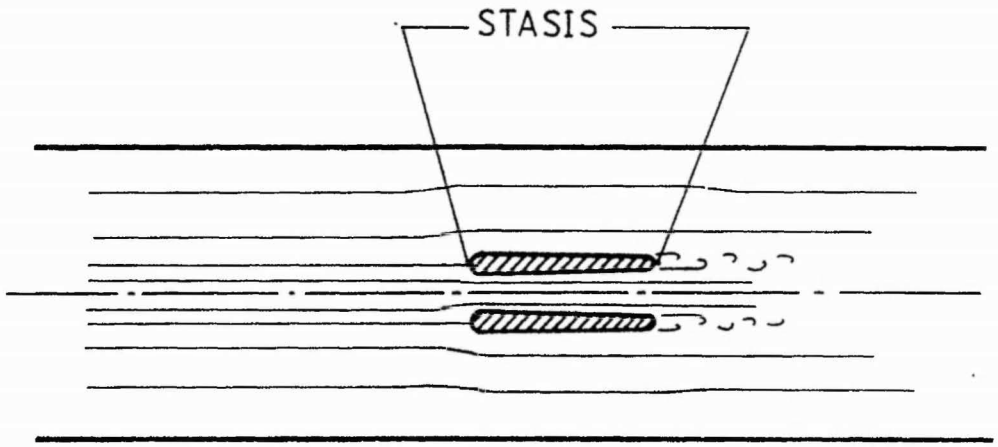

Fig 4

Tansley, G.D., Edwards, R.J., Leefe, S.E., Gentle, C.R. 1986

Ball occluder instability during forward flow through prosthetic heart valve conduits.

Proceedings of the 13th Annual General Meeting of the European Society for Artificial Organs. Life Support Systems, 4, Supplement 2, 169-191

BALL OCCLUDER INSTABILITY DURING FORWARD FLOW THROUGH PROSTHETIC HEART VALVE CONDUITS

G.D. Tansley, R.J. Edwards, S.E. Leefe, C.R. Gentle

Department of Mechanical Engineering, Trent Polytechnic, Nottingham, UK

Preliminary tests on a ball occluder heart valve conduit (1,2) have highlighted a potentially problematic design area. The valve consists of a conical inlet section against which the ball seats during the reverse pressure phase and a downstream tapered section with three rigid struts which restrain the occluder during forward flow. However, axial oscillation of the ball was noted at all but the very lowest forward flow rates. This is in keeping with the findings of other researchers (3,4,5). Streak photography has revealed that this was not the result of vortex shedding. Further investigation by Benjamin (2) has suggested that the cause was an increase in flow area immediately downstream of the ball centre where there is a transition from annular flow around the ball to pipe flow down the exit section, producing a decrease in velocity and a rise in pressure sufficient to cause a resultant backwards force on the ball, lifting it clear of the struts and towards the inlet section. This would cause a back-pressure which, in turn, would force the ball downstream again for the cycle to repeat itself. An "order of magnitude" calculation using Bernoulli's equation revealed that a sufficient pressure rise was indeed produced. A new outlet section was designed to provide constant flow area with the ball in the fully open position and was found to have cured ball vibration. In addition, a numerical simulation of flow through the improved conduit was developed and checked against the prototype by comparing predicted and measured overall pressure drops. This model confirmed the absence of a net upstream force on the ball in the fully open position.

Methods

Based on the considerations outlined above, an further prototype was developed, manufactured and tested. The aim was to maintain a constant flow area around the downstream side of the ball. Since the "flow area" means that part of a given cross-section "seen by" the flow or alternatively the area of the surface which is everywhere normal to the velocity but whose outer edge defines the cross-section, it should be apparent that to keep this quantity constant required a prior knowledge of the velocity field that the new profiled downstream section would produce. Since this could not be attained, calculation had to proceed on conservatively assumed velocity directions. The flow area was defined as normal to the ball surface (hence conical in form) and the velocity taken as everywhere normal to the flow area. Referring to figure 1, it can be seen that flow area was given by

$$A = \int_{Rb}^{Ro} 2\pi r \sin\theta \, dr$$

and hence

$$Ro = \sqrt{A/(\pi \sin\theta) + Rb^2}$$

where A is kept constant and equal to the area of the inlet pipe. The locus of Ro fixed the downstream conduit profile until it intersected with the cylindrical outflow pipe. An estimate was made of the error of the "constant flow area" assumption by taking a revised assessment of the flow direction as being everywhere parallel to the bisector of the angle between the tangents to the ball and the conduit inner surface (i.e. the flow was somewhere between tangential to the ball and tangential to the conduit surface). The

assumed flow area was thus determined to be no worse than 1.2% in error and was such as to produce a reduced area, resulting in slightly lower pressure and hence further counteracting the cause of the problem. A prototype was manufactured and tested under steady flow conditions using water. It was also decided to provide pressure tappings the same distances upstream and downstream of the inlet section as for the original design in order to investigate the effect of the modification on pressure drop characteristics.

The new design was also modelled mathematically using the "Phoenics" finite difference procedure running on a Vax 11/785 mainframe computer. This was for two reasons. Firstly, a check was required that the pressure distribution was no longer such as to produce a net backwards force. Secondly, if the pressure drop predicted by "Phoenics" for a given flow rate agreed with that determined experimentally for the conduit, then one could simply run the program to test subsequent design modifications without the necessity of building a prototype for each, until the design was finalised. A three-dimensional grid was fitted to the conduit dimensions. Figure 2 illustrates a typical diametral half-plane. The restraining struts were omitted for ease of modelling, whilst the ball was specified as fixed in space at the fully open position. A uniform inlet velocity of 0.625 m/s was specified, corresponding to a volumetric flowrate of 7.54 l/min and a Reynolds number based on conduit inner diameter of 10 000, which was within the test range. Accordingly, a turbulent solution was produced. A long inlet section was specified to allow for the development of a reasonable velocity profile. Fluid properties were taken as those of water since the instability problem was observed during tests in water. A downstream pressure of zero was specified so that the pressure drop across the valve could be read directly as the pressure at the valve entry section. The output file contained a list of pressures and two velocity components, axial and radial, for each grid cell of a diametral half-plane. Circumferential velocity was not required since the flow was assumed axi-symmetric. A program was written to calculate the net pressure and shear forces on the ball from the pressure and velocity distributions around it. Clearly, the resultant ball force was the sum of the two.

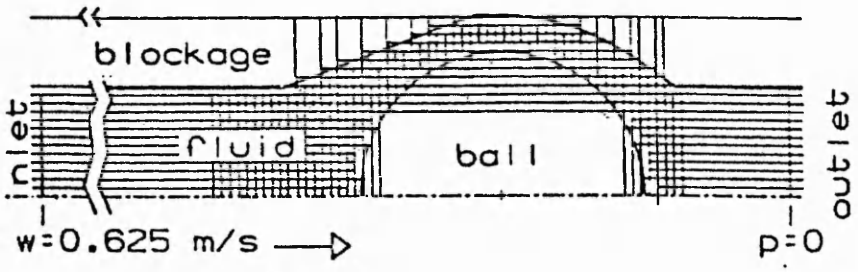

Figure 2

Results

Tests of the improved prototype over the entire range of flowrates to which the original was subjected and found to be unstable showed that there was no tendency for the ball occluder to oscillate nor lift off the restraining struts. Figure 3 shows the pressure drop measurements for the two valves.

Discussion

The primary object of the investigation was to establish the cause of ball occluder instability and to effect its cure. Clearly this has been achieved. However, the pressure drop experiments have indicated that the re-profiled downstream section has resulted in a reduction in forward flow efficiency of

the valve. The cause could well be separation at the abrupt direction change where the outflow pipe meets the valve body. Clearly, this can be prevented by further streamlining - either a blending radius or a small Venturi throat. Further scope for improvement is offered by the incorporation of a similar design procedure for the inlet section, based on maintaining a constant flow area around the upstream side of the fully-open ball, improving the streamlining here and thus reducing separation losses and ensuring that a sensibly predictable pressure pattern is maintained throughout. Closing function would not be jeopardised since backward displacement of the ball would immediately result in the reduction of the upstream flow area causing increased velocity and decreased pressure upstream, thus assisting closure.

Whilst the foregoing is encouraging for the conduit designer, unfortunately it is of little comfort to the manufacturer of prosthetic heart valves for straightforward replacement operations, since here there is no scope for profiling the heart or the major vessels. Indeed, the aortic root would seem to be a case in point, where flow between the orifice and occluder is at high velocity which drops downstream where flow is into the sinuses of Valsalva, which constitute a significant enlargement.

