DISSERTATION

Title:

Surgical techniques used for closure of perimembranous ventricular septal defects

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Declaration

I, Zeead Karani, hereby declare that the work on which this dissertation is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, or is being, submitted for another degree in this or any other university.
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Abbreviations

Ave.	average
ANOVA	analysis on variances
BSA	body surface area (m^2)
Chi sq	chi square test for calculation of *p* value
CPB	cardiopulmonary bypass
CXR	chest x-ray
DRV	divided Right ventricle
D/C	discharge
e.g.	for example
ff. up	follow up
Fig.	figure
FE	fisher exact test for calculation of *p* value
I.E.	infective endocarditis
JET	junctional ectopic tachycardia
Kg	kilograms
MDM	mid-diastolic murmur
Min.	minutes
Neurol.	neurologic
PR	pulmonary regurgitation
PSM	pansystolic murmur
P2	pulmonary component of the second heart sound
PAB	pulmonary artery band
PAP	Pulmonary artery pressure
P_{pa}	measured pressure in the pulmonary artery
Perm.	permanent
PHT	pulmonary hypertension
P_{la}	measured pressure in the left atrium
PPM	permanent pacemaker
PPS	peripheral pulmonary stenosis
Preop	preoperative
PS	pulmonary valvular stenosis
Pts	patients
PVR	pulmonary vascular resistance

Q_p	pulmonary blood flow ($l.min.m^2$)
Q_s	systemic blood flow ($l.min.m^2$)
RV	right ventricle
Res.	residual
RVF	right ventricular failure
RXH	red Cross Children's Hospital
Tab.	table
VSD	ventricular septal defect
Viz.	namely
Wt.	weight
x-clamp	cross clamp

TABLES **page**

FIGURES

GRAPHS: page

Abstract

Objective:

To compare different surgical techniques of closing ventricular septal defects (VSDs) and to ascertain the advantages and disadvantages of the various techniques.

Methods:

A retrospective study was done at Red Cross Children's Hospital, Rondebosch, Cape Town in which two separate case-matched groups of children undergoing different surgical closure techniques for their isolated perimembranous ventricular septal defects were compared.

Group I consisted of 77 children who had their VSDs closed between 1987 to 1990, mainly with a double velour dacron patch using interrupted alternating 5.0 silicone coated braided polyester suture (Ticron®) and 5.0 polypropylene (Prolene®) pledgetted sutures (n=71). Five patients in Group I had bovine pericardium used and 1 patient's VSD was closed by a direct suture technique

Group II consisted of 93 children operated on between 1995 to 1998, and had their VSDs closed with a 0.6% glutaraldehyde-treated autologous pericardial patch using 5.0 polypropylene suture material in a continuous horizontal mattress suture without pledgets.

Surgical time, discharge echocardiograms and follow up records were reviewed to assess the incidence of complications, reoperation or residual VSD needing further follow up. Ten patients in Group I and 23 patients in Group II were lost to follow-up.

Results:

The median follow up time was 40 months (range = 2-153 months) in Group I and 18.5 months (range = 1-60 months) in Group II. A summary of the results is shown in the following table:

Parameter	**Group 1**	**Group II**	**p-value**
Cross-clamp time in minutes (median & range)	50(22-110)	33(99-117)	<0.001
CPB time in minutes (median & range)	86(45-231)	59(29-139)	<0.001
Mortality* (in hospital)	7%	1%	=0.04
Spontaneous closure of residual VSD.	36%	76%	<0.001
Reoperation**	4%	1%	
Heart block (permanent pacemaker)	4%	0%	

* Five out of six deaths occurred in children less than one year old (There was 1 late death in group I)

** A third patient needing reoperation declined surgery in group I

Conclusions:

The present technique of VSD closure (Group II) had a shorter surgical and cross-clamp time, lower morbidity and mortality with a marked incidence of spontaneous closure of residual VSD when compared to previously used surgical techniques (Group I). These advantages are particularly beneficial to the high risk under-one-year-old patient group undergoing primary closure of their VSDs.

TABLE OF CONTENTS

CHAPTER 1

INTRODUCTION

Ventricular septal defect (VSD) is a defect in the interventricular septum of the heart resulting in the left to right shunting of blood. This defect can be either in the perimembranous or in the muscular component of the interventricular septum (see *Morphology*).

VSD is the most common congenital defect in children; an incidence of up to 50% of all congenital heart defects has been reported.[1] It comprises 0.3 - 0.5 / 1000 live births and premature infants have a tenfold increase in incidence.[1]

Perimembranous VSDs account for 75% of all VSDs, muscular VSDs for approximately 15% and endocardial cushion defects the least common VSDs at 5 - 10% incidence.[1]

The natural history of ventricular septal defects is a tendency towards spontaneous closure, especially for small defects lying close to the tricuspid valve.[1]

The probability of spontaneous closure of a large perimembranous VSD according to the age of the patient is illustrated in Fig. 1.1 [2, 3]

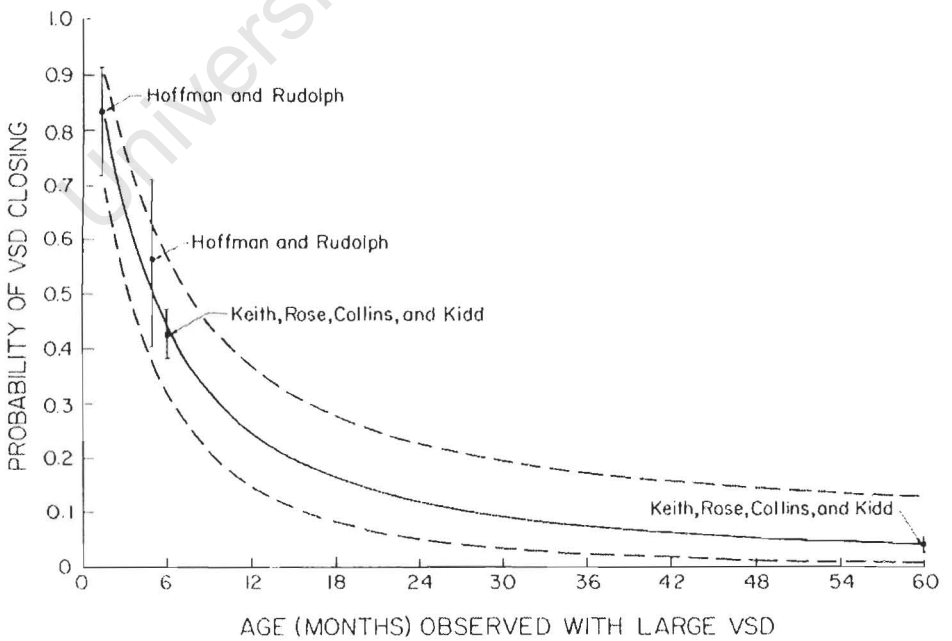


Fig. 1.1 Probability of spontaneous closure of large VSD according to age
(From : Kirklin J W, Barret-Boyes B G. Cardiac surgery: Morphology, Diagnostic criteria, Natural history, Techniques, Results and Indications. Second Edition, page 777. Churchill Livingstone, New York, 1993.)

A recent prospective study by Mehta et al [4] showed an overall spontaneous closure rate of a large isolated perimembranous VSD to be 34% at one year and 60% at 5 years. Only 25% of children with perimembranous VSDs required surgery by 5 years of age and a further 22% needed follow up after the 5th year of life. The same study showed a 9% mortality at one year of life associated with undiagnosed / untreated VSDs.[4]

Late deaths are attributed to "Eisenmenger" syndrome (irreversible pulmonary hypertension with reversal of the shunt from right to left), leading to cyanosis and death in 20% of undiagnosed / untreated patients with VSD. [5]

Five percent of patients with VSD will develop aortic incompetence and a further 5% will develop a discrete subpulmonic stenosis ("divided right ventricle") in the first decade of life.[3, 5] Both these latter complications are indications for surgical intervention (see Chapter 2).

Various surgical closure techniques have been used for closure of perimembranous VSDs (see Chapter 5). This study aims to compare closure techniques for VSDs without major associated heart defects, that were previously used at Red Cross Children's Hospital, viz. a double velour knitted Dacron ("de Bakey") patch, sewn into place with interrupted, alternating 5.0 polypropylene and 5.0 Ticron® pledgetted sutures (Group I), with the technique currently in use, viz. a 0.6% glutaraldehyde-treated autologous pericardial patch sewn into place with a 5.0 polypropylene continuous horizontal mattress suture (Group II).

The current technique at Red Cross Children's Hospital of using a 0.6% glutaraldehyde-treated autologous pericardial patch has theoretical advantages in that the patch is readily available and cheap (free) and the bypass and cross clamp times are shorter, with a resultant reduction in morbidity and mortality. The latter advantage is particularly important in the under-one-year-old age group when closure of a VSD is contemplated as a higher risk.

The use of the 0.6% low dose glutaraldehyde solution overcomes the potential problem of post-operative aneurysmal patch formation. [6,7]

There have been fears of tissue toxicity associated with the use of glutaraldehyde, but the safety of low dose glutaraldehyde is accepted[6] and is again demonstrated in this study. The reported incidence of complete heart block following VSD closure is 1.3-5%.[1, 8, 10]

The current suture technique of choice at Red Cross Children's Hospital, is a continuous, horizontal mattress suture. This suture technique is compared to the well-described interrupted pledgetted suture technique still used in many centres.[1, 3](see chapter 5).

With the trend towards earlier closure of VSDs in younger children and infants, a short surgical time is an important advantage in reducing the morbidity and mortality. This is particularly true in the under-one-year-old high risk age group, who are especially vulnerable to the effects of cardiopulmonary bypass. [2, 10, 11, 12, 13, 16]

PART I
LITERATURE REVIEW

CHAPTER 2

INDICATION AND TIMING OF OPERATION

Seldom is a single indication responsible for surgical intervention to repair a VSD; more commonly a variety of indications are present. The following are potential indications for surgery in VSD and they will be discussed individually: [3]

i. Congestive cardiac failure

ii. Failure to thrive, secondary to cardiac failure

iii. Recurrent respiratory tract infections

iv. Elevated pulmonary vascular resistance or development of pulmonary hypertension

v. Aortic incompetence associated with the VSD

vi. Infective endocarditis

vii. Development of subpulmonic stenosis ("divided" or "double-chambered" right ventricle)

viii. Low probability of closure (large left to right shunt)

i. CONGESTIVE CARDIAC FAILURE

Large VSDs leading to congestive cardiac failure and difficult to control, are usually seen in neonates and infants. Closure of these large isolated perimembranous VSDs is advisable. [2, 8, 10, 11, 12, 14] Although this subgroup of patients (usually under one year of age) is regarded as a high risk group, surgical closure is reported to carry a peri-operative mortality from as low as 1.6%.[14] However, mortalities as high as 24% have been reported,[13] whilst most centres report a peri-operative mortality of between 3 and 6%.[2, 8, 11, 12, 13]

Infants especially at risk are those who have additional risk factors; particularly, low birth weight and associated pulmonary problems (pneumonia, aspiration, bronchospasm, ventilator-dependent).

A two-stage approach to VSD closure in small infants was advocated in the past, where a pulmonary artery band was placed as a first stage with VSD closure done as a second stage procedure when the PA band is removed.

One-stage surgical closure of an isolated VSD is now generally considered advantageous over a two-stage approach for the following reasons:

- the first stage (palliative pulmonary artery banding) in the two-stage approach to isolated VSDs carries a peri-operative mortality of 8-10%, and even higher (20 - 50%) if done under 3 months of age.[12, 15]
- one operation lessens the psychological and economic burden on a family.[11]
- no risk of increase in pulmonary artery pressures noted at follow-up i.e. regression in pulmonary hypertension is well documented in patients who had pulmonary hypertension at the time of their VSD closure.[11, 10, 13]
- "catch-up" growth is well documented with early closure .[10, 13]

The two-stage approach should be limited to: [3, 12, 16]

- VSDs with major associated congenital cardiac abnormalities.
- Multiple muscular VSDs (to avoid a possible ventriculotomy).
- The very sick infant who is ventilated for severe pulmonary oedema or pneumonia.

In summary, primary closure of an isolated perimembranous VSD under 1-year of age, carries a mortality of 1.5-3%. Pulmonary artery banding as a first-stage approach in isolated VSDs carries a mortality of 10% (up to 17% with second stage operation) and medical treatment of intractable congestive cardiac failure due to an isolated VSD, carries a mortality of 27% under the age of one. [1, 8, 10, 12, 13]

ii. FAILURE TO THRIVE, SECONDARY TO CCF

Failure to thrive, or failure to gain weight satisfactorily according to the growth chart, as a result of an isolated large perimembranous VSD, is an indication for surgical closure of the VSD. Accelerated growth has been shown to occur with VSD closure in infants which is important for normal physical and mental development. [10, 13]

iii. RECURRENT RESPIRATORY TRACT INFECTIONS

The increase in pulmonary blood flow from the left to right shunt caused by the VSD, leads to pulmonary congestion or "wet lungs", increasing the likelihood of bacterial colonization and pulmonary infection.

Recurrent pulmonary infection can predispose to lung injury and adversely affect pulmonary function, apart from the added risk of increasing the pulmonary vascular resistance, with failure to thrive and repeated hospitilization.

Early closure of the VSD will reverse or limit the progression of the above complications.[10]

iv. ELEVATED PULMONARY VASCULAR RESISTANCE

Pulmonary hypertension or pulmonary vascular disease in patients with a large VSD tends to worsen with age (Fig. 2.1).[3]

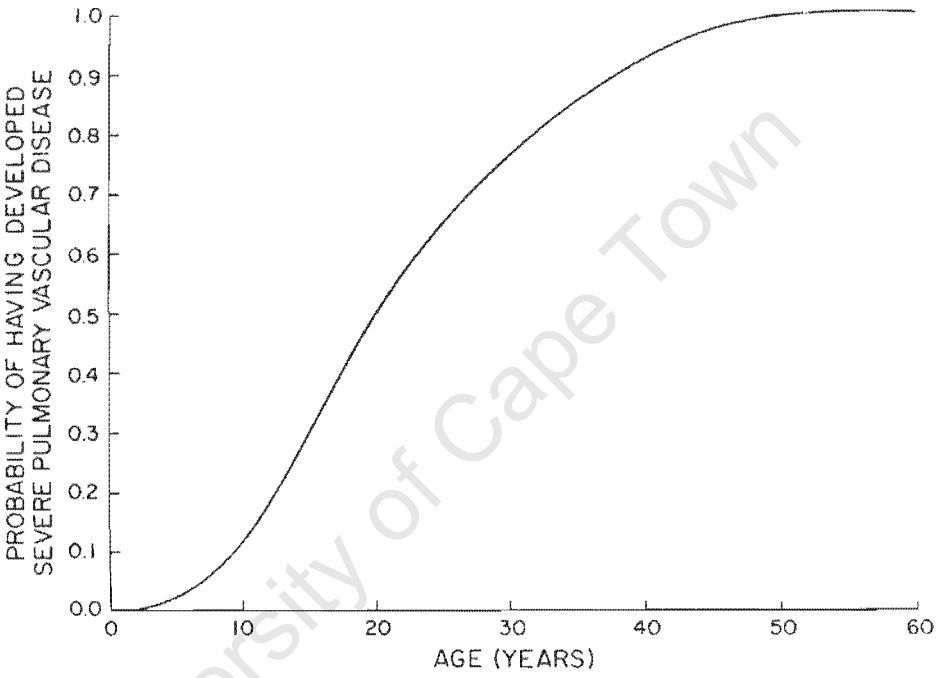


Fig. 2.1 Probability of developing severe pulmonary vascular disease in patients with large VSD according to age.

(From: Kirklin J W, Barret-Boyes B G. Cardiac surgery: Morphology, Diagnostic criteria, Natural history, Techniques, Results and Indications. Second Edition, page 778. Churchill Livingstone, New York, 1993.)

Prompt closure of large VSDs for clinically suspected pulmonary hypertension or documented elevation in pulmonary vascular resistance at cardiac catheterization, is indicated to avert inoperability (see fig 2.2)[3] and Eisenmenger Syndrome, which will lead to premature death.[1, 5]

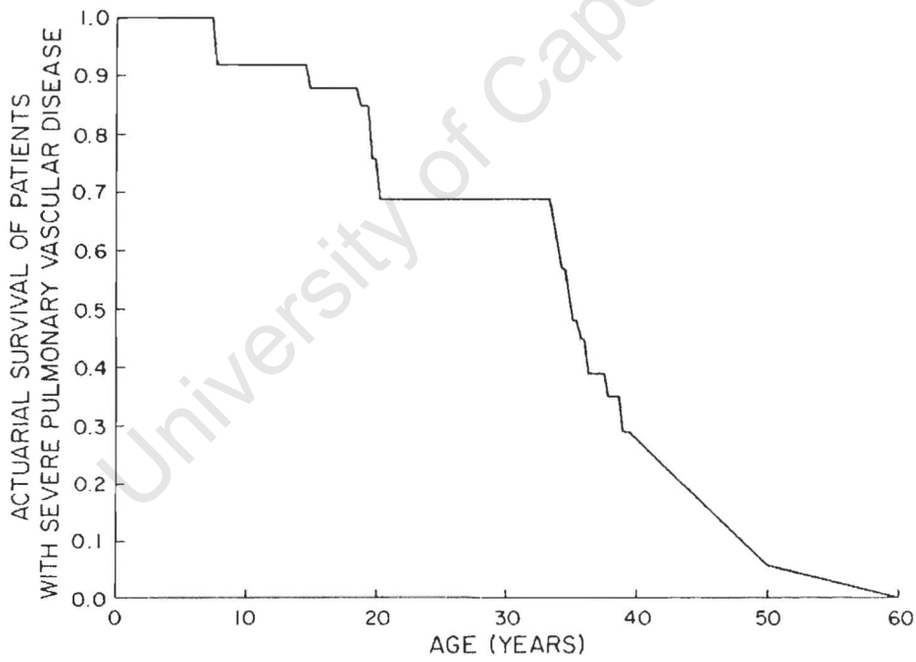


Fig. 2.2 Actuarial survival of patients with large VSDs after developing severe PHT.

(From Kirklin J W, Barret-Boyes B G. Cardiac surgery: Morphology, Diagnostic criteria, Natural history, Techniques, Results and Indications. Second Edition, page 779. Churchill Livingstone, New York, 1993.)

Table 2.1 shows the figures as measured at cardiac catheterization that are generally accepted to quantify the severity of pulmonary vascular disease.[3]

Table 2.1 Grading of pulmonary vascular resistance
$(PVR = (P_{pa} - P_{la}) / Q_p) \times B.S.A)$

PVR (units.m^2)	Description
< 4	Normal
4 – 5	Mildly elevated
5 – 8	Moderately elevated
> 8	Severely elevated

(From: Kirklin J W, Barret-Boyes B G. Cardiac surgery: Morphology, Diagnostic criteria, Natural history, Techniques, Results and Indications. Second Edition, page 771. Churchill Livingstone, New York, 1993.)

Patients with elevated PVR at cardiac catheterization are further subjected to oxygen or Nitric oxide to assess reversibility and feasibility of surgical cure.

