postdilatation. All procedures were safely performed with good results (mean gradients < 20 mmHg with no residual leaks).

Although THV durability appears favorable in the setting of native aortic valve stenosis, there are concerns about its durability in underexpanded ViV implants and the occurrence of specific procedural complications. Certain surgical valves have been identified as having a higher risk of coronary obstruction (the main mechanism being occlusion of the ostia by a dislodged leaflet after deployment of the THV). Thus, detailed preprocedural computed tomography study is crucial to identify high-risk features (such as low coronary heights, shallow sinuses of Valsalva, and short virtual THV to coronary distance).[4] Although very few reports are available, we extend this experience with 5 patients, none of whom developed coronary obstruction. Although the risk of coronary occlusion seemed low (high coronary heights, wide sinuses, and virtual THV to coronary distance > 4 mm), this type of valve may have a lower risk of coronary occlusion than other prostheses due to its implantation technique and valve design (the metallic stent has sinusoidal struts that fit Valsalva sinuses). Regarding conduction disturbances, a recent propensity-matched analysis reported lower rates of pacemaker implantation with ViV vs redo surgery for the management of failed prostheses.[5] Data on sutureless valves are lacking, but among our patients, 2 patients with Percevals with stent deformation treated with self-expandable valves presented with atrioventricular block requiring pacemaker implantation, 2 patients with Percevals S and M treated with Edwards SAPIEN 3 presented with new onset left bundle branch block that was treated conservatively, and 1 patient with Perceval XL did not have any conduction abnormalities.

One of the advantages of sutureless valves is the absence of a sewing ring, which provides larger EOAs and reduces the risk of the development of patient-prosthesis mismatch compared with other bioprostheses. As described in table 1, acceptable gradients where achieved in our patients after the procedure and at mid-term follow-up. Paravalvular leaks were not a major concern in any of the patients in our series.

To conclude: *a)* ViV transcatheter aortic valve implantation in sutureless valves was feasible and safe; *b)* challenging cases such as small degenerated valves were successfully treated with self-expandable valves and acceptable gradients, and *c)* the rate of procedural complications was low and good in-hospital and mid-term outcomes were acheived with different types of transcatheter aortic valves.

Currently, procedural or mid-term results for ViV procedures in sutureless aortic valves are lacking. However, because sutureless valves are becoming more widely used and ViV is increasing, thorough knowledge will be essential and the features described above provide insights into the safety and feasibility of these procedures.

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REFERENCES

1. Attia R, Papalexopoulou N, Hancock J, et al. Successful treatment of failing biological prosthesis due to 'stent creep' with Valve-in-Valve Transcatheter Aortic Valve Implantation. *Catheter Cardiovasc Interv.* 2015;86:119–125.
2. Meco M, Montisci A, Miceli A, et al. Sutureless Perceval Aortic Valve Versus Conventional Stented Bioprostheses: Meta-Analysis of Postoperative and Midterm Results in Isolated Aortic Valve Replacement. *J Am Heart Assoc.* 2018;7:e006091.
3. Balghith MA. Degenerated Suturless Perceval with (Paravalvular Leak and AS) Treated by Valve in Valve using S3 Edward Valve. *Heart Views.* 2019;20:166–169.
4. Bernardi FLM, Dvir D, Rodes-Cabau J, Ribeiro HB. Valve-in-Valve Challenges: How to Avoid Coronary Obstruction. *Front Cardiovasc Med.* 2019;6:120.
5. Tam DY, Dharma C, Rocha RV, et al. Transcatheter ViV Versus Redo Surgical AVR for the Management of Failed Biological Prosthesis. *JACC Cardiovasc Interv.* 2020;13:765–774.

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Changes in the microbiological etiology of infective endocarditis in our region in the last 3 decades (1987-2019)

Cambios en el espectro microbiológico causal de la endocarditis infecciosa en nuestro medio en las últimas 3 décadas (1987-2019)

To the Editor,

Infective endocarditis (IE), first described by William Osler in 1885, is a serious disease.[1] Classic forms of IE are mainly caused by *Streptococcus viridans* or *Staphylococcus aureus*.[2] Numerous sociocultural and health-related changes (eg, population aging, complex cardiac surgery, a greater use of implanted pacemakers and defibrillators, and a higher prevalence of health care-associated bacteremia)[3] have changed the face of IE in recent decades.[4] As suggested in recent studies, one of the possible consequences of these changes is a shift in the microbiological profile of IE.[4,5] The aim of this study was to analyze the causative microorganisms identified in native and prosthetic forms of IE at our hospital over a period of 33 years and to examine the changes that have taken place. We analyzed a cohort of patients with IE whose data were prospectively recorded between 1987 and 2019. The cohort included all patients diagnosed with IE during this period except persons who inject drugs. The study period was divided into 3 periods: 1987 to 1997, 1998 to 2008, and 2009 to 2019.

