

Cardiovascular Topics

Prosthetic valve obstruction at Tygerberg Hospital between January 1991 and February 2001

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Summary

Background: Prosthetic valve obstruction is a relatively rare, but potentially fatal complication in patients with prosthetic heart valves. The diagnosis and appropriate management of these patients present a challenge to both the cardiologist and the cardiac surgeon. Despite efforts over the last 30 years to prevent this complication, it remains a lifelong risk. Obstruction is caused by pannus formation, thrombus formation or a combination of pannus and thrombus. Valve replacement has traditionally been the treatment of choice.

Methods: Patients were selected from echocardiography and surgical reports between January 1991 and February 2001. All patients were analysed with regard to demographic information, clinical features, imaging results, treatment and outcome data. INR values on presentation were obtained from haematology archives.

Results: A total of 32 patients presented on 34 occasions. There were 25 women and seven men. Obstruction occurred in the mitral position in 56% of cases and in the

aortic position in 44% of cases. All but two valves were St Jude bileaflet valves. Patients generally presented with severe dyspnoea (NYHA class IV in 64.7%) and poor anticoagulation control (INR < 2.5 in 75.8%). The initial imaging modality used in all cases was transthoracic echocardiography. Fluoroscopy was used in five cases and transesophageal echocardiography in only two cases. Valve replacement was performed on 20 patients, six patients received thrombolysis and the remaining eight patients did not receive any treatment. Outcome was poor with an overall mortality of 64.7%.

Conclusions: Given the extremely high mortality rate with current management, the treatment of prosthetic valve obstruction with thrombolysis in selected patients deserves consideration in a prospective study.

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Prosthetic valve obstruction is a relatively rare, but potentially fatal complication in patients with prosthetic valves. The diagnosis and appropriate management of these patients present a challenge to both the cardiologist and the cardiac surgeon. Since the introduction of cardiac valve replacement surgery in 1960 by Dwight Harken from Boston, prosthesis thrombogenicity remained a constant source of post-operative morbidity and mortality.¹ Efforts over more than three decades to prevent this problem include the development of bileaflet valves and less thrombogenic material, improvement of surgical techniques, optimisation of post-operative care and long-term anticoagulation. Despite these efforts, valve obstruction remains a lifelong risk in patients who have a valve replacement.

The reported incidence of prosthetic valve obstruction ranges from 0.2 to 6% in aortic and mitral valves, and as high as 20% in tricuspid valve prosthesis.^{2,6} The most commonly reported causes for prosthetic valve obstruction in Europe and the United States are pannus formation with thrombosis (35–45%), pannus formation only (31–59%) or thrombosis only (15–24%).^{2,3} Reports from South Africa and India suggest an increased incidence of primary thrombus formation of 54%.^{4,5}

The pathogenesis of intracardiac thrombus formation occurring with or without the implantation of prosthetic material is complex.¹ It is often the direct result of the thrombogenic surface of the prosthesis, altered trans-prosthetic blood flow or inadequate anticoagulation, but other causes of hypercoagulability must always be excluded (Table I).

The diagnosis of obstruction is generally based on the presence of specific clinical, echocardiographic and fluoroscopic features. The clinical presentation may be acute, sub-acute or chronic. Common presenting symptoms and signs are listed in Table II.^{4,5,7,8} The most useful imaging modalities for confirmation of prosthetic valve obstruction include cinefluoroscopy, transthoracic echocardiography (TTE) and transoesophageal echocardiography (TEE) (Table III). Cinefluoroscopy is an excellent method to detect prosthetic valve dysfunction. It requires minimal expertise and is read-

TABLE I. THE PATHOGENESIS OF PROSTHETIC VALVE THROMBOSIS

1. Molecular interaction between plasma components and prosthesis
 - adsorption of plasma proteins
 - adhesion of molecules
2. Transprosthetic blood flow
 - turbulent flow with shear stress and recirculation downstream
 - subclinical haemolysis – release of ADP, PF-4 and thromboglobulin
3. Inadequate anticoagulation
4. Other pro-thrombotic factors
 - loss of atrial contractions
 - drugs, e.g. contraceptives
 - malignant tumours
 - systemic diseases, e.g. SLE
 - incomplete endothelialisation of the valve ring