Early closure of a large VSD causing elevation in pulmonary vascular resistance is imperative. Numerous studies have consistently documented a reduction in pulmonary vascular resistance and normalization of pulmonary artery pressures after closure of the VSD.[2, 3, 6, 9, 11, 14]

However, with persistent or irreversible PVR of > 8 units/m^2, the closure of a VSD is not advised, as severe RV failure in the immediate post operative period could prove fatal.[3] Even so, some centres have reported operating successfully on patients with PVR of 10 units/m^2. [1]

v. AORTIC INCOMPETENCE ASSOCIATED WITH THE VSD

Up to five percent of patients with a perimembranous VSD, regardless of size, will develop aortic incompetence during the first decade of life which gradually worsens during the second decade of life. It can be severe when only diagnosed in the second decade of life.[3, 5]

This aortic incompetence is said to be secondary to a *Venturi* effect beneath the aortic valve caused by a small to moderate sized subarterial VSD (see *Morphology*).[16] It has also been attributed to a weak or poorly supported aortic annulus.[16]

Surgical closure of the VSD should be undertaken as soon as aortic incompetence is detected so as to avoid the possible need for replacement of the aortic valve. [1]

vi. INFECTIVE ENDOCARDITIS

Infective endocarditis is very rare (0.15 - 0.3%) in patients with VSD and usually occurs in patients with small defects.[4]

A documented incidence of infective endocarditis requires surgical intervention once the endocarditis has been appropriately treated so as to prevent recurrence of septic emboli.[3]

vii. DEVELOPMENT OF SUBPULMONIC STENOSIS

A discreet midcavity right ventricular stenosis, termed "divided right ventricle", occurs in a further 5% of patients with a large ventricular septal defect. [3, 5]

The pathogenesis is thought to be due to turbulence caused by the left to right shunt, thereby promoting hypertrophy of the complex muscle bundles within the right ventricle. The stenosis is semi-protective towards the development of pulmonary hypertension, as it reduces the left to right shunt. Elective surgical closure of the VSD and relief of the RV stenosis should be carried out.[3]

viii. LOW PROBABILITY OF CLOSURE

(Large left to right shunt without pulmonary hypertension)

In the case of incidental late diagnosis, or follow-up of children with large (uncomplicated) perimembranous VSDs which show no inclination towards getting smaller or closing, elective VSD closure should be planned, particularly when there is evidence of a large left to right shunt (Q_p:Q_s ratio >2:1), as this could lead to the development of pulmonary vascular disease.[3]

Furthermore, the spontaneous closure rate of VSDs diagnosed later in life is low. For example, if diagnosed at 3 years of age, the spontaneous closure rate of a VSD is less than 10%.(fig. 1.1)[3]

The timing of surgery in this group of patients is elective, but should be undertaken within one year of diagnosis. It is further recommended that closure of these VSDs should be performed by age 5, or before school going age.[3, 17]

CHAPTER 3

DIAGNOSIS

A detailed clinical evaluation (table 3.1),[18] ECG (fig. 3.1.1) and chest radiograph (fig. 3.2) are done.

Clinical

At Red Cross Children's Hospital, VSDs are initially classified according to clinical evidence of left to right shunting and of pulmonary hypertension, as depicted in Table 3.1. This classification was developed at the University of Cape Town by Schrire in the 1960s and is described in his book Clinical Cardiology.[18] It is still the classification used at Red Cross Children's Hospital, Cape Town.

Table 3.1: Clinical evaluation of a VSD after Schrire

Group (degree of PHT)	A	B	C
I (no PHT)	3/6 PSM, Normal P2	4/6 PSM, Normal P2	RV lift, Normal P2
II (moderate PHT)	Symptomatic clinically, RV lift, **Loud P2**		
III (severe PHT)	3/6 PSM , EDM of PR, Loud P2, **MDM @ apex**	Eisenmenger syndrome: Cyanosed, RV lift, Soft PSM, Loud P2	

(Modified from Chesler E: " Schrire's Clinical cardiology".
Fourth Edition. pp. 154-157. John Wright & sons, Bristol, 1981.)

In **Grade I** of the Schrire classification, **A, B & C** refer to the size of left to right shunt as evidenced by a systolic murmur, with a normal P2 (i.e. normal pulmonary artery pressures). Thus, grade IA is characterized by a short systolic murmur which radiates poorly and varies little with respiration. Grade IB is characterized by a pansystolic murmur, radiating more widely and increasing on inspiration and grade IC, by additional signs of ventricular hypertrophy but still with a normal sounding P2.

Grades II and III refer to increasing pulmonary hypertension:

Grade II is characterized by the clinical signs of IC, together with a loud P2 with normal splitting, indicating the development of pulmonary hypertension.

Grade IIIA is characterized by a 3/6 PSM + the EDM of PR, as well as an additional MDM at the apex, together with a loud P2 which is widely split.

.

In Grade IIIB, there is no longer any murmur, or very soft PSM, with marked signs of pulmonary hypertension and the development of cyanosis (reversed shunt, Eisenmenger syndrome).

VSDs in grades IC, II, and IIIA (Table 3.1) require surgery. Those in grades II and IIIA may need prior cardiac catheterization to assess pulmonary hypertension.

A grade IA VSD does not require surgery because it will likely close spontaneously.

A grade IB VSD may be observed for a time to see if it will get smaller and close, whilst a grade IIIB VSD is inoperable.

Electrocardiogram:

The electrocardiogram shows a normal axis with biventricular enlargement (fig. 3.1)

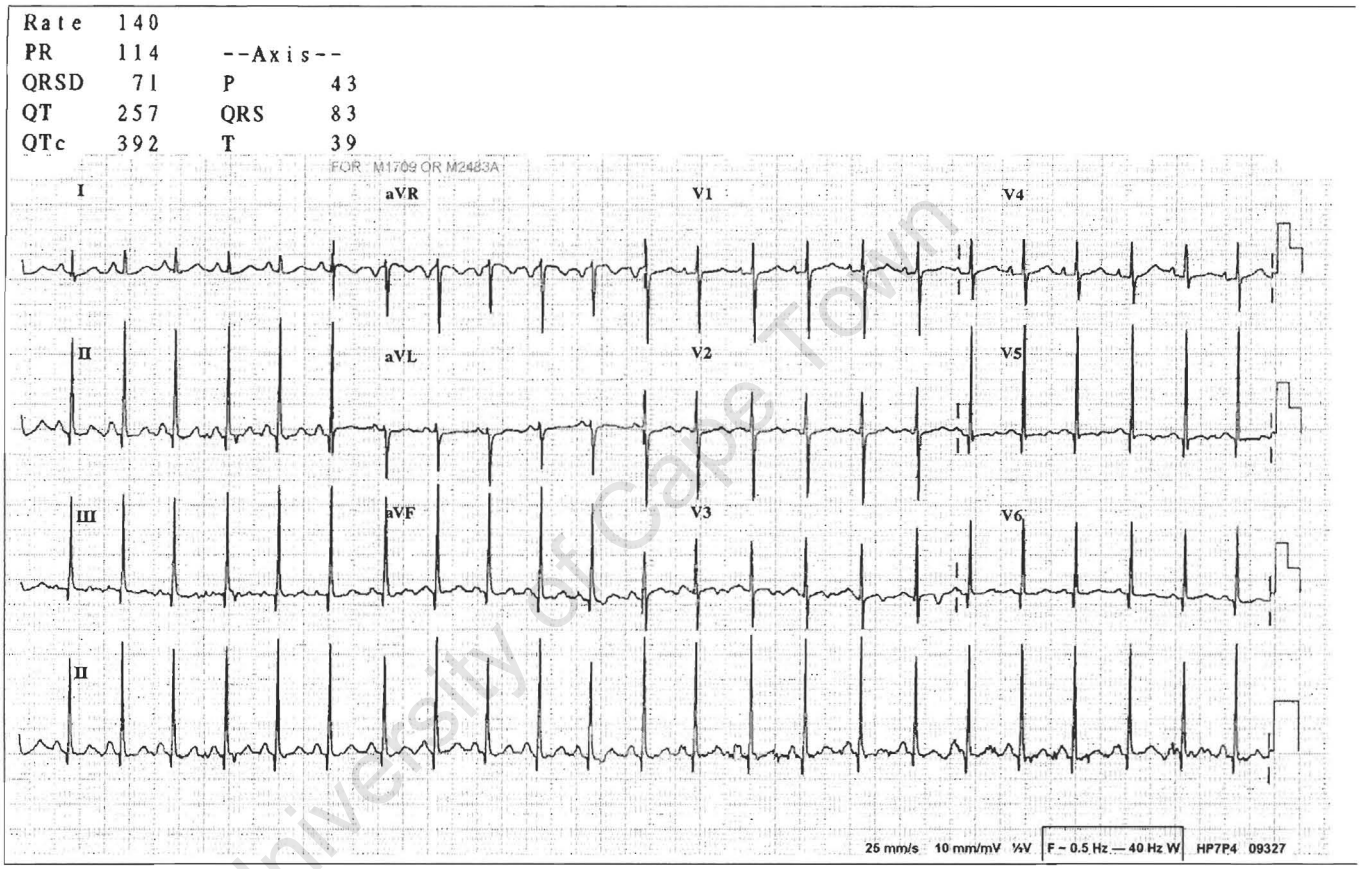


Fig. 3.1 Typical ECG of patient with VSD showing normal axis and biventricular enlargement

Chest radiograph:

A posterolateral and lateral CXR shows pulmonary plethora, biventricular enlargement and enlarged pulmonary artery (fig. 3.2.1/2)

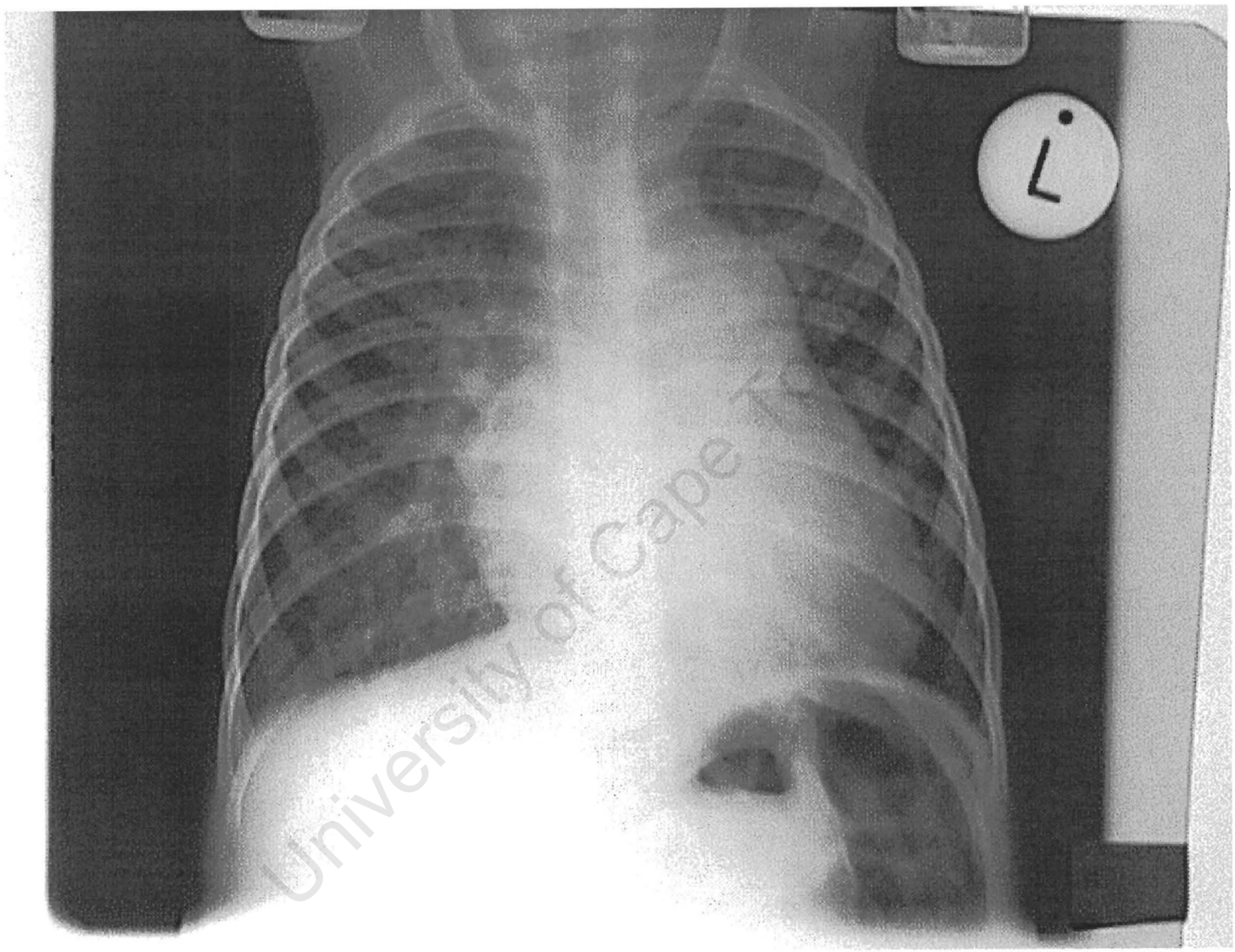


Fig. 3.2.1: PA CXR of patient with VSD showing biventricular enlargement, pulmonary plethora and enlarged main pulmonary artery (incidental associated hyperinflated lung fields noted)

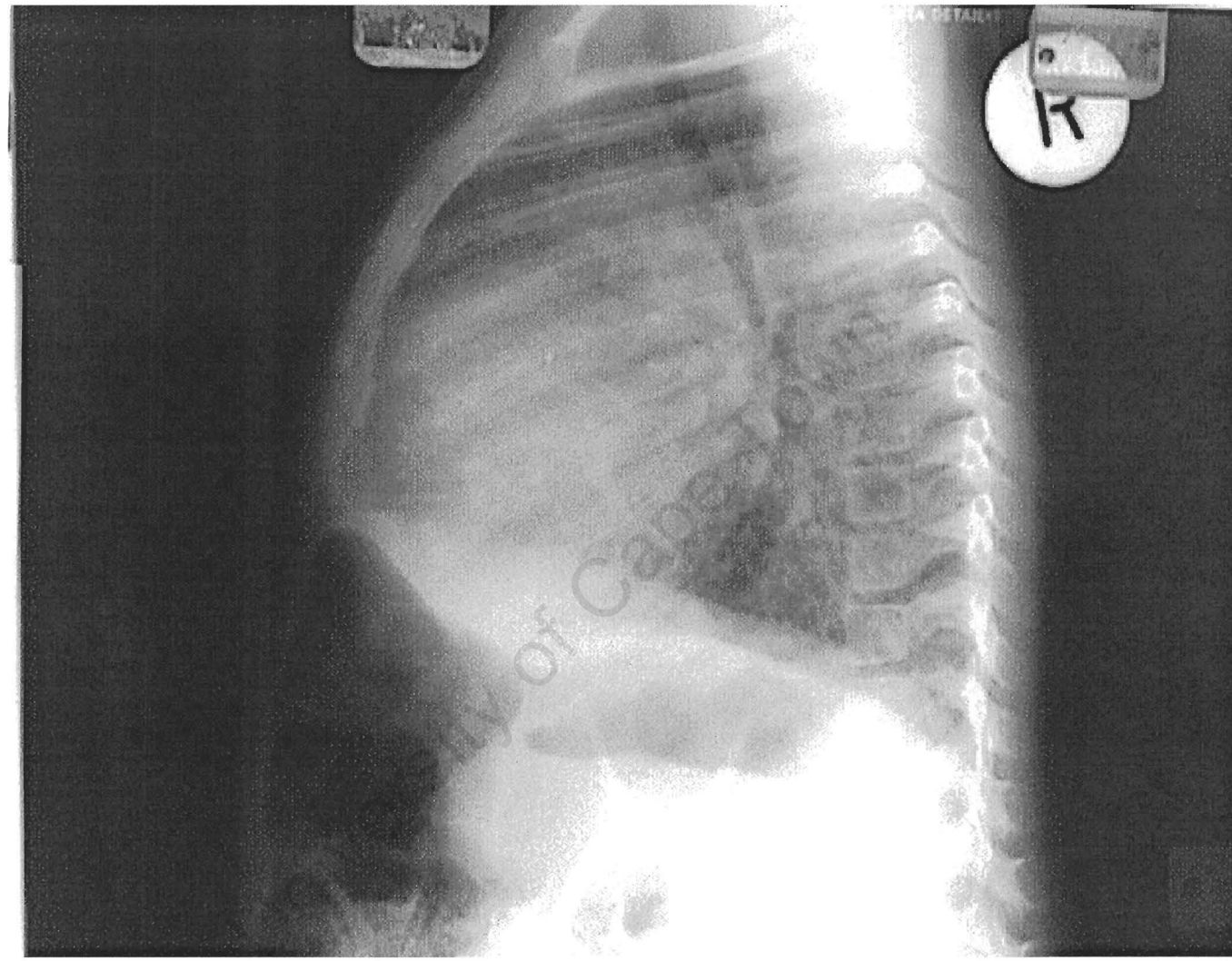


Fig. 3.2.2: Lateral CXR of patient with VSD showing biventricular enlargement, pulmonary plethora and enlarged main pulmonary artery (incidental associated hyperinflated lung fields noted)

Echocardiogram :

The echocardiogram can reveal the following important information regarding the VSD : [1]

- the anatomical position of the defect (two-dimensional echo)
- the estimated right ventricular pressure
- the magnitude of left to right shunt (see table 3.2)
- the presence of additional VSDs (particularly colour flow Doppler)
- the presence of associated cardiac anomalies

A subjective echocardiographic assessment can be obtained by the cardiologist, whereby the left to right shunt is labelled as *trivial (insignificant), small, moderate or large.*

The information provided by two-dimensional and Doppler echocardiography is usually sufficient to proceed confidently with surgical correction of most perimembranous VSDs.

It is important to mention that the advent of colour-flow Doppler revolutionized echocardiography and is one of the main reasons catheterization can be so confidently dispensed with in most cases now. Another result is the very high incidence of "residual VSDs" now seen on early postoperative echo, most of them disappearing within weeks. These trivial leaks are often called "stitch leaks", seen only on colour Doppler and they soon close. They were presumably previously undetected and close by clotting.

Cardiac Catheterization:

The indications for diagnostic cardiac catheterization in patients with an isolated larger perimembranous VSD are: [1]

- Moderate to severe pulmonary hypertension (to quantify pulmonary blood flow and to derive an accurate assessment of the pulmonary vascular resistance and the reversibility thereof).

- The suspected or known presence of major associated cardiac anomalies which may warrant detailed haemodynamic study before surgery is undertaken.

Cardiac catheterization is an invasive diagnostic modality not; without complications, which are related to: [1]

- The duration and difficulty of the procedure; subjecting the infant to hypothermia (exposure) and hypovolaemia (blood losses and sampling) which could precipitate acidemia.

- Direct damage from the catheter such as femoral vessel laceration, myocardial irritation and arrhythmias, or even myocardial perforation.

- Toxicity of the contrast medium, which could lead to myocardial, renal or cerebral dysfunction due to its osmolarity, pH and sodium content.

Cardiac catheterization as part of the diagnostic evaluation of an isolated perimembranous VSD has been used less and less over the two decades, as improvements in echocardiographic techniques have been made available to clinicians.

A recent comparative study, measuring outcomes in two groups of patients undergoing preoperative evaluation and surgery in isolated ventricular septal defects, found a significant reduction in early mortality, peri-operative complications and diagnostic mistakes in a recent group (1991-1996), compared to an earlier group (1976-1990), even though less invasive diagnostic procedures were being used. [9]

CHAPTER 4

MORPHOLOGY

4.1 Embryology of the interventricular septum

The basics of the embryological development of the interventricular septum help to classify the anatomical location of isolated ventricular septal defects. The ventricular septum is derived from three components: [19, 20]

i. *The muscular septum* develops from the primitive interventricular septum.

ii. *The membranous septum* is derived from a downgrowth from the side of the AV endocardial cushions onto the muscular septum.

iii. *The outflow parts of the ventricles* (infundibulum of the right ventricle and vestibule of the left) are separated by the proximal part of the bulbospiral septum, the distal part of which separates the aorta from the pulmonary artery in the truncus. The bulbar septum fuses with the membranous and muscular septa at the crista supraventricularis.

