ELSEVIER

European Journal of Cardio-thoracic Surgery 11 (1997) 46–52

EUROPEAN JOURNAL OF
CARDIO-THORACIC
SURGERY

Long-term results of surgery for active infective endocarditis[1]

Y. d'Udekem, T.E. David*, C.M. Feindel, S. Armstrong, Z. Sun

From the Division of Cardiovascular Surgery of The Toronto Hospital and the University of Toronto, 200 Elizabeth Street, 13EN219 Toronto, Ontario M5G 2C4, Canada

Received 5 October 1995; revised 18 March 1996; accepted 17 April 1996

Abstract

Objective: This paper was undertaken to determine the long-term outcome of active infective endocarditis treated with antibiotic and radical excision of infected tissues by surgery. *Methods:* From October 1978 to August 1994, 122 consecutive patients were operated on during the acute phase of infective endocarditis. There were 85 men and 37 women whose mean age was 50 years, ranging from 20 to 79. Surgery was needed because of one or more of the following complications: cardiogenic/septic shock in 19 patients, congestive heart failure in 68, persistent sepsis in 64, peripheral embolization in 20, and cerebral embolization in 10. The offending microorganism was identified in 110 patients, staphylococci were the most common ones. Seventy-six patients had native valve endocarditis and 46 had prosthetic valve endocarditis. Simple valve replacement or repair was performed in 60 patients and radical resection of the valve and surrounding tissues with reconstruction of the heart with either fresh autologous pericardium or glutaraldehyde-fixed bovine pericardium was performed in 62 with paravalvular abscess. Pulmonary autograft and aortic homograft were used in only three patients, the remaining patients had either bioprostheses or mechanical heart valves if valve repair was not feasible. *Results:* There were nine deaths, for an operative mortality of 7.4%. Logistic regression analysis identified preoperative shock and renal failure as predictors of operative mortality. Operative survivors were followed up from 4 to 173 months, mean of 56.4. The actuarial survival at 10 years was $61 \pm 6\%$. Logistic regression analysis identified preoperative New York Heart Association functional class IV and perioperative renal failure as predictors of late mortality. Eight patients developed recurrent endocarditis 10–102 months postoperatively. The freedom from recurrent endocarditis at 10 years was $79 \pm 9\%$. All patients who developed this late complication had paravalvular abscess at the time of original operation. *Conclusions:* These data suggest that surgery for active infective endocarditis yield a high probability of eradicating the infection with relatively low operative mortality and good long-term results.

Keywords: Abscess; Endocarditis; Follow-up studies; Heart valve prosthesis

1. Introduction

Although antibiotics are the primary therapy for infective endocarditis, adjunct surgical treatment is necessary in a large proportion of patients [8,17,19]. Experience with heart valve surgery in patients with active infective endocarditis has shown that timing of surgical intervention is an important determinant of outcome because operative deaths are often related to metastatic infections, multiorgan failure, and recurrent infection with prosthetic valve dehiscence [2,4,8,13,17,19]. Thus, it is important to consider surgical intervention in all patients with prosthetic valve endocarditis and in patients with native valve endocarditis who develop signs of valve dysfunction and heart failure, persistent sepsis, large vegetations and recurrent emboli [2–4,8,12,13,17,20].

This paper is a review of the experience of two attending surgeons at The Toronto Hospital who have surgically treated patients with active infective endocarditis in the same fashion.

* Corresponding author. Tel.: +1 416 3405062; fax: +1 416 3403803.

[1] Presented at the Ninth Annual Meeting of the European Association for Cardiothoracic Surgery, Paris, France, 24–27 September 1995.