Our hospital is a tertiary care center with a cardiac surgery department that serves as an IE referral center for 3 regional hospitals in the province. Native IE accounted for 66.1%

Table 1
Distribution of causative microorganisms in patients with IE for the period 1987 to 2019 (overall and by type)

	Native IE (n = 338)	Early-onset prosthetic IE (n = 64)	Late-onset prosthetic IE (n = 83)	PM/ICD-associated IE (n = 27)	Total (n = 512)
Staphylococci	108 (31.9)	35 (54.7)	27 (32.5)	24 (88.9)	194 (37.9)
Staphylococcus aureus	73 (21.6)	9 (14.1)	13 (15)	15 (72.8)	110 (21.5)
Coagulase-negative staphylococci	35 (10.3)	26 (40.6)	14 (17.5)	9 (16.1)	84 (16.4)
Oral streptococci	78 (23.1)	3 (4.7)	18 (21.7)	0	99 (19.3)
Streptococcus viridans	74 (21.9)	3 (4.7)	18 (21.7)	0	95 (18.6)
Others[a]	4 (1.2)	0	0	0	4 (0.7)
Streptococcus gallolyticus (*Streptococcus bovis*)	10 (2.9)	3 (4.7)	6 (7.2)	0	19 (3.7)
Enterococci	61 (18.1)	6 (9.4)	11 (13.2)	3 (11.2)	81 (15.8)
Corynebacterium spp	7 (2.1)	3 (4.7)	2 (2.4)	0	12 (2.3)
Brucella spp	3 (0.9)	0	1 (1.2)	0	4 (0.8)
Coxiella burnetii	8 (2.3)	0	7 (8.4)	0	15 (2.9)
Fungi	7 (2.3)	3 (4.7)	1 (1.2)	0	11 (2.2)
Other microorganisms[b]	12 (3.5)	1 (1.6)	0	0	13 (2.6)
Unknown	44 (13.0)	10 (15.5)	10 (12.2)	0	64 (12.5)

ICD, implantable cardioverter defibrillator; IE, infective endocarditis; PM, pacemaker.
Values are expressed as No. (%).
[a] Other oral streptococci: *Abiotrophia* (3 cases), *Granulicatella* (1 case).
[b] Other microorganisms: *Listeria monocytogenes* (1 case), *Lactobacillus* spp. (1 case), *Propionibacterium* spp (1 case), enterobacteria (2 cases), HACEK microorganisms (*Haemophilus*, *Aggregatibacter*, *Cardiobacterium*, *Eikenella*, and *Kingella* (5 cases), and multiple pathogens (polymicrobial infection) (2 cases).

(n = 512) of all IE cases over the 33-year study period, prosthetic IE for 28.7% (12.5% early-onset and 16.1% late-onset), and pacemaker/implantable cardioverter defibrillator (PM/ICD)-associated IE for 5.2%. The mean ± SD age of the patients was 55.3 ± 17.9 years and 66.2% were men. There was a significant increase in IE cases detected in more recent years (138 in 1987-1997, 180 in 1998-2008, and 194 in 2009-2019; $P < 001$). The causative microorganisms by period are shown in table 1. The most common microorganisms (accounting for 37.9% of all causative agents over the 33 years) were staphylococci (21.5% *S aureus* and 15.8% coagulase-negative staphylococci), streptococci (23%; 19.3% oral streptococci and 3.7% *Streptococcus gallolyticus*), enterococci (16.2%), and other (11.3%). The cause was unknown in 12.5% of cases. Staphylococci were the most common causative agents in all forms of IE, but they were particularly common in early-onset prosthetic IE—where they were responsible for 54.7% of all cases (40.6% coagulase-negative staphylococci)—and ICD/PM-associated IE, where they were responsible for 88.9% of all cases (72.8% *S aureus*) (table 1). Of the 110 cases of *S aureus*, 25 (22.7%) were methicillin-resistant. This high rate of *S aureus* infection is important, as recent studies have shown that *S aureus* is an independent predictor of poor outcome in IE.[6] The microorganisms responsible for native IE and late-onset prosthetic IE were similar (table 1). Of note, a relatively high proportion of native IE and late-onset prosthetic IE cases were caused by *Coxiella burnetii* (2.3% and 8.4%, respectively).