The membranous area of the interventricular septum is the fibrous continuity between the leaflets of the aortic and tricuspid valves. A further fibrous continuity between the aortic and pulmonary valves is formed by the bulbospiral septum and this is closely related to the membranous component of the interventricular septum.[1, 20]

A defect in the fibrous continuity of the interventricular septum is therefore termed a *perimembranous* defect and results from a deficiency of the muscular septum forming the circumference of the membranous areas.[12, 20]

The interventricular septum has a large muscular component (posterior 2/3) and a small fibrous membranous component (anterior 1/3) extending from the inlet to the outlet of the right ventricular cavity (fig. 4.1).[1] A defect fully within the muscular septum is termed a *muscular* defect.[20]

The inlet component of the septum extends from the tricuspid valve septal annulus to the medial papillary muscle (of *Lansici*), whilst the outlet component extends from the medial papillary muscle to the aortic valve annulus.[20]

The trabecular component of the septum is the portion in between and extending towards the apex.[20]

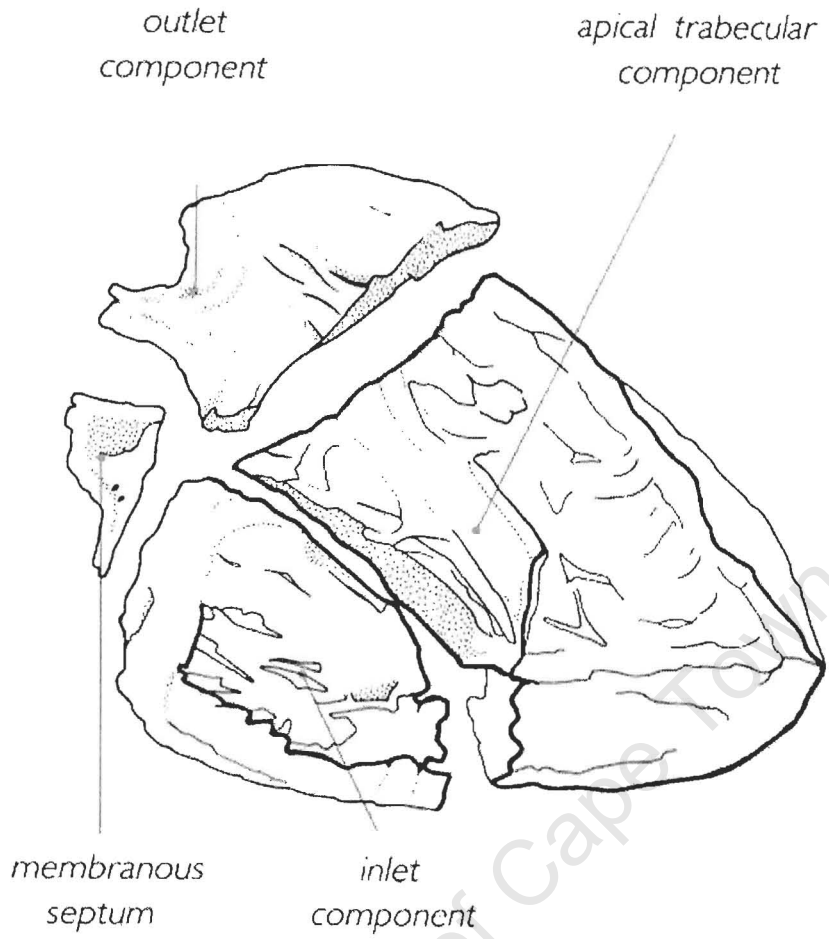


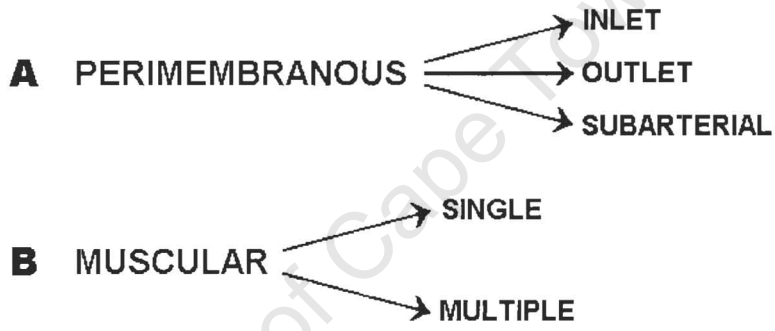
Fig. 4.1 Different components of the interventricular septum

(From: Anderson R H, Becker Anton E. The Heart, structure in health and disease. page 7.1. Gower Medical Publishing, London, 1992.)

4.1 Anatomical classification of VSDs

There is no real consensus on a standard classification of VSDs, since terminology and definitions vary considerably between anatomists.[1,3,20]

Anderson and Bekker's classification gives the surgeon a good practical understanding of the anatomical location of isolated VSDs and classifies ventricular septal defects as follows: [1,20]



The subdivisions of both perimembranous and muscular VSDs are based on the anatomical site in the interventricular septum (fig. 4.2).[20]

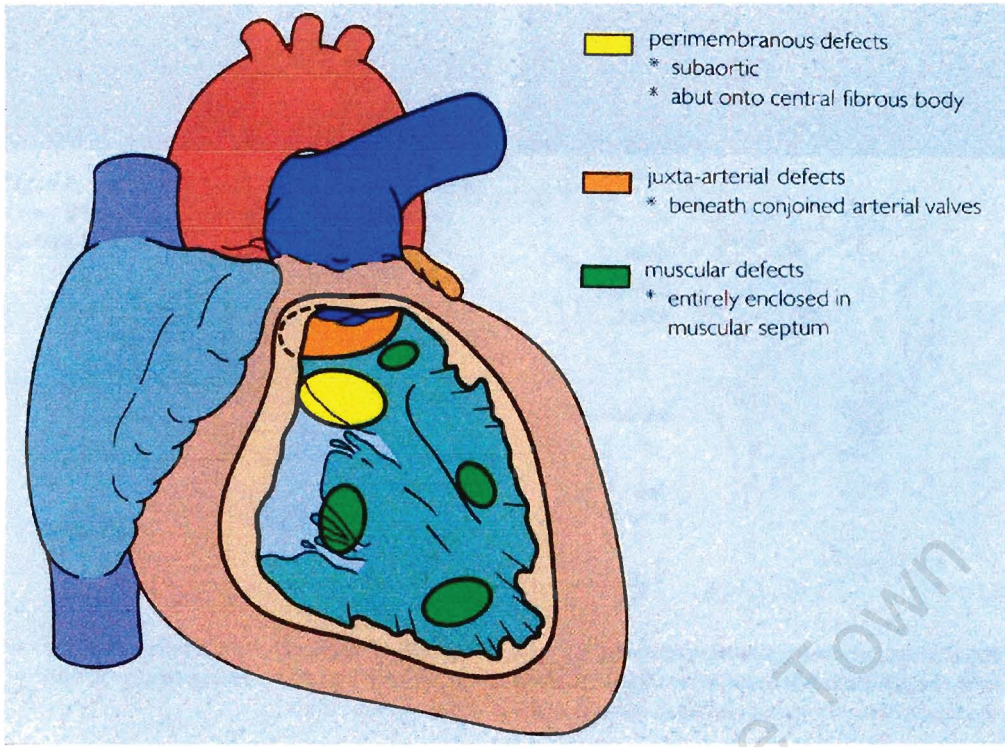


Fig. 4.2 Subdivision of perimembranous and muscular VSDs

(From: Anderson R H, Becker Anton E. The Heart, structure in health and disease. page 7.1. Gower Medical Publishing, London, 1992.)

A. THE PERIMEMBRANOUS VSD

i. Perimembranous inlet VSD

This VSD is the result of a failure of the primitive interventricular septum to fuse with the endocardial cushions.[19]

The anatomic location of the VSD is beneath the septal leaflet of the tricuspid valve, with the medial papillary muscle (*muscle of Lansici)* above and to the left of the defect when viewed from the right atrium.[1]

The septal leaflet may, on occasion, harbour a cleft, though this is generally associated with an AV canal (endocardial cushion) defect.[1, 20]

An important consideration in the repair of perimembranous inlet VSDs, is the particular vulnerability of the penetrating atrio-ventricular bundle (bundle of HIS) located on the postero-inferior aspect of this VSD.[1, 20]

ii. Perimembranous outlet VSD

This defect probably results from a failure of fusion between the primitive interventricular septum and fibrous membranous continuity of the aortic valve.[19]

The VSD is typically located underneath the antero-septal commisure of the tricuspid valve, with the medial papillary muscle of Lansici to the right of the VSD when viewed from the right atrium.[1, 20]

An important consideration during surgical closure, is the close relationship of the aortic valve to the VSD because damage to the right or non-coronary cusps of the aortic valve may occur.[1, 20]

iii. Perimembranous subarterial VSD

The basis of this defect is failure of the bulbospiral septum to fuse with the muscular septum at the crista supraventricularis, hence another term used is a *supracristal* defect. Other descriptive terms for this defect are *juxta-arterial* and *doubly committed* VSD.[1, 3, 20]

An important feature of this defect is the potential for development of aortic incompetence, due, possibly, to lack of support to the right (and less commonly the non-coronary) cusp of the aortic valve, or from a V*enturi* effect as mentioned earlier.[16]

B. THE MUSCULAR VSD [1]

Muscular VSDs have entirely muscular rims.[1, 20]

These defects are often multiple and they can co-exist with perimembranous VSDs. When there are several small muscular VSDs, the condition has been termed a "Swiss Cheese" defect.[1, 3]

The classification of muscular VSDs is simplified by considering the defect's position in the inlet, apical trabecular, or outlet components of the right ventricle.[20]

CHAPTER 5

SURGICAL OPTIONS

Operations on VSDs are employed hrough a midline sternotomy. Aortic and bicaval cannulation is carried out and cardiopulmonary bypass instituted, before cold cardioplegic arrest at moderate hypothermia is effected. The cavae are snared prior to aortic cross clamping. Deep hypothermic arrest may rarely be needed in a small infant (< 3kg) if bulky venous cannulae impede exposure.[1, 3]

Various surgical methods have been used and these will be discussed under the following headings:

5.1 *Approach*

5.2 *Patch Material*

5.3 *Suture Techniques*

5.4 *The Red Cross method*

5.1 APPROACH

5.1.1 Transatrial approach

i. Indication:

The transatrial (or right atrial) approach (fig 5.1.1) is the preferred approach to most isolated perimembranous or inlet muscular VSDs.[1, 3]

Trabecular muscular and infundibular muscular defects may also be accessible through the right atrium, but not always.[1]

ii. Incision:

The usual approach is an oblique incision in the right atrial wall, anterior to the sulcus terminalis, extending inferiorly toward the inferior vena cava.

Access to the VSD is by gentle retraction on the anterior margin of the right atrium and the anteroseptal leaflet of the tricuspid valve, using small eyelid retractors (fig. 5.1.1).[1]

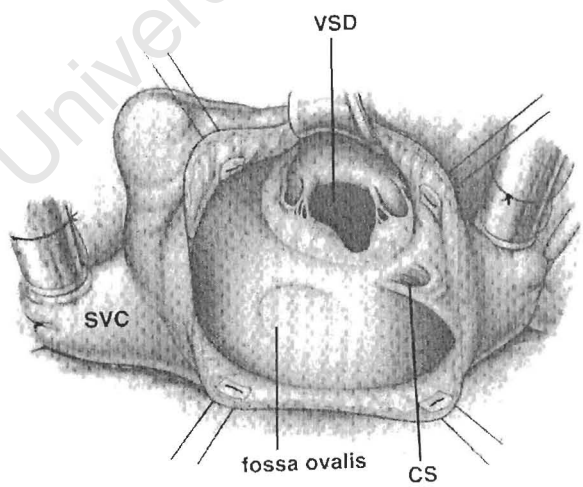


Fig. 5.1.1 Right atrial approach showing a perimembranous VSD

(From : Stark J, de Leval M. Surgery for congenital heart defects. Second Edition, page 357. W.B. Saunders Company, Philadelphia,1994.)

If need be, exposure of the VSD may be facilitated by detachment of the overlying tricuspid valve leaflets from the annulus, with minimal disruption of post-operative tricuspid valve function.[21]

The transatrial approach is the preferred approach for closure of isolated perimembranous VSD, with a few exceptions. [12, 21, 22]

5.1.2 Right Ventricular Approach

i . Indication:

If possible, a right ventriculotomy is avoided because of the potential for RV failure and arrhythmias [5], but is occasionally necessary for a subarterial perimembranous or a trabecular muscular VSD. [1]

This approach is also occasionally required for other reasons e.g. enlargement of the right ventricular outflow tract or for resection of subpulmonic stenosis. [1,3]

ii. Incision:

A transverse or longitudinal (if the right ventricular outflow tract needs a patch or a valved conduit) incision across the infundibulum is made about 5-10 mm away from the left anterior descending coronary artery. Exposure is facilitated by stay stitches and retraction (fig. 5.1.2).[23]

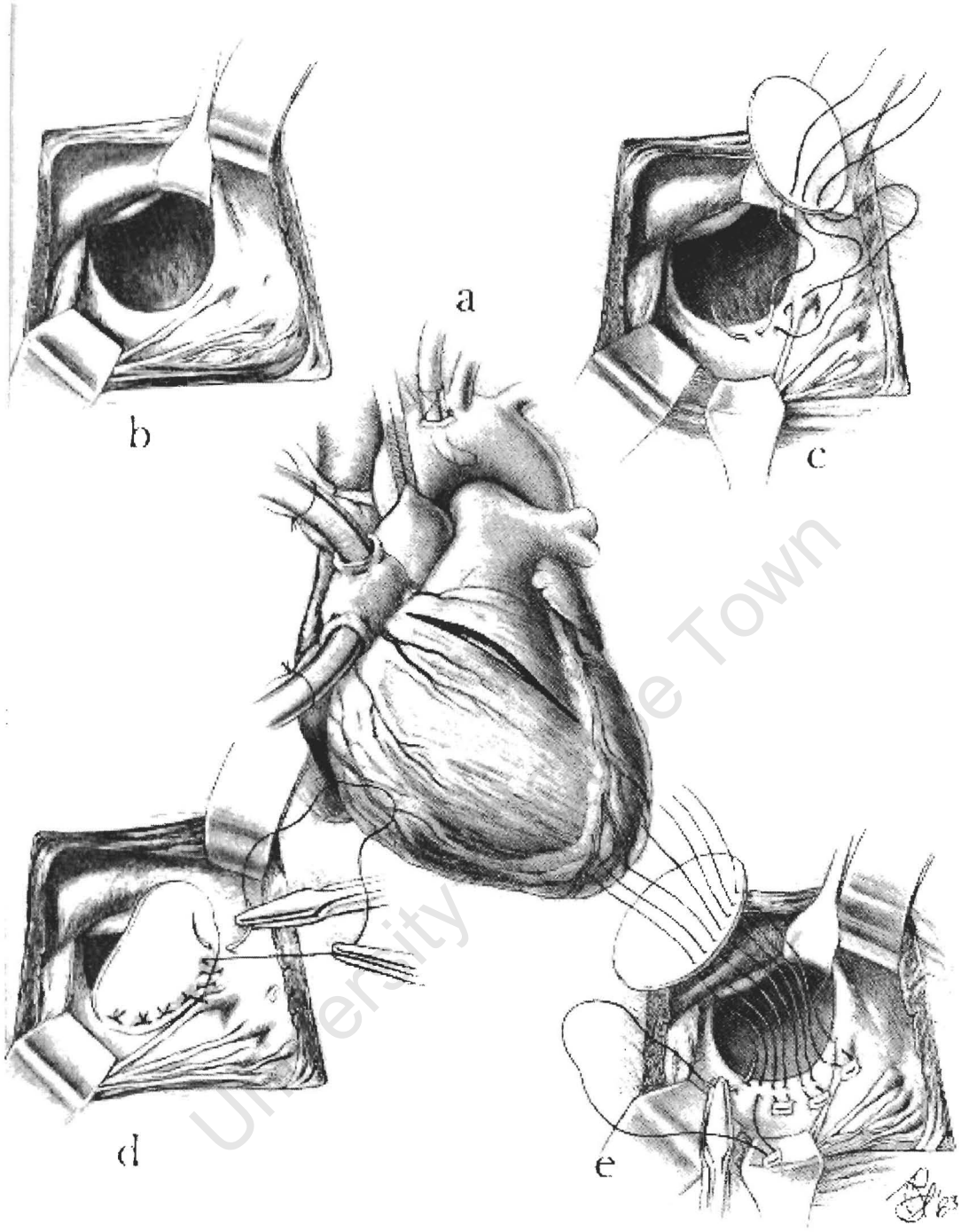


Fig. 11B–5.— *Technique of transventricular repair of VSD. a, the incision is made in the outflow tract of right ventricle, avoiding interruption of major coronary artery branches. b, the VSD is exposed by traction on the tricuspid valve apparatus and on the superior margin of the VSD. c, simple horizontal mattress sutures may be used, placed along the inferior margin of VSD. d, VSD closure using a combination of interrupted mattress sutures and a continuous suture. e, technique of suture placement with interrupted pledgeted sutures.*

Fig. 5.1.2 Right ventricular approach to subarterial perimembranous VSD

(From: Arciniegas E. Pediatric Cardiac Surgery. pp. 148. Year Book Medical Publishers, Chicago, Ill,1985.)

5.1.3 Left Ventricular Approach

i. Indication:

This approach is to be avoided, especially in infants, but is indicated for apical / trabecular muscular VSDs, particularly when there are multiple defects.[1]

ii Incision:

After elevation of the heart with a swab, a short 'fish-mouth' incision is made at the apex of the left ventricle, away from and parallel to the left anterior descending coronary artery. Care must be taken to avoid damage to the anterior papillary muscle of the mitral valve.[1]

There is always a degree of left ventricular dysfunction following this approach and it is therefore not advised in the infant or small child (< 4 years of age), when the incision is proportionately larger relative to the LV size in the small heart. [1]

An associated perimembranous VSD should be closed through a separate transatrial approach, as its repair through the left ventriculotomy increases the risk of postoperative heart block.[3]

5.1.4 Pulmonary Artery Approach

i. Indication:

The perimembranous subarterial VSD can sometimes be better accessed via the pulmonary artery.[1]

ii. Incision:

A longitudinal pulmonary arteriotomy is made; stay sutures are placed in the pulmonary valve leaflets and the retraction on the pulmonary artery incision is facilitated with eyelid retractors.[22]

This may be a useful alternative to a right ventriculotomy, especially in the young child or infant without subpulmonic stenosis.[1]

5.1.5 Aortic Approach

i. Indications:

This approach is sometimes useful in patients with a subarterial perimembranous VSD, with associated aortic valve incompetence; especially in an older child.[1]

ii. Incision:

An oblique aortotomy incision is made in the ascending aorta. A subarterial perimembranous VSD is exposed using eyelid retractors and stay sutures.[22]

This is a difficult approach to most VSDs and when the aortic root is opened for other reasons, it is still generally easier to use an atriotomy for the VSD as well. Special care should be taken to avoid damage to the aortic valve leaflets.[1, 3]

5.2 PATCH MATERIAL

Patch closure of a VSD is the recommended method of closure (as opposed to direct suture closure), in order to offer a tension-free, non-distorted, anatomical and secure closure. The patch should be sized 10-20% bigger than the actual defect.[1, 3]

The following patch materials are commonly used for closure of VSDs:

5.2.1 Biological patches

i. Autologous pericardium
   - Treated (with gluteraldehyde)
   - Untreated

ii Xenograft pericardium (bovine)

5.2.2 Synthetic Patches

i. Double velour-knitted dacron (*"de Bakey"* patch)

ii. Expanded Poly-tetra-fluoro-ethylene (ePTFE) (Goretex®)

The prosthetic (Dacron) patches are the most widely used for VSD closure.[1, 3, 8, 11, 12] These patches and the xenograft pericardium patches are expensive, may not always be readily available and most importantly, consist of foreign material permanently implanted in the heart, with the attendant risks of infection.

Untreated autologous pericardium has not been widely favoured due to a fear of postoperative aneurysm formation in the patch.[6, 24] However, a recent study discounts this complication, provided the patch is properly sized.[6]

We have chosen to use 0.6% glutaraldehyde-treated autologous pericardium as a cheap and logical alternative. The fixation also makes the patch easier to work with.

5.3 SUTURE TECHNIQUES

The following techniques are used for attaching the selected patch to the interventricular septum when closing a VSD:

5.3.1 Continuous Suture

i. Simple suture

ii. Horizontal mattress suture

5.3.2 Interrupted Sutures with pledgets

5.3.3 *Combination: Interrupted Pledgetted and Continuous Suture*

Although direct suture closure techniques of isolated perimembranous defects have been described, this option is discouraged, as it commonly results in undue tension on the sutures, with distortion of the anatomy and potential for breakdown of the repair.[1, 3] Direct suture closure should be reserved for slit-like muscular defects or for tiny defects with a fibrous rim. [1]

For the continuous suture technique, a 5.0 polypropylene (Prolene®) double-armed suture on a 10mm needle is commonly used. The following points are relevant to placement of sutures:

i. The bundle of HIS lies at the posterior-inferior angle of the perimembranous inlet VSD. Sutures in this area should be superficially placed, approximately 5-7 mm away from the inferior edge of the VSD, on the right side of the septum to avoid damaging the conduction tissue.[1,3,13] One study has suggested superficial suture placement along the *rim* of the defect is possible without danger of complete heart block. [21]

ii. The right and non-coronary aortic cusps are vulnerable during suture placement along the ventricular infundibular fold in perimembranous outlet and subarterial VSDs.[1, 20]

iii. The free muscular edge of a perimembranous VSD anterior to the medial papillary muscle of Lansici can tear easily and deep securing sutures should be placed in this area.[1, 3] Simple sutures have a greater propensity to tear through muscle and a horizontal mattress suture is theoretically a better alternative.

iv. Horizontal mattress sutures are placed through the base of the tricuspid valve septal leaflet, 1-2 mm from the annulus, as suture placement through the central fibrous body can also damage the penetrating bundle.[1, 3]

Many centres advocate reinforcing the simple continuous suture technique with interrupted mattress sutures using Teflon pledgets, especially along the posterior-inferior margin (so sutures may be placed more superficially) and on the tricuspid valve (where the leaflet tissue may be thin).[1, 3]

5.4 THE RED CROSS METHOD

A. The Incision:

Rather than the usual oblique incision which is lower on the RA wall—see fig. 5.1.1, the incision is made *parallel to* the atrioventricular groove, anteriorly on the RA free wall. It begins at the *RA appendage* about 8-10mm from the AV groove and extends parallel to the groove, *anterior* to the IVC cannula (fig. 5.4.1). The reasons for this approach are:

i. Theoretical advantage over the oblique incision in terms of late atrial arrhythmias.

ii. Improved exposure of the tricuspid valve. Access to the whole atrial septum is excellent, so that atrial defects can also be dealt with via this route.