1010-7940/97/$17.00
PII S1010-7940(96)01020-2

2. Patients and methods

From October 1978 to August 1994, 122 consecutive patients were operated on during the active phase of infective endocarditis, that is, surgical intervention was deemed necessary before completion of a course of intravenous antibiotic therapy. Table 1 shows the clinical data of these patients. Only four patients were intravenous drug users. Seventy-six patients had native and 46 had prosthetic valve endocarditis. Table 2 shows the pathology of the infective endocarditis. The 46 patients with prosthetic valve endocarditis had a total of 62 previous valve operations. Twelve of them had also had one or more previous composite replacements of the ascending aorta and aortic valve with either a homograft or a valved Dacron conduit. Paravalvular abscess was present in 28 patients (38%) with native valve endocarditis and 34 patients (74%) with prosthetic valve endocarditis.

The offending microorganism was identified by preoperative blood cultures or surgical specimens in 110 patients. The remaining 12 patients had clinical and pathologic evidence of endocarditis but no microorganism could be cultured and they were treated as culture negative endocarditis. Table 3 lists the microorganisms responsible for the endocarditis.

Nineteen patients (15%) were moribund at the time of surgery because of cardiogenic and/or septic shock. The remaining 103 patients were operated on because of one or more of the following problems: symptoms of congestive heart failure due to valve dysfunction in 68 (56%), persistent sepsis in 64 (53%), peripheral embolization in 20 (16%), and cerebral embolization in 10 (8%). Preoperative coronary angiography was performed in 58 patients and 13 were found to have significant coronary artery disease.

2.1. Operative management

Radical resection of all infected valvular and paravalvular tissues was the basic operative principle employed in all patients. If the infection was limited to the leaflets of the valve, simple valve replacement was performed. However, mitral valve repair was feasible in eight patients (four isolated and four combined with aortic valve replacement). If the infection had extended into the annulus or surrounding structures, a radical resection of the abscess and patch reconstruction of the annulus and adjoining structures with either fresh autologous pericardium or glutaraldehyde-fixed bovine pericardium was performed. We have described these operative techniques in detail in previous publications [5–7,16]. Surgical instruments, suction equipment, local drapes and surgical gloves were discarded after extirpation of the infected tissues and new ones used for the reconstruction of the heart.

Simple valve replacement or repair was performed in 60 patients and complex reconstructions in 62. Table 4 lists the operations performed.

Patients received intravenous antibiotic therapy for a total of 6 weeks. No patient was kept on antibiotics permanently.

2.2. Statistical analyses

Clinical, laboratorial, and pathological variables were tested by chi-square and Fisher's exact test to determine their statistical value as predictors of operative and late mortality. The statistically significant ones were entered into a stepwise logistic regression model to determine their independent value. Postoperative events were estimated using the Kaplan-Meier method. Results were expressed in percentages of the means and percentages of the standard errors of the means. SAS (Statistical Analysis System Institute, Carry, NC) and BMDP (BMDP Statistical Software, Los Angeles, CA) softwares were used for these analyses.

3. Results

There were nine operative deaths for an overall operative mortality of 7.4%. Of the nine patients who died, six had prosthetic and three had native valve endocarditis. The six patients with prosthetic valve endocarditis who died had paravalvular abscesses and were moribund before surgery: three died in the operating room due to technical problems, one died of respiratory failure (she had suffered a preoperative hemorrhagic stroke), one died in hepatic failure, and one died because of gastrointestinal bleeding. Of the three patients with native valve endocarditis, two died because of multi-organ failure and one due to bleeding caused by

Table 1
Clinical data

Number of patients		122 (%)
Age:	Mean ± S.D. (years)	50 ± 17
	Range	20–79
Sex:	Male	85 (70)
	Female	37 (30)
ECG:	Sinus rhythm	95 (78)
	Atrial fibrillation/flutter	21 (17)
	Complete heart block	6 (5)
NYHA functional class		
1 and 2		21 (17)
3		20 (16)
4		81 (66)
Cardiogenic and/or septic shock		19 (15)

S.D., standard deviation of mean; ECG, electrocardiogram; NYHA, New York Heart Association.

