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Early Surgery for Endocarditis Complicated by Preoperative Cerebral Emboli Is Not Associated With Worsened Outcomes

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Abstract

Background—Valve surgery for patients presenting with infective endocarditis (IE) complicated by stroke is thought to carry elevated risk of postoperative complications. Our aim is to compare outcomes of IE patients who undergo surgery early after diagnosis of septic cerebral emboli with patients without preoperative emboli.

Methods—All patients undergoing surgery for left-sided IE between 1996–2013 at our institution were reviewed. Patients undergoing surgery > 14 days after embolic stroke diagnosis (n=11) and those with purely hemorrhagic lesions were excluded from analysis (n=7). In total, 308 were included in the study and stratified according to the presence (STR, n=54) or absence of a preoperative septic cerebral embolus (NoSTR, n=254). Primary outcomes of interest were development of new postoperative stroke and 30-day mortality.

Results—Mean time to surgical intervention from stroke onset was 6.0 ± 4.1 days. *S. aureus* (39% STR vs. 21% NoSTR, $p = 0.004$) and annular abscess at surgery (52% STR vs. 27% NoSTR, $p < 0.001$) were more prevalent in STR patients. There was no significant difference in 30-day mortality (9.3% STR vs. 7.1% NoSTR, $p = 0.57$) or rate of new postoperative stroke [5 (9.4%) STR vs. 12 (4.7%) NoSTR, $p = 0.19$] between groups. Additionally, there was no difference in 10-year survival between groups (log rank $p = 0.74$).

Conclusions—Early surgical intervention in patients with IE complicated by preoperative septic cerebral emboli does not lead to significantly worse postoperative outcomes. Early surgery for IE following embolic stroke warrants consideration, particularly in patients with high-risk features such as *S. aureus* and/or annular abscess.

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Keywords

Endocarditis; Heart Valves; Stroke; Surgery (complications)

INTRODUCTION

Septic cerebral embolic events frequently occur in left-sided infective endocarditis (IE) with a reported incidence of clinically significant lesions in approximately 20–40% of patients (1–2). These lesions place patients at increased risk of morbidity and mortality compared with their counterparts who have not suffered an embolic event (3–4). While guidelines recommend brain imaging when cerebral emboli are suspected, some studies suggest that asymptomatic or “silent” emboli may affect up to 50–60% of patients. Therefore, the majority of patients presenting with IE may have an embolic cerebral lesion, whether it be clinical or subclinical (5–8). Such lesions have the potential to undergo hemorrhagic transformation due to what is believed to be ischemic breakdown of the blood-brain barrier, and this transformation may be a fatal occurrence in the setting of intraoperative anticoagulation for cardiopulmonary bypass (9–11).

The traditional, conservative perspective is that surgery should be postponed for at least 4 weeks following diagnosis of a septic brain embolus in order to allow for healing of the peri-infarct tissue and restoration of the blood-brain barrier. However, postoperative degeneration of preoperatively identified cerebral lesions has been found to be a fairly rare event (1, 12–13). The combination of a low postoperative incidence of hemorrhagic stroke and the persistent risk of further embolization in medically treated endocarditis creates a strong argument to consider early surgical therapy. Therefore, the aim of our study was to evaluate perioperative and late outcomes of patients undergoing early valvular surgery for left-sided IE complicated by cerebral emboli compared to patients who present without evidence of cerebral emboli.

PATIENTS AND METHODS**Patient Selection**

All patients undergoing valvular surgery for left-sided endocarditis at our center from 1996 to 2013 (n = 326) were retrospectively reviewed. Patient charts and radiologic studies were reviewed for the presence or absence of an acute or early subacute preoperative endocarditis-related cerebral embolic event in patients with an appropriate clinical description (eg. new onset focal neurologic deficit) and/or imaging in the presence of confirmed endocarditis. The date of either the onset of neurologic symptoms (symptomatic event) or new lesions on CT or MRI (asymptomatic event) was recorded in order to determine time from event to surgery. Patients who presented with a purely hemorrhagic stroke (n=7) and those undergoing surgery more than 14 days after the onset of embolic stroke (n=11) were excluded from the analysis. Remaining patients were stratified according to their preoperative neurologic status: patients with no evidence of preoperative acute stroke (Group NoSTR, n=254) versus those with evidence of septic cerebral emboli (Group STR, n=54) undergoing surgery within 14 days of diagnosis of stroke. The study was

approved by the Columbia University Institutional Review Board and need for informed consent was waived.

