to confirm the safety and to establish the most appropriate timing for this double treatment strategy in this challenging group of patients who combine significant aortic and mitral valve disease.

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SUPPLEMENTARY MATERIAL

Supplementary material associated with this article can be found in the online version available at http://dx.doi.org/10.1016/j.rec.2017.05.001

David del Val,[a,b] Ander Regueiro,[a] Tania Rodríguez-Gabella,[a] Jean Michel Paradis,[a] Rishi Puri,[a] and Josep Rodés-Cabau[a,*]

[a]*Cardiology Department, Quebec Heart & Lung Institute, Laval University, Quebec City, Quebec, Canada*

[b]*Servicio de Cardiología, Hospital Universitario Ramón y Cajal, Madrid, Spain*

*Corresponding author:
E-mail address: josep.rodes@criucpq.ulaval.ca (J. Rodés-Cabau).

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Paravalvular Leak Correction: Searching for a Balance Between Surgical and Percutaneous Techniques

Corrección de fugas paravalvulares: buscando el equilibrio entre las técnicas quirúrgicas y percutáneas

To the Editor,

Paravalvular leak (PVL) is a complication after valve replacement surgery, with an incidence ranging from 2% to 10% in the aortic position and from 7% to 17% in the mitral position.[1] Although most cases have a benign course, 1% to 5% of linked might be linked to serious clinical consequences such as heart failure or hemolytic anemia.[1,2] Mortality following re-do surgery has been reported to be high (10%-15%) and rises with the number of previous surgeries.[2] Percutaneous treatment of PVL has emerged as a promising alternative to surgery,[3] although data comparing the results of surgical vs percutaneous PVL correction are scarce. The purpose of the present study was to describe the outcomes of surgical and percutaneous PVL correction in a contemporary series of patients.

Between January 2006 and December 2015, all patients undergoing isolated PVL through either surgery or the percutaneous approach at our institution were analyzed. The selection of percutaneous or surgical treatment was at the discretion of the treating medical team. However, percutaneous PVL correction became available in 2012 and was performed by experienced operators. Since then, all technically feasible PVLs were initially approached by percutaneous techniques after a Heart Team discussion. To avoid bias, corrective procedures other than isolated PVL, such as combined interventions with coronary revascularization or adjunctive treatment of another cardiac valve, were excluded from the analysis. Patients with active endocarditis were excluded. All patients gave informed consent before the intervention.

A total of 50 patients (32 percutaneous and 18 surgical) underwent isolated PVL correction and were therefore included in the study (Table). Procedural success was achieved in 94% and 87% of the surgical and percutaneous patients, respectively. Major adverse events and in-hospital mortality were balanced between groups and patients undergoing percutaneous correction had shorter admission periods. At 1-year of follow-up, no significant differences between groups were found in all-cause mortality, hospital readmissions for PVL symptoms, and reintervention (Table).

The main findings of the present study were: *a)* both percutaneous and surgical PVL correction techniques were associated with a high rate of procedural success (> 85%) with a trend toward more complete sealing in patients undergoing surgery; *b)* in-hospital major adverse events were comparable between groups; *c)* patients treated with percutaneous techniques had shorter in-hospital admissions; and *d)* at 1-year of follow-up, clinical outcomes remained balanced between groups.

In our series, surgical patients showed a trend toward more complete PVL sealing but procedural success with percutaneous techniques was still high and similar to that achieved with surgery. These results are in agreement with those of previous publications reporting similar outcomes after percutaneous PVL correction.[3] Although our surgical in-hospital mortality might be considered high (11%), it reflects the high surgical risk of the treated population and is in agreement with previous series reporting mortalities between 6% and 22%.[1–3] However, it is important to highlight that surgical PVL repair might be the only therapeutic alternative, especially in large or multiple PVLs. Communication within the Heart Team and discussion about the technical complexity of percutaneous repair as well as the proposed

Table
Baseline, Procedural and Clinical Outcomes Comparing Percutaneous and Surgical PVL Repair