Conclusion

It has been demonstrated that the problem of instability is closely connected with pressure distribution around the occluder and that the designer can successfully guard against this by careful selection of the conduit profile surrounding the occluder. However, there remains a potential problem with the use of caged-occluder type prostheses for straightforward valve replacement, where the downstream area cannot be shaped.

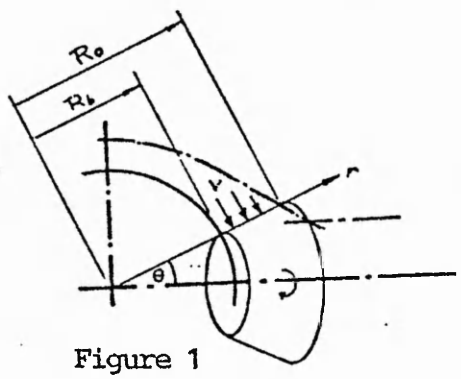

Figure 1

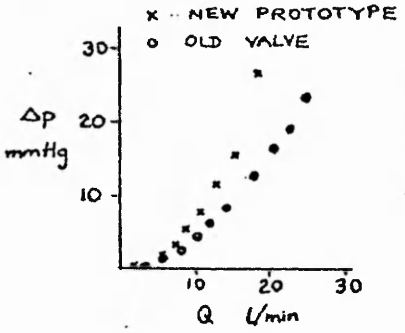

Figure 3

References

1. Gentle, C.R. (1983) Minimization of pressure drop across heart valve conduits: a preliminary study. Life Support Systems, 1, 263.
2. Benjamin, E.L. (Unpublished) Steady flow investigations of valved heart conduits. MSc Thesis, Trent Polytechnic, Nottingham, pp51-52,72-88.
3. Uglov, F.G. et al (1978) Distribution of pressure on the surface of the closing elements of artificial heart valves. Biomedical Engineering, 12, 61.
4. Uglov, F.G. et al (1984) Investigation of self-induced vibrations of the ball closing an artificial aortic valve. Biomedical Engineering, 18, 45.
5. Viggers, R.F., Robel, S.B. and Sauvage, L.R. (1967) A hydraulic figure of merit for heart valve prostheses. Journal of Biomedical Materials Research, 1, 103.

Address for reprints

Dr. C.R. Gentle, Department of Mechanical Engineering, Trent Polytechnic, Burton Street, Nottingham NG1 4BU, UK

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Pulsatile flow testing of prosthetic heart valve conduits.

Heart Valve Engineering,
Seminar at the Institution of Mechanical Engineers 4-5 December 1986

Pulsatile flow testing of prosthetic heart valve conduits

S E LEEFE, G D TANSLEY and C R GENTLE
Department of Mechanical Engineering, Trent Polytechnic, Nottingham, UK

SYNOPSIS A pulse duplicator for testing prosthetic heart valve conduits is described. The model left ventricle permits the testing of a conduit either as a straightforward valve replacement or as an apico-aortic shunt. Drive is provided by a microcomputer-controlled servomotor, operating a piston forcing an hydraulic fluid into a chamber surrounding the model ventricle, thus providing direct programmable control of ventricular volume throughout the cycle. Pressure and flow signals are digitised, stored and processed in a microcomputer to yield performance data in terms of energy loss.

The first phase of the research programme concerns the testing of existing conduit designs. The second will incorporate particle image velocimetry for flow visualisation to highlight regions of stasis and high shear rate. These findings, in conjunction with the results of the tests oulined above, will form the basis for the development of a new valved conduit.

1 INTRODUCTION

Artificial heart valves have been the subject of extensive testing and development work over the years. Consequently, there is a large array of prostheses available to the surgeon and the choice of replacement valve can be determined by referring to fairly exhaustive in vitro and clinical performance data as well as considering cost. Unfortunately, the choice of prosthesis for conduit operations is not so scientific. Not only is there a far more restricted choice available, but the extent of published performance data is limited. Indeed, the authors know of no references containing pulsatile flow test results for prosthetic heart valve conduits. This results from a lack of development work, which is surprising, given the scope for improvement afforded by the absence of geometrical constraints which the surrounding vessels impose on the design of valves for straightforward replacement. Instead, the strategy has been to incorporate a valve , which has been designed to separate two chambers, into a length of conduit tubing. Clearly, this is a different fluid mechanical situation and, as such, is unlikely to be optimal. This approach also unnecessarily restricts the type of valve which can be employed in a conduit. For example, if a Starr-Edwards valve were to be sewn into straight tubing, in the open position the ball occluder would still occupy nearly all of the cross-section: a downstream enlargement is essential for its efficient operation. Thus we can see that a pulsatile flow test facility for valved conduits is necessary both to provide the surgeon with performance data and to enable development work to proceed.

Since valved conduits are employed in a variety of ways it is logical to test their performance in different modes of usage. For this reason the pulse duplicator allows a conduit to be tested as an apico-aortic shunt by providing the model ventricle with an optional second outflow. The fluid circuit compliance and resistance can be adjusted to model either pulmonary or systemic load conditions. Great flexibility is also afforded by the use of computer-generated driving waveforms for ventricular volume. One is not constrained to a sinusoidal function, although, should one wish to use this as a suitable standardised basis for testing, one is at liberty to vary its amplitude and frequency virtually continuously. One can employ a physiological-type waveform, vary its amplitude and frequency, alter the ratio of filling to emptying time or even study the effect of arrhythmia. By designing versatility into the pulse duplicator one has a valuable research tool.

2 DESCRIPTION OF APPARATUS

2.1 Fluid circuit

The heart is essentially a positive displacement pump. Changes in ventricular volume determine the flow of blood into or out of the chamber and pressures throughout the system are a function of the load impedance. This standpoint justifies the pulse duplicator design. See Figures 1 and 2. A flexible membrane, semi-elliptical in cross-section, cast in cold cure clear silicone rubber (MDX-4-4210, Dow Corning) models the ventricle walls. This sits inside a cylindrical acrylic enclosure and is sealed from above by a plate containing inlet and outlet ducts. A pipe carries hydraulic fluid from a master cylinder to the chamber surrounding the membrane. As this fluid is pumped into the enclosure it squeezes the membrane, emulating the squeezing action of ventricular systole. Further, the volume pumped in equals the reduction of 'ventricular' volume, so, by controlling piston position or velocity, one may directly control ventricular volume or its rate of change. There is a hole through the base of the membrane and the cylindrical container. This is normally plugged but may be used as a second outflow for testing a conduit as an apico-aortic shunt. For

the basic pulsatile flow testing the conduit is attached in the aortic position. Compliance is provided by a pressure chamber into which air may be pumped through a car tyre valve. Resistance is provided by a simple screw clamp. All interconnecting tubing is of 22 mm bore clear plastic. (Since all the conduits obtained for testing are of nominal 22 mm diameter this choice of bore is used throughout the circuit.) Downstream of the impedance elements, the flow drains into an open reservoir which feeds the return to the pulse duplicator's atrial chamber. A Bjork-Shiley mitral valve separates the atrium from the ventricle. All circuit elements are fitted with standardised flange connectors for ease of interchangeability. Interconnecting pipe lengths are terminated in the same connectors, the tubing sliding over an external taper so that the bore remains constant throughout the joint. See Figure 3. Most conduits are flexible and somewhat porous. This raises two problems: firstly, how to secure them into position in the test apparatus and secondly, how to prevent leakage through the conduit walls. Both these difficulties may be overcome by mounting the conduit inside a length of 25 mm bore plastic tubing, securing it by sewing o-rings to both ends of the conduit and locating them in grooves machined into the tubing. See Figure 4. The stiffness of the tubing holds the conduit in position, whilst the o-rings provide axial location within the sheathing and prevent leakage of the fluid which seeps through the conduit walls.

The original working fluid was a buffered Ringer's solution (as used by Juden (1)), but this had to be abandoned since it caused unacceptable zero drift of the electromagnetic flowmeter probe. Eventually, a solution of 50g/dm^3 of sodium chloride in distilled water was mixed with glycerol in the ratio 70:30 by volume, giving a conductive working fluid, compatible with the flowmeter, with a kinematic viscosity (at 19.5°C) of 3.00×10^{-6} m^2/s, which is comparable to that of blood, and a density of 1.10×10^{-3} kg/m^3.