Clinical and follow-up data were collected from the electronic medical record. Mortality data for patients lost to follow-up was collected from the Social Security Death Index. Baseline demographics, co-morbidities, culture results, echocardiographic data, surgical indications, presenting symptoms, cerebral lesion characteristics, operative details, concomitant procedures, postoperative complications, length of stay, and survival data were collected for analysis.

Cerebral Embolic Events

Symptomatic preoperative stroke was defined as the persistence of symptoms > 24 hours and the presence of an acute or subacute cerebral infarction on neuroimaging as determined by a neuroradiologist. Asymptomatic preoperative strokes were characterized by the presence of acute or subacute cerebral infarct on neuroimaging studies without any noticeable neurologic symptoms. A new postoperative stroke was defined as new, focal, reproducible neurologic symptoms lasting > 24 hours with or without the presence of a cerebral lesion on non-contrast head CT or MRI, as determined by a neurologist.

Endocarditis Diagnosis and Therapy

All patients were diagnosed with IE according to the modified Duke's criteria (14). Following diagnosis, blood cultures were drawn and broad spectrum antibiotics were instituted. Changes in antibiotic regimen were made according to the causative organism and antibiotic sensitivities. Length of antibiotic therapy duration was determined in consultation with the infectious disease service with the most common regimen being a total treatment period of 4–6 weeks. Postoperative antibiotic therapy was continued after surgery except in patients who underwent surgery following the prescribed long-term antibiotic course and with negative intraoperative cultures.

Operative Methods

Indications for surgical intervention included new severe valvular regurgitation, congestive heart failure, large vegetation (> 10 mm), abscess formation, persistent positive blood cultures or emboli despite appropriate antibiotic therapy, prosthetic valve dehiscence, or the presence of highly resistant organisms. In cases of mitral valve involvement, valve repair was attempted if feasible and left to the discretion of the operating surgeon. Valve replacement was accomplished with a bioprosthetic valve, mechanical valve, homograft, or pulmonary autograft, and prosthesis selection was also left to the discretion of the surgeon. In cases with involvement of the tricuspid valve, repair or replacement was performed concomitantly. During surgery, radical debridement of infected tissues was performed and the operative field was sterilized with glutaraldehyde.

Statistical Analysis

Analysis was conducted using SPSS version 22 (IBM corporation, Armonk, NY). Continuous variables are presented as mean $\pm$ standard deviation and compared using independent samples t-tests or median and interquartile range and compared using Mann-

Whitney U test depending on distribution of the data. Categorical variables are presented as total count and percentage of the group and compared using Pearson's chi-square test or Fisher's exact test where applicable. Kaplan-Meier analysis was used for comparison of 10-year survival, and survival curves were compared using the log-rank test. All *p*-values 0.05 were considered statistically significant.

RESULTS

Patient Characteristics

A total of 308 patients met criteria for inclusion into the study. Baseline characteristics and co-morbidities are presented in Table 1. Mean age for the study was 58.8 ± 15.7 years (range 18–93 years) and 201 (65%) were male with no intergroup differences. Patients in the STR group had a significantly higher left ventricular ejection fraction than NoSTR ($p < 0.001$). There were no differences in the rates of preoperative co-morbidities, including end-stage renal disease. In total, 127 (41%) underwent reoperative surgery, with 73 (23%) having undergone prosthetic valve replacement within 5 years prior to presentation. There was no significant correlation between a history of a cerebrovascular accident/transient ischemic attack and the development of a preoperative embolic stroke.

Endocarditis Presenting Characteristics

Specific endocarditis characteristics including causative organism are presented in Table 2. In total, the aortic valve was affected in 122 (40%) patients, mitral valve in 105 (34%) patients, and both the aortic and mitral valve in 81 (26%) patients with no differences according to preoperative embolic stroke status. The tricuspid valve was concomitantly affected in 26 (8%) of patients. Prosthetic valve endocarditis was present in 111 (36%) of patients with no difference between study groups.