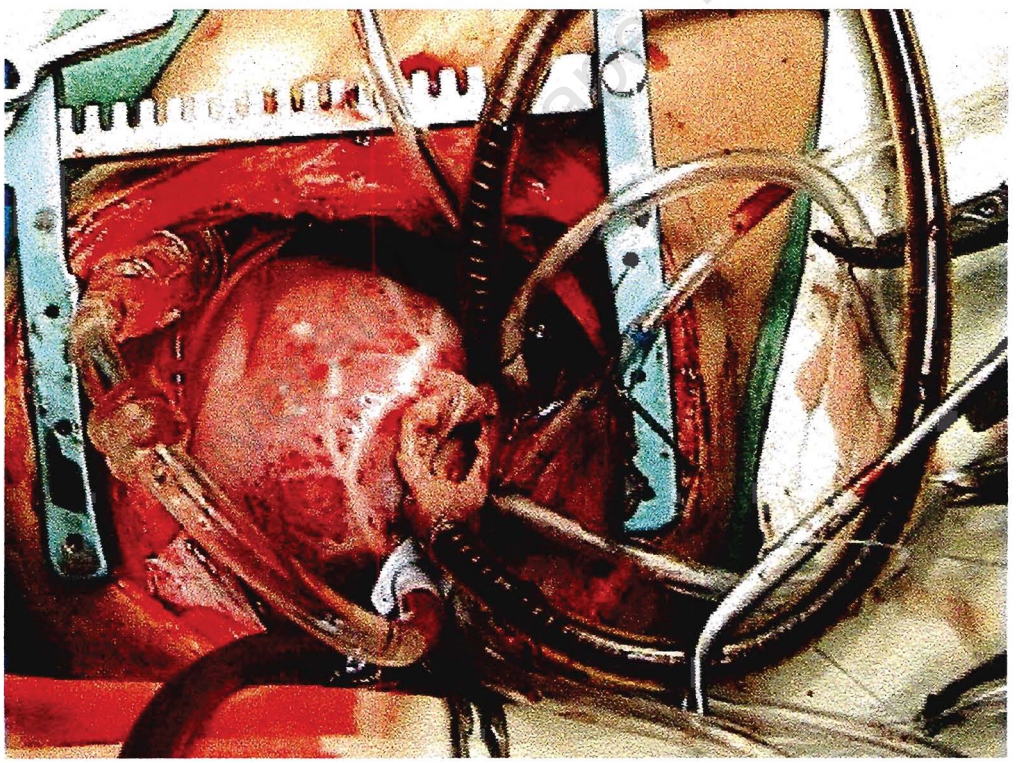


Fig. 5.4.1 Parallel right atrial approach, between the caval cannulae

The preferred patch material at Red Cross Children's Hospital for the closure of VSDs, is autologous pericardium harvested after opening the sternum. (fig. 5.4.2 –5.4.3).

It is held taut with forceps in a 0.6% glutaraldehyde solution for 15 seconds (fig. 5.4.3 - 5.4.4) to reduce contraction and is then left to soak in the solution for a further 5 minutes.

It is then washed and kept in normal saline until needed (fig. 5.4.5).

The resultant patch is stiffened and easy to use. After tailoring the patch to a size at least 20% larger than the defect, it is implanted. (fig. 5.4.6)

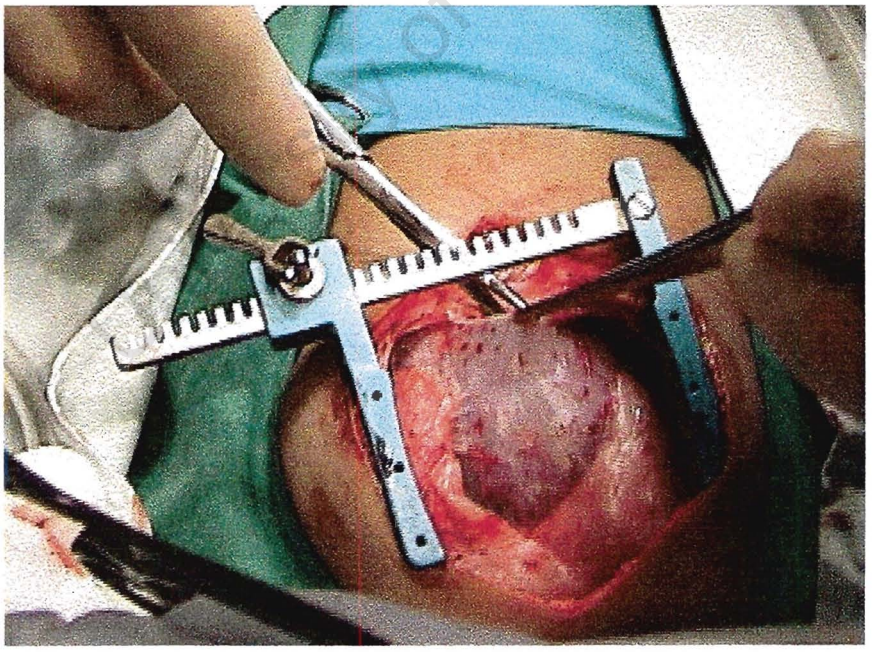


Fig. 5.4.2 – Harvesting of autologous pericardial patch after sternotomy

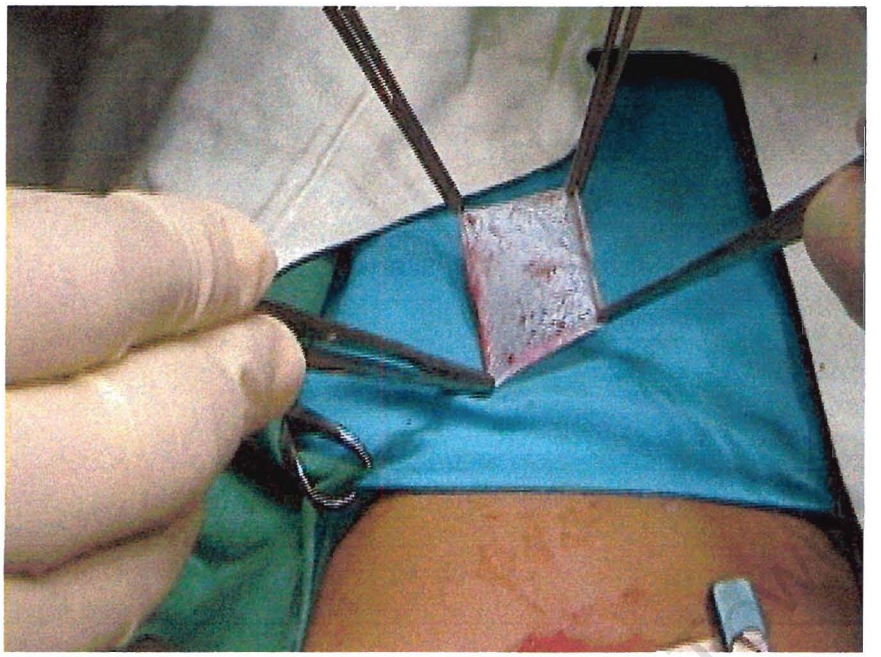


Fig 5.4.3 Freshly harvested autologous pericardial patch

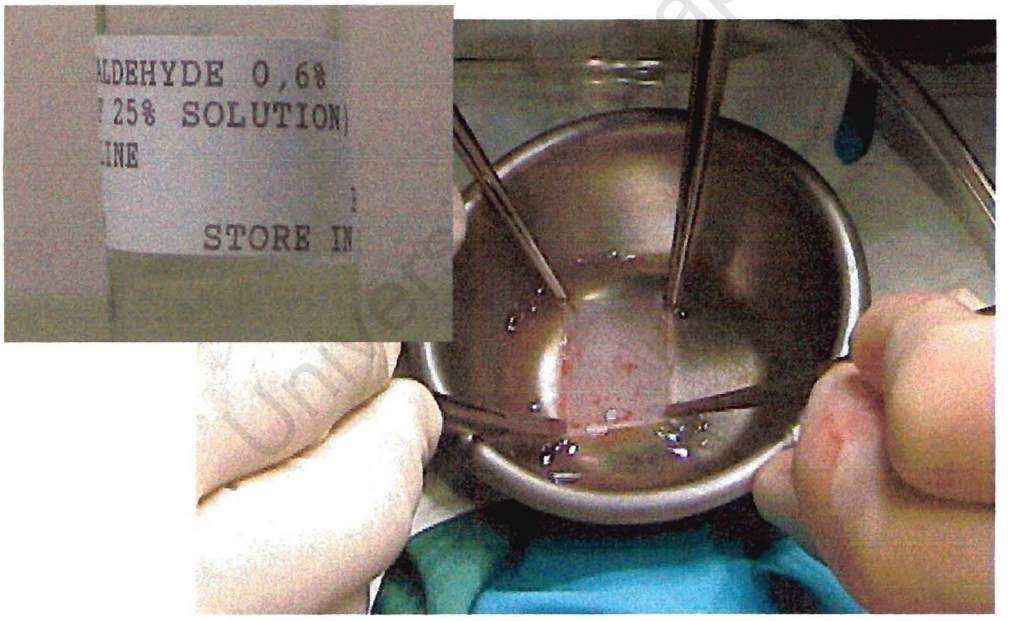


Fig. 5.4.4 Soaking the taut pericardial patch in 0.6% glutaraldehyde solution for 15-20 seconds

Fig 5.4.5 Treated pericardial patch is soaked and kept in normal saline while surgeon prepares for bypass

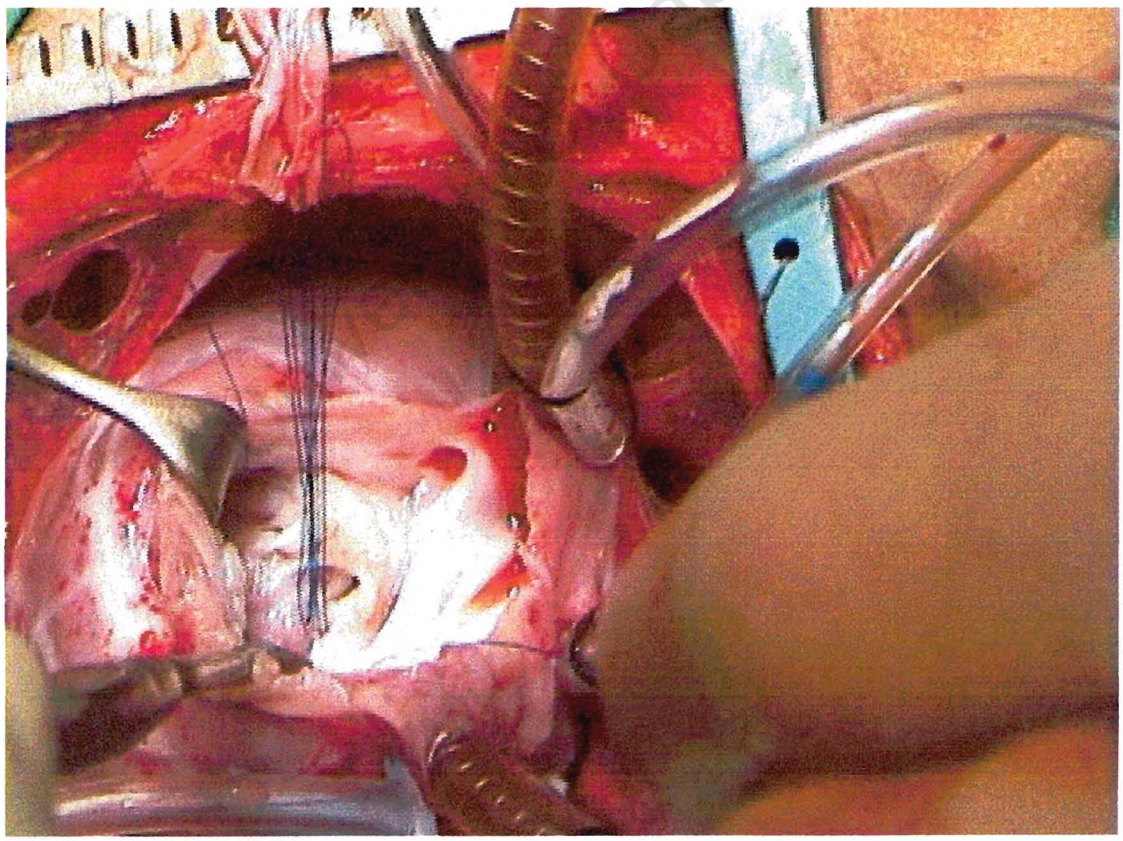


Fig 5.4.6 Implanting the treated autologous pericardial patch

C. *The Suture Technique: Continuous horizontal mattress*

This is a continuous horizontal mattress suture (fig. 5.4.7). Pledgetts are considered unnecessary because of the inherently better purchase of this suture on the muscle.

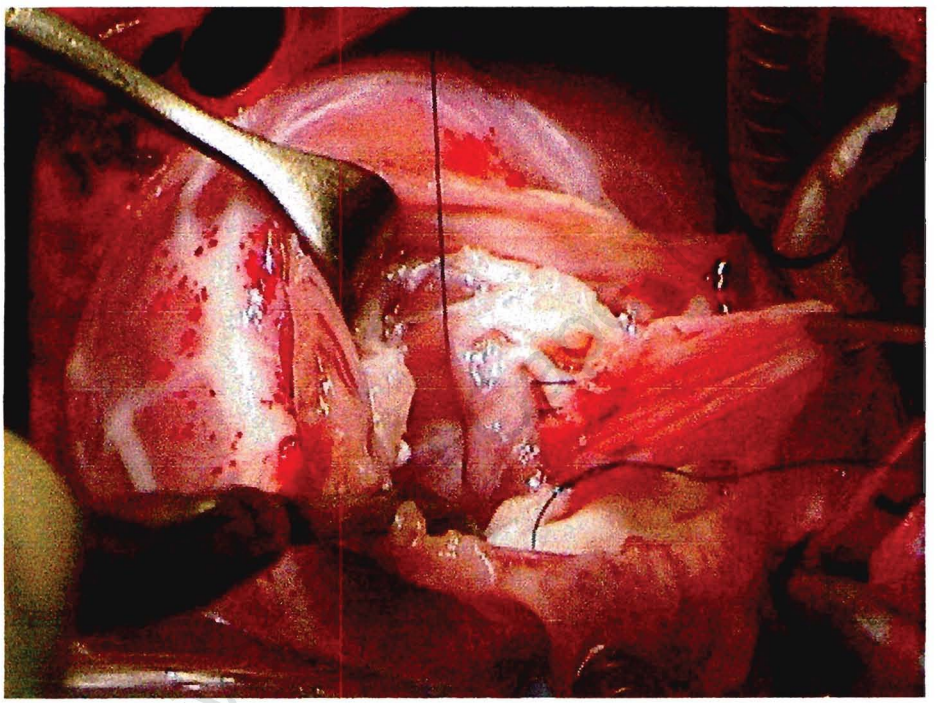


Fig 5.4.7 Horizontal mattress suture technique presently in use at Red Cross Children's hospital

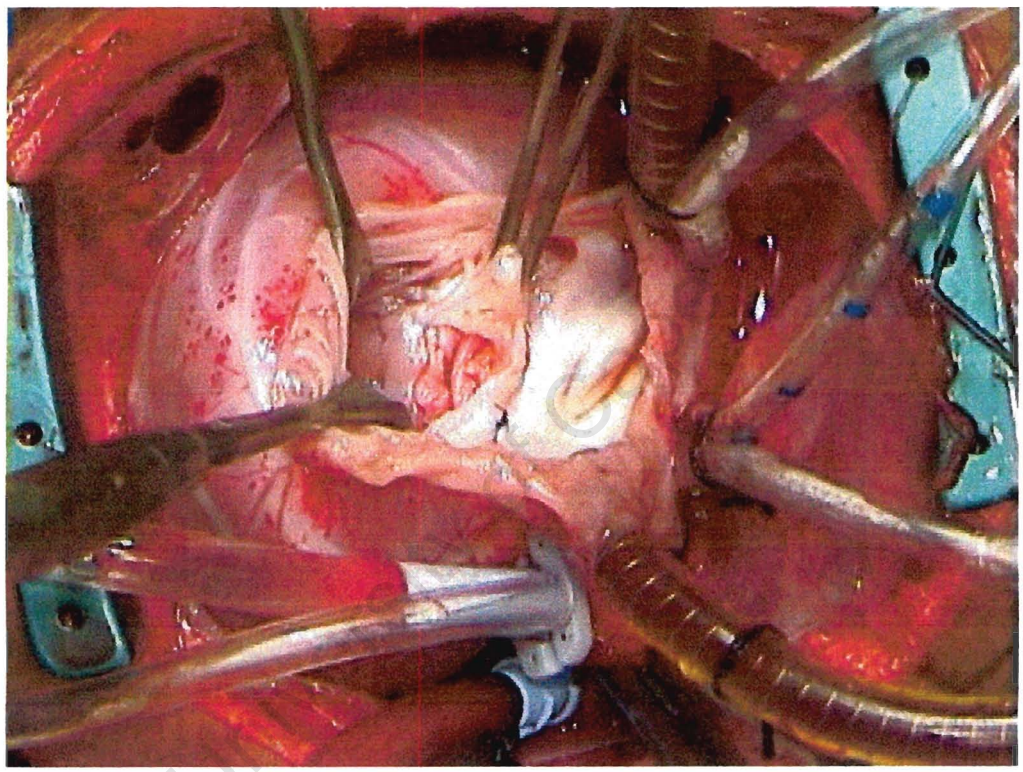


Fig 5.4.8 Suture line along the annulus of the septal leaflet of the tricuspid valve (enlarged detail)

PART II
Comparative analysis of surgical techniques used in the closure of perimembranous VSD

CHAPTER 6

The surgical technique used in the closure of perimembranous ventricular septal defects at Red Cross Children's Hospital since 1994 has not been described in the literature. This technique involves the use of glutaraldehyde-treated autologous pericardium to fashion a patch which is sewn into place with a continuous, horizontal mattress suture without any pledgets, using a 5.0 polypropylene (Prolene®) suture and approaching the VSD through a right atrial incision, parallel to the AV groove.

The technique most often employed prior to 1990 at Red Cross Children's Hospital consisted of a double velour knitted Dacron synthetic patch, sewn into place with interrupted pledgetted sutures, using alternating 5.0 Ticron® and 5.0 Prolene® sutures. Other variations in technique during this period, included a RV and a PA approach. The introduction of bovine pericardium also made its appearance at the latter stages of this period and gained popularity after 1991.

Between 1991 and 1993, the Dacron patch technique was mostly replaced by bovine pericardium to close VSDs. The bovine pericardium was secured with continuous simple horizontal suturing technique using 5.0 Prolene® begun on a Teflon pledget; the TV septal annulus reinforced with a strip of autologous pericardium and the final knot tied onto a Teflon pledget.

From 1994 the use of the autologous pericardial patch was the preferred mode of VSD closure by all surgeons at Red Cross Children's Hospital.

6.1 OBJECTIVES

It was decided to compare the current surgical technique used for the closure of perimembranous ventricular septal defect without major associated anomalies (0.6% glutaraldehyde treated autologous pericarial patch), to the previously used technique (double velour dacron patch) in such patients at Red Cross Children's Hospital.

The purpose of this comparative analysis is to ascertain the advantages and disadvantages of the two techniques, looking particularly at the incidence of postoperative residual VSD requiring repeat surgery or long-term follow-up.