TABLE II. CLINICAL PICTURE OF PROSTHETIC VALVE THROMBOSIS ON PRESENTATION^{4,5,7,8}

Symptoms	Dyspnoea (NYHA class II–IV)	81–97%
	Cough	66–73%
	Chest pain	24–44%
	Embolisation (central/peripheral)	13–19%
	Asymptomatic	0–8%
Signs	Decreased/absent valve click	56–100%
	Atrial fibrillation	20–38%
	Pulmonary oedema	26–80%
	New/changing murmur	21–26%
	Hypotension	21–22%
	Cardiac arrest	3–5%
	Acute myocardial infarction	2–6%

TABLE III. SPECIAL INVESTIGATION USED FOR DIAGNOSIS OF VALVE OBSTRUCTION

Cinefluoroscopy	Decreased or absent leaflet motion Decreased or absent opening and closing angles
Transthoracic echocardiography	2-D echo: May visualise thrombus or decreased valve motion but resolution is poor Doppler: increased transvalvular gradients may be overestimated Doppler: decreased valve area may be underestimated.
Transesophageal echocardiography*	Optimal resolution Increased ability to detect thrombi on mitral prosthesis and in atria

*Method of choice for diagnosing specific nature of the obstructive process.

ily available in tertiary centres. Two-dimensional TTE may visualise thrombus or decreased valve motion but resolution is usually poor. Doppler TTE may overestimate increased transvalvular gradients and underestimate decreased valve area. Transoesophageal echocardiography has an increased ability to detect thrombi on mitral prosthesis and in atria.^{3,5,6,8–11}

It is difficult to diagnose the specific morphological process responsible for obstruction preoperatively, based on the clinical, echocardiographic and fluoroscopic features. A short duration of symptoms (< 15 days) and poor anticoagulation status (INR < 2.5) suggest primary thrombus formation.^{12,13} Fluoroscopic evidence of abnormal valve motion and echocardiographic evidence of increased transvalvular gradients confirm a clinical diagnosis of obstruction, but do not provide any further information concerning the nature of the obstruction. In a recent publication, Vitale *et al.* suggest that incomplete obstruction or abnormal movement of only one leaflet is more in favour of thrombus formation.¹² Due to its superior resolution, TEE is widely recognised as the method of choice for diagnosis of the specific nature of the obstructive process. The impression of decreased ultrasound intensity, i.e. as soft mass, strongly suggests a thrombus.¹³ When a thrombus is seated on a normal functioning prosthesis it is a strong indication of primary thrombotic obstruction.⁹

Appropriate treatment should be started immediately after diagnosis of the obstruction, the therapeutic goal being rapid restoration of prosthetic function and elimination of thrombotic material. Surgery has traditionally been the treatment of choice. This may include either thrombectomy or valve replacement. Valve replacement is preferable to thrombectomy because it has a lower incidence of rethrombosis.¹⁴ Surgical mortality increased from 2 to 4% in low-risk patients, to 25 to 40% in high-risk patients. Factors associated with increased postoperative mortality include valve prosthesis in the mitral position, severely ill patients, complicated surgery and co-morbid conditions (Table IV).¹⁵

Due to high mortality rates in the high-risk group, thrombolysis has emerged as an alternative to surgical intervention in selected patients. It was first described as successful

TABLE IV. FACTORS ASSOCIATED WITH INCREASED POST-OP MORTALITY^{15,16}

Mitral valve position
NYHA class IV symptoms
Emergency surgery
Need for multiple procedures
Long cross-clamp times
Associated non-cardiac surgical risks