2.2 Drive System

Mechanical power to the pulse duplicator is derived from a linear motion reciprocating pump comprising a piston and cylinder arrangement. The choice of drive system was governed by two factors: smoothness of operation and availability of existing resources. These considerations led to the use of analogue command of a servo-motor to control piston velocity. Figure 5 shows a schematic diagram of the control system. Drive is taken from a low-friction nut running on a lead screw which is coupled to a d.c. servo-motor with integral tacho-generator for velocity feedback (M670 TE, by McLennon Servo). Velocity control is provided by a servo-amplifier (EM 200, McLennon Servo) taking its command signal from a 14-bit digital-to-analogue converter (RS 7534, Radio Spares). The signal is generated by a dedicated single board computer (1087/05E, Antronics Ltd) using sampled waveform data stored sequentially in EPROM. The system is capable of pumping at a maximum ejection rate of 1 dm^3/s at pulse rates between 30 and 200/min. Thus, within these limitations, any waveform may be output by selecting an appropriately programmed EPROM. One such option is a 'physiological' waveform derived from a graph of left ventricular volume versus time for a typical healthy subject, presented in Strand (2). This was reproduced as a 25-term Fourier series, from which was derived a smooth and continuous curve of the rate of change of ventricular volume. (The volume curve and its inverted derivative are shown in Figure 6.) This was sampled at 1024 equally spaced points and the values stored in EPROM to generate the piston velocity control signal.

A further feature of the control system is the 'reset' function, which stops the piston dead. This can be effected manually or on receipt of signals from opto-sensors at the lead screw's extremities, preventing the possibility of mechanical damage caused by the nut running into either end. There is also a manual override so that the nut can be re-positioned after a reset. Finally, trigger signals are available to initiate and stop a data acquisition cycle and to operate a laser or a camera for flow visualisation studies.

2.3 Data acquisition system

For reasons discussed by Leefe and Gentle (3), conduit performance is to be assessed in terms of 'energy loss'. This requires the continuous measurement, throughout a cycle, of two pressures (or one differential) and one volumetric flowrate. For tests on a conduit used as an apico-aortic shunt, three pressures (or two differentials) and two flows are needed. Instrumentation is comprised of pressure transducers (22 A-005-G IC-Sensors, Computer Controls Ltd), an electromagnetic flowmeter (SP2202 Research Blood Flowmeter with SP4005 22 mm i.d. cannulating probe suppled by Gould Medical) and a turbine flowmeter (B / 5/8 / 8, A.O.T Systems). The pressure transducers were calibrated statically, whilst the flowmeters were calibrated for steady flows in the range -30 to +30 dm^3/min in the working fluid. The data acquisition system is based around an eight channel, programmable gain, 12-bit analogue-to-digital converter (ADC) unit (U-A/D, U-Microsystems Ltd) under the control of a dedicated Apple IIe microcomputer. For rapid sampling the ADC gain needs to be kept constant, but the electromagnetic flowmeter signal is two orders of magnitude larger than the pressure transducer signals. Also, the turbine flowmeter's output is a small a.c. signal whose frequency is proportional to flow. Further, some of the signals are negative-going, but the ADC unit is unipolar. For these reasons a signal conditioning unit was built which also includes a facility for auto-calibration under computer control. Signal sampling is at the rate of ten kHz (with five channels this is two kHz per channel) so that data for a typical one second cycle, stored in bit-form occupies about 20 kilobytes of RAM. Data acquisition must, therefore, be controlled from an assembly language subroutine, since BASIC is too slow and floating point numerical data is wasteful of memory. The acquisition cycle is initiated and terminated by trigger pulse inputs generated by the drive system's computer. At the end of a cycle, data is retrieved from memory, one channel at a time, converted into values of process variables by means of calibration equations and stored on disc for subsequent plotting, manipulation and analysis. The system is shown schematically in Figure 7.

3 PROGRAMME OF WORK

The aim of the programme of research into prosthetic heart valve conduits, being undertaken at

Trent Polytechnic, is to develop a prototype which demonstrably improves on existing designs. To this end, a base of performance data is being established for comparison. This primarily entails the calculation of 'energy loss' per pulse for each conduit in the aortic position, with a given ventricular forcing function. Performance may also be evaluated for the conduit used as an apico-aortic shunt for the relief of a given degree of aortic valve stenosis, by calculating the 'energy loss' for a system in which the aortic outflow tract incorporates, for example, a 70 per cent occlusion representing the stenosed valve, and the conduit provides a second outflow tract.

The proposed idea for prototype development is a variant of the caged ball valve idea, in which there is a rigid, ceramic valve-bearing section attached at either end to flexible conduit tubing. The central section would consist of an inlet tract, sealed, under adverse pressure gradient, by a spherical occluder, which, under forward flow conditions, would move out into a diverging section, where it would be restrained from sealing the outflow duct by three struts, integral to the converging outlet section. See Figure 8. Preliminary steady flow tests on a rigid mock-up (4) have indicated that considerable improvements in forward flow pressure drop over existing designs are possible, although attention is needed with regard to the problem of ball instability. An investigation into this problem (5), however, has pinpointed the cause. Development work will be augmented both by the use of numerical flow simulation with the 'Phoenics' finite difference package and flow visualisation studies on transparent prototypes, using particle image velocimetry, to highlight regions of stasis and high shear rate. This technique relies upon the exposure of small particles (typically five microns) to double pulsed ruby laser radiation, for the creation of two laterally displaced image matrices on a photograpic negative. Local velocity vectors are derived by analysing the Young's fringes produced by the pointwise interrogation of a contact printed positive transparency in laser light. The magnitude of the velocity vector is inversely proportional to the fringe spacing, whilst its direction is gleaned from the fringe direction.

REFERENCES

(1) JUDEN, H. Wear studies of prosthetic heart valves, PhD Thesis, Trent Polytechnic, 1983.

(2) STRAND, F.L. Physiology: a regulatory systems approach, Macmillan, 1983, p233.

(3) LEEFE, S.E., GENTLE, C.R. A theoretical evaluation of energy loss methods in the analysis of prosthetic heart valves. Submitted August 1986 to: Journal of Biomedical Engineering.

(4) GENTLE, C.R. Minimization of pressure drop across heart valve conduits: a preliminary study. Life Support Systems, 1983, 1, 263-270.

(5) TANSLEY, G.D., EDWARDS, R.J., LEEFE, S.E., GENTLE, C.R. An investigation into ball occluder instability in prosthetic heart valve conduits. Presented at 13th annual meeting of the European Society for Artificial Organs, Avignon, France, September 18-20, 1986.

	PART No.	NAME OF PART	MATERIAL	FINISH	No. OFF
	22	HYDRAULIC FLUID INLET/OUTLET	COPPER		1
	21	BASE PLATE	PERSPEX		1
	20	SECURING RING	BRASS		1
	19	SECURING COLLAR	BRASS		1
	18	PLUG	PERSPEX	POLISH	1
	17	O-RING, 1/16" SECTION	RUBBER		1
	16	C'S'K SCREW 8 BA × 1/4"	BRASS		14
	15	C'S'K SCREW 4 BA × 3/8"	BRASS		6
	14	WASHER	FIBRE		1
	13	VENT PLUG, M3 × 6	STEEL		1
	12	FLEXIBLE MEMBRANE	RUBBER		1
	11	PRESSURE TAPPING TUBE	COPPER		1
	10	O-RING, 1/16" SECTION	RUBBER		1
	9	OUTLET STUB PIPE	PERSPEX		1
	8	SECURING COLLAR	PERSPEX	POLISH	1
	7	INLET STUB PIPE	PERSPEX	POLISH	2
	6	GRUB SCREW, M4×4			*
	5	INLET/OUTLET MANIFOLD	PERSPEX	POLISH	1
	4	22mm DIA BJÖRK-SHILEY VALVE			1
	3	C'S'K SCREW, 8 BA 3/8	BRASS		27
	2	VALVE RING	PERSPEX	POLISH	*
	1	PUMP BODY	PERSPEX	POLISH	*