The mortality rate of the two groups was analysed with particular reference to the under-one-year-old age group. Recommendations with regards to primary closure in the under-one-year-old, high risk age group[2, 10, 11, 12, 13] will be made in relation to the surgical technique used.

6.2. METHODOLOGY

A retrospective study was done at the Red Cross Children's Hospital, Rondebosch, Cape Town, in which two separate case-matched groups of children undergoing different surgical closure techniques for their isolated perimembranous ventricular septal defects were compared.

6.2.1 PATIENTS

Group I consisted of 77 children operated on between January 1987 and December 1990.

Group II consisted of 93 children operated on between January 1995 and December 1998.

The period between 1990-1994 was excluded since a wide variety of techniques were used, including the use of a *bovine* pericardial patch. This period did not offer an easily defined group.

Thus, the study focussed on the Dacron patch[1, 3, 8, 11, 12] technique (Group I), used extensively during the 1980's and early 1990's at Red Cross Children's Hospital, comparing it to the present technique of VSD closure (Group II), excluding the period 1990-1994.

Seventy-one of the seventy-seven children (93%) in Group I had VSDs closed with a double velour-knitted dacron synthetic patch ("de Bakey" patch), sewn in place with interrupted pledgetted sutures, using alternating 5.0 Ticron® and Prolene® sutures (alternated to enable easy identification when tying). Of these, 67 (87%) were done through an oblique right atrial incision, 2 (3%) via a right ventricular approach (infundibular) and 2 (3%) via a pulmonary artery approach.

A further 5 (6%) in Group I had VSDs closed using a bovine pericardial patch sewn into place with a simple continuous 5.0 Prolene® suture, using autologous pericardium to reinforce the TV annulus suture line.

One patient (1%) in Group I had a VSD closed directly with interrupted pledgetted sutures of 5.0 Prolene®.

Group II consisted of 93 children all of whom had closure of isolated perimembranous VSDs; a 0.6% glutaraldehyde-treated autologous pericardial patch was sewn into place using a continuous horizontal mattress suture, without pledgets, with a 5.0 Prolene® suture via a right atrial incision, parallel to the AV groove.

Only children with an isolated perimembranous ventricular septal defect were entered into the study. Children with associated major congenital cardiac anomalies (e.g. Tetralogy of Fallot, double outlet right ventricle, endocardial cushion defects, etc.) were excluded.

Children with the following minor associated anomalies were included in the study:

- Patent ductus arteriosus	Group I: 1/77 Group II: 3/93
- Patent foramen ovale	Group I : 10/77 Group II: 15/93
- Minor pulmonary stenosis	Group I: 0/77 Group II: 4/93

Children who had previous pulmonary artery banding (3/77 in Group I and 8/93 in Group II) to prevent severe pulmonary hypertension associated with VSD were *included* in the study.

All children were operated on at Red Cross Children's Hospital and they were all under the age of 13 years.

A further subdivision was made for the *under one year age group*:

- Group I: 43/77
- Group II: 36 /93

This higher risk sub-group was subjected to separate analysis to assess mortality.[2, 10, 11, 12, 13]

The combination of indications for operation in both groups were as outlined in chapter 2, viz.:

i. Intractable congestive cardiac failure

ii. Controlled cardiac failure with failure to thrive

iii. Recurrent respiratory tract infection

iv. Elevated pulmonary vascular resistance

v. Aortic incompetence associated with the VSD

vi. Infective endocarditis

vii. Development of subpulmonic stenosis (double chambered right ventricle)

viii. Low probability of closure viz. large right to left shunt without pulmonary hypertension

6.2.2 MATERIALS

The patients in Group I were identified from the Red Cross Children's Hospital theatre registers and the diagnosis and surgical management confirmed by reviewing the patients' hospital folders and operation notes.

The patients in Group II were identified through a computer-generated list from the existing Cardio-thoracic surgery database at Red Cross Children's Hospital.

All patients who had closure of an isolated VSD were noted and cross-checked with the patients' hospital folders to ensure accuracy of diagnosis and surgical management.

The relevant preoperative, intraoperative and postoperative information was extracted from the patients' hospital folders. Follow-up data was available in 54/77 patients in Group I and 83/93 patients in Group II. This was obtained through the Cardiology department at Red Cross Children's Hospital, which keeps separate follow-up records for local patients. Follow-up data for patients referred from the Eastern Cape was obtained with the help of the attending paediatricians at Frere and Cecilia Makiwane Hospitals in East London. Thirteen patients in Group I and six patients in Group II from the Eastern Cape were lost to follow-up.

A further 10 patients in Group I and 4 patients in Group II were either foreign patients referred back to their country of origin, or patients from rural parts of South Africa with no contact details and were lost to follow-up.

Cardiopulmonary bypass and cross-clamp times could not be obtained for 4 patients in Group I and 2 patients in Group II.

All information compiled was tabulated in the *Microsoft Excel* computer programme under the following headings:

A. Group

B. Name

C. Folder number

D. Date of birth

E. Weight in kilograms

F. Degree of pulmonary hypertension

G. Date of operation

H. Age (in days)

I. Aortic cross clamp time

J. Cardiopulmonary bypass time

K. Degree of residual VSD at discharge

L. Degree of residual VSD at last follow-up

M. Need for re-operation for residual VSD

N. Date of re-operation

O. Mortality

P. Date of death

Q. Sternal wound infection needing resuturing and debridement

R. Complete heart block after repair requiring permanent pacing

S. Postoperative junctional ectopic tachycardia

T. Postoperative tamponade or bleeding requiring re-operation

U. Permanent postoperative neurologic deficit

V. Postoperative right ventricular failure

W. Postoperative infective endocarditis

X. Operative surgeon

Y. Date of last follow-up

Z. Follow-up in months

AA. Associated / alternative procedure done with VSD closure

A follow-up of 70% (54/77) in Group I and 90% (83/93) in Group II was obtained . A total of 23 patients and 10 patients (total = 43) were lost to follow-up in each group respectively.

A further analysis relating complications and mortality to the individual surgeon was also analysed.

6.2.3 STATISTICAL ANALYSIS[25, 26]

The data was captured using the *Microsoft Excel* computer programme. The *Stata 6 Software Programmme- 6*[27] was used to analyse univariate and bivariate statistics; means, standard deviation, medians and range and percentages were calculated where applicable, using the *Excel* programme as well as the *Stata 6 Software Programme- 6.*[27]

Comparisons for age, weight, cross clamp time, CPB time and follow-up period were made by the non parametric, *Wilcoxon rank-sum (Mann-Whitney) test* using the *Stata 6 Software Programme- 6*[27]; this test was chosen due to the skewed data distribution of variables (see box and whisker plot). Statistical significance of residual VSD, mortality and other complications between Groups I and II was determined, using the *Chi square* and / or the *Fisher Exact* test through the *Sata 6 Software Program- 6.*[27]

A p-value of <0.05 was deemed to be statistically significant - the test that tended towards statistical significance was chosen between the two tests.[25, 26]

Graphs (bar, line, scatter and pyramid) were used to depict the differences between Groups I and II, using the *Microsoft Excel* computer programme (see *Results*).

6.3 RESULTS

See Table 6.3.1 for a summary of results. The results will be further detailed under the following headings:

6.3.1 Patient profile (preoperative factors)

6.3.2 Cross-clamp and cardiopulmonary bypass time

6.3.3 Residual VSD

6.3.4 Mortality

6.3.5 Postoperative complications

6.3.6 Follow-up

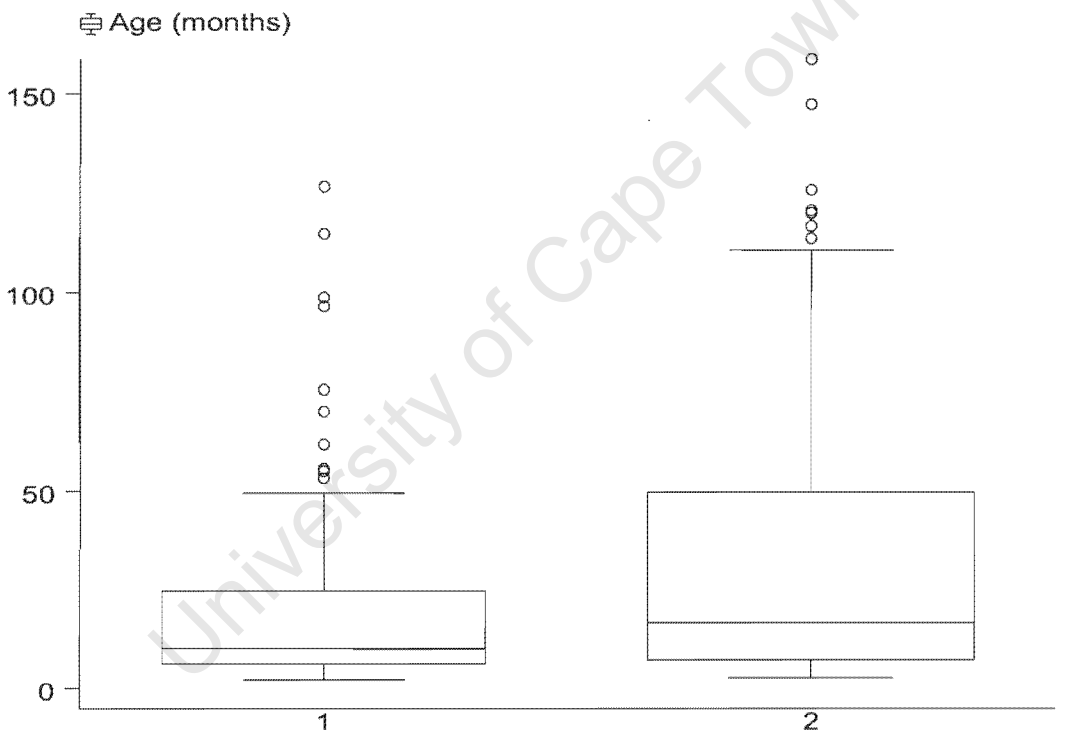
Table 6.3.1 Summary of Results

VARIABLE	GROUP I	GROUP II	COMBINED	*p* VALUE
	n =77	*n* =93	*n* =170	ns
Age (mnths))(median & range)	10.2 (2.2-127)	16.8 (2.7-159)	14 (2.2- 159)	= 0.0355 (Wilcoxon)
Children < 12 mnths	**56%(n=43)**	**39%(n=37)**	**47%(n=80)**	**= 0.04** (chi sq)
Weight (kg)(median & range)	6 (3.1-23)	8 (2.6-31)	7 (2.6-31)	= 0.06 (Wilcoxon)
wt < 12 mnths (kg) mean+- 2SD	**5.1 ± 1.1**	**5.3 ± 2.7**	**5.2 ± 2**	**ns**
Children < 5 kgs (<12 mnths subgroup)	**30%(n=23)**	**23%(n=17)**	**27%(n=40)**	**ns**
Preop. Cardiac cath.	100%(n=77)	28%(n=26)		<0.001 (chi sq)
Pulm HPT	78%(n=61)	66%(n=61)		= 0.07 (chi sq)
X-clmptm (min) (median&range)	**50(22-110) (n=67)**	**33(117-99) (n=91)**	**45(22-110) (n=158)**	**< 0.001** (Wilcoxon)
CPBtime (min) (median&range)	**86(45-231) (n=67)**	**59(29-139) (n=91)**	**75(29-231) (n=158)**	**< 0.001** (Wilcoxon)
Res. VSD @ D/C	**35%(n=25)**	**63%(n=58)**		**<0.001** (chi sq)
Res. VSD@ D/C trivial	0%(n=0)	62%(n=57)		<0.001 (chi sq)
Res.VSD@D/C-small LtoR shunt	33%(n=24)	1%(n=1)		<0.001 (chi sq)
Res. VSD @ D/C-large shunt	1%(n=1)	0%(n=0)		ns
Res.VSD @last ff up	27%(n=13)	17%(n=14)		ns
trivial VSD @ ff up	0%	0%		ns
small L to R shunt @ ff. Up	21%(n=10)	16%(n=13)		ns
large L to R shunt @ ff. Up	6%(n=3)	1%(n=1)		ns
Spont closure rate of res. VSD	**36%(n=9)**	**76%(n=44)**		**<0.001** (chi sq)
Reop. for res. VSD	**4%(n=2)**	**1%(n=1)**		**ns**
Total incidence of reop.	6%(n=2)	1%(n=1)		ns
Mortality (<30day)	**7%(n=5)**	**1%(n=1)**		**= 0.04** (Fisher Exact)
Mortality (late)	2%(n=1)	0%(n=0)		ns
Mortality (total)	11%(n=6)	1%(n=1)		= 0.02 (Fisher Exact)
Mortality in <1yr subgroup	7%(n=4)	1%(n=1)		= 0.08 (Fisher Exact)
sternal infx	4%(n=3)	1%(n=1)		ns
PPM insertion	4%(n=2)	0%(n=0)		ns
JET	4%(n=2)	2%(n=2)		ns
Tamponade	6%(n=3)	0%(n=0)		= 0.06 (Fisher Exact)
Cardiac arrest leading to perm neurological damage	4%(n=2)	0%(n=0)		ns
RVF	1%(n=1)	0%(n=0)		ns
I.E.	4%(n=3)	0%(n=0)		= 0.06 (Fisher Exact)
Ff up mnths (median&range)	**40(2-153)**	**18.5(1-60)**	**21(1-153)**	**< 0.001** (Wilcoxon)
Ff up *(no.)*	**54**	**83**	**137**	
Pts lost to ff up *(no.)*	**23**	**10**	**33**	
pts ff up (%)	**70%**	**89%**	**81%**	

6.3.1. PATIENT PROFILE

The median age for Group I was less than Group II,
10.2months (range= 2.2-127 months) vs 6.8 months (range= 2.7 -159 months),
respectively. p=0.03. (see box and whisper plot)(Graph 6.3.1.1)

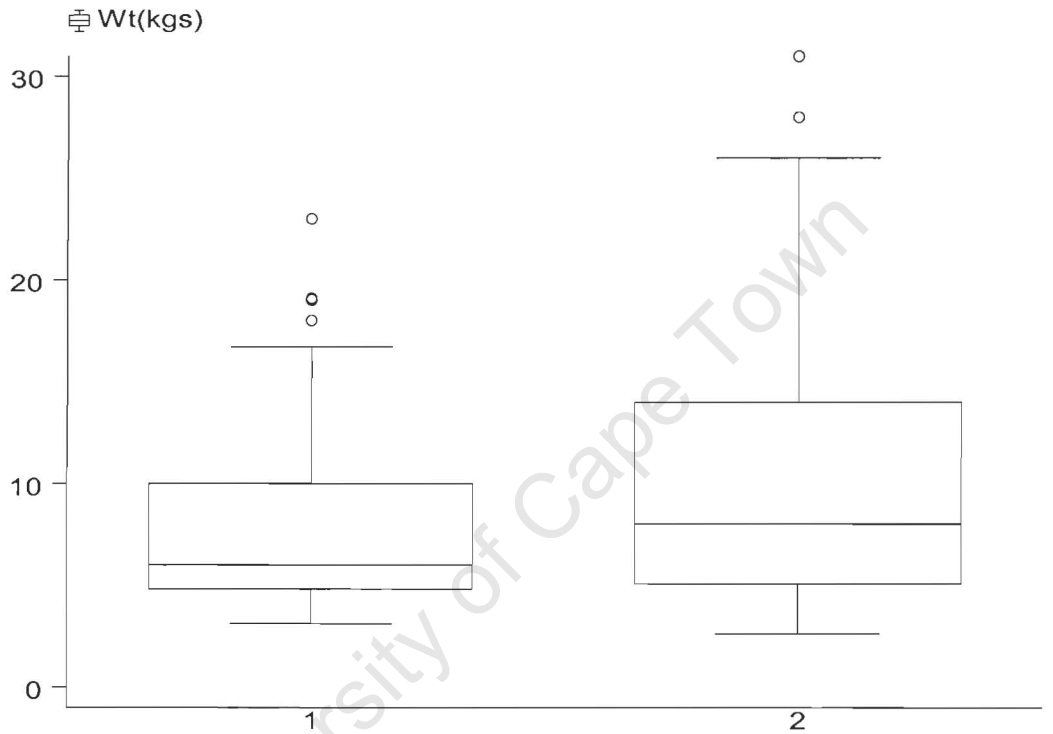
T*he Wilcoxon rank-sum* non-parametric test was used for skewed distribution of age variables between groups I and II.



Graph 6.3.1.1
Box and whisker plot showing skewed distribution of age between groups I and II

The median weight for Group I was 6.0 kgs (range= 3.1 -23 kgs) and for Group II the median weight was 8 kgs (range =2.6-31 kgs). (see graph 6.3.1.2)

T*he Wilcoxon rank-sum* non-parametric test was used for skewed distribution of weight variables between groups I and II..



Graph 6.3.1.2
Box and whisker plot showing skewed distribution of weight between groups I and II

The under-one-year-old subgroup had a mean weight of 5.1 ± 1.1 kg in Group I and 5.3 ± 2.7 kg in Group II (p=0.06). (see Table 6.3.1)
This variable was not statistically significant; uniform distribution of variables allows the use of the *t-test* to determine statistical significance.

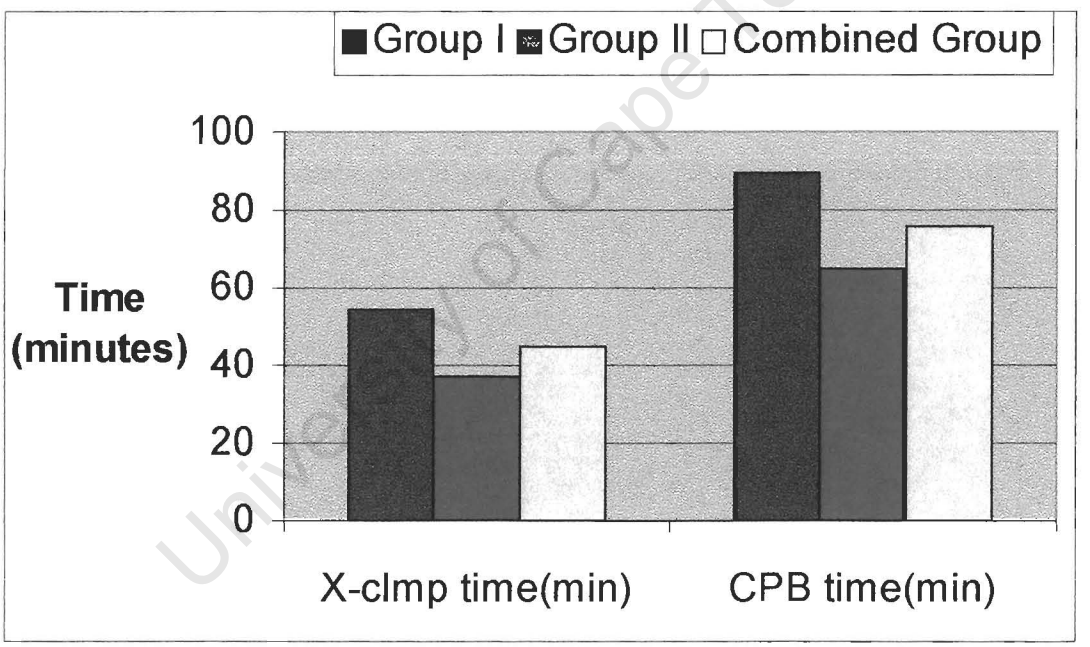
Forty-three patients (56%) in Group I and 37 patients (39%) in Group II were under 12 months of age, while 23 (30%) patients and 17 (23%) patients were less than 5 kg in weight respectively. (Table 6.3.1)

Preoperative pulmonary hypertension was present in 61 patients (78%) in Group I and 61 patients (66%) from Group II (p=0.07). Preoperative cardiac catheterization was done in all Group I patients, but was done in only 26 patients (28%) in Group II (p<0.001). (Table 6.3.1)

6.3.2 CROSS-CLAMP AND CARDIOPULMONARY BYPASS TIME

The median cross-clamp time for Group I was 50 minutes (range = 22-110minutes) compared to 33 minutes (range=17-99) for Group II ($p<0.001$), according to the Wilcoxon rank-sum test. (Table 6.3.1 and Graph 6.3.2)

The median CPB time was also longer for Group I at 86 minutes (range = 45-231min) compared to Group II at 59 minutes (range = 29-139min), with a p value of <0.001, according to the Wilcoxon rank-sum test. (Table 6.3.1 and Graph 6.3.2)



Graph 6.3.2 Difference in operative times of groups I and II

6.3.3 RESIDUAL VSD

At the time of discharge, 25 patients (35%) in Group I and 58 patients (63%) in Group II were noted on echocardiography to have residual VSDs ($p<0.001$).