TABLE V. COMPLICATIONS OF THROMBOLYTIC THERAPY^{3,9,15,18,19,20,21}

Thrombo-embolism	8–22%
Bleeding	5–25%
Allergic reaction/anaphylaxis	0–4%
Re-thrombosis	12–40%

therapy for right-sided thrombosis by Luluaga *et al.* in 1971.¹⁶ In 1974 Baille *et al.* reported successful treatment of left-sided prosthetic thrombosis.¹⁷ Success rates with thrombolytic therapy in the range of 73 to 86%,^{18,19} and even 100% in one series²⁰ have been reported. Complications of thrombolytic therapy (Table V) are usually not of catastrophic nature and can be limited by careful patient selection.^{3,9,17–20} Systemic embolism, however, remains a concern with figures of up to 22% reported in the USA and Europe.^{1,6,17,18} Lower rates of embolism (12%) have been reported from India.^{17,18}

The choice of thrombolytic agent (streptokinase, urokinase or t-PA) does not seem to have any influence on the success rate, although thrombolysis may be more rapid and duration of treatment shorter if t-PA is used.¹ Fibrinolysis in selected patients has also been reported to be more cost-effective than surgery.^{4,19–21} Some authors have proposed specific indications for thrombolytic therapy.^{9,12} Patients with reasonable proof of primary thrombus formation, right-sided prosthetic valve thrombosis, and co-morbid conditions with a high surgical risk are ideal candidates for thrombolysis.^{2,6,9,12,17,18} Conflicting ideas exist on the appropriate management of the severely compromised patient. Although these patients have a high surgical risk, time lost waiting for thrombolytic therapy to be effective may also be fatal.

Objective

Our aim is to document our experience of the diagnosis and management of prosthetic valve obstruction at Tygerberg Hospital between January 1991 and February 2001. This study is the first of its kind to be conducted at Tygerberg Hospital and the data may provide us with a platform to direct further prospective research in the field.

Study design

This is a retrospective, descriptive study of all the patients presenting at Tygerberg Hospital between January 1991 and February 2001, with a final diagnosis of prosthetic heart valve obstruction.

Methods

All the echocardiography reports between January 1991 and February 2001 were systematically reviewed for patients with a confirmed diagnosis of prosthetic valve obstruction. Surgical reports of prosthetic valve re-operation over the same period were also screened for patients with a diagnosis of valve obstruction.

Folders and microfilms of the relevant patients were obtained from hospital records.

The following data were gathered from the clinical notes, post-operative reports and imaging reports:

Demographic information: Age, gender, position of clotted valve(s), and type of valve.

Clinical presentation: Interval between valve surgery and presentation, type and duration of symptoms, compliance with anticoagulation, findings on physical examination, and co-morbid conditions.

Special investigations: ECG changes, chest X-ray reports, and INR results of relevant patients were obtained from the haematology archives (adequate anticoagulation was defined as an INR value of > 2.5²²).

Imaging: Fluoroscopy – leaflet motion
Echocardiography – position and size of valve(s)
– transvalvular gradients
(adjusted for valve size)
– leaflet motion
– pulmonary hypertension
– ejection fraction
– nature of obstructive process.

Treatment and outcome: Surgery or thrombolysis, time from presentation to treatment, complications, and duration of hospital stay.

Results

Clinical presentation

Between January 1991 and February 2001, 32 patients presented on 34 occasions with left-sided valve obstruction. This included seven men and 25 women, with a mean age of 36 years (range: 17.9–62.9 years). Thirty patients had their initial valve replacement performed at Tygerberg Hospital. Eleven patients received double valve replacement.

The interval between implantation and obstruction of the prosthetic valve ranged from 45 days to 188 months (mean: 67 months). The mean time from the onset of symptoms to presentation was 6 days (range: 1–30 days). Obstruction occurred in the mitral position in 19 patients (56%) and in the aortic position in 15 patients (44%). With the exception of two patients, all cases had bileaflet prosthetic valves (St Jude Medical). One patient had a Björk-Shiley tilting-disc valve and one patient a Carpentier-Edwards bioprosthesis.