There were significantly more cases of *S. aureus* endocarditis in the STR group (38.9% STR vs. 20.5% NoSTR, $p = 0.004$) and significantly more culture negative endocarditis in the NoSTR group (20.5% NoSTR vs. 5.6% STR, $p = 0.009$). There were no other differences in causative organism between groups. Significantly more patients presented with fever or leukocytosis $> 10,000$ cells/microliter in the STR group, and there was a trend towards more immunologic phenomenon (Osler's nodes, Janeway lesions, Roth spots, and glomerulonephritis) in the STR group ($p = 0.08$).

Stroke Variables

Cerebral lesion characteristics are presented in Table 3. The majority of embolic lesions were present bilaterally (61%). Presenting neurologic symptoms were focal in 39% of cases (hemiparesis, aphasia, facial droop, diplopia, and vertigo), non-focal in 32% (altered mental status, headache, seizure), and asymptomatic in 22%. There was evidence of microhemorrhagic conversion in 15% of patients as seen on gradient echo series of preoperative MRI. Patients in the STR group underwent surgery a mean of 6.0 ± 4.1 days after diagnosis of the cerebral embolic event. Of all 308 patients included in the study, 117 (38%) underwent preoperative CT or MRI; all patients in the STR group underwent preoperative cerebral imaging. Of patients in NoSTR who underwent preoperative

neuroimaging (n=63) for generalized alterations in mental status, evidence of non-cerebral systemic emboli, or as a screening tool, 4 patients (6.3%) suffered a postoperative stroke compared to 8 postoperative strokes (4.2%) in the remaining patients in NoSTR who had no preoperative neuroimaging ($p = 0.50$).

Operative Indications and Details

Surgical indications and operative details are listed in Table 2 and Table 4. There were significantly more patients in NoSTR that underwent surgery for severe valvular regurgitation ($p < 0.001$), while patients in STR more commonly underwent surgery for persistent bacteremia ($p < 0.001$) or the presence of annular abscess ($p < 0.001$). Many patients had more than one indication for surgery as presented in the table.

In total, there were 129 aortic valve replacements (41.9%), 92 mitral valve replacements (29.9%), 51 aortic and mitral valve replacements (16.6%), 18 aortic valve replacement and mitral valve repairs (5.8%), and 18 mitral valve repairs (5.8%). There were significantly more mitral valve replacements in the STR group compared to NoSTR ($p = 0.02$). The majority of patients underwent bioprosthetic valve implant, and there were no intergroup differences in concomitant procedures performed. Additionally, there was no significant difference in cardiopulmonary bypass time or aortic cross-clamp time between groups.

Postoperative Complications and Survival

Perioperative survival and postoperative complications are shown in Figure 1. One patient from the STR group died in the operating room and was not included in the calculation of postoperative complication rates. Overall, 5 patients in STR (9.4%) and 12 patients in NoSTR (4.7%) developed a new postoperative stroke ($p = 0.19$) at a mean of 4.9 ± 4.9 days post surgery (range 1–21 days). Of those strokes, hemorrhagic lesions were confirmed in 1 STR patient and 2 NoSTR patients. Of the 5 new strokes in the STR group, 4 patients had new radiographic brain lesions that were thought to be responsible for their symptomatology, and the fifth had a hemorrhagic conversion of his previously noted embolic lesion. There were no significant differences in the rates of postoperative atrial fibrillation, respiratory failure, new need for hemodialysis, re-operation for bleeding, new permanent pacemaker, unplanned procedures, deep sternal wound infection, or need for postoperative mechanical circulatory support between groups. Median postoperative length of stay was significantly longer in the STR group than in the NoSTR group (15 days (IQR 10–28 days) STR vs. 13 days (IQR 8–21 days) NoSTR, $p = 0.03$). Thirty-day mortality was 7.1% in NoSTR and 9.3% in STR patients ($p = 0.57$). Kaplan-Meier survival analysis is presented in Figure 2. Long-term survival up to 10 years showed no differences between groups (log rank $p = 0.74$).