However, a subanalysis of residual VSDs at the time of discharge, demonstrates a 62% (57/92) incidence of "trivial" VSDs in group II, with none in group I. Group I patients, however, had a 33% (24/72) incidence of a small left to right shunt, as opposed to 1% (1/92) incidence in group II.

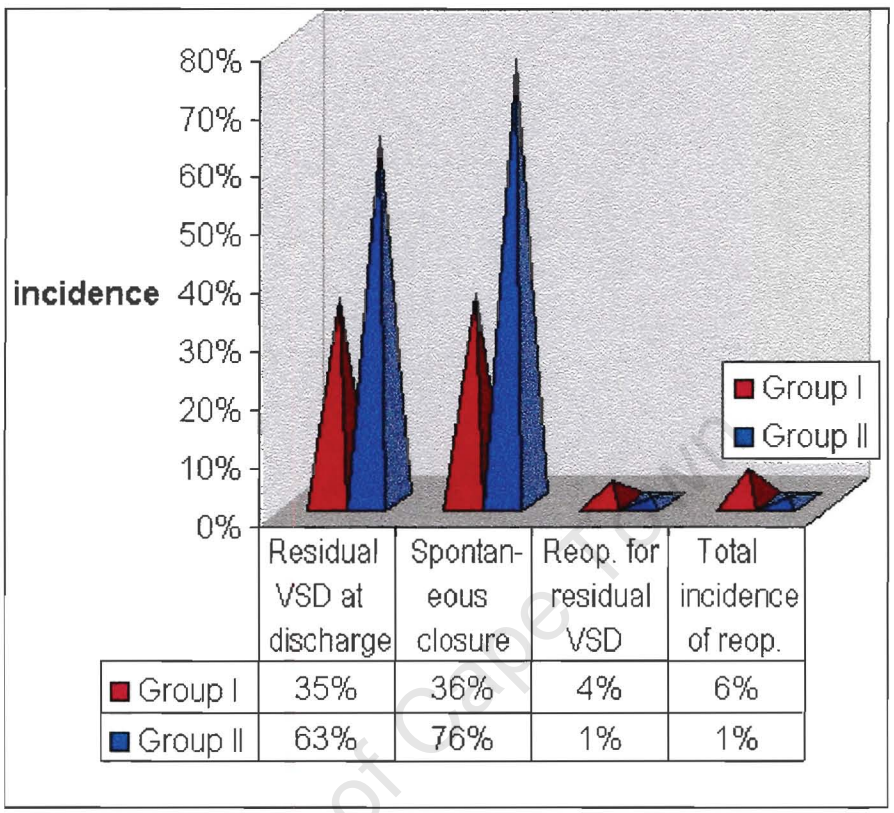
One patient (1%) in Group I had a large residual VSD at the time of discharge.

At the time of last follow-up, 10 patients (21%) in Group I and 3 patients (16%) in Group II were found to have a small left to right shunt.

Three patients (6%) in Group I and 1 patient (1%) in Group II had a large left to right shunt at the time of their last follow-up visit.

Spontaneous closure of residual VSDs after discharge had occurred in 9 out of 25 patients (36%) in Group I and in 44 out of 58 patients (76%) in Group II ($p < 0.001$).

Three patients (6%) in Group I and one patient (1%) in Group II needed reoperation for large residual VSDs. The parents of one of the Group I patients refused reoperation, so actual reoperation rate in Group I was only 4% (2 patients).
(See Graph 6.3.3)



	Residual VSD at discharge	Spontan-eous closure	Reop. for residual VSD	Total incidence of reop.
Group I	35%	36%	4%	6%
Group II	63%	76%	1%	1%

Graph 6.3.3 Residual VSD: Outcome

The 2 patients in Group I who underwent reoperation for their residual VSDs originally had dacron patch closure and direct suture closure of their VSDs respectively. The third patient in the group requiring reoperation, whose parents refused surgery, originally had dacron patch closure of her VSD.

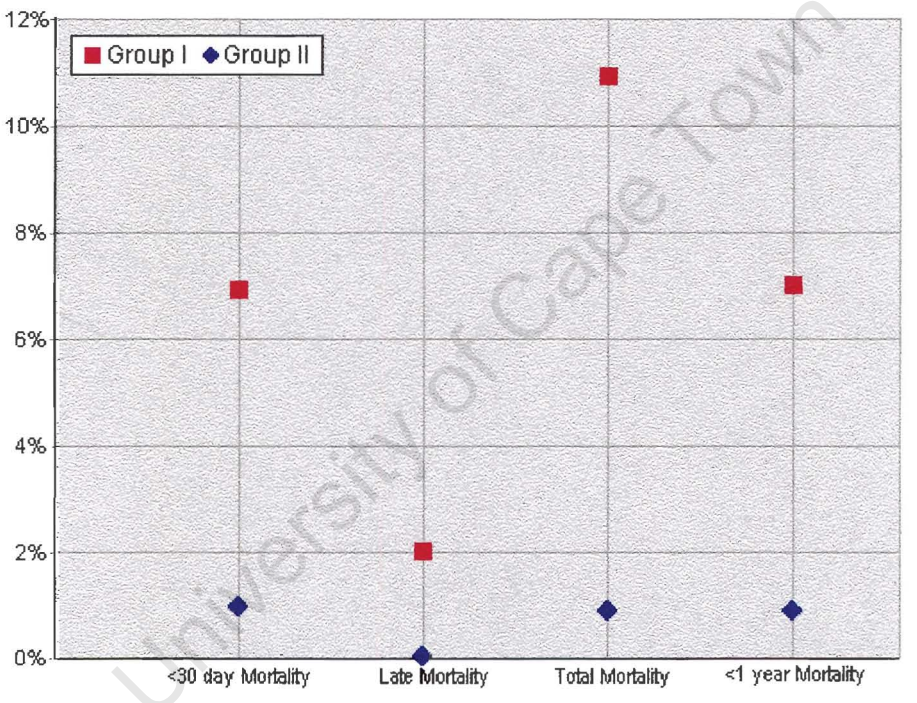
6.3.4 MORTALITY

In-hospital or 30-day mortality occurred in 5 patients (7%) in Group I and only 1 patient (1%) in Group II died (p=0.04).

There was one late death (2%) in Group I. Thus total mortality for Group I was 11% (6/54) and for Group II, 1% (p=0.02).

A subanalysis of the under-one-year-old group revealed a 7% (4/54) mortality rate in Group I, compared to 1% (1/83) mortality rate in Group II (p=0.08).

Further analysis of the combined mortality of groups I and II, showed that 71% (5/7) of all deaths occurred in the under-one-year-old subgroup. The combined mortality rate for the under-one-year-old subgroup was thus 4% (5/137). (See Graph 6.3.4.1)



Graph 6.3.4.1: VSD mortalities

The record of each of the patients who died was examined for possible risk factors, and 10 were identified (see table 6.3.4.1)

Table 6.3.4.1 Risk factors related to mortality

Risk factor	No of patients	% of total deaths	*P* value
Age <12 months	5	71%	
Mod. to severe PHT	5	71%	
Cross clamp time >60 min.	4	57%	= 0.02
CPB time > 110 min.	4	57%	=0.02
Surgical technique (dacron)	6	86%	
Weight ≤ 5 kg.	3	43%	
Single surgeon	3	43%	=0.02
Debanding for previous PAB	2	29%	=0.02
Infective endocarditis	2	29%	< 0.001
Junctional ectopic tachycardia	1	14%	=0.04

6.3.5 POSTOPERATIVE COMPLICATIONS

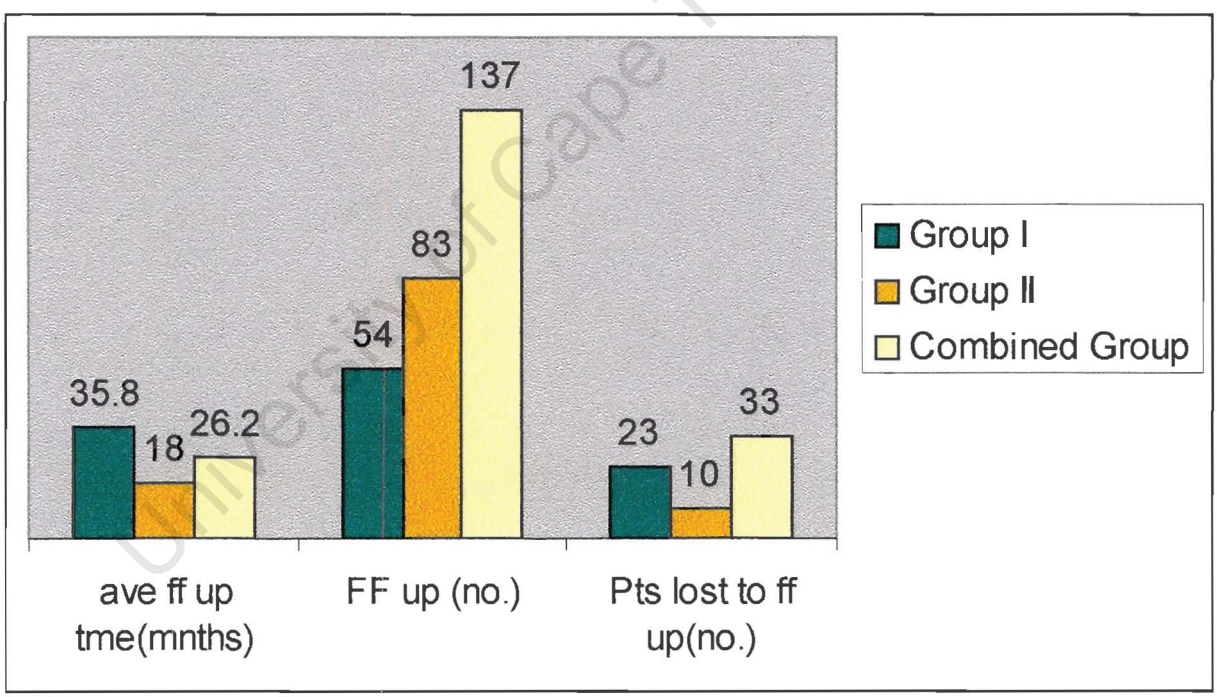
Table 6.3.1 shows a higher incidence of post operative complications in Group I of which tamponade and infective endocarditis were tending towards statistical significance ($p < 0.06$) between Group I (4%; 3/54) and Group II (0%).

6.3.2 FOLLOW-UP

The 70% (54/77) follow-up of patients in Group I was for a median period of 40 months (range = 2-153 months), while the 89% (83/93) follow-up of patients in Group II was for a median period of 18 months (range = 1-60 months)($p<0.001$).

The combined follow-up number was 137 patients (81%), with a combined median follow-up period of 21 months (range = 1-153months). (Table 6.3.1 and graph 6.3.6)

Twenty three patients in Group I and ten patients in Group II were lost to follow-up, i.e.33 of the total 170 patients were lost to follow-up; these patients were either foreign patients whose follow up records were not accessible, or local patients who were well and discharged early. Still others were rural patients (from outside Cape Town), where compliance is a major problem for various social and economic reasons.



Graph 6.3.6: Follow-up data

CHAPTER 7

DISCUSSION

Ventricular septal defect is the most common congenital heart defect[12] with a tendency towards spontaneous closure.[3] VSDs that are referred for surgical closure are subjected to various surgical techniques.[1, 3]

The current surgical technique for closure of perimembranous VSDs at Red Cross Hospital, is that of a 0.6% glutaraldehyde-treated autologous pericardial patch, secured with a continuous horizontal mattress suture, using a single 5.0 polypropylene (Prolene®) suture (without pledgets), via a right atrial approach.

The technique of using a double velour knitted dacron (*'De Bakey'*) patch,[3, 8, 16, 22] sewn in place with alternating 5.0 Prolene® and Ticron® pledgetted sutures, was the favoured option between 1987-1990 after which time various modifications and different techniques were introduced. Towards the latter part of 1990, bovine pericardium was used (n=5/77) to close perimembranous VSDs. These patches were sewn in place with a simple continuous suture, with pledgets used to start and end the suture line. Further pledgetted reinforcements were made along the tricuspid annulus. From 1994 the current technique was adopted and used exclusively; hence the chosen study period for group II of 1995-1998.

The dacron patch technique used in Group I was mainly done through a right atrial approach (n=67/77), with total circulatory arrest being commonly used when the patient was less than 8kg. A right ventricular (n=2/77) or pulmonary artery approach (n=2/77) was also used for subarterial perimembranous VSDs. The surgical approach in Group II was exclusively (n=93/93) through the right atrium using bicaval cannulation and moderate hypothermia with an arrested heart. None of the Group II patients needed total circulatory arrest. This can be partly attributed to the improved design in venous cannulae; not limiting exposure as much as previously.

Although Group II patients fared better than Group I patients, it must be borne in mind that Group I was statistically a higher risk group than Group II, in terms of age, weight and pulmonary hypertension (Table 7.1). Hence better patient selection for primary repair in Group II is partly responsible for the better postoperative outcome seen in this group of patients.

Table 7.1 Preoperative profile

	Group I	*Group II*	*p value*
Age (mnths))(median & range)	10.2 (2.2-127)	16.8 (2.7-159)	= 0.035
Weight(kg)(median & range)	6 (3.1-23)	8 (2.6-31)	=0.06
Pulmonary hypertension	66/77	66/93	= 0.07

Closer analysis of the high risk, under-one-year-old subgroup, [2,3,10,11,14] showed no statistical significance between the two groups with regard to weight (approximately 5kg for both groups), although there were more under one year old patients (n=43/77) in Group I than Group II (n=37/93)(Table 6.3.1)

Pulmonary hypertension was difficult to quantitate between the two groups, as the diagnosis in Group II was largely a clinical one.[17] Only 28% (26/93) of patients undergoing preoperative cardiac catheterization, whereas in Group I, all 77 patients underwent preoperative cardiac catheterization for objective measurement of their pulmonary artery pressures. With the advent and ease of administration of nitric oxide, pulmonary hypertension should become less of a risk factor in determining the postoperative outcome of patients undergoing primary VSD closure.[28]

A highly significant statistical difference ($p < 0.001$) in cross clamp and cardiopulmonary bypass times was noted between the Dacron patch technique in Group I and present surgical technique of Group II. The 5 patients who had VSDs closed with bovine pericardium and the 1 patient who had direct suture closure of a VSD, were excluded from the calculations of Group I (see Table 7.2).

Table 7.2 Difference in cross-clamp and CPB times *(median & range)*

	Group I	*Group II*	*p value*
X-clamp time (min)	50(22-110) (n=67)	33(117-99) (n=91)	<0.001
CPB time (min)	86(45-231) (n=67)	59(29-139) (n=91)	<0.001

The shorter ischemic and bypass times seen with the operative technique in Group II supports the lower morbidity and mortality in this group when compared to Group I, where the dacron patch technique is used. (Table 6.3.1)

The use of the 0.6% glutaraldehyde-treated autologous pericardium patch in Group II showed superior results in terms of spontaneous closure of residual VSDs detected at time of discharge. The spontaneous closure rate was 36% (9/25) for Group I and 76% (44/58) for Group II ($p<0.001$). The spontaneous closure rate of 76% in Group II is better than the spontaneous closure rate quoted in the literature of 20-50%.[2, 10, 21]

However the residual VSDs detected at time of discharge in Group II were almost all trivial (stitch leaks) (57/58), whilst those detected in Group I were mostly larger, graded as small left to right shunts (24/25); further supporting the efficacy of VSD closure with the technique used for Group II (see Graph 6.3.3).

One patient (1%) in Group II required reoperation for a large residual VSD, compared to 3 patients in group I (6%). As mentioned before, only 2 of the Group I patients actually had reoperation.

The reported reoperation rate for residual VSD in the literature is from 3 to 5% with the use of a Dacron patch.[2, 8, 11, 22] Group II still fares better with the autologous pericardial patch at 1% incidence of reoperation.

A further 7 complications have been identified between Groups I and II (Table 6.3.1). Only 2 out of these 7 complications featured in Group II: sternal wound infection (1/82), and junctional ectopic tachycardia (JET) (2/82). Table 7.3 tabulates the postoperative complications related to the various surgical approaches.

Table 7.3 Complications related to surgical approach

	Number	*Reoperation*	*Sternal infection*	*Pacemaker inserted*	*JET*	*Tamponade*	*Neurological deficit*	*RVF*	*Infective endocarditis*
Autologous pericardium	93	1	1	0	2	0	0	0	0
Dacron patch	67	1	2	1	2	2	2	0	2
Bovine pericardium	5	0	0	1	0	0	0	0	0
RV approach	2	0	0	0	0	0	0	1	0
PA approach	2	0	0	0	0	0	0	0	0
Direct suture	1	1	1	0	0	0	0	0	1

Group I showed a 6% incidence (3/48) of sternal wound infection and 4% (2/49) incidence of JET. A 4% (2/48) incidence in complete heart block, requiring permanent pacemaker insertion, was also seen in group I. One of these patients had a Dacron patch repair while the other had bovine pericardium with a continuous suture technique. The reported incidence of PPM insertion for complete heart block after VSD repair is 1.3 to 5% and is reportedly directly related to the suturing technique.[1, 8, 10, 20, 22]

Two patients in Group I (4%) also had severe permanent neurological deficit following cardiac arrest in the postoperative period. Rein et al [11] showed a 1% incidence in neurologic dysfunction following cardiac arrest after VSD repair, while Richardson et al [8] showed a 6% incidence in neurologic complications.

Other postoperative complications:

Group I showed a 6% (3/48) incidence of cardiac tamponade, a 1% (1/48) incidence of right ventricular failure on follow-up (this patient had a dacron patch VSD closure via a right ventriculotomy) and a 6% incidence of early infective endocarditis.

It is worth noting that the double velour dacron was supplied in large sheets, cut to the appropriate sizes and *resterilized* to curtail costs. This practice came under scrutiny in 1990 when 3 patients died from infected dacron patches used for the closure of their VSDs; Staphlococcus Aureus and Epidermidis were isolated from supposedly sterile patches (information supplied by a senior surgeon operating at the time and verified by the theatre matron who was also the cardiac scrub sister at the time). This is the reason for the period of exploring other materials; notably, bovine pericardium.

Resterilization of synthetic material could have potentially disastrous complications if not carried out according to documented sterilization protocols.[29, 30, 31]

A breakdown in the sterilization protocol in 1989/90 could well have contributed in the increased incidence of infective endocarditis in the Group I patients.

The overall mortality in Group I was 11% (6/54), compared to 1% (1/83) in Group II ($p<0.02$). This significant difference in mortality could be explained by the higher operative risk of the Group I patients as mentioned earlier (younger overall age, lower weight and possibly higher incidence of pulmonary hypertension). This higher risk group was subjected to longer cross-clamp and cardiopulmonary bypass times compared to Group II ($p <0.001$). The mortality currently reported in the literature for isolated VSD closure is <1%. [6, 9, 21, 22]

The under-one-year-old subgroup had a mortality rate of 7% (4/54) in Group I, compared to 1% (1/83) in Group II ($p = 0.08$). One-stage VSD closure in the under-one-year-old age group is being increasingly advocated as the mortality in this group has been considerably reduced from 6-14% [2, 10, 11, 13] to between 1.5-3%[1, 8, 12, 13] in recent times. This is attributed to better patient selection, improved operative technique, better myocardial preservation, improved devices, management of cardiopulmonary bypass and improved postoperative care. [8, 10, 11, 13]
The low under-one-year mortality in Group II supports this.

The one late death in Group I was not related to the VSD repair *per se,* but was due to respiratory failure, which may, in turn, have been related to high pulmonary artery pressures. This patient had a two-stage repair due to severe pulmonary hypertension, requiring a pulmonary artery band as a first stage. Richardson et al reports a potential combined mortality of 12 to 31% for the two-stage procedure. [8]

The overall mortality of both groups was further analysed to identify risk factors as predictors of mortality, particularly in selecting patients for closure of a VSD in the under-one-year-old age group.