Table 2
Pathology of the infective valve endocarditis

Etiology of valve disease	
Aortic valve:	
Bicuspid	21
Calcific degeneration	5
Annuloaortic ectasia	5
Rheumatic	3
Normal	10
Prosthetic valve	39*
Mitral valve:	
Myxomatous degeneration	14
Rheumatic	10
Normal	3
Prosthetic valve	17*
Tricuspid valve:	
Normal	2
Ventricular septal defect	2
Pulmonary valve:	
Tetralogy of Fallot	1
Native valve endocarditis:	
Infection limited to leaflets	46
Paravalvular abscess	28
Prosthetic valve endocarditis:	
Infection limited to valve	12
Paravalvular abscess	34
Type of valve infected:	
Aortic homograft	3
Bioprostheses	24
Mechanical prosthesis	19
Timing of infection:	
Early (<61 days)	6
Late (>60 days)	42

*Ten patients had prosthetic valves in both positions.

dehiscence of a patch used to reconstruct the outflow tract. Table 5 shows the operative mortality in various subgroups of patients. The following variables were associated with higher operative mortality: preoperative

Table 3
Offending microorganisms

	Native valve	Prosthetic valve
Staphylococcus aureus	22	13
Staphylococcus epidermis	3	12
Streptococcus viridans	29	0
Streptococcus faecalis	5	4
Other *streptococci*	6	4
Enterobacter	0	2
Escherischia coli	0	1
Pseudomonas aeruginosa	0	1
Pseudomonas stutzeri	0	1
Hemophilus parainfluenzae	1	1
Actinobacillus	0	1
Cardiobacterium hominis	1	1
Coxiella bunnetii	1	0
Proprionibacterium	0	1
Culture negative endocarditis	8	4

Table 4
Operative data

	Native valve	Prosthetic valve
AVR	28	10
Bentall	3	14
AVR + MVR	13	12
AVR + MV repair	4	0
AVR + TVR + PVR	0	1
Bentall + MVR	0	2
MVR	19	7
MV repair	4	0
TV repair	4	0
PVR	1	0
Additional procedures:		
Reconstruction of LVOFT	18	31
Reconstruction of MV annulus	14	13
Reconstruction of RVOFT	1	1
Coronary artery bypass	7	6
Repair of tetralogy of Fallot	1	0
Repair of ventricular septal defect	2	0
Prosthetic valves:		
Aortic homograft	1	1
Pulmonary autograft	1	0
Bioprostheses	38	18
Mechanical valves	28	27

AVR, aortic valve replacement; Bentall, composite replacement of the aortic valve; MVR, mitral valve replacement; TVR, tricuspid valve replacement; PVR, pulmonary valve replacement; LVOFT, left ventricular outflow tract; RVOFT, right ventricular outflow tract.

shock, New York Heart Association (NYHA) functional class IV, and postoperative renal failure. Although the mortality in patients with prosthetic valve endocarditis was higher than in patients with native valve endocarditis, the difference did not reach statistical significance ($P = 0.062$). A stepwise logistic regression analysis indicated that only preoperative shock and postoperative renal failure were predictive of operative death. The operative mortality rate was not affected by the year that the operation was performed.

Approximately one-third of the patients experienced one or more postoperative complications: 13 (11%) had to be reexplored because of bleeding; three (2.4%) suffered a perioperative stroke; 15 (12%) developed low cardiac output syndrome; 12 (10%) required renal dialysis; 23 (19%) required insertion of a permanent transvenous pacemaker; and, one patient required reoperation three weeks after the original procedure because of persistent sepsis. One patient required splenectomy and two needed leg amputation because of extensive limb necrosis due to septic emboli.

The mean postoperative length of stay in hospital was 24 ± 15 days, ranging from 6 to 78 days. The mean length of stay in the intensive care unit was 4.6 ± 5.3 days, ranging from 1 to 27 days.