DRN.	S.F.L.	SCALE 1:1	MODEL HEART PUMP GENERAL ARRANGEMENT
APPD.		SHEET	DRG. No.
DATE	3-6-85	SHEET No.	TRENT POLYTECHNIC

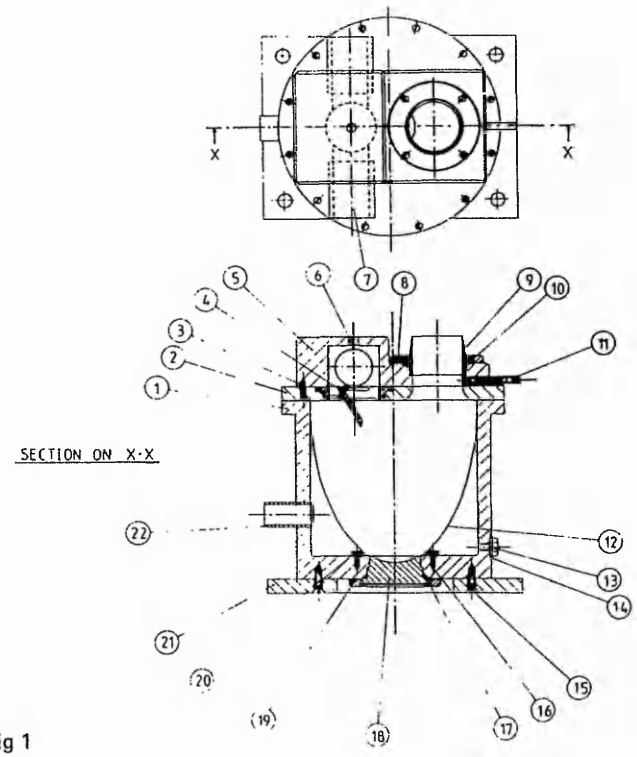

Fig 1

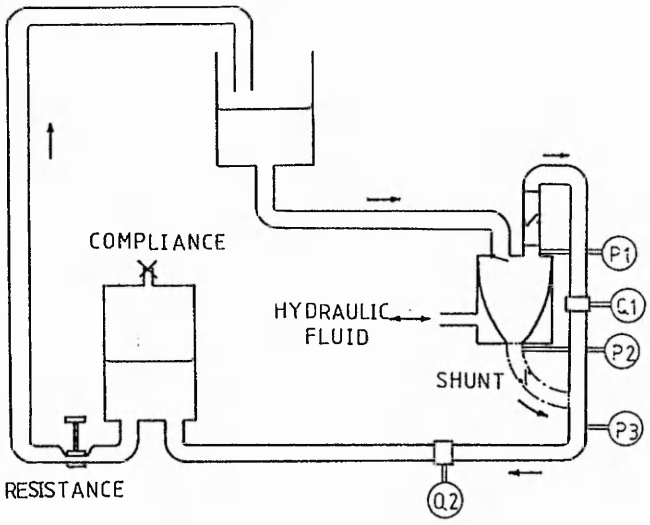

Fig 2

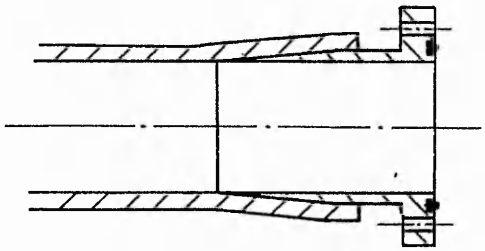

Fig 3

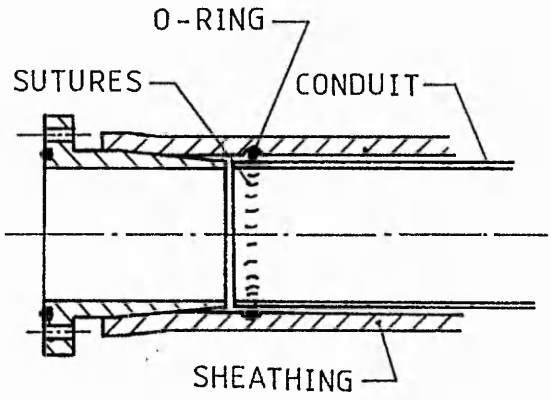

Fig 4

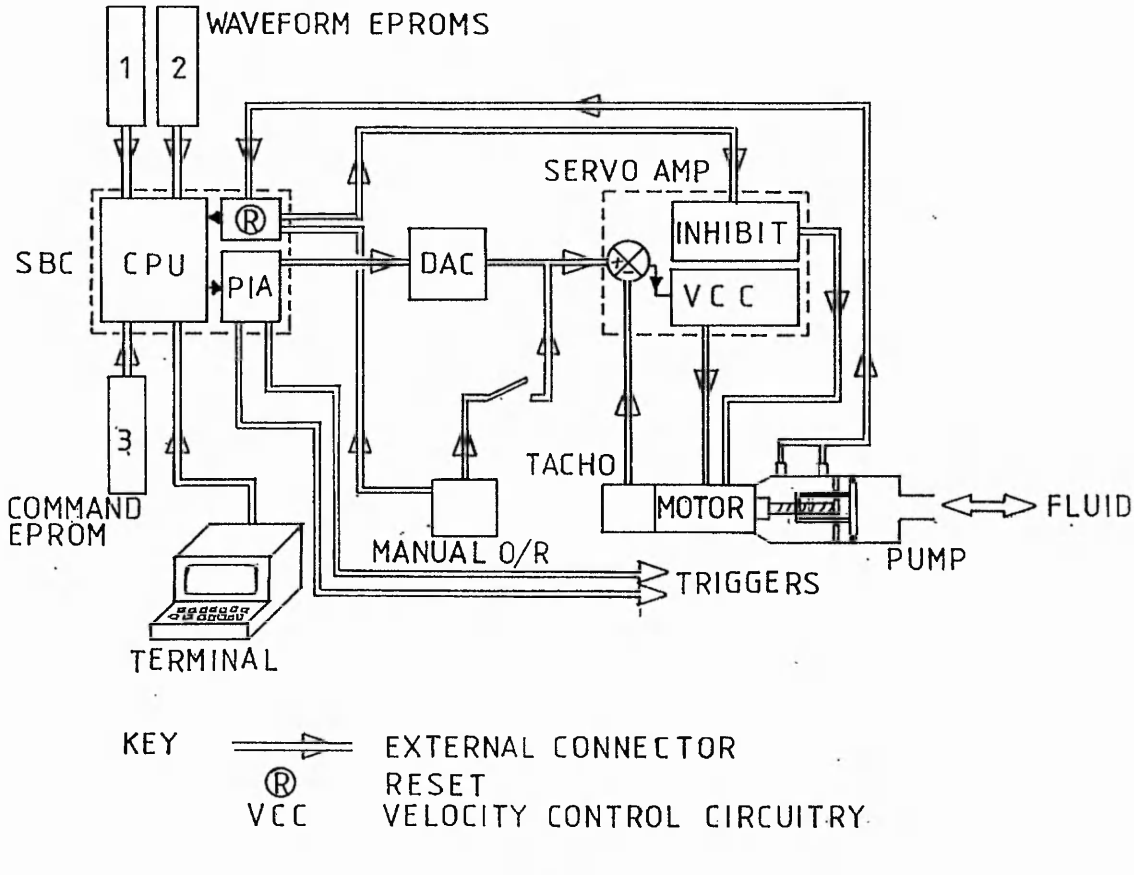

Fig 5

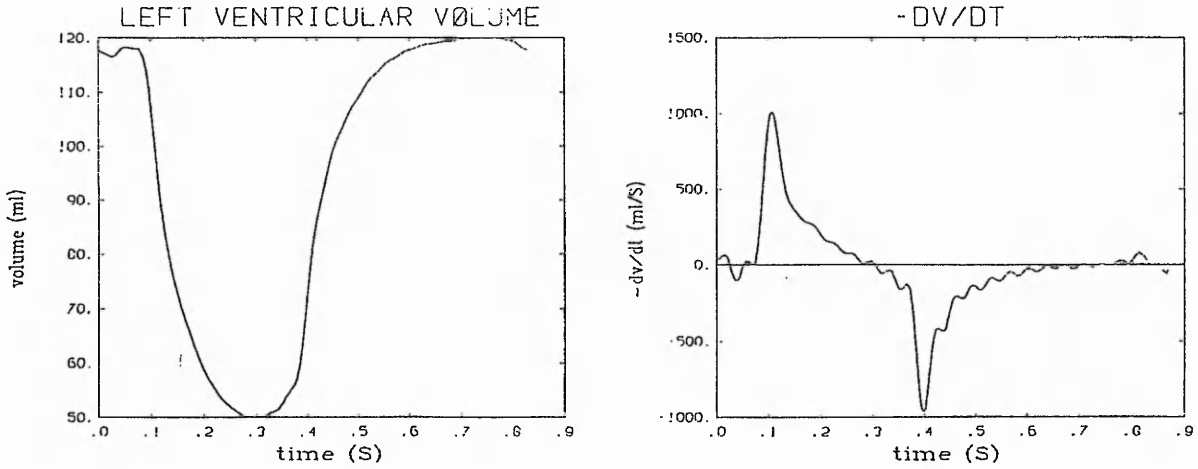

Fig 6

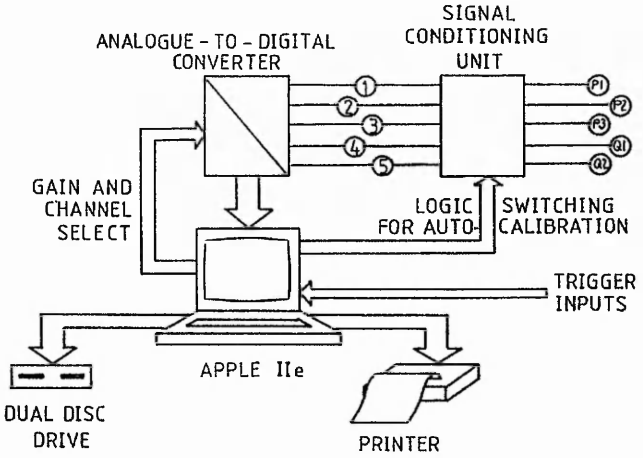

Fig 7

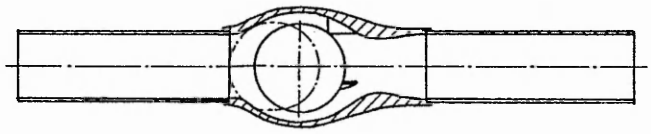

Fig 8

Leefe, S.E. and Gentle, C.R. 1987

Theoretical evaluation of energy loss methods in the analysis of prosthetic heart valves.

Journal of Biomedical Engineering, 9, 121-127

THEORETICAL EVALUATION OF ENERGY LOSS METHODS IN THE ANALYSIS OF PROSTHETIC HEART VALVES

S.E. Leefe and C.R. Gentle

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ABSTRACT

The purpose of this paper is to clarify certain aspects of the 'energy loss' approach to the analysis of artificial heart valves. This involves the discussion of what is meant by 'energy loss'; the derivation of an appropriate energy equation; the scrutiny of the conditions under which it may be applied and of the assumptions involved in deriving a useful formulation in terms of readily measurable parameters; consideration of the extent to which these assumptions are valid in reported experiments using this formulation and the demonstration of the suitability and versatility of this approach when applied specifically to heart valve conduits.

Keywords: Circulatory system, heart, heart valves, prostheses, mathematical model

INTRODUCTION

When prosthetic heart valves were first introduced the only assessment of their performance which was important was whether or not they were an improvement on the diseased valves which they replaced. It was only when it was realized that these early valves themselves could be improved upon that a quantitative system of assessment became necessary. For gauging flow performance this took the form of a measurement of the pressure drop across the valve in forward flow, in the same way that clinical practice is to measure the pressure gradient *in vivo* for natural valves. A lowering of pressure drop, under fixed conditions, denotes an improvement in the valve's performance since it leads to a higher exercise tolerance, reduced haemolysis and, probably, a reduced risk of thromboembolism.

As valves progressed further, the simple quoting of pressure drop was replaced by more sophisticated systems of assessment such as the 'figure of merit' of Viggers *et al.*[1] or the 'hydraulic efficiency' of Gentle[2]. These sought to take into account the variation of pressure drop with orifice diameter and flow so that a single figure could represent a complete size range of a particular design of valve, and could apply to any physiological flow. Hence it was possible to predict approximately what the pressure drop would be for a patient fitted with a specific size of valve and undertaking a specific level of exercise, given only a single figure for that model of valve. The method has been extended from its original steady-flow *in vitro* measurements to encompass pulsatile flow, (see, for example, Gentle[3]) and it is still probably the most widely used and understood valve assessment.

Department of Mechanical Engineering, Trent Polytechnic, Burton Street, Nottingham NG1 4BU, UK
Reprints from Dr C.R. Gentle

Unfortunately, the pressure drop method cannot deal effectively with the regurgitant flow when the valve is closing, or with leakage flow when the valve is fully closed. A modified figure of merit could be contrived, based on a net forward flow, but this would hardly be sufficiently elaborate to meet the needs of current valve flow development. Therefore, engineers have moved towards a more rigorous measure of the overall impedance to flow caused by prosthetic valves, which is the determination of the energy loss per pulse of blood through the valve. Examples of the use of this method are the work of Kaster *et al.*[4], Mohnhaupt *et al.*[5], Walker *et al.