COMMENT

Septic brain emboli frequently complicate IE leading to neurologic symptoms in up to 40% of patients and asymptomatic cerebral ischemic lesions in an additional 20–40% of patients (1, 2, 7, 8). Morbidity and mortality remain elevated in this high-risk subset of the IE population in comparison to patients without cerebral emboli, and few therapeutic

interventions exist to decrease the rate of these complications (3–4). For patients who require valvular surgery, the traditional approach suggests that surgery should be delayed approximately 4 weeks if septic emboli are present in order to decrease the risk of catastrophic hemorrhagic conversion following anticoagulation for cardiopulmonary bypass (5). However, given that the actual rate of fatal postoperative brain hemorrhage is low, it is possible that early surgical intervention may actually: 1) limit mortality that might occur during the delay prior to surgery in patients with severe cases of IE, and 2) prevent patients with already existing embolic lesions from suffering further embolic events, which may, therefore, limit permanent disability.

In this study, we compared postoperative outcomes of patients with IE complicated by preoperative septic emboli who underwent early (< 14 days) surgery with patients undergoing surgery for IE without any known neurologic complications. We found no significant difference in the rate of any postoperative stroke, 30-day mortality, or long-term survival between groups. We identified 3 postoperative brain hemorrhages out of the entire study population, 2 of which occurred in patients who did not have preoperative septic emboli. Additionally, we found that patients presenting with IE complicated by stroke were more likely to have *S. aureus* as their causative organism and more likely to present with an annular abscess, suggesting that these are both risk factors for cerebral embolic events. Finally, although we were unable to identify any difference in postoperative stroke rate or perioperative survival, patients with preoperative strokes had a significantly longer postoperative length of stay, which suggests higher healthcare resource utilization in this group compared to patients without a preoperative stroke.

Several prior studies have addressed the timing of surgery in IE complicated by cerebral emboli. The two largest studies evaluating patients based on time of surgery after diagnosis of cerebral infarct concluded that the rate of neurologic deterioration following surgery falls well below 5% after 4 weeks of delay, but remains approximately 20–50% in the first two weeks following the infarct and, therefore, concluded that surgery should be delayed if possible (15, 16). However, several other studies have reported deterioration rates far lower, in the range of 6–9%, which approach the 5% postoperative stroke rate seen in our series in patients without a preoperative stroke (3, 8, 13, 17). Although it is unclear why there was such a large discrepancy in the reported rates of neurologic deterioration between these studies, it should be noted that the two large studies with high early rates of neurologic deterioration relied on data compiled either from other studies or questionnaire data from several centers, suggesting that the discrepancy may be due to a difference in definition of neurologic deterioration between centers. In addition, these studies likely suffered from selection bias since patients who died while awaiting surgery were almost certainly not captured. Such issues make it difficult to make concrete conclusions regarding optimal timing of valve surgery following stroke.

Yoshioka et al recently examined this issue in detail and came to conclusions similar to ours (8). They suggest that the hemorrhagic conversion rate is low (2.9%) in patients undergoing early surgery after stroke, but that ectopic intracranial hemorrhage (i.e. hemorrhage that occurs independent of preoperative embolic infarct) may occur and is independent of the timing of surgery. This group's observations suggest that postoperative lesions that were the

result of prior infarct progression should be distinguished from lesions which are simply the result of the inherent risk of surgery for endocarditis. Such an observation, combined with the fact that approximately one-third of patients with preoperative cerebral emboli are asymptomatic, may lead one to conclude that preoperative cerebral imaging should be performed in *all* patients undergoing surgery for endocarditis. Similar to Yoshioka's observations, our study suggests that the majority of postoperative cerebral lesions are not due to progression of pre-existing lesions.

Valve surgery for endocarditis complicated by septic brain lesions should: 1) address the anatomical or hemodynamic indication for surgery, and 2) eliminate the risk of further septic emboli by removal of the embolic source without placing the patient at an unnecessarily high risk of postoperative morbidity or mortality. In a study by Kim et al. on the impact of early surgery on embolic events in IE, the authors demonstrated that patients who undergo early surgery suffered significantly fewer embolic events than patients who underwent delayed surgery (18). Although stroke patients were excluded from the early surgery group, the overall conclusion of the study remains applicable to IE patients with embolic stroke undergoing surgery: early removal of the embolic source prevents further, potentially devastating, embolic events. Thus, assuming that early surgery can be performed safely with a low rate of postoperative events -- as our group and others have shown -- this population of patients may benefit from earlier surgery in order to prevent further neurologic events.