The main predictors of mortality in this group reported in the literature are: [10, 11, 13]

i. weight for age < 3rd centile

ii. respiratory failure needing preoperative ventilation

iii age less than 6 months

iv pulmonary hypertension (severe)

This study identified the following risks for mortality:

Preoperative risks:

i. Age < 12 months – 5/7 deaths

ii. Moderate to severe PHT – 5/7 deaths

iii. Weight < 5kg – 3/7 deaths

Intraoperative risks:

iv. Surgical technique (Dacron patch)– 6/7 deaths

v. X-clamp time > 60 min – 5/7 deaths

vi. CPB time > 110 min – 5/7 deaths

vii. Surgeon – 3/7 deaths

viii. Associated debanding – 2/7 deaths

Postoperative complications:

ix. infective endocarditis – 2/7 deaths

x. JET – 1/7 deaths

Six out of the 7 deaths overall had four or more risk factors.

Six out of 10 complicating factors noted were statistically significant (see table 6.3.4.1)

As mentioned earlier, 5 out of 7 patients who died were all under one year old, while the two remaining patients were 22 and 15 months old, respectively. One patient had chronic respiratory problems; this was the late death in the series, from respiratory failure.

The one death in Group II had only 2 risk factors, but this patient was 2.7 months old and weighed only 3kg (below the 3rd centile for age). These factors were in keeping with risks identified in the literature [10, 11] as strong predictors of mortality following closure of a VSD.

Four other patients were in the under-one-year-age group (average age 7.3 ± 2.5 months) with an additional 4 or more risk factors contributing to their death.

The incidence of residual VSD and 30-day mortality was statistically significant between the different *surgical techniques* in groups I and II (Table 6.3.1); supporting the conclusion of a better outcome with the use of the 0.6% glutaraldehyde-treated autologous pericardial patch using a continuos horizontal mattress suture technique of 5.0 polypropylene suture material.

CHAPTER 8

SUMMARY

The current surgical technique of using a 0.6% glutaraldehyde-treated autologous pericardial patch, with a continuous horizontal mattress suture of 5.0 polypropylene without pledgets, through a right atriotomy, has demonstrated superior results with regards to a lower morbidity and mortality. The high rate of spontaneous closure of trivial residual VSDs and the low rate of reoperations for residual VSD supports the use of the current technique when compared to the use of a synthetic double velour knitted Dacron patch using interrupted, alternating pledgetted sutures of 5.0 Prolene® and 5.0 Ticron®.

The technique of using autologous pericardium treated with 0.6% glutaraldehyde washed off with normal saline is cheap, non- toxic and obviates the need for the implantation of foreign material as a possible site for bacterial seeding.

A major advantage with this technique is its simplicity and speed with a relatively short x-clamp and bypass time, thereby reducing the deleterious effects of cardiopulmonary bypass as evidenced by the low postoperative complication rate in group II.

Primary closure of isolated perimembranous VSD without associated major cardiac anomalies, can be safely undertaken in the under-one-year-old, high risk age group.

The mortality rate can be reduced to well below the 1.5 to 3% presently quoted in the literature (1% morality in Group II; 7% mortality in group I). (Table 6.3.1).

To achieve a low mortality in the under-one-year-old high risk group, careful patient selection is required by identifying and minimizing pre- and intraoperative risks which have been listed in this study.

Patients in the under-one-year-old age group should not be selected for closure of isolated perimembranous VSD if their identifiable risk factors cannot be reduced to below 4. Optimizing nutritional status (aim for weight above 3rd centile), respiratory function (treat congestive cardiac failure aggressively and treat pneumonia before the operation) and offering a quick surgical procedure by an experienced surgeon are strong recommendations to ensure a low mortality in the high risk under-one-year-old age group. A two-stage approach remains an option for select patients.

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APPENDIX

i. Data sheets of group I and group II

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P
1	Group 1('87-'90)	Name	Fol.no.	D.O.B	Wt(kgs)	PHT(clinical/pvri)	Op. Date	Xclmp tm	CPB tm	Res vsd D/C	Res vsd on ffup	reop	reop dte	mortality	D.O.D	stn debrid
2	1	Smit H	70485685	04/20/87	3.8	yes(2/1.9)	10/13/87	77	136	small	no	no		no		yes(11/3/87)
3	2	Nkqayi K	70446828	04/10/87	4.6	yes(2/3)	10/06/87	72	105	no				no		no
4	3	May Z	749071106	12/10/89	4.6	no	07/31/90	65	90	small						no
5	4	Grey G	69152916	12/02/86	5.4	yes(2/3.2)	07/14/87	42	64	small	no	no		no		no
6	5	Davids Mo	70110945	06/16/87	4	yes(2/0.4)	10/06/87	45	116		no	no		YES	10/30/87	no
7	6	Wesso S	69992022	03/09/87	4.8	yes(2/2)	07/01/87	42	60	no	no	no		no		no
8	7	Davids Mic	69599991	06/02/87	4	yes(2/2.7)	05/26/88	62	84	small	small	no		no		no
9	8	Davids J	70067772	03/06/87	5.8	yes(-/1.9)	01/05/88	67	90	no	no	no		no		no
10	9	Manhoe J	68763036	08/15/86	4.4	no	01/27/87	45	82	small	small	no		no		no
11	10	Hector M	69257152	02/14/87	6.6	yes(3a/4)	09/29/87	59	62	no	no	no		no		no
12	11	Solomon N	69933190	10/07/86	4.8	yes(3a/2)	07/07/87	43	65	no				no		no
13	12	Smith D	69283349	01/08/87	4.7	yes(2/2.5)	06/16/87	56	84	no	no	no		no		no
14	13	Fati J	68673367	08/01/86	6.3	no	03/18/87	45	63	no	small	no		no		no
15	14	Jonkers L	70585690	04/24/87	5.6	yes(2/-)	11/03/87	56	99	no	no	no		no		no
16	15	Fowler C	71809768	04/21/88	4	yes(2/4.8)	10/25/88	48	115	small	no	no		no		no
17	16	Nkamana V	71835987	06/06/88	4.4	yes(3a/4.40	09/20/88	42	52	small	no	no		no		no
18	17	Ntshoyiya	71864011	05/13/88	5.1	yes(-/1.4)	09/06/88	50	75	no	no	no		no		no
19	18	Terblanche	70302112	08/11/87	6	*yes(PAB)*	03/16/88	46	90	small	no	no		no		no
20	19	Rixana N	71012421	02/19/87	4.7	yes(-/4.5)	02/03/88	38	47	no				no		no
21	20	Van der Lir	72347834	06/26/88	6	yes(-3.4)	12/14/88	34	90	small	no	no		no		no
22	21	Lakay T	71862742	06/25/88	4	no	11/22/88	50	80	no	no	no		no		no
23	22	Job L	71289409	04/11/88	5.7	yes(3a/2.5)	11/15/88	86	130	no	no	no		no		no
24	23	Petersen J	71683429	02/17/88	5.4	yes(2/4.8)	11/08/88	72	150	small	no	no		no		no
25	24	Brown Y	71570840	05/24/88	5	yes(3a/4.5)	10/11/88	51	69	no	no	no		no		no
26	25	Harmse T	71566590	04/20/88	5.1	yes(-/1.3)	12/13/88	43	45	no	no	no		no		no
27	26	Jordaan B	71614259	08/28/87	6	yes(3a/2.4)	07/19/88	50	93	no				no		no
28	27	Beukes L	71161244	03/13/88	4	yes(3a/3.9)	06/21/88	50	90	no	no	no		no		no
29	28	Peters J	70240692	06/27/87	4.5	*yes(PAB)*	03/22/88	110	203		no	no		YES	03/22/88	no
30	29	Moguduka	71012421	11/13/87	3.3	yes(2/-)	02/15/88	53	82	no	no	no		no		yes(2/23/88)
31	30	Van der Sc	73889842	05/26/89	5	yes(1c/2)	12/04/89	49	102	small	small	no		no		yes(12/6/89)
32	31	Kotze B	72540016	12/09/88	5.1	yes(2/3.5)	06/27/89	60	70		no	no		YES	06/28/89	no
33	32	Willemse V	72662299	07/11/88	4.3	yes(2/-)	03/06/89	93	153	small	small	no		no		no
34	33	Arendse A	63905483	01/29/89	4	no	10/03/89	45	67	no	no	no		no		no
35	34	Cloete L	71912216	07/16/88	4.8	yes(3a/4.8)	01/20/89	57	110	no	no	no		no		no
36	35	Ngcelwane	74556325	03/20/90	4.5	yes(3a/2.8)	09/05/90	45	87	no	no	no		no		no
37	36	Ganief N	74904228	05/20/90	3.1	yes(-/3.2)	07/24/90	37	108	small	small	no		no		no
38	37	Mgijima K	74482837	06/07/89	6.5	yes(3a/5)	04/17/90	100	157		no	no		YES	05/01/90	no
39	38	Baqwa O	74344508	10/04/89	4.9	yes(3a/2.8)	04/03/90	53	90	no				no		no
40	39	Spanenber	74934993	10/23/89	6.3	yes(3a/2.2)	08/14/90	45	78	no				no		no
41	40	Josephe A	73867384	08/21/89	4	no	04/15/90	66	112	small	no	no		no		no
42	41	Bhushula S	74064015	02/01/90	5.5	yes(-/1.5)	10/03/90			small				no		no
43	42	Fouti D	73624173	05/03/89	6	yes(3a/-)	01/17/90	70	102	no	no	no		no		no
44	43	Susa	69054559	09/13/86	8	yes(-/3.2)	09/09/87	79	118	small	small	no		no		no
45	44	Mahomet F	68898329	09/22/86	6.5	yes(3a/2.1)	10/27/87	57	77	no	no	no		no		no
46	45	Baba X	70340073	08/20/85	11.7	yes(2/3.4)	09/01/87	33	47	no				no		no
47	46	Maselana I	69340073	06/12/85	10	yes(-/1.6)	01/13/87	38	75	no				no		no

	Q	R	S	T	U	V	W	X	Y	Z	AA	AB
1	PPM	J.E.T	tampnde	cardiac arrst v	RVF	I.E.	surgeon	dte of last ff	ff up(mnths	associated defect/procedure	event free	age(mnths
2	no	no	no	no	no	no	UVO	12/27/92	62	no	1874	5.866667
3	no	no	no	no	no	no	UVO	*10/16/97*	0	no	10	5.966667
4	no	no	no	no	no	no	UVO	*08/07/90*	0	no	7	7.766667
5	no	no	no	no	no	no	JH	07/16/90	36	no	1082	7.466667
6	no	no	no	no	no	yes	BR	10/30/87	0	no	24	3.733333
7	no	no	no	no	no	no	JO	02/19/88	7	no	229	3.8
8	no	no	no	no	no	no	JO	02/17/92	57	no	1731	11.96667
9	no	no	no	no	no	no	BR	07/22/96	112	RV app	3377	10.16667
10	no	no	no	no	no	no	UVO	03/03/98	123	no	3696	5.5
11	no	no	no	no	no	no	JO	03/24/94	30	no	895	7.566667
12	no	no	no	no	no	no	JO	*07/26/87*	0	PFO	19	9.1
13	no	no	no	no	no	no	JO	03/20/00	153	no	4592	5.3
14	no	no	no	no	no	no	JO	03/28/88	12	AI	359	7.633333
15	no	no	no	yes(4/11/87)	no	no	UVO	03/01/91	40	no	1198	6.433333
16	no	no	no	no	no	no	UVO	08/03/95	81	no	2438	6.233333
17	no	no	no	no	no	no	BR	09/18/89	12	no	358	3.533333
18	no	no	no	no	no	no	UVO	04/03/92	42	PFO	1257	3.866667
19	no	no	no	no	no	no	SV	03/28/90	24	no	708	7.266667
20	no	no	no	no	no	no	BR	*02/24/88*	0	PFO	21	11.63333
21	no	no	no	no	no	no	JO	02/04/91	46	no	1370	5.7
22	no	no	no	no	no	no	JB	09/05/94	69	no	2052	5
23	no	no	no	no	no	no	JB	11/26/98	120	no	3611	7.266667
24	no	no	no	no	no	no	UVO	09/10/98	118	PDA	3542	8.833333
25	no	no	no	no	no	no	JO	08/26/99	129	no	3885	4.666667
26	no	no	no	no	no	no	BR	02/19/90	14	no	426	7.9
27	no	no	no	no	no	no	UVO	*08/01/88*	0	no	13	10.86667
28	no	no	no	no	no	no	UVO	02/20/95	80	no	2399	3.333333
29	no	no	no	no	no	no	UVO	03/22/88	0	debanding/PFO	0	8.966667
30	no	no	no	no	no	no	SV	04/30/88	2	PA app	75	3.133333
31	no	no	no	no	no	no	BR	06/19/90	6	no	195	6.4
32	no	no	no	no	no	no	JB	06/28/89	0	no	1	6.666667
33	no	no	no	no	no	no	JB	02/20/92	35	no	1064	7.933333
34	no	no	no	no	no	no	JO	03/23/90	5	no	170	8.233333
35	no	no	no	no	no	no	UVO	12/24/99	131	no	3934	6.266667
36	no	no	no	no	no	no	SV	02/11/97	77	Bovine pericardium with simp	2316	5.633333
37	no	no	no	no	no	no	SV	08/01/98	96	Bovine pericardium with simp	2888	2.166667
38	no	no	no	no	no	yes	UVO	05/01/90	0	PFO	14	10.46667
39	no	yes	no	no	no	no	UVO	*04/14/90*	0		11	6.033333
40	no	no	no	no	no	no	SV	*08/29/90*	0	Bovine pericardium with simp	14	9.833333
41	no	no	no	no	no	no	JO	12/20/90	8	no	257	7.9
42	no	no	yes(10/3/9	no	no	no	JO	*10/20/90*	0	no	17	8.133333
43	no	no	no	no	no	no	UVO	01/23/96	72	no	2166	8.633333
44	no	no	no	no	no	no	JO	10/01/93	72	no	2182	12.03333
45	no	no	no	no	no	no	JO	01/23/95	87	PFO	2606	13.33333
46	no	no	no	no	no	no	JO	*09/14/87*	0	no	13	24.73333
47	no	no	no	no	no	no	UVO	*01/21/87*	0	no	8	19.33333

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P
48	47	Setlhabi K	69854347	05/13/85	9.6	yes(2/1.4)	05/12/87	38	48	no				no		no
49	48	Sijaji B	67636373	06/13/85	8	yes(-/1.5)	09/22/87	35	55	small	no	no		no		no
50	49	Mlauzi I	70674585	10/22/85	12.5	no	11/17/87	57	96	no				no		no
51	50	Rapu N	70749684	09/17/81	16.7	yes(-/4.7)	12/01/87	65	85	no				no		no
52	**51**	**Malase M**	69488617	07/29/83	12.5	no	02/24/87	22	50	small	large	**YES**	**06/23/87**	no		no
53	52	Jooste L	63644603	07/07/79	18	yes(-/1.80	08/17/87	50	78	small	no	no		no		no
54	53	Ngonzo N	70157920	10/26/83	14.2	yes(2/1.4)	07/21/87	68	100	no	no	no		no		no
55	54	Philander S	67160911	07/12/85	12	no	08/04/87	32	57	small	no	no		no		no
56	55	Sebapalwa	69887826	09/18/85	6.8	yes(2/2.1)	05/19/87	60	84	no				no		no
57	**56**	**Mcay R**	72036627	11/29/84	13.9	yes(2/2.6)	10/04/88	69	97	small	**large**	**YES**	**pt decline**	no		no
58	57	Mhlontlon	68449735	06/17/85	13	yes(1c/-)	11/01/88	42	60	small	small	no		no		no
59	58	Rossouw A	69280444	01/07/87	10	*yes(PAB)*	07/26/88			no	no	no		no		no
60	59	Jooste D	64508260	12/05/83	16	no	06/13/88	56	89	small	no	no		no		no
61	60	Williams G	70289483	09/01/83	13.2	no	03/29/88	33	48	no	no	no		no		no
62	61	Matthews I	71143606	05/30/82	19.1	yes(-/2.5)	03/01/88	94	119	no				no		no
63	**62**	**Tywaku B**	71510424	04/26/83	14.3	yes(-/4.4)	05/24/88	59	231	**large**	**large**	**YES**	**05/26/88**	no		no
64	63	Brown J	71517841	03/03/87	6.2	yes(2/4.50	05/24/87	40	64	no				no		no
65	64	Maqenuka	71607980	01/13/87	9.7	*yes(PAB)*	12/06/88			no	no	no		**YES(resp**	**11/11/89**	no
66	65	Liwani P	71889877	06/20/84	15.9	no	11/08/88	66	93	no				no		no
67	66	Septembe	57384323	10/15/79	23	no	03/21/89	60	87	no	no	no		no		no
68	67	Van Schall	67796128	04/05/85	12	yes(-/1.9)	04/25/89	46	70	no	small	no		no		no
69	68	Qekema Z	72592330	04/13/88	7.3	yes(-/3)	07/25/89	70	112		no	no		**YES**	**07/31/89**	no
70	69	Zokufa S	72677404	07/09/88	9.4	yes(2/1.8)	12/11/89	47	68	no				no		no
71	70	Bisai S	72540891	12/28/84	14.5	yes(2/2.4)	01/25/89	51	114	no				no		no
72	71	Lottering L	63624647	10/12/81	19	no	09/18/89	65	86	no				no		no
73	72	Joos N	74112186	02/02/89	8.7	no	01/30/90			small	small	no		no		no
74	73	Haisoswi T	74686361	02/05/88	12	yes(2/2.7)	06/12/90	67	100	no				no		no
75	74	White S	72905672	11/05/88	5.7	yes(2	02/13/90	32	48	small				no		no
76	75	Smit M	74646233	10/26/89	6.2	yes(2/-)	11/14/90	39	77	no	no	no		no		no
77	76	Ryneveld F	58923707	06/02/80	5.9	no	10/30/90	45	90	no	no	no		no		no
78	77	William N	74868811	06/24/89	7.6	yes(-/3.6)	12/11/90	41	60	no	no	no		no		no

	Q	R	S	T	U	V	W	X	Y	Z	AA	AB
48	no	no	no	no	no	no	SV	*05/22/87*	0	**PA app**	10	24.3
49	no	no	no	no	no	no	JO	06/04/90	20	no	582	27.7
50	**yes(11/17/**	no	no	no	no	no	UVO	*12/25/87*	0	no	8	25.2
51	no	no	no	no	no	no	UVO	*12/28/87*	0	no	27	75.53333
52	no	no	no	no	no	**yes**	**JO**	07/24/87	5	**Direct suture closure with ple**	149	43.53333
53	no	no	no	no	no	no	UVO	01/11/88	6	no	185	98.76667
54	no	no	no	no	no	no	SV	01/11/87	4	no	110	45.46667
55	no	no	no	no	no	no	JO	03/17/89	19	no	583	25.1
56	no	no	no	no	no	no	JO	*05/06/87*	0	no	17	20.26667
57	no	no	no	no	no	no	**JO**	*10/28/88*	0	no	24	46.83333
58	no	no	no	no	no	no	JB	05/22/89	6	no	180	41.1
59	no	no	no	no	no	no	UVO	10/31/97	108	debanding	3245	18.86667
60	no	no	no	no	no	no	JO	04/12/91	34	no	1019	55.06667
61	no	no	no	no	no	no	JO	04/10/90	24	PFO	723	55.7
62	no	no	no	no	no	no	BR	*03/15/88*	0	no	14	70.06667
63	no	no	**yes(5/24/8**	no	no	no	**UVO**	06/30/95	84	no	2	61.83333
64	no	no	no	no	no	no	JB	*03/06/88*	0	no	10	2.733333
65	no	no	no	no	no	no	**JO**	11/11/89	11	debanding	335	23.1
66	no	no	no	no	no	no	SV	*11/19/88*	0	DCRV	18	53.4
67	no	no	no	no	no	no	JB	12/12/96	93	DCRV	1105	114.8333
68	no	no	no	no	no	no	JB	01/22/90	9	PFO	267	49.36667
69	no	**yes**	no	no	no	no	**UVO**	07/31/89	0	PFO	6	15.6
70	no	no	no	no	no	no	UVO	*12/22/89*	0	no	11	17.33333
71	no	no	no	no	**yes**	no	JB	*02/15/89*	0	**RV app**	21	49.63333
72	no	no	no	no	no	no	JO	*10/13/89*	0	no	23	96.6
73	no	no	no	no	no	no	JB	08/16/00	127	no	3796	12.06667
74	no	no	no	no	no	no	UVO	*06/24/90*	0	no	12	28.6
75	no	no	no	no	no	no	JO	*02/26/90*	0	no	13	15.5
76	no	no	no	no	no	no	SV	01/10/91	2	**Bovine pericardium with simp**	56	12.8
77	**yes(11/4/9**	no	no	no	no	no	SV	10/01/02	23	**Bovine pericardium with simp**	661	126.7333
78	no	no	**yes(12/11/**	**yes(12/11/90)**	no	no	JO	04/25/96	63	PFO	1904	17.83333