*[6], Reul[7] and Swanson[8]. The technique uses the same pressure drop measurement as the earlier method, but in addition requires a measure of volumetric flow obtained non-occlusively *in vitro*. The great advantage of the technique is that it takes into account the leakage flow and the regurgitant flow, which have to be treated separately by the pressure drop method. It would seem, however, that the full potential of the energy loss method has not yet been realized, for there is a second advantage which apparently has not been widely recognized. The method could go some way to overcoming the problem found with the pressure drop method of obtaining widely differing assessments of a valve's performance simply by measuring the pressure at different points on a changing flow geometry (see, for example Tindale *et al.*[9]). The energy loss method presented here takes this into account by recognizing that a pressure drop may be accompanied by a flow velocity increase due to a reduced cross-section and therefore the total energy of the blood may in fact remain almost constant. The problem still remains to a certain extent, however, because in some cases it is the pressure drop of a valve which is of paramount clinical importance, even if the energy loss could be made minimal. An example of this is given by Gentle[10], who envisaged two mitral valves, both with the

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properties of ideal orifices and hence with lossless flow through the orifices themselves, but with one having an absurdly tiny orifice size. This would produce such a large pressure drop between the atrium and ventricle, because of dissipation associated with the downstream jet, that it would be unusable because of the passive nature of ventricular diastole, a feature which the energy loss method could miss, depending on the choice of control volume.

Thus, although the energy loss method seems set to become the principal assessment method for the rising number of valve development engineers, care must be taken to recognize its limitations. The purpose of this paper is partly to explore these limitations by referring back to the original theory, something which has not previously been considered in applications of the method to heart valves, and partly to develop the theory further so that the technique can be used for a general deformable control volume. This development is necessary so that the energy loss method can be used to assess valved conduits. Here the pressure drop method has been used, for example by Gentle and Benjamin[11], but the conduit really needs to be viewed as part of a flow system and for this the energy loss method is essential. Ultimately, because there is no restriction on the number of flow inlets and outlets for the control volume of the energy loss method as developed here, the technique will be applicable to conduits used as apico–aortic shunts to provide a double outflow tract from the left ventricle. The shunt system's energy loss can then be compared with that of the severely stenosed system it replaces.

THEORETICAL DEVELOPMENT

Description of theory

The analysis starts with the consideration of a convenient form of the energy equation for a Lagrangian fluid element. This incorporates a term which represents 'energy loss' in the sense that it quantifies energy which can no longer be made available as mechanical work. It is an expression for the irreversible dissipation of heat by fluid friction. The equation is then presented in integral form for a system of particles moving with the fluid. Finally, a transformation is applied to convert the equation to an integral form over a general, deformable control volume through which the fluid flows.

The reasons for this approach are twofold: firstly, it enables the development of a truly general equation; secondly, when considering heart valves, it allows for the possibility of a flexible, distensible vessel on whose walls work may be done which, if the walls are not perfectly elastic, may not be recoverable. Other benefits accrue from the development of the general equation. Principally, as we work from the general to the specific, we are forced to make note of our simplifying assumptions and therefore to be aware of the limitations of our mathematical model (a point which will be amply demonstrated below) and the stage of the development at which these assumptions need to be made. Thus we are well placed to relax the assumptions, as necessary, being fully aware of the implications of so doing.