The symptoms and signs of all patients on presentation are listed in Table VI. Twenty-two patients presented in New-York Heart Association functional class IV; nine of them being in shock, three were in class III and four in

TABLE VI. CLINICAL PRESENTATION OF PROSTHETIC VALVE THROMBOSIS AT TYGERBERG HOSPITAL BETWEEN JANUARY 1991 AND FEBRUARY 2001 (34 ADMISSIONS OF 32 PATIENTS)

Symptoms	Dyspnoea (NYHA class III–IV)	74%
	Chest pain	50%
	Embolisation (cerebral/limb)	12%
	Asymptomatic	15%
	Dyspnoea (NYHA class II)	12%
Signs	Pulmonary oedema	91%
	Decreased/absent valve click	50%
	Abnormal murmur	29%
	Hypotension	26%
	Atrial fibrillation	18%
	Acute myocardial infarction	6%

class II (Fig. 1). Five patients denied any symptomatology. In two patients, the diagnosis was suspected on clinical grounds due to sinus tachycardia, an absent prosthetic click and a changing murmur. The remaining three asymptomatic patients were diagnosed on routine follow-up transthoracic echocardiography. Chest pain was a presenting complaint in 17 cases (50%). Seven patients had typical angina pectoris. Although cough is mentioned in the literature as a prominent presenting complaint (see Table II), we could not find consistent documentation of its presence or absence. In view of the high percentage of patients presenting with pulmonary oedema though, one would expect it to be a prominent and early symptom of prosthetic valve obstruction.

Co-morbid conditions included SLE with nephrotic syndrome (1), diabetes mellitus (2), coronary artery disease (7), pneumonia (3), gout (1), sero-negative arthritis (1), pregnancy (1), haemoptysis (1) and one patient who tested positive for HIV. Four patients presented with a thrombo-embolic event; two had cerebral emboli and two presented with acute myocardial infarction, presumed to be of embolic nature. One patient had an embolism to his right leg while waiting for re-operation. The majority of the patients (82%) were in sinus rhythm. Of the six patients in atrial fibrillation, only one presented with a thrombo-embolic event. An absent prosthetic click was reported in 50% of cases. Almost all the patients (31), including one asymptomatic patient, had basal crepitations on auscultation of the lungs.

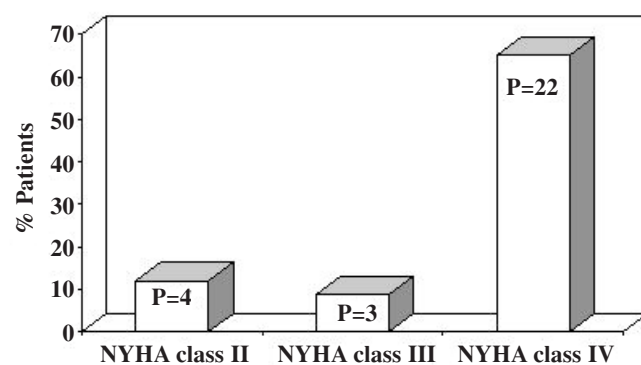


Fig. 1. Severity of dyspnoea on presentation.

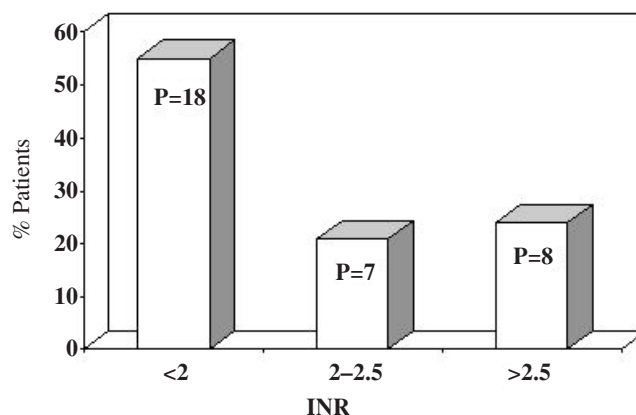


Fig. 2. Anticoagulation status on presentation (available in 33 of 34 admissions).

Special investigations

Anticoagulation was considered inadequate (INR < 2.5) in 75.8% of the admissions (Fig. 2). Anticoagulant administration was discontinued before presentation on six occasions (18%).

Chest X-rays showed evidence of pulmonary oedema in 29 cases. ECG abnormalities included sinus tachycardia, 1st degree AV-block, atrial fibrillation, bi-atrial enlargement, left ventricular hypertrophy, left and right bundle branch block and myocardial infarction changes. Two ECGs were reported as normal.