Table 5
Operative mortality

	No. patients	No. deaths (%)	*P*-value (chi-square)
Age			
<66	94	8 (8)	0.380
>65	28	1 (3)	
Sex			
Male	85	7 (8)	0.583
Female	37	2 (5)	
NYHA: Classes I, II, and III	41	0	0.023
Cardiogenic/septic shock			
Yes	19	5 (26)	0.001
No	103	4 (4)	
Native valve endocarditis	76	3 (4)	0.062
Prosthetic valve endocarditis	46	6 (13)	
Paravalvular abscess			
Yes	62	7 (11)	0.083
No	60	2 (3)	
Postoperative renal failure			
Yes	12	4 (33)	0.000
No	110	5 (5)	
Staphylococcus aureus			
Yes	35	4 (11)	0.222
No	87	5 (6)	
Number of valves infected			
One	34	3 (9)	0.704
Two or more	88	6 (7)	

Only one patient was lost to follow-up. The length of the follow-up extended from 4 to 173 months, mean 56.4. There have been 20 late deaths. Eight deaths were valve related: late recurrent infective endocarditis in three, late valve dehiscence in one, stroke in one, anticoagulant-related hemorrhage in one, and sudden death in two. Four deaths were non-valvular cardiac deaths, and eight were non-cardiac deaths. Fig. 1 shows the actuarial survival of all patients, at 5 years it was $76 \pm 4\%$ and at 10 years it was $61 \pm 6\%$. A stepwise logistic regression analysis demonstrated that preoperative New York Heart Association functional class IV and perioperative renal failure were predictive of late death. Of the survivors, 91% of them were in NYHA functional classes I or II.

Eight patients had recurrent endocarditis, 10–102 months (mean of 47 months) after the initial operation for endocarditis. Six were treated medically and three died. Two were treated surgically and survived. It is noteworthy that all patients who had late recurrent endocarditis had an abscess at the initial operation. Fig. 2 shows the freedom from recurrent infective endocarditis at 5 years was $91 \pm 3\%$ and at 10 years was $79 \pm 9\%$.

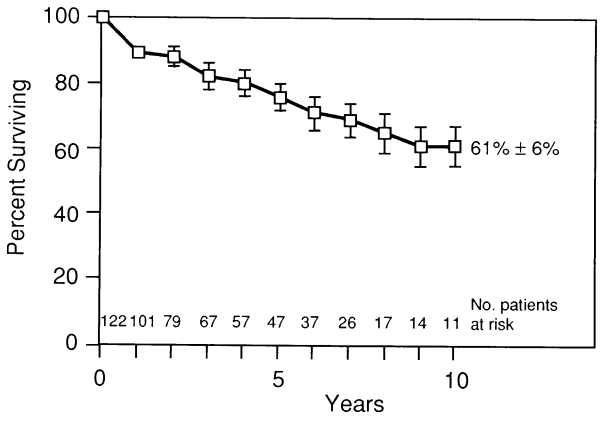

Fig. 1. Actuarial survival of all patients.

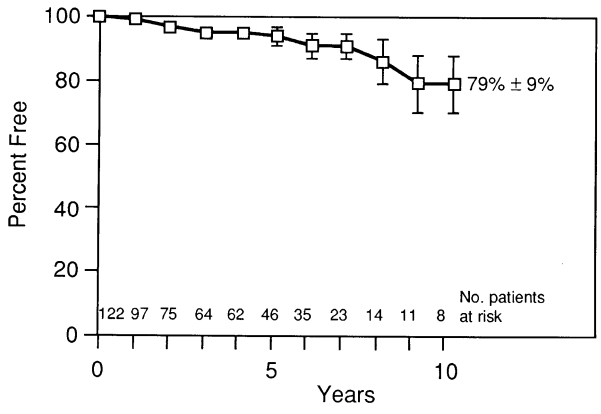

Fig. 2. Freedom from recurrent infective endocarditis.

Ten patients needed reoperation: six for bioprosthetic degeneration, two for endocarditis, one for failed mitral valve repair, and one for paravalvular leak. The freedom from reoperation at 5 years was $96 \pm 2\%$ and at 10 years was $86 \pm 5\%$.

Thromboembolic complications occurred in four patients: three had transient ischemic attacks and one had a fatal stroke. The freedom from thromboembolism at 5 years was $95 \pm 2\%$ and at 10 years was $92 \pm 3\%$.