Derivation of the 'energy loss' equation

Let x, y, z be a set of cartesian coordinates and consider a Lagrangian fluid element of volume δV and density ρ moving relative to the cartesian axes with velocity q, whose components are u, v and w. We can write an energy equation for this element, thus:

$$\rho\delta V\frac{\mathrm{D}}{\mathrm{D}t}\left(e+\frac{p}{\rho}\right) = \delta V\left(\frac{\mathrm{D}p}{\mathrm{D}t}+\frac{DQ}{\mathrm{D}t}+\dot{\phi}\right) \quad (1)$$

(see, for example, White[12], p. 76.) Here, e is the specific internal energy, p is the thermodynamic pressure and Q is the heat transferred to the element per unit volume. The quantity $\dot{\phi}$ is the dissipation function and represents the rate at which the fluid in the element dissipates energy as irrecoverable heat through fluid friction. In this sense it can be taken as 'energy loss' per unit volume (since this energy is no longer available to do mechanical work) and as such forms the subject matter of this paper. For full derivations and discussions of its meaning see White[12], pp. 72–76, and Raudkivi and Callander[13], pp. 71–76. When equation (1) is rearranged as:

$$\rho\delta V\frac{De}{\mathrm{D}t} = \delta V\left(\frac{p}{\rho}\frac{\mathrm{D}\rho}{\mathrm{D}t}+\frac{\mathrm{D}Q}{\mathrm{D}t}+\dot{\phi}\right)$$

we can see that it has the following physical interpretation. The rate of increase of internal energy equals the rate of storage of elastic strain energy plus the rate at which heat is added plus the rate of viscous dissipation. The elastic strain is recoverable. Of the heat transferred, the second law of thermodynamics shows that some of this may be irrecoverable. Thus the total rate of conversion of energy to irrecoverable form is the irreversible part of the heat transfer rate plus the rate of dissipation of fluid friction heat. We shall concern ourselves with this last term, which is the dissipation function, $\dot{\phi}$. It should be emphasized at this stage that this is not, for a general element, the total energy loss, since some energy loss is associated with irreversibilities in the heat transfer between element and surroundings. To find the total energy loss, this term should be added.

Equation (1) can be more conveniently written in terms of the work done by the element per unit volume, W, thus:

$$\dot{\phi}\delta V = -\frac{\mathrm{D}}{\mathrm{D}t}\left(gh+\frac{1}{2}q^2\right)\rho\delta V-\left(\frac{p}{\rho}\frac{\mathrm{D}\rho}{\mathrm{D}t}\right)\delta V-\frac{\mathrm{D}W}{\mathrm{D}t}\delta V$$

where h is the elevation of the particle above some arbitrary horizontal datum plane. But $-DW/\mathrm{D}t\ \delta V$ the rate of work done on the element can be shown (see, for example, White[12], p. 74) to be equal to:

$$\text{div} \begin{Bmatrix} (u\sigma_{xx} + v\tau_{xy} + w\tau_{xz}) \\ (u\tau_{yx} + v\sigma_{yy} + w\tau_{yz}) \\ (u\tau_{zx} + v\tau_{zy} + w\sigma_{zz}) \end{Bmatrix} \delta V$$

where σ_j is the normal stress on the element in direction j and τ_{ij} is the shear stress in direction j on the element face normal to direction i.

Also,

$$\frac{p\mathrm{D}\rho}{\rho \mathrm{D}t}$$

is equal to $-p.\text{div q}$ (using the continuity equation). The divergences in both these expressions are taken as those at the centre of the element. Thus we have for our element:

$$\dot{\phi}\delta V = -\frac{\mathrm{D}}{\mathrm{D}t}\left(gh + \frac{1}{2}q^2\right)\rho\delta V + p\,\text{div q}\,\delta V + \text{div}\begin{Bmatrix} (u\sigma_{xx} + v\tau_{xy} + w\tau_{xz}) \\ (u\tau_{yx} + v\sigma_{yy} + w\tau_{yz}) \\ (u\tau_{zx} + v\tau_{zy} + w\sigma_{zz}) \end{Bmatrix} \delta V$$

We now integrate this equation for a system of particles moving with the fluid, to obtain:

$$\iiint_{SYS} \dot{\phi}\mathrm{d}V = -\frac{\mathrm{D}}{\mathrm{D}t}\iiint_{SYS}\left(gh + \frac{1}{2}q^2\right)\rho\mathrm{d}V + \iiint_{SYS} p\,\text{div q}\mathrm{d}V + \iiint_{SYS}\text{div}\begin{Bmatrix} (u\sigma_{xx} + v\tau_{xy} + w\tau_{xz}) \\ (u\tau_{yx} + v\sigma_{yy} + w\tau_{yz}) \\ (u\tau_{zx} + v\tau_{zy} + w\sigma_{zz}) \end{Bmatrix} \delta V$$

Let us consider this last term in more detail. Applying Gauss' theorem this is:

$$\oiint \begin{Bmatrix} (u\sigma_{xx} + v\tau_{zy} + w\tau_{xz} \\ (u\tau_{yx} + v\sigma_{yy} + w\tau_{yz}) \\ (u\tau_{zx} + v\tau_{zy} + w\sigma_{zz}) \end{Bmatrix} \cdot \mathrm{dA}$$

which can be shown to be equivalent to:

$$\oiint_{SYS} (\sigma_n + \tau) \cdot \mathrm{q}\mathrm{d}A$$

where σ_n is the outward normal direct stress and τ is the in-plane shear stress acting over the surface element, $\mathrm{d}A$, of the system.

We now need an integral expression for the energy dissipation rate over a generalized, deformable control volume. This can be derived by defining the 'system' as occupying the same region of space as the 'control volume' at a given instant and applying a transformation to relate the 'system' integral to the 'control volume' integral, namely:

$$\frac{\mathrm{D}}{\mathrm{D}t}\iiint_{SYS} f\rho\mathrm{d}V = \frac{\mathrm{d}}{\mathrm{d}t}\iiint_{C.V.} f\rho\mathrm{d}V + \oiint_{C.V.} f\rho\mathrm{q}_r\cdot\mathrm{dA}$$

where f is any specific fluid property (i.e. per unit mass), and q_r is the velocity of the fluid relative to that of the surface element, $\mathrm{d}A$, of the control volume. Since the system and the control volume are coincident:

$$\iiint_{SYS} B\mathrm{d}V = \iiint_{C.V.} B\mathrm{d}V$$

and

$$\oiint_{SYS} B\mathrm{d}A = \oiint_{C.V.} B\mathrm{d}A$$

where B is any quantity. For details see Hansen[14], chapters 4 and 5 especially p. 110 and p. 124, or for a fuller discussion of the relationship between system and control volume analyses, see Hansen[15].

Then we have:

$$\iiint_{C.V.}\dot{\phi}\mathrm{d}V = -\frac{\mathrm{d}}{\mathrm{d}t}\iiint_{C.V.}\left(gh + \frac{1}{2}q^2\right)\rho\mathrm{d}V - \oiint_{C.V.}\left(gh + \frac{1}{2}q^2\right)\rho\mathrm{q}_r\cdot\mathrm{dA} + \iiint_{C.V.} p\,\text{div}\mathrm{q}\mathrm{d}V + \oiint_{C.V.}(\sigma_n + \tau)\cdot\mathrm{q}\mathrm{d}A \qquad (2)$$

This equation is completely general. The only assumption embodied here is that weight is the only body force. Let us be clear, however, about what it means. It describes the rate at which the mechanical energy in an arbitrary, time-varying control volume is degraded into irrecoverable heat. Only in the absence of heat transfer, or if heat transfer between the region and the surroundings can be considered reversible can this be taken as the total energy loss rate, where 'energy loss' is in the sense of irrecoverable energy conversion.

The assumptions

In this section all simplifying assumptions will be itemized and numbered as they arise.

The first point to note is that since heart valves are situated in the middle of the thorax we may assume the following.

1. *Negligible heat transfer between the control volume and its surroundings.* In view of the concluding paragraph of the previous section, we may take equation (2) to represent the total instantaneous rate of 'energy loss' (in the sense already defined) within the control volume.

2. *The fluid is incompressible.* This means that ρ is a constant and also, because of the continuity equation, we have $\text{div q} = 0$ so that the third integral on the right-hand side of equation (2) vanishes.