Transthoracic echocardiography was performed in 33 of the 34 cases. Abnormal leaflet motion was observed in all but one patient. Pressure gradients, as measured by doppler echocardiography on presentation, were increased in all cases. Serial measurements were only available in five patients and did not show a gradual increase in gradients over the time before the obstruction. Echocardiographic evidence of pulmonary hypertension was reported in 25 cases (74%); two asymptomatic patients had increased pulmonary pressures. On transthoracic echocardiography, thrombus was suspected in 20 patients and pannus formation in one patient. No comment was made on the presence of thrombus or pannus in the remaining 13 cases. Primary thrombus formation was wrongly suspected in three cases and the patient proposed to have pannus formation turned out to have a combination of pannus and thrombus.

Cinefluoroscopic examination was utilised on only five occasions. All the patients had abnormal leaflet motion. One of these patients had normal leaflet motion reported on transthoracic echocardiography.

Diagnostic transoesophageal echocardiography was performed on only two occasions. The nature of the obstruction was correctly diagnosed in both patients – pannus in one case, and thrombus in the other.

Treatment

Treatment options included valve replacement surgery (20), thrombolysis (6) or no treatment (8). The interval from hospital admission to treatment varied from 3 hours to 18 weeks (Table VII). This interval depended mainly on the patients' condition on presentation and the time necessary to establish the diagnosis.

TABLE VII. INTERVAL FROM HOSPITAL ADMISSION TO TREATMENT

Interval	No. of patients
< 24 hours	13 (50%)
1 day – 1 week	6 (23%)
1 week – 1 month	5 (19%)
> 1 month	2 (8%)

Twenty patients (59%) underwent prosthetic valve replacement consisting of St Jude bileaflet mechanical prosthesis. Reasons for obstruction, reported by the surgeon on macroscopic examination of the explanted valves included thrombus only (55%), pannus only (15%) or a combination of thrombus and pannus (30%). Complications occurred in 50% of cases. Intra-operative acute heart failure and cardiac arrest was the cause of death in four patients. Post-operative complications included pneumonia (2), septicaemia (2), transient acute renal failure (2) and one patient needed evacuation of an intrathoracic haematoma. Eleven patients died (55%); 10 of these patients presented in NYHA class IV. In the group that survived surgery, only one patient presented in NYHA class IV.

A total of six patients (17.6%) received thrombolytic treatment. Four patients were considered too ill for surgery. In one patient who tested HIV positive, the surgeons declined to offer re-operation. In another case, surgery was not available due to a lack of postoperative ICU beds. Three patients (50%) had complications associated with the thrombolytic treatment. One had transient epistaxis, one had upper gastrointestinal bleeding and a stroke, and one re-obstructed after two months. The patient who re-obstructed turned out to have primary pannus formation on re-operation. Four patients in the thrombolytic group died (66.6%), including the patient turned down for surgery due to an ICU bed shortage. This was also the only patient in the thrombolytic group whose death could at least partially be contributed to the complications of thrombolytic treatment (GIT bleed and stroke).

Eight patients (23.5%) did not receive either of the above treatments. Four of these patients died before emergency surgery could be carried out; three were not considered for either treatment due to severe haemoptysis, a large stroke, and chronic renal failure, respectively. The only survivor in the group recovered after embolisation of the obstructing thrombus to his right leg.

Discussion

The incidence of left-sided prosthetic valve thrombosis is reported in the current literature to be between 0.5 and 6% per patient year. Although relatively rare, it remains the most frequent valve-related complication of mechanical prostheses.³ This study included only patients who presented to Tygerberg Hospital with obstructed prosthetic valves. We have no knowledge of the non-presenters, and included in the study two patients who received their valves at other institutions. We therefore cannot comment on the incidence of prosthetic valve obstruction at Tygerberg Hospital during the last 10 years.