4. Discussion

This series of patients with infective endocarditis differs from others probably because of bias in referral patterns to and within a large tertiary care facility. This is the reason why we had so many patients who were in shock when transferred to our hospital, and also so many patients with paravalvular abscesses. Previous reports on surgically treated patients with infective endocarditis indicate that paravalvular abscess was present in 11–28% of the cases of native valve endocarditis [1,11,13,19], and in 40–63% of the patients with prosthetic valve endocarditis [9,11,12]. In our present series, 38% of the patients with native and 74% of those with prosthetic valve endocarditis had paravalvular abscess. These incidences of paravalvular abscess are similar to those we reported previously [3,4]. Since most of our patients had the diagnosis of abscess made preoperatively by Doppler echocardiography, this high incidence of abscess is partly due to referral bias rather than the true incidence of abscess in patients with infective endocarditis who fail to respond to medical therapy. Additionally, we had a high proportion of cases of endocarditis due to *Staphylococcus aureus* which is a very virulent microorganism that rapidly destroys the valve leaflets and extends into the paravalvular tissues.

Staphylococci and *streptococci* were the most common microorganisms responsible for the endocarditis in our patients. These microorganisms were also the most common agents in most series of surgically treated patients [2,8,12,17]. However, we had a higher proportion of *Staphylococcus aureus* than other series [2,8,12,17]. This microorganism has become the commonest offending agent in patients with endocarditis [20]. In the past, *Staphylococcus aureus* endocarditis was associated with higher mortality and morbidity [2,17], but this no longer appears to be the case at the present [20]. We had no case of fungal endocarditis probably because of the small number of intravenous drug users among our patients. It is said that fungal endocarditis almost invariably requires surgical treatment, is difficult to eradicate, and is associated with high mortality and morbidity [18,20].

The overall operative mortality in our series (7.4%) as well as the mortality in patients with native valve endocarditis (4%) and prosthetic valve endocarditis (13%) are among the lowest operative mortality rates ever reported in patients with active infective endocarditis. Most reports indicate a mortality rate of 6–26% for native valve and 8–67% for prosthetic valve endocarditis [2,12–15,17]. These wide ranges in operative mortality rates must reflect the clinical conditions that the patients were in at the time of surgery, the offending microorganisms, and also the operative approach employed. For instance, in aortic valve endocarditis the use of aortic valve homografts has been shown to decrease the operative mortality as well as the risk of late recurrent infection [10–15]. Our data suggest that equally good clinical results can be obtained by the approach used in this series. We firmly believe that there is no substitute for radical extirpation of the infected tissues and meticulous reconstruction of the heart to obtain good clinical results in patients with active infective endocarditis. All our patients received bactericidal antibiotics for a total of 6 weeks during the perioperative course [4,5,7,16,18]. Prosthetic valve dehiscence and persistent endocarditis are usually caused by inadequate debridement of all infected tissues or gross contamination of the prosthetic valve implanted.

The long-term survival of patients surgically treated for active infective endocarditis are quite satisfactory [4,8,12]. The actuarial survival in this series was 61% at 10 years. However, late recurrent endocarditis remains a problem in these patients. In our series, all cases of late recurrent endocarditis occurred in patients who had a paravalvular abscess at the time of the first surgery for active infective endocarditis. The freedom from recurrent endocarditis was 79% at 10 years. These findings were also observed by other investigators [8,12]. It is not known why these patients are at a higher risk of recurrent infection.

In conclusion, patients with infective endocarditis of the native valves should be operated at the earliest signs of severe valve dysfunction, paravalvular abscess or recurrent embolization. Radical resection of all infected tissues, reconstruction of the annuli with biological materials and careful implantation of prosthetic valves yield good operative and long-term results.

References

[1] Arnett EN, Roberts WC. Valve ring abscess in active infective endocarditis. Frequency, location, and clues to clinical diagnosis from the study of 95 necropsy patients. Circulation 1976;54:140–145.

[2] D'Agostino RS, Miller DC, Stinson EB, Mitchell RS, Oyer PE, Jamieson SW, Baldwin JC, Shumway NE. Valve replacement in patients with native valve endocarditis: what really determines operative outcome? Ann Thorac Surg 1985;40:429–438.