On analyzing the data by periods, we observed that the proportion of IE cases caused by staphylococci increased from 31.2% in 1987 to 1997 to 42.1% in 2009 to 2019; there was also a significant increase in coagulase-negative staphylococci (7.3% to 22.1%, $P = .001$) and a trend toward an increase in enterococci (10.8% to 19.5%, $P = .089$). There was no change in the proportion of cases caused by *S aureus*, oral streptococci, *S gallolyticus*, or unidentified microorganisms over the years (table 2). We did, however, observe a reduction (from 22.1% to 9.3%) of cases caused by rare microorganisms such as *Brucella* spp, *C burnetii*, *Corynebacterium* spp, and fungi ($P < .001$) (table 2). There was a significant increase in native IE cases due to coagulase-negative staphylococci (4.3% in 1987-1997 to 14.3% in 2009-2019) and a significant decrease in those due to *S aureus* (29.8% to 19.8%) and rare microorganisms (17.0% to 10.2%). The proportion of other causative microorganisms did not change (table 2). In the case of early-onset prosthetic IE, we observed a notable increase in cases due to coagulase-negative staphylococci (15% to 52.2%) and a decrease in those due to *Corynebacterium* spp (15% to 0%) and unidentified microorganisms (20% to 8.7%). The proportion of cases caused by other microorganisms did not change (table 2). Finally, in the case of late-onset prosthetic IE, there was an increase in cases caused by staphylococci (*S aureus* and coagulase-negative staphylococci) and enterococci (from 0% to 21.9%) and a notable decrease (36.4% to 12.5%) in those caused by oral streptococci (table 2).

Our findings from a large series of IE cases spanning 33 years shows how the microbiological profile at our hospital has changed very significantly over time, with a notable increase in cases caused by coagulase-negative staphylococci and enterococci, practically no change in cases caused by oral streptococci, and a reduction in cases caused by rare microorganisms, such as *Brucella* spp, *C burnetii*, and Corynebacterium spp, which were all relatively common in the last century. Despite some slight differences, the changes in microbiological profile affected all types of IE. Our findings may have prognostic implications and lead to changes in the choice of empirical antibiotic therapy.

Table 2
Distribution of causative microorganisms (overall and by type) in patients with infective endocarditis for 1987 to 1997, 1998 to 2008, and 2009 to 2019

All cases of infective endocarditis	1987-1997 (n = 138)	1998-2008 (n = 180)	2009-2019 (n = 194)	P[a]
Staphylococci	43 (31.2)	71 (39.4)	80 (42.1)	.152
Staphylococcus aureus	33 (23.9)	39 (21.7)	38 (20.0)	.637
Coagulase-negative staphylococci	10 (7.3)	32 (17.7)	42 (22.1)	.001[d]
Oral streptococci	29 (16.7)	37 (20.5)	33 (17.0)	.579
Streptococcus viridans	29 (16.7)	35 (19.4)	31 (15.9)	.642
Other[b]	0	2 (1.1)	2 (1.1)	.816
Streptococcus gallolyticus (Streptococcus bovis)	6 (4.3)	6 (3.3)	7 (3.6)	.748
Enterococci	15 (10.8)	29 (16.1)	37 (19.5)	.089
Other microorganisms[c]	29 (22.1)	10 (5.4)	16 (9.3)	< .001[d]
Not identified	16 (11.6)	27 (15.0)	21 (11.1)	.486
Native infective endocarditis	1987-1997 (n = 94)	1998-2008 (n = 118)	2009-2019 (n = 126)	P[a]
Staphylococci	32 (34.1)	33 (27.9)	43 (34.1)	.364
S aureus	28 (29.8)	20 (16.9)	25 (19.8)	.048[d]
Coagulase-negative staphylococci	4 (4.3)	13 (11.0)	18 (14.3)	.049[d]
Oral streptococci	20 (21.3)	31 (26.3)	27 (21.4)	.486
S viridans	20 (21.3)	29 (24.6)	25 (19.8)	.524
Other	0	2 (1.7)	2 (1.6)	.841
S gallolyticus (S bovis)