The clinical characteristics of prosthetic valve thrombosis have been well described by other investigators. Classically

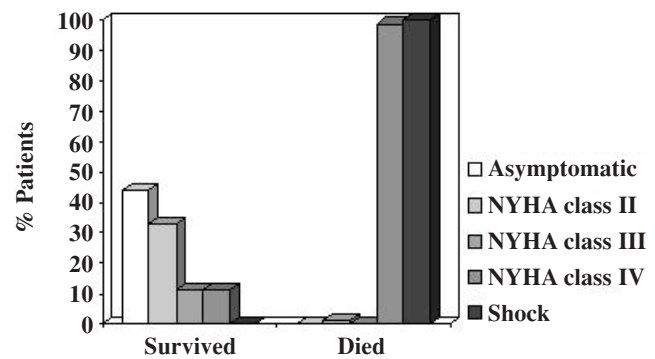


Fig. 3. Relationship between mortality and presenting status.

the patient's condition deteriorates rapidly to become life threatening in a short period of time. Some patients, however, may present in a sub-acute, slowly progressive manner. In another group of patients, usually with non-obstructive thrombosis, presentation may be insidious or limited to peripheral embolism.

Kontos *et al.* evaluated 27 patients presenting to the Mayo Clinic with prosthetic valve thrombosis over a 19-year period, and reported that 70% of patients had clinical evidence of prosthetic valve malfunction for 1 week to 6 months prior to presentation.⁸ In our study, most patients (66%) presented with a history of onset of symptoms of 1 week or less before presentation. A short duration of symptoms may suggest thrombus formation. In this series though, there was no statistical association between the duration of symptoms and the pathological process causing obstruction. A careful and detailed history should always be taken from patients with prosthetic valves. Even relatively minor symptoms should prompt a more intensive search for prosthetic valve malfunction in an attempt to diagnose these patients earlier. The most prominent symptoms were dyspnoea and chest pain. The majority of patients presenting to our unit already had severe disability. Of the 25 patients in NYHA class III and IV, only four survived. When complicated by cardiac shock, the mortality was 100% (Fig. 3).

Chest pain was a common complaint. Although a relatively high percentage of patients (21%) had a history of underlying coronary artery disease, only one patient in this subgroup complained of chest pain. The reasons why patients develop atypical chest pains with prosthetic valve obstruction are unclear.

Co-morbid conditions that may have contributed to thrombus formation were present in 12 patients and included atrial fibrillation (6), previous myocardial infarction (6), nephrotic syndrome (1), SLE (1), pregnancy (1) and type 2 diabetes mellitus (2). The only case in this series with obstruction of a bioprosthesis was due to a combination of pannus and thrombus formation. Because the Carpentier-Edwards bioprosthesis is non-thrombogenic, the presence of atrial fibrillation and an atrial size of 71 mm must have made a major contribution to thrombus formation in this case. An important finding was the absence of a prosthetic click on auscultation in 50% of cases. This supports the 56% reported by Kontos *et al.*⁸ Most other studies reported the absence of prosthetic clicks in up to 100% of patients.^{4,5,7} Although

the presence of an abnormal murmur and basal crepitations on auscultation are non-specific findings, it should be sought, and when present investigated appropriately.

Chest X-ray and EGG findings were non-specific, confirmed clinical findings only and did not contribute additional diagnostic or prognostic information.

The anticoagulation status of our patients was generally suboptimal (Fig. 2), suggesting that sub-therapeutic INR levels contribute to thrombosis of the prosthetic valve. Because we do not know the INR values of the non-presenters, the data cannot be analysed to statistically express the risk factor associated with a sub-therapeutic INR. One must also keep in mind that a single INR value on presentation does not reflect the patient's anticoagulation status during the weeks or months prior to presentation. It is therefore very important to keep the INR values within the therapeutic range at all times. This may be achieved by regular follow-up, preferably at a dedicated clinic.

Transthoracic echocardiography proved to be adequate in confirming abnormal or absent leaflet motion in all but one patient. Although increased transvalvular pressure gradients were present in all patients, it is of less value as a single measurement due to overestimation of the net pressure gradient across bileaflet prosthetic valves. Serial measurements with progressively increasing gradients are more specific.