There were several limitations to this study. First, the low sample size in both groups as well as low stroke rate postoperatively require that further studies be performed to validate the current study findings. The study was retrospective in nature and likely suffers from the institutional bias of our cardiologists, surgeons, and infectious disease specialists. Only 38% of the total cohort underwent preoperative neuroimaging at our institution. Given that the incidence of symptomatic and asymptomatic cerebral emboli has been shown to be well above 50% in the literature, it is possible that some patients who were grouped in the NoSTR group in fact had *asymptomatic* cerebral emboli. For patients who had asymptomatic preoperative embolic events documented by neuroimaging, the duration of time from onset to surgery was difficult to determine given the absence of symptoms. We attempted to address this issue by using time from discovery (i.e. neuroimaging) to surgery as a surrogate measure. However, it is possible these lesions were present for a longer period of time and, therefore, those patients have been inappropriately grouped into the STR group undergoing early surgery. In the postoperative period, we identified patients with clinical evidence of new strokes and did not perform postoperative neuroimaging in all patients. Therefore patients with new, asymptomatic cerebral lesions may have been missed.

In conclusion, we found no significant differences in new postoperative stroke rate, perioperative survival, or long-term survival between patients undergoing early valve surgery for IE complicated by cerebral emboli and those patients without preoperative cerebral complications. Our findings suggest that most patients can safely undergo valve surgery for IE in the early period following embolic stroke onset with a low risk of significant neurological deterioration in the postoperative period. Larger, multicenter studies are required in order to more definitively determine if early surgery is safe in IE patients with preoperative stroke.

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Figure 1.

Perioperative survival and postoperative complications stratified by the presence of preoperative embolic stroke (Abbreviations: DSWI=deep sternal wound infection, MCS=mechanical circulatory support, PPM=permanent pacemaker)

Figure 2.
Kaplan-Meier 10-year survival analysis stratified by study group

Table 1

Baseline patient characteristics and co-morbidities

	NoSTR	STR	p-value
<i>Demographics</i>			
Total, n	254	54	---
Age, years (mean $\pm$ SD)	59.2 $\pm$ 15.6	56.8 $\pm$ 16.6	0.32
Male, n (%)	168 (66.1)	33 (61.1)	0.48
BMI, kg/m ² (mean $\pm$ SD)	27.3 $\pm$ 5.8	26.6 $\pm$ 6.2	0.46
LVEF, % (mean $\pm$ SD)	49.7 $\pm$ 11.1	54.7 $\pm$ 8.2	0.001
<i>Co-morbidities, n (%)</i>			
Myocardial infarction	18 (7.1)	1 (1.9)	0.22
Coronary artery disease	66 (26.0)	9 (16.7)	0.15
Hypertension	138 (54.3)	24 (44.4)	0.19
Atrial Fibrillation	42 (16.5)	5 (9.3)	0.18
Stroke/TIA	29 (11.4)	4 (7.4)	0.39
Hemodialysis	25 (9.8)	5 (9.3)	0.90
COPD	10 (3.9)	1 (1.9)	0.70
Diabetes	54 (21.3)	9 (16.7)	0.45
Prior IV drug use	11 (4.3)	5 (9.3)	0.17
Prior cardiac surgery	109 (42.9)	18 (33.3)	0.19
Valve Replacement w/in 5 years	59 (23.2)	14 (25.9)	0.67

Abbreviations: BMI=body mass index, COPD=chronic obstructive pulmonary disease, LVEF=left ventricular ejection fraction, TIA=transient ischemic attack