[3] David TE. The surgical treatment of patients with prosthetic valve endocarditis. Sem Thorac Cardiovasc Surg 1995;7:47–53.
[4] David TE, Bos J, Christakis OT, Brofman PR, Wong D, Feindel CM. Heart valve operations in patients with active infective endocarditis. Ann Thorac Surg 1990;49:701–705.
[5] David TE, Feindel CM, Armstrong S, Sun Z. Reconstruction of the mitral annulus. A ten-year experience. J Thorac Cardiovasc Surg 1995;110:1323–1332.
[6] David TE, Feindel CM, Ropchan GV. Reconstruction of the left ventricle with autologous pericardium. J Thorac Cardiovasc Surg 1987;94:710–714.
[7] David TE, Komeda M, Broffman PR. Surgical treatment of aortic root abscess. Circulation 1989;80(suppl I):269–274.
[8] Delahaye F, Ecochard R, De Gevigney C, Malquarti V, Saradarian W, Delaye J. The long term prognosis of infective endocarditis. Eur Heart J 1995;16(Suppl B):48–53.
[9] Fernicola DJ, Roberts WC. Frequency of ring abscess and cuspal infection in active infective endocarditis involving bioprosthetic valves. Am J Cardiol 1993;72:314–323.
[10] Haycock D, Barratt-Boyes B, Macedo T, Kirklin JW, Blackstone E. Aortic valve replacement for active infectious endocarditis in 108 patients. A comparison of freehand allograft valves with mechanical prostheses and bioprostheses. J Thorac Cardiovasc Surg 1992;103:130–139.
[11] John RM, Pugsley W, Treasure T, Sturridge MF, Swanton RH. Aortic root complications of infective endocarditis – influence on surgical outcome. Eur Heart J 1991;12:241–248.
[12] Larbalestier RI, Kinchla NM, Aranki SF, Couper GS, Collins JJ Jr, Cohn LH. Acute bacterial endocarditis. Optimizing surgical results. Circulation 1992;86(suppl II):68–74.
[13] Middlemost S, Wisenbaugh T, Meyerowitz C, Teeger S, Essop R, Skoularigis J, Cronje S, Sareli P. A case for early surgery in native left-sided endocarditis complicated by heart failure: results in 203 patients. J Am Coll Cardiol 1991;18:663–667.
[14] Otaki M. Prosthetic valve endocarditis. An analysis of the outcome in 32 cases. ASAIO J 1994;40:176–180.
[15] Petrou M, Wong K, Albertucci M, Brecker SJ, Yacoub MH. Evaluation of unstented aortic homografts for the treatment of prosthetic aortic valve endocarditis. Circulation 1994;90(part 2):198–204.
[16] Ralph-Edwards A, David TE, Bos J. Infective endocarditis in patients who had replacement of the aortic root. Ann Thorac Surg 1994;58:429–433.
[17] Richardson JV, Karp RB, Kirklin JW, Dismukes WE. Treatment of infective endocarditis: a 10-year comparative analysis. Circulation 1978;58:589–597.
[18] Rubinstein E, Noriega ER, Simberkoff MS, Holzman R, Rahal JJ. Fungal endocarditis: analysis of 24 cases and review of the literature. Medicine 1975;54:331–344.
[19] Verheul HA, van den Brink RBA, van Vreeland T, Moulijn AC, Duren DR, Dunning AJ. Effects of changes in management of active infective endocarditis on outcome in a 25-year period. Am J Cardiol 1993;72:682–687.
[20] Watanakunakorn C, Burkert T. Infective endocarditis at a large community teaching hospital, 1980–1990. A review of 210 episodes. Medicine 1993;72:90–102.

Appendix A. Conference discussion

Dr Lange (*Heidelberg*, *Germany*): I noted that you have a very high percentage of patients who were operated on in shock. Apparently those patients have a very high risk. Would you elaborate on how you define shock?