The major advantage of using transoesophageal echocardiography in prosthetic valve obstruction lies in its superior resolution. More accurate diagnosis of the nature of the obstruction may prove to be invaluable in cases where thrombolytic treatment is considered.

Cinefluoroscopic examination was utilised only five times, and was diagnostic in all cases. The one case in our study with an increased pressure gradient across the mitral valve, but with apparently normal leaflet motion on transthoracic echocardiographic examination, was correctly diagnosed with cinefluoroscopy, which is an excellent method to show abnormal leaflet motion, is non-invasive, easy to perform and readily available in many hospitals. It may also be of great value when echocardiographic services are not immediately available, i.e. after hours or in a peripheral hospital setting.

Currently all the patients presenting at our unit with prosthetic valve obstruction are considered for re-operation as the first choice of treatment. In this series, only 59% eventually received surgery. The main reason for not receiving surgery was that the patients were too ill (11 out of 14). Four patients in this group received streptokinase and four died before receiving any treatment. The remaining three patients were not considered for any treatment due to severe haemoptysis, a large cerebral infarct, and advanced chronic renal failure, respectively. Only one patient in the streptokinase group survived but re-obstructed two months later. With subsequent surgery, pannus formation was reported as the reason for failure of thrombolysis. Other reasons cited for not performing surgery included positive HIV status (1) and a lack of post-operative intensive-care beds (1). Both of these patients received streptokinase. Only the HIV-positive patient survived. The only survivor in the group of patients who did not receive any treatment had embolisation of the obstructing thrombus to the right leg while awaiting surgery. Follow-

up echocardiography confirmed resolution of the thrombus.

The fact that only six patients were treated with four different regimes of streptokinase probably reflects the lack of clear guidelines on selecting patients for thrombolysis and limited experience with this treatment modality. Commonly recommended treatment regimes include streptokinase 250 000 U loading dose over 30 minutes, followed by an infusion of 100 000 U/hr, or tissue plasminogen activator (t-PA) 10 mg as a bolus, followed by an infusion of 5 mg/hr. When full valve mobility is achieved, thrombolytic therapy may be stopped. All patients should receive an intra-venous heparin infusion at a rate of 1 000 U/hr after thrombolysis for a variable period until adequate anticoagulation is achieved with warfarin.¹⁹⁻²¹

Intra-operative macroscopic examination of the explanted prosthetic valves showed a clear predominance of primary thrombus formation and only a small number of patients with pannus formation. These results contradict several well-performed studies from Europe and the USA which cited pannus formation as the most frequent cause of obstruction.^{2,3} Our results though, are very similar to other studies from the developing world.^{4,5} One reason for the difference may be a higher rate of inadequate anticoagulation in developing countries, due mainly to socio-economic factors. Other reasons may be the different ways of diagnosing the process, i.e. macroscopic versus microscopic examination, and differences in study designs.

Overall mortality of patients presenting to our unit with prosthetic valve obstruction was 64.7%. This extremely high mortality rate does not compare well with most studies quoted in the literature. The main reason for our high mortality rate appears to be the poor condition of patients on presentation to the unit, therefore requiring emergency surgery, the large number of patients not receiving any form of treatment, and the lack of criteria to decide on the most appropriate form of treatment, i.e. valve replacement or thrombolysis. Although the study was not designed to identify specific risk factors for mortality, cardiogenic shock and NYHA class IV on presentation were clearly associated with a poor prognosis. Other important factors, which we were unable to investigate in more detail during this study, and that may have contributed to the high mortality include: time between presentation and surgery, time to make definitive diagnosis, cross-clamp times, surgical techniques, experience of the surgeon and specific post-operative care factors. It is well documented that optimisation of these factors improves outcome.

We believe the best way to prevent prosthetic valve thrombosis is to keep the INR within the therapeutic range and avoid periods of inadequate anticoagulation. Careful selection of patients for valve replacement by a team including a cardiologist, a cardio-thoracic surgeon and a social worker also plays a major role.