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P
1	Group 2('95-'98)	Name	Fol.no.	D.O.B	Wt(kgs)	PHT(clinical grde/pvri)	Op. Date	Xclmp tm	CPB tm	Res. Vsd on d/c	Res vsd on ff up	reop	reop dte	mortality	D.O.D	stn debrid
2	1	Pendu A	81557100	07/04/94	7.9	yes(2/2.2)	06/27/95	54	84	trivial	small	no		no		no
3	2	Present Tw	81596884	12/31/94	4	yes(2/0.8)	08/08/95	26	44	no	no	no		no		no
4	3	Kakaza S	81613440	01/02/95	6.8	yes(2/2.8)	08/28/95	24	50	no						no
5	4	Nelani E	82026667	02/09/95	5.1	yes(2)	10/31/95	28	65	trivial	small	no		no		no
6	5	Potgieter S	80883721	12/06/94	7.5	yes(2)	06/14/95	25	66	trivial	no	no		no		no
7	6	Brown T	82124967	06/05/95	5.1	no	12/19/95	28	65	trivial	no	no		no		no
8	7	Oosthuizer	80503246	01/16/94	9.3	yes(2)	05/16/95	43	79	no	no	no		no		no
9	8	Fielies E	79818175	01/21/94	9.2	no	01/10/95	34	69	no	no	no		no		no
10	9	Kortjie PE	81780769	04/19/95	4.7	yes(2/1.2)	11/02/95	60	90	no	no	no		no		no
11	10	Koopman L	82132630	10/20/95	4.4	no	02/28/96	30	53	trivial	no	no		no		yes(5/22/96
12	11	Nesbitt X	82279191	06/14/95	3	yes(2)	01/10/96	26	75	trivial						no
13	12	Daniels M	81424699	04/14/95	6.1	yes(2)	01/09/96	58	82	trivial	no	no		no		no
14	13	Jacobs RA	82940974	04/20/96	3	no	07/23/96	68	104	no	no	no		no		no
15	14	Boas K	83083741	07/12/96	3.7	no	11/13/96	20	53	trivial	no	no		no		no
16	15	Tetyana A	82220005	10/31/95	6	no	08/12/96	39	70	trivial	no	no		no		no
17	16	Engelbrecl	82071754	06/16/95	6.3	yes(2/5)	05/20/96	25	54	no	no	no		no		no
18	17	Stadler L	83182170	08/01/96	3.8	no	11/12/96	29	58	trivial	no	no		no		no
19	18	Thomas A	82947979	07/01/96	4	no	11/25/96	33	52	no	no	no		no		no
20	19	Standaar D	83307561	08/29/96	3.6	no	11/18/96	20	60	no	no	no		no		no
21	20	Pietersen J	83136135	05/28/96	3.2	yes(2)	12/09/96	41	64	trivial	no	no		no		no
22	21	Mba S	83394155	09/24/96	5.7	yes(2)	02/25/97	32	74	trivial	no	no		no		no
23	22	Muller B	83468132	03/19/96	6.7	*yes(PAB)*	03/11/97			trivial	no	no		no		no
24	23	Manuel B	82659038	04/21/96	6	yes(2/1.3	03/24/97	37	72	trivial	no	no		no		no
25	24	Gonyela L	83809483	03/07/97	3.2	yes(1c)	06/04/97	30	55	trivial	no	no		no		no
26	25	Tom O	83073833	08/15/96	4.2	yes(2)	01/13/97	35	65	trivial	small	no		no		no
27	26	Magijima N	83826420	11/24/96	6.5	no	06/25/97	22	36	no	no	no		no		no
28	27	**Walters M**	83733212	02/15/97	5.5	no	06/24/97	34	71	small	large	yes x 1	8/18/97& 1	no		no
29	28	Yokwe B	83756890	02/25/97	2.6	no	05/29/97	24	48	no				no		no
30	29	Fani W	84333350	12/17/97	4.5	yes(3a)	03/09/98	23	59	no	no	no		no		no
31	30	Rothman H	84502871	09/13/97	5	yes(3/0.1)	05/12/98	29	45	trivial	small	no		no		no
32	31	Ndziba H	84647478	04/30/98	3	no	07/22/98	40	71					YES	07/23/98	
33	32	Mralati Z	84857291	10/18/97	7	yes(2/1.4)	10/27/98	31	58	trivial				no		no
34	33	Qavane k	84582261	05/06/98	3.2	no	08/18/98	31	69	no	no	no		no		no
35	34	Bathini N	81464224	12/16/94	7.5	yes(1c)	09/05/95	26	52	trivial	no	no		no		no
36	35	Mbenyane	84346501	12/27/97	3.2	yes(2/1)	08/31/98	37	53	trivial	no	no		no		no
37	36	Khumalo K	80904972	10/28/94	3.8	yes(2/1.5)	05/23/95	44	67	no	no	no		no		no
38	37	Pitcher J	82019357	07/22/95	3.6	yes(2)	11/28/95	57	91	trivial	no	no		no		no
39	38	Makhola B	80258932	05/27/94	9.5	yes(2)	08/21/95	24	47	no	no	no		no		no
40	39	Swanepoel	76132315	01/05/91	5.1	no	05/09/95	57	78	trivial	no	no		no		no
41	40	Guga S	81796047	05/21/94	8.8	yes(2/4)	09/12/95	19	45	trivial				no		no
42	41	Carelse JV	78746526	08/20/91	15.5	no	09/26/95	47	71	trivial	no	no		no		no
43	42	Gabriels R	71528962	10/28/94	18.5	yes(1c)	09/19/95	17	29	no	no	no		no		no
44	43	Paulsen	70169008	11/16/86	17.9	yes(1c)	01/17/95	18	42	no	no	no		no		no
45	44	Du Preez F	69375384	09/13/86	21	yes(1c)	10/24/95	42	71	trivial				no		no
46	45	Geelbooi A	73034382	12/24/88	19.5	yes(2)	06/12/95	30	49	trivial	small	no		no		no
47	46	Zita B	81000424	01/05/82	28	yes(2/01)	01/17/95	40	68	no				no		no

	Q	R	S	T	U	V	W	X	Y	Z	AA	AC	AD
1	PPM	J.E.T	tampnde	cardiac arrst with perm CNS dysfx	RVF	I.E.	surgeon	dte of last ff up	ff up(mnths)	associated defect/procedure	Event free	age(mnths)	
2	no	no	no	no	no	no	MW	07/22/99	49	no	1463	11.93333	
3	no	no	no	no	no	no	JH	07/04/96	13	no	386	7.333333	
4	no	no	no	no	no	no	JH	*09/12/95*	0	no	15	7.933333	
5	no	no	no	no	no	no	JH	04/01/99	41	PFO	1200	8.8	
6	no	no	no	no	no	no	JH	06/15/00	60	no	1801	6.333333	
7	no	no	no	no	no	no	JH	03/13/97	33	no	984	6.566667	
8	no	no	no	no	no	no	MW	11/03/95	6	no	167	16.16667	
9	no	no	no	no	no	no	MW	02/10/97	25	PFO	750	11.8	
10	no	no	no	no	no	no	MW	06/19/97	29	PFO	887	6.566667	
11	no	no	no	no	no	no	JH	09/22/97	19	no	564	4.366667	
12	no	no	no	no	no	no	JH	*01/18/96*	0	PFO	8	7	
13	no	no	no	no	no	no	MW	10/06/97	21	no	627	9	
14	no	no	no	no	no	no	JH	09/09/96	2	PFO	46	3.133333	
15	no	no	no	no	no	no	JH	11/18/99	36	no	1085	4.133333	
16	no	no	no	no	no	no	JH	11/15/99	39	PFO	1173	9.533333	
17	no	no	no	no	no	no	JH	10/04/99	40	PFO	1184	11.3	
18	no	no	no	no	no	no	JH	09/29/97	11	PDA	347	3.433333	
19	no	no	no	no	no	no	JH	11/26/98	24	PFO	719	4.9	
20	no	no	no	no	no	no	JH	06/22/00	43	no	1294	2.7	
21	no	no	no	no	no	no	JH	01/20/97	1	no	41	6.5	
22	no	no	no	no	no	no	JH	05/29/97	3	no	94	5.133333	
23	no	no	no	no	no	no	JH	03/23/98	12	debanding	372	11.9	
24	no	no	no	no	no	no	JH	03/07/00	35	no	1033	11.23333	
25	no	no	no	no	no	no	JH	02/04/99	28	PFO	840	2.966667	
26	no	no	no	no	no	no	JH	05/22/97	4	PFO	129	5.033333	
27	no	no	no	no	no	no	JH	11/09/98	17	no	494	7.1	
28	no	no	no	no	no	no	JH	10/18/99	28	no	55	4.3	
29	no	no	no	no	no	no	JH	*06/18/97*	0	no	20	3.1	
30	no	no	no	no	no	no	JH	11/25/99	19	no	586	2.733333	
31	no	yes	no	no	no	no	JH	01/27/00	20	no	615	8.033333	
32							JH	07/23/98	0	PFO	1	2.766667	
33	no	no	no	no	no	no	JB	*11/10/98*	0	no	14	12.46667	
34	no	no	no	no	no	no	JH	01/24/00	17	no	516	3.466667	
35	no	no	no	no	no	no	JH	12/05/96	15	no	450	8.766667	
36	no	no	no	no	no	no	JH	03/09/00	18	no	562	8.233333	
37	no	no	no	no	no	no	MW	07/28/95	2	no	95	6.9	
38	no	no	no	no	no	no	JH	11/30/96	12	PFO	362	4.3	
39	no	no	no	no	no	no	JH	10/29/98	34	no	1028	15.03333	
40	no	no	no	no	no	no	MW	02/02/96	9	PFO	983	52.83333	
41	no	no	no	no	no	no	JH	*09/26/95*	0	no	14	15.96667	
42	no	no	no	no	no	no	JH	02/28/00	53	AI	1592	49.93333	
43	no	no	no	no	no	no	JH	02/01/96	4	PDA	104	10.86667	
44	no	no	no	no	no	no	JH	11/24/97	58	no	1747	99.46667	
45	no	no	no	no	no	no	JH	*11/03/95*	0	AI	10	110.9333	
46	no	no	no	no	no	no	JH	02/08/95	2	no	56	78.7	
47	no	no	no	no	no	no	JH	*01/28/95*	0	no	11	158.6667	

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P
48	47	Bizi O	81250508	02/06/90	18	yes(2/1.8)	05/30/95	40	67	trivial	small	no		no		no
49	48	Wani N	81969537	04/13/90	18.1	yes(2/1.8)	10/17/95	27	48	trivial	no	no		no		no
50	49	Abrahams	81635252	04/02/95	5.4	yes(2/1.5)	09/10/96	24	58	no	no	no		no		no
51	50	Levack E	81256091	10/18/94	9.2	yes(2)	07/24/96	19	44	no	no	no		no		no
52	51	Damons S	81635690	06/02/90	20	no	11/26/96	29	60	trivial	no	no		no		no
53	52	Vika N	80551765	08/11/94	10	yes(3a/4)	08/13/96	62	84	trivial	no	no		no		no
54	53	Gobinngca	83120568	03/15/93	16.3	yes(20	09/16/96	33	56	trivial	no	no		no		no
55	54	Funda Y	81160608	05/02/94	8.1	yes(2)	02/13/96	34	51	trivial	small	no		no		no
56	55	Majolla G	81667057	09/01/86	24	yes(2)	04/09/96	28	53	no	no	no		no		no
57	56	George JS	76638022	09/04/91	11.4	*yes(PAB)*	02/13/96	54	117	trivial	no	no		no		no
58	57	Long E	79308417	07/21/93	12.5	no	02/27/96	46	73	no	no	no		no		no
59	58	Stoffels S	77845667	07/03/92	12.9	no	04/17/96	40	69	no	no	no		no		no
60	59	Alexander	76614445	04/14/91	9.5	yes(2)	07/15/96	36	56	trivial	small	no		no		no
61	60	Fekema S	82934233	06/18/95	4	no	07/23/96	51	78	no	no	no		no		no
62	61	Luyima M	83759589	03/24/97	6.3	no	10/27/97	20	41	trivial	no	no		no		no
63	62	Mentoor R	78068483	12/15/92	22	no	11/24/97	19	44	no	no	no		no		no
64	63	Amon J	82747650	03/21/96	6.3	no	07/09/97	24	37	no	no	no		no		no
65	64	Njokweni S	83900894	11/24/95	10	yes(2)	08/24/97	18	36	trivial				no		no
66	65	Ngxanga S	80696859	08/18/94	12	*yes(PAB)*	07/22/97	33	59	trivial	no	no		no		no
67	66	Langeveldt	66118530	01/12/85	31	yes(1b)	02/18/97	33	52	no	no	no		no		no
68	67	Valtain G	81935322	09/24/95	6.3	yes(2/3.1)	02/10/97	39	57	trivial	no	no		no		no
69	68	Mbena S	81755878	03/23/95	10.5	*yes(PAB)*	01/21/97	42	58	trivial	no	no		no		no
70	69	Fredericks	80898778	12/10/94	10.7	*yes(PAB)*	03/26/97	91	133	trivial	no	no		no		no
71	70	Daniels P	76220151	06/21/91	14	no	04/29/97	38	67	no	no	no		no		no
72	71	Ennes D	81966202	04/20/95	10	*yes(PAB)*	05/26/97			no	no	no		no		no
73	72	Davids D	73558587	01/12/89	20	no	03/25/97	62	88	trivial	no	no		no		no
74	73	Kiel D	79208161	08/05/93	12	no	03/12/97	53	95	trivial	small	no		no		no
75	74	Kenny A	72433782	12/19/88	25	no	11/02/98	81	134	trivial	no	no		no		no
76	75	Ntiyane M	84918747	01/09/93	17	no	10/20/98	52	76	no	small	no		no		no
77	76	Ngqase A	84361807	07/23/97	4.8	yes(2/4.2)	07/20/98	36	56	trivial	no	no		no		no
78	77	Felix M	84239789	11/04/96	12	yes(2/2.6)	07/20/98	18	35	trivial	no	no		no		no
79	78	Saul M	72476443	05/20/88	23	yes(2)	09/22/98	40	81	trivial	small	no		no		no
80	79	Malgas A	80738446	06/10/94	15	yes(1b)	06/29/98	23	43	no	no	no		no		no
81	80	Edon E	82636374	02/21/96	8	yes(1b)	04/28/98	26	51	trivial	small	no		no		no
82	81	Van Wyk N	82537481	01/04/96	9	*yes(PAB)*	11/04/98	63	90	trivial	no	no		no		no
83	82	Mama B	73431728	04/27/89	19	yes(1c)	09/02/98	36	60	trivial	no	no		no		no
84	83	Dastile N	75883645	03/06/90	25	no	09/08/98	76	97	no	no	no		no		no
85	84	Sauls C	72109838	08/24/88	26	no	07/28/98	30	54	trivial	no	no		no		no
86	85	Kalamdien	81717837	03/17/95	4.8	*yes(PAB)*	06/15/98	68	102	trivial	no	no		no		no
87	86	Kotze F	84345966	06/20/90	21.2	no	05/25/98	37	56	trivial	no	no		no		no
88	87	Van Stade	84068543	10/03/97	6.5	yes(2/4)	12/07/98	30	52	trivial	no	no		no		no
89	88	Beukes A	83126672	09/26/96	7.4	yes(2/2.3)	04/06/98	40	64	no	no	no		no		no
90	89	Siyibane N	84162700	12/15/96	6.4	yes(3a/2.4)	03/02/98	30	48	trivial				no		no
91	90	Milityalwa	84329242	11/13/96	10.9	yes(1c/2)	08/05/98	99	139	trivial	no	no		no		no
92	91	Naude T	82760000	05/01/96	8	yes(1c)	04/29/98	27	45	trivial	small	no		no		no
93	92	Lottering W	82660218	03/21/95	13.4	yes(1b)	07/20/98	30	50	no	no	no		no		no
94	93	Lookmanja	84306356	11/24/93	16	no	01/26/98	24	50	trivial				no		no

	Q	R	S	T	U	V	W	X	Y	Z	AA	AC	AD
48	no	no	no	no	no	no	JH	07/11/95	1	no	40	64.63333	
49	no	no	no	no	no	no	JH	01/11/96	2	no	71	67.1	
50	no	no	no	no	no	no	JH	04/10/97	7	PDA	210	17.56667	
51	no	no	no	no	no	no	JH	11/05/99	39	no	1151	21.5	
52	no	no	no	no	no	no	MW	01/11/99	25	AI	756	78.96667	
53	no	no	no	no	no	no	JH	12/11/98	28	no	838	24.43333	
54	no	no	no	no	no	no	JH	06/01/98	21	no	615	42.7	
55	no	no	no	no	no	no	JH	11/26/96	9	no	283	21.73333	
56	no	no	no	no	no	no	JH	02/28/97	10	AI	319	116.9333	
57	no	no	no	no	no	no	JH	05/08/00	52	debanding	1555	54.1	
58	no	no	no	no	no	no	JB	01/03/97	11	mild PS	305	31.7	
59	no	no	no	no	no	no	JH	05/20/99	37	DCRV	1113	46.13333	
60	yes(9/17/9	no	no	no	no	no	JH	08/01/97	12	DCRV	346	63.96667	
61	no	no	no	no	no	no	JH	11/23/99	40	no	1200	13.36667	
62	no	no	no	no	no	no	JH	11/05/99	25	PPS	728	7.233333	
63	no	no	no	no	no	no	JH	02/19/99	17	AI	505	60.16667	
64	no	no	no	no	no	no	JH	05/08/00	34	no	1019	15.83333	
65	no	no	no	no	no	no	JH	*09/09/97*	0	no	16	21.3	
66	no	no	no	no	no	no	JH	03/03/00	31	debanding	909	35.63333	
67	no	no	no	no	no	no	JH	04/17/00	38	no	1139	147.3333	
68	no	no	no	no	no	no	JH	05/01/00	39	no	1161	16.83333	
69	no	no	no	no	no	no	JH	08/02/97	6	debanding	161	22.33333	
70	no	no	no	no	no	no	JH	05/14/98	14	debanding	408	27.9	
71	no	no	no	no	no	no	JB	11/29/99	19	AI	570	71.3	
72	no	no	no	no	no	no	JH	07/03/98	12	debanding	337	25.56667	
73	no	no	no	no	no	no	JH	06/03/99	26	AI	758	99.8	
74	no	no	no	no	no	no	JH	05/05/00	38	AI	1133	43.83333	
75	no	no	no	no	no	no	ZK	01/17/00	14	AI	435	120.1667	
76	no	no	no	no	no	no	ZK	05/25/00	19	AI	575	70.33333	
77	no	yes	no	no	no	no	JH	08/31/98	1	PFO	41	12.06667	
78	no	no	no	no	no	no	JH	11/16/98	4	no	116	20.76667	
79	no	no	no	no	no	no	ZK	05/11/00	20	no	589	125.9	
80	no	no	no	no	no	no	JH	08/18/99	14	AI	409	49.33333	
81	no	no	no	no	no	no	JH	11/23/98	7	DCRV	205	26.56667	
82	no	no	no	no	no	no	ZK	12/10/98	1	debanding	36	34.5	
83	no	no	no	no	no	no	ZK	10/18/99	13	mild PS	406	113.8333	
84	no	no	no	no	no	no	ZK	09/06/99	12	mild PS	358	103.6	
85	no	no	no	no	no	no	JH	05/11/00	21	DCRV	622	120.8333	
86	no	no	no	no	no	no	JB	04/10/00	22	debanding	655	39.53333	
87	no	no	no	no	no	no	JB	03/11/99	14	DCRV	406	96.53333	
88	no	no	no	no	no	no	JH	04/22/99	4	no	135	14.33333	
89	no	no	no	no	no	no	JH	08/26/99	16	DCRV	500	18.56667	
90	no	no	no	no	no	no	JH	*03/10/98*	0	PFO	8	14.73333	
91	no	no	no	no	no	no	ZK	01/19/00	17	no	524	21	
92	no	no	no	no	no	no	JH	04/12/99	12	AI	375	24.26667	
93	no	no	no	no	no	no	JH	03/27/00	28	DCRV	847	40.56667	
94	no	no	no	no	no	no	JH	*01/31/98*	0	DCRV	5	50.8	