Dr d'Udekem: Shock was defined as a blood pressure persistently under 80 mmHg, even with continuous inotropic support, oliguria or anuria and signs of peripheral shut-down. Four of these patients with severe mitral insufficiency needed a preoperative insertion of an intra-aortic balloon pump.

Dr Rizzoli (*Padova*, *Italy*): I congratulate you for beautiful results. You presented us an actuarial curve of your reoperated patients, which shows an excellent survival. I would like to know if you have done separate curves for those reoperated on of natural valve endocarditis and of prosthetic valve endocarditis. This because, in our experience, late survival of patients with prosthetic valve endocarditis was much worse than late survival of those with natural valve endocarditis.

Dr d'Udekem: In the present paper, we did not look at the actuarial survival of the patients operated on for native valve endocarditis and for prosthetic valve endocarditis as separate groups. In a previous publication, the 5 year actuarial survival of our patients operated on for prosthetic valve endocarditis was 62%, while in the present study the 5 year actuarial survival of all patients was 76%. So, effectively, the late survival seems better for patients operated on for native compared to prosthetic valve endocarditis.

Dr Bauernschmitt (*Heidelberg*, *Germany*): Did you follow up your patients with abscesses, because it is surprising that the late occurrence of endocarditis in these patients was higher. My precise question is, did you find more late postoperative perivalvular leaks in your patients with abscesses as compared to the others?

Dr. d'Udekem: We believe that one of the main advantages of the radical execision is the extremely low incidence of post-operative paravalvular leak. Only one patient had to be reoperated on for paravalvular leak. Six years after the operation, a patch used to reconstruct the aortic root became partly calcified and at that level, occurred a small dehiscence of the valve. I have recently received the reports of all the follow-up echos of all the patients who had an abscess at the time of the operation. None of them had a paravalvular leak. This is a much lower incidence than in other series, probably because Dr T. David and Dr C. Feindel are so aggressive in their excision that all the valves implanted are either sutured to healthy tissue or to pericardial patches, leaving no space for paravalvular leak.

Dr O'Brien (*Brisbane*, *Australia*): You identified the one risk factor of abscess. Were there other factors such as a *staphylococcal* infection or previous endocarditis, influencing the development of recurrent endocarditis. If so, is your policy of management, particularly for patients with root abscesses, to continue antibiotics for some months after surgery?

Dr d'Udekem: We could not identify any risk factors for the development of recurrent endocarditis apart from the presence of an abscess at the time of the operation. None of these patients were on antibiotic therapy for longer than 6 weeks. The policy in the hospital is to give an intravenous course of antibiotics for 6 weeks for all patients having active, infective endocarditis. When they had quite a lot of antibiotics preoperatively and the valve is found to be sterile, only four weeks of intravenous antibiotics are then given, but no continuous antibiotic treatment.

Dr O'Brien: We would recommend that for some 3 months when an extensive abscess is present. Thank you.

Dr Turina (*Zurich*, *Switzerland*): You have stressed—very commendably—the total excision of the infected tissue. Can you tell us in some detail how do you propose to resect the upper ventricular septum below the right coronary artery, underneath the membranous septum? In my opinion, this just cannot be done; when there is a destruction in this position, it can only be covered with a tissue patch. Every larger series of endocarditis has some patients with septal rupture due to infection. In our own material the incidence is between 1–2%.

Dr d'Udekem: As you can see, I am not a senior surgeon so I cannot give you as many good details as Dr T. David can but I have reviewed all the OR notes of Dr David, and he really does en bloc

excision. Sometimes even the right coronary ostia is taken down and a bypass graft is done when the right coronary or the left coronary ostia is found to be infected. When the IV septum is involved, and sometimes even the infection is going into the tricuspid annulus and the tricuspid valve has to be resected, then you need more than one patch. When it is just for the mitral and the aortic annuli, usually it can be reconstructed with a single patch for the LV and one for the roof of the left atrium. When the IV septum and RV are involved, usually a third patch is necessary to repair the right ventricle and/or right atrium.