	Surgical n = 18	Percutaneous n = 32	*P*
Baseline characteristics			
Age, y	64.9 ± 10.9	69.6 ± 105	.148
Sex, male	6 (33.3%)	7 (21.8%)	.375
Diabetes mellitus	6 (33.3%)	7 (21.8%)	.375
Atrial fibrillation	10 (55.5%)	16 (50%)	.706
Coronary artery disease	2 (11.1%)	7 (21.8%)	.342
Creatinine, mg/dL	1.4 ± 0.8	1.1 ± 0.4	.089
EuroSCORE-2	15.7 ± 10.6	15.9 ± 15.1	.947
Left ventricular ejection fraction, %	55.2 ± 9.2	54.1 ± 11.8	.744
Systolic pulmonary artery pressure, mmHg	65.9 ± 20.7	49.9 ± 18.6	.021
Procedural characteristics			
Leak location			.186
Aortic	5 (28%)	15 (47%)	
Mitral	13 (72%)	17 (53%)	
Previous cardiac surgeries			.700
1	12 (67%)	23 (72%)	
2	6 (33%)	9 (28%)	
Clinical presentation			.584
Heart failure alone	11 (61%)	22 (69%)	
Hemolysis alone	0 (0%)	3 (9%)	
Heart failure + hemolysis	7 (39%)	7 (22%)	
Procedural outcomes			
Procedural success[a]	17 (94.4%)	28 (87.5%)	.432
Residual leak (echocardiography)			.268
None or mild	16 (94%)	25 (78%)	
Moderate	0 (0%)	4 (12.5%)	.149
Severe	1 (6%)	3 (9.3%)	
Complete PVL sealing[b]	16 (94%)	25 (79%)	
Procedural major adverse events	4 (16.6%)	4 (12.5%)	.684
Death	2 (11.7%)	1 (6%)	.254
Stroke	1 (5.5%)	0 (0%)	.178
Myocardial infarction	0 (0%)	0 (0%)	-
Major bleeding/vascular	1 (5.5%)	3 (6%)	.090
Device embolization	0 (0%)	0 (0%)	-
1-Year follow-up outcomes			
Hospital admission, d	16.7 ± 13.6	5.2 ± 5.1	< .001
In-hospital mortality	2 (11.7%)	1 (3.1%)	.230
Mortality at 1 y	2 (11.7%)	3 (9.3%)	.873
Readmission at 1 y	1 (6.2%)	6 (19.3%)	.232
Reintervention at 1 y	1 (6.2%)	4 (16.1%)	.336

PVL, paravalvular leak.
[a] Procedural success: surgical or percutaneous correction resulting in an immediate 1-grade regurgitation reduction. In addition, for percutaneous interventions, the device must not interfere with the movement of the prosthetic valve.
[b] Complete PVL sealing, defined as no or mild residual PVL.

surgical strategy is pivotal in these patients. In fact, this communication strategy generally tends to initially offer a percutaneous correction when technically feasible, considering the less invasive nature of the intervention. After a Heart Team discussion, a failed percutaneous PVL correction should therefore not be seen as a "decision mistake" but rather as proof that the move from a less to a more invasive strategy is justified. In addition, surgical techniques might combine additional corrective interventions other than PVL repair that might provide further benefit to the patient. Indeed, in our series, 3 patients with mitral PVLs and percutaneous repair failure underwent corrective mitral surgery and had excellent clinical outcomes. In all 3 cases, the patients and surgeons were informed about the technical complexity of PVL repair beforehand, and surgical repair was conducted during the same hospital admission.

Another relevant finding of the study was the durability of both percutaneous and surgical PVL correction as reflected by the relatively low incidence of 1-year reinterventions and hospital

readmissions. Although the relatively small number of patients is a limitation in this regard, some other series seem to confirm this finding.[3]

In conclusion, our series suggest that percutaneous treatment of isolated PVL seems to be a valid alternative to surgery. Nonetheless, surgical correction should always be considered, as it might be the only option with favorable outcomes in patients with PVLs not suitable for percutaneous repair, after failed percutaneous procedures, or for those patients in need of additional surgical interventions. Larger series will be necessary to confirm these findings.