It is likely that earlier diagnosis of prosthetic valve malfunction may improve prognosis significantly. Taking a good history and examining the patient thoroughly with every follow-up visit is mandatory. The use of cinefluoroscopy may speed up the diagnostic process and facilitate earlier treatment. Transoesophageal echocardiography is the imaging modality of choice for diagnosing thrombus or pannus formation.

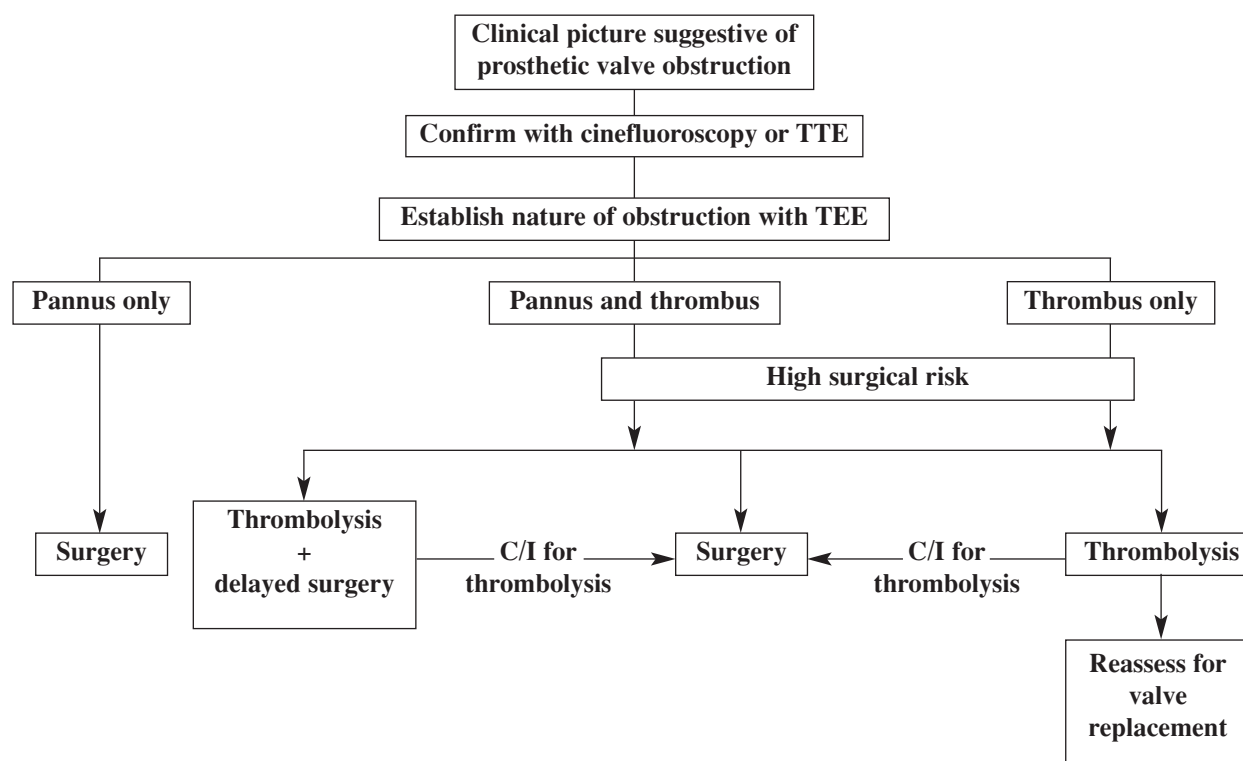


Fig. 4. Proposed algorithm for the management of prosthetic valve obstruction.

From this study it appears that thrombolysis as a treatment modality is under-utilised, possibly due to a lack of guidelines for selecting suitable candidates (Fig. 4). Thrombolytic therapy may prove to be particularly beneficial in patients considered to have high non-cardiac surgical risks and in patients presenting with NYHA class IV symptoms or cardiogenic shock. Primary thrombus formation was the predominant cause of prosthetic valve obstruction at Tygerberg Hospital during the last 10 years and a large number of patients were poor candidates for surgery. Given these factors and the very high mortality rate with the current management of prosthetic valve obstruction, treatment with thrombolysis in selected patients deserves consideration in a prospective study.

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