We sub-divide the boundary into the inlet/outlet sections, 'IN/OUT', across which there is a flow and the 'WALLS', across which there is no flow and hence over which $\mathbf{q}_r = 0$ and note that the surface force integrals over the walls simply represent the rate of work done on the fluid by the solid boundary, $-\dot{W}_{walls}$, where $\dot{W}_{walls}$ is the rate of work done on the walls by the fluid. Denoting the integral on the left-hand side of equation (2) by $\dot{\Phi}$, the rate of energy dissipation inside the control volume, we have:

$$\dot{\Phi} = -\frac{d}{dt}\iiint_{C.V.}(gh + \tfrac{1}{2}q^2)\rho dV - \iint_{IN/OUT}(gh + \tfrac{1}{2}q^2)\rho \mathbf{q}_r \cdot d\mathbf{A} + \iint_{IN/OUT}(\sigma_n + \tau)\cdot \mathbf{q}\, dA - W_{walls} \quad (3)$$

3. *Assume that our fluid is Newtonian.* This means that the direct stress can be written as:

$$\sigma_n = -p - \frac{2}{3}\mu \mathrm{div}\mathbf{q} + 2\mu\frac{\partial q}{\partial n}$$

where μ is the dynamic viscosity and the derivative is taken along the normal direction but, since the fluid is incompressible and div $\mathbf{q} = 0$ this becomes:

$$\sigma_n = -p + 2\mu\frac{\partial q}{\partial n}$$

In order to equate the normal stress to the pressure, we need to assume that

4. *$2\mu\, \partial q/\partial n$ is negligible in comparison to typical pressures.* This is a very good approximation for low viscosity fluids such as water or blood where convective accelerations are not excessive. Thus we may replace σ_n with $-p$. Since $\sigma_n dA$ is identical with $\sigma_n d\mathbf{A}$ we can replace $\sigma_n \cdot \mathbf{q}dA$ with $\sigma_n \mathbf{q} \cdot d\mathbf{A}$ and hence, by the preceding argument, with $-p\mathbf{q}\cdot d\mathbf{A}$. We may now integrate equation (3) with respect to time, t, over one cardiac cycle to obtain:

$$\Phi = -\left[\iiint_{C.V.}(gh + \tfrac{1}{2}q^2)\rho dV\right]_0^T - \int_0^T\left\{\iint_{IN/OUT}(\rho gh + \tfrac{1}{2}\rho q^2)\mathbf{q}_r\cdot d\mathbf{A} + \iint_{IN/OUT}\rho \mathbf{q}\cdot d\mathbf{A} - \iint_{IN/OUT}\tau\cdot \mathbf{q}dA\right\}dt - W_{walls}$$

where Φ is the total energy dissipated per cycle inside the region of interest, W_{walls} is the work done per cycle on the walls and T is the cycle time.

5. *Assume steady cyclic conditions.* The amount of kinetic plus potential energy contained inside the control volume at any instant of the cycle is the same at the corresponding instant of any other cycle and, specifically, of the next cycle so that the first term on the right-hand side vanishes.

6. *Assume all inlet and outlet sections are fixed in space.* $\mathbf{q}_r = \mathbf{q}$ over IN/OUT. This allows us to combine the pressure integral with the preceding term and, using the notation p^* for piezometric pressure we can write:

$$\Phi = -\int_0^T\left\{\iint_{IN/OUT}\left(p^* + \tfrac{1}{2}\rho q^2\right)\mathbf{q}\cdot d\mathbf{A} - \iint_{IN/OUT}\tau\cdot \mathbf{q}dA\right\}dt - W_{walls} \quad (4)$$

7. *The walls are rigid or perfectly elastic.* This gives $W_{walls} = 0$. When we are testing valves *in vitro* we may employ rigid-walled test sections since we are interested in losses associated with the valve rather than those arising from its anatomical situation.

So far, all the assumptions are fairly realistic. However, to obtain the formulation usually employed by 'energy loss' researchers, namely:

$$\text{energy loss per cycle} = \int_0^T Q\Delta p\, dt$$

(where Δp is the instantaneous pressure drop across the valve and Q is the instantaneous flow through it), we need to embody the following further assumptions, some of which our discussion will reveal to be wholly unjustifiable in the circumstances in which the equation has been employed.

To eliminate the shear force work term, flow over the inlet/outlet sections must be normal to the (fixed) surface at all instants. In this way $\tau\cdot\mathbf{q} = 0$ throughout the cycle. This means that:

8. *Flow must be parallel to the axis.* Also, turbulent components perpendicular to the mean flow direction must be zero for all time. Clearly, this will only hold if:

9. *Flow over the inlet/outlet sections is laminar.* With the last three assumptions, equation (4) has reduced to:

$$\Phi = \int_0^T\left\{\iint_{IN}\left(p^* + \tfrac{1}{2}\rho q^2\right)q\, dA - \iint_{OUT}\left(p^* + \tfrac{1}{2}\rho q^2\right)q\, dA\right\}dt$$

understanding that the orientation of $\mathbf{q}$ and $d\mathbf{A}$ is the same and $\mathbf{q}\cdot d\mathbf{A} = -q dA$ over inlet sections and $+q dA$ over outlet sections.

10. *The piezometric pressure is regarded as constant over a cross-section.* The definition of volumetric flow rate, $Q = \iint q dA$ is used and we assume:

11. *There is just one inlet and out outlet.* This is as in a simple flow path from one point to another. (It is worth noting that this need not be the case. Indeed, the 'energy loss' approach is equally applicable to the analysis of heart valve conduits used as apico–aortic shunts to provide the left ventricle with a double outflow tract, see *Figure 1*). These assumptions yield:

$$\Phi = \int_0^T Q\Delta p^* \mathrm{d}t + \frac{1}{2}\rho \int_0^T \left\{ \iint_{\mathrm{IN}} q^3 \mathrm{d}A - \iint_{\mathrm{OUT}} q^3 \, \mathrm{d}A \right\} \mathrm{d}t$$

where Δp^* is the drop in piezometric pressure across the valve. Since the first integral may be rewritten:

$$\int_0^T Q\Delta p \mathrm{d}t + \rho g \Delta h \int_0^T Q \mathrm{d}t$$

(where Δh is the difference in elevation from inlet to outlet), we see that:

12. *Differences in elevation are assumed negligible from inlet to outlet.* We may arrange for this to be the case by the design of our *in vitro* test rig. In order for the last integral to vanish we need to assume:

13. *No change in mean velocity or velocity profile from inlet to outlet.* This last point warrants some further comment since it is, in the authors' view, a potentially serious omission. To draw an analogy, with regard to the mean velocity, to omit this term is akin to inferring a head loss in a venturi meter because one observes a large pressure drop from inlet to throat: if the channel area decreases, continuity dictates an increase in velocity and hence kinetic energy, and this appears at the expense of pressure, but this, in itself, certainly does not constitute a loss of head. Also, a change in velocity profile from inlet to outlet means that different amounts of kinetic energy are being convected into and out of the control volume.

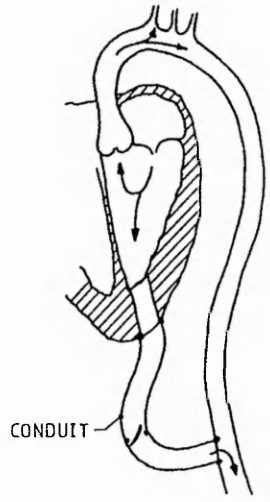

Figure 1 Valve-bearing conduit used as an apico–aortic shunt

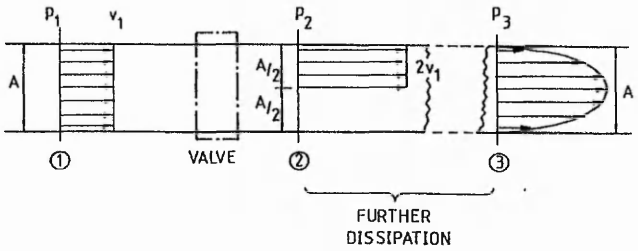

Figure 2 The effect of a hypothetical valve on the velocity distribution to illustrate local asymmetry and consequent downstream dissipation. The choice of downstream measurement station can affect the result of the various 'loss' computations in different ways

Finally, it should be noted that the equation with which we are left is:

$$\Phi = \int_0^T Q\Delta p \mathrm{d}t$$

where Φ is the energy dissipated per cycle within the control volume. To identify this with 'energy loss' per cycle is to assume that:

14. *All energy loss associated with the valve takes places within the control volume.* This may not, in practice, be the case. For example, if a biased velocity distribution is produced downstream of the valve, as in the Bjork–Shiley valve, clearly there will be some viscous dissipation further downstream as the velocity distribution rearranges itself to the fully developed shape. (See *Figure 2.*) Thus, although dissipation is outside the control volume, it is a result of the presence of the valve and hence is an energy loss associated with the valve.