CONFLICTS OF INTEREST

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Marco Hernández-Enríquez, Xavier Freixa,* Eduard Quintana, Daniel Pereda, Elena Sandoval, and Manel Sabaté

Instituto Clínico Cardiovascular (ICCV), Hospital Clínic de Barcelona, Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), Barcelona, Spain

* Corresponding author:
E-mail address: freixa@clinic.ub.es (X. Freixa).

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Long-term Outcome of Patients With Tachycardia-induced Cardiomyopathy After Recovery of Left Ventricular Function

Evolución a largo plazo de pacientes con taquimiocardiopatía tras la recuperación de la función ventricular

To the Editor,

Tachycardia-induced cardiomyopathy (TIC) is a heart disease characterized by ventricular dysfunction and dilatation secondary to sustained tachyarrhythmia that is reversible with heart rate control. It is diagnosed after exclusion of other causes of cardiomyopathy and recovery in left ventricular ejection fraction (LVEF) of at least 15% after heart rate control. The ventricular dysfunction generated by TIC is sometimes extremely serious, leading to heart failure, arrhythmias, and sudden death.[1] TIC is frequently associated with atrial fibrillation. Because TIC is generally considered a benign and reversible condition, it is probably underdiagnosed. However, recent studies indicate that it may cause persistent subclinical damage.[2–4] The true prognosis of the disease is unknown, as well as the mechanisms underlying its reversibility and whether it causes an irreversible subclinical condition.

The present study analyzes the baseline clinical, electrocardiographic, and cardiac imaging characteristics of patients with TIC, their long-term outcomes, and the association of these characteristics with adverse events during follow-up. The study comprises a retrospective analysis of a series of patients diagnosed with TIC and evaluated and followed up in our center between March 2006 and March 2016. Patients with other heart diseases and/or possible triggers were excluded. Clinical treatment was provided according to clinical practice guidelines and at the discretion of the treating physician. LVEF relapses (an LVEF $< 50\%$ or a reduction $\geq 15\%$) during follow-up were analyzed after their complete or partial recovery, as well as their association with prognostic factors. Delayed relapses were those that occurred from the fifth year of follow-up onward. Statistical comparisons between groups were performed using a chi-square test, the Student *t* test, and the Mann-Whitney *U* test; survival analysis was performed using a Cox regression model and Kaplan-Meier estimator. $P < .05$ was considered statistically significant.

In total, 36 patients (23 men) were evaluated with a mean follow-up of 3.2 ± 2.9 years (Table). The most frequent cause of TIC was atrial fibrillation (72%). In 70% of the patients, their symptoms were not directly attributable to their arrhythmia. Eleven LVEF reductions were detected during follow-up (30% of patients; median time from treatment initiation to relapse, 3.08 [0.32-8.03] years) due to arrhythmic relapse or poor control of the original arrhythmia; of these relapses, 5 were delayed (14%). In patients who had a relapse, there were no significant differences in LVEF at treatment initiation or after ventricular function recovery (Figure A). Nonetheless, these patients did show slower LVEF recovery from disease initiation (0.39 [0.21-0.75] vs 1.13 [0.36-4.10] years; $P = .041$) (Figure B) and their clinical follow-up was significantly longer (2.1 ± 2.0 vs 5.6 ± 3.1 years; $P = .007$). In contrast, patients treated with ablation of the triggering arrhythmia were nonsignificantly less likely to have a relapse ($P = .076$), regardless of the type of arrhythmia ablated. There were no significant differences in the relapse-free survival curves between patients with atrial fibrillation and those with other arrhythmias (Figure C). Nonetheless, Cox regression analysis showed that atrial fibrillation multiplied the relapse risk during follow-up by 2.42, although the difference was again not statistically significant (95% confidence interval, 0.29-20.4; $P = .416$). Only 1 death occurred, from noncardiovascular causes.

The present study represents the most extensive series of patients with TIC. Our data show that these patients have a significant future likelihood of relapse. These findings might be related to studies indicating residual subclinical damage in the form of interstitial fibrosis that causes relapses and/or