Table 2**Endocarditis Characteristics**

	NoSTR	STR	p-value
<i>Affected Valve, n (%)</i>			
Aortic	104 (40.9)	18 (33.3)	0.30
Mitral	82 (32.3)	23 (42.6)	0.15
Aortic + Mitral	68 (26.8)	13 (24.1)	0.68
Tricuspid involvement	21 (8.3)	5 (9.3)	0.79
Pulmonary involvement	1 (0.4)	0 (0)	1.00
Prosthetic valve infection	92 (36.2)	19 (35.2)	0.89
<i>Causative Organism, n (%)</i>			
<i>S. aureus</i>	52 (20.5)	21 (38.9)	0.004
Coagulase-negative staphylococci	41 (16.1)	13 (24.1)	0.16
Viridans streptococci	25 (9.8)	3 (5.6)	0.44
Enterococcus species	38 (15.0)	6 (11.1)	0.46
HACEK organism	2 (0.8)	0 (0)	1.00
Candida species	8 (3.1)	0 (0)	0.36
Culture negative endocarditis	52 (20.5)	3 (5.6)	0.009
<i>Presenting Characteristics, n (%)</i>			
Fever	140 (55.1)	39 (72.2)	0.02
Angina	42 (16.5)	6 (11.1)	0.31
WBC count > 10,000/microliter	102 (40.2)	30 (55.6)	0.04
Immunologic phenomenon	44 (17.3)	15 (27.8)	0.08
Septic shock	40 (15.7)	13 (24.1)	0.14
<i>Indication for Surgery, n (%)</i>			
Highly resistant organism	31 (12.2)	10 (18.5)	0.22
Severe valvular regurgitation	96 (37.8)	6 (11.1)	<0.001
Congestive heart failure	38 (15.0)	4 (7.4)	0.14
Persistent bacteremia	44 (17.3)	23 (42.6)	<0.001
Dehiscence of prosthetic valve	24 (9.4)	2 (3.7)	0.28
Vegetation > 10mm	98 (38.6)	27 (50.0)	0.12
Annular abscess	68 (26.8)	28 (51.9)	<0.001

Abbreviations: WBC=white blood cell

Table 3**Cerebral Embolic Characteristics**

<i>Embotic Location, n (%)</i>	
Right	8 (14.8)
Left	9 (16.6)
Bilateral	33 (61.1)
Unknown	4 (7.4)
<i>Presenting Symptoms, n (%)</i>	
Focal	21 (38.9)
Non-focal	17 (31.5)
Asymptomatic	12 (22.2)
Unknown	4 (7.4)
<i>Lesion Details</i>	
Maximum diameter, mm (mean $\pm$ SD)	17.1 $\pm$ 9.9
Evidence of microrhemorrhage, n (%)	8 (14.8)
Mycotic aneurysm present, n (%)	2 (3.7)
1 lesion, n (%)	8 (14.8)
2–3 lesions, n (%)	18 (33.3)
4–5 lesions, n (%)	14 (25.9)
>5 lesions, n (%)	14 (25.9)
<i>Days from stroke to surgery (mean $\pm$ SD)</i>	6.0 $\pm$ 4.1

Table 4**Operative Details**

	NoSTR	STR	p-value
<i>Cardiopulmonary Bypass Details (mean ± SD)</i>			
CPB time, minutes	151 ± 67	154 ± 76	0.84
XCL time, minutes	107 ± 46	112 ± 56	0.51
<i>Procedure, n (%)</i>			
AVR	110 (43.3)	19 (35.2)	0.27
MVR	69 (27.2)	23 (42.6)	0.02
AVR+MVR	43 (16.9)	8 (14.8)	0.70
AVR+MVr	16 (6.3)	2 (3.7)	0.75
MVr	16 (6.3)	2 (3.7)	0.75
Any TVR/r	24 (9.4)	6 (11.1)	0.71
Any PVR	1 (0.4)	0 (0)	1.00
<i>Prosthesis Type, n (%)</i>			
Bioprosthesis	157 (61.8)	37 (68.5)	0.35
Mechanical	64 (25.2)	11 (20.4)	0.45
Homograft	17 (6.7)	4 (7.4)	0.77
Annuloplasty ring only	13 (5.1)	1 (1.9)	0.48
Vegetomy only	1 (0.4)	1 (1.9)	0.32
Pulmonary autograft	2 (0.8)	0 (0)	1.00
<i>Concomitant Procedures, n (%)</i>			
CABG	24 (9.4)	6 (11.1)	0.71
Aortic root replacement	18 (7.1)	4 (7.4)	1.00
VSD repair	2 (0.8)	0 (0)	1.00
Septal myomectomy	1 (0.4)	0 (0)	1.00

Abbreviations: AVR=aortic valve replacement, CABG=coronary artery bypass grafting, CPB=cardiopulmonary bypass, MVR=mitral valve replacement, MVr=mitral valve repair, PVR=pulmonary valve replacement, TVR/r=tricuspid valve replacement/repair, VSD=ventricular septal defect, XCL=aortic cross-clamp