EXAMPLE

We now include an example which, we would hasten to emphasize, is not intended to be physiologically realistic. Rather, we are presenting a simplified model to illustrate the problems already highlighted with the conventional 'pressure drop' approach and the hitherto accepted 'energy loss' formulation.

Consider the steady flow of fluid down a rectangular channel of constant cross-sectional area, A. This channel contains a partial blockage such as may be presented by an open valve. Let us assume that this produces a biased velocity distribution immediately downstream, because it obstructs half of the channel area. If the upstream velocity is uniform with value v_1 then we can imagine that downstream there could be a uniform velocity of $2v_1$ over half of the channel area and zero over the other half. This situation is depicted in *Figure 2.*

A 'pressure drop' proponent may well place pressure transducers at stations (1) and (2), measure the pressure drop as $\Delta p = p_1 - p_2$ and report the results. The problem is that further investigation would reveal that at some station further downstream where the flow has readjusted itself to occupy the whole channel, the pressure will have

risen to a larger value, p_3. So what is the 'pressure drop' across this valve? Further, if the downstream area of the flow channel is not constant the poor researcher is really in trouble and will have to admit that the velocity should be taken into account, since this will vary with the flow area and the pressure will adjust accordingly.

A '$Q\Delta p$ energy loss' proponent will fare little better. The measured flow will, of course, be $Q = Av_1$ and continuity dictates that this will be the same at any section down the duct. So the results reported in this case will be the same as those of the 'pressure drop' team except that they will differ by a constant factor of Q! Thus, measuring between sections (1) and (2 a loss of $Q(p_1 - p_2)$ would be assumed, although if stations (1) and (3) were chosen a lower loss of $Q(p_1 - p_3)$ would be inferred, implying a negative loss between sections (2) and (3).

A more sophisticated approach might suggest that Bernoulli's equation should be applied between sections (1) and (2) to take into account the variation in effective flow area. The 'energy loss' equation in its fuller form of:

$$\dot{\Phi} = Q\Delta p + \Delta\left(\iint \frac{1}{2}\rho q^3 \, dA\right)$$

would yield:

$$\text{LOSS} = Q\Delta p + \frac{1}{2}\rho\,(v_1^3 \cdot A - (2v_1)^3 \cdot (A/2)) \text{ per second}$$

or:

$$\text{LOSS} = Q\left(\Delta p - \frac{3}{2}\rho v_1^2\right) \text{ per second}$$

Bernoulli's equation would then give:

$$p_1 + \frac{1}{2}\rho v_1^2 = p_2 + \frac{1}{2}\rho(2v_1)^2$$

or:

$$\Delta p - \frac{3}{2}\rho v_1^2 = 0$$

so that incorporating this result in the energy equation would merely yield:

$$\text{LOSS} = 0$$

which is hardly surprising since this is one of the assumptions made in applying Bernoulli's equation in the first place.

The point is that the pressure drop is not related to the velocity in such a simple way. Both need to be determined independently for a given situation and both need to be included in the calculation of the 'energy loss'.

APPLICABILITY TO VALVES AND CONDUITS

If the downstream station is taken too close to the valve, flow disturbances will almost certainly be encountered. This problem is particularly acute for mitral valves, where downstream flow is swirling into a large chamber and, to a lesser extent, with aortic valves where the blood is discharged into the sinuses of valsalva. This raises serious doubts as to the applicability of the assumptions of laminar, parallel flow and of no change in mean velocity or velocity profile from inlet to outlet. One may well also question the practice of ignoring losses as these eddies are damped out downstream, outside the control volume. Therefore, the authors feel that if 'energy loss' methods are to be applied to prosthetic heart valves a fuller expression for the loss should be employed.

The problems are not so serious, however, when the 'energy loss' approach is applied to heart valve conduits. The assumptions which cause problems for valves, namely 8, 9, 13 and 14, will be discussed individually.

Where a conduit is employed, flow is away from a ventricle so that the flow converges into a parallel entry section. It is thus essentially parallel and laminar at the upstream end of the conduit, which we may take as the inlet section to the control volume. With regard to the downstream station, firstly, flow is no longer discharging into the ventricle or the sinus cavities and therefore does not diverge. This means that not only is outlet flow parallel, but also that separation is unlikely. Secondly, since we may take the outlet section of the control volume at the downstream extremity of the conduit, the eddying is likely to have subsided sufficiently for the assumption of parallel laminar flow here to be more reasonable than for the valve alone. This also means that since the most vigorous eddy motion is now inside the control volume, it is more valid to identify the 'energy loss' associated with the valve with the 'energy loss' inside the control volume, in line with assumption 14. The analysis of the conduit alleviates the problem of assuming no change in mean velocity from inlet to outlet since, being a duct of constant cross-sectional area, this condition must be satisfied with an incompressible fluid. It is, however, the authors' opinion that since upstream is essentially an entry flow and downstream the flow may not be axi-symmetric, neither can be considered fully developed and hence the velocity distribution is unlikely to be the same at the two stations and should ideally be investigated experimentally. This work forms part of the research programme into heart valve conduits being undertaken in the Department of Mechanical Engineering at Trent Polytechnic.

CONCLUSION

We have called into question the applicability of the form of the 'energy loss' equation most commonly employed by heart valve researchers, with particular reference to the location of the downstream measurement site and the omission of the effect of changes in velocity from inlet to outlet. We have also shown that these considerations are far less problematic when the approach is applied to the analysis of losses associated with heart valve conduits. In view of the additional benefits

afforded by this mode of analysis, it is suggested that the 'energy loss' method forms a suitable basis for the comparison of existing designs and the development of improved heart valve conduits.

REFERENCES

1 Viggers, R.F., Robel, S.B., Sauvage, M.D. A hydraulic figure-of-merit for heart valve prostheses, *J. Biomed. Mat. Res.* 1967, **1**, 103–112

2 Gentle, C.R. A limit to hydraulic design of heart valve prostheses, *Eng in Med,* 1977, **6**, 17–21

3 Gentle, C.R. The role of simulation studies in cardiac valve prosthesis design, *Eng in Med,* 1978, **7**, 101–106

4 Kaster, R.L., Mantini, E., Tanaka, S., Lillehei, C.W. Analysis of dynamic characteristics of heart valves in a pulse duplication analog computer system, *Proc. 5th Nat. Biomed. Sci. Inst. Symp.,* 1967, (Ed. Allison, R.D.): Plenum Press, New York, 1968, **4**, 112–118

5 Mohnhaupt, A., Affeld, K., Mohnhaupt, R., Bucherl, E.A. A comparative performance analysis of heart valve prostheses, *Trans. E.S.A.O.,* 1975, **2**, 39–45

5 Walker, D.K., Scotten, L.N., Modi, V.J., Brownlee, R.T. In vitro assessment of mitral valve prostheses, *J. Thorac. Cardiovasc. Surg.,* 1980, **79**, 680–688

7 Reul, H. Influence of the degree of stenoses on pressure-loss energy-dissipation and flow in coronary arteries. In: *Advances in Cardiovascular Physics, vol. 5 (Part I),* Karger, Basel, 1983, 185–194

8 Swanson, W.M. Relative performance of artificial heart valves based on power measurements. *Med. Instrum.,* 1984, **18**, 314–325

9 Tindale, W.B., Martin, T.R.P., Van Noort, R., Black, M.M. Problems of measurement in aortic valve in vitro evaluation, *Proc. 8th Ann. Meeting E.S.A.O.,* 1981, 356–360

10 Gentle, C.R. Assessment of the performance of heart valves, *J. Med. Eng. Tech.,* 1977, **1**, 229

11 Gentle, C.R., Benjamin, E.L. Measurement of steady flow characteristics of valved conduit prostheses, *Proc. 10th Ann. Meeting E.S.A.O.,* 1983, 324–327

12 White, F.M. *Viscous Fluid Flow,* McGraw-Hill, 1974, 61–77

13 Raudkivi, A.J., Callander, R.A. *Advanced Fluid Mechanics,* Edward Arnold, London, 1975, 8–12, 20–34, 70–76

14 Hansen, A.G. *Fluid Mechanics,* John Wiley & Sons, New York, 1967, 95–134

15 Hansen, A.G. Generalized control volume analyses with application to the basic laws of mechanics and thermodynamics, *Bull. Mech. Eng. Educ.,* 1965, **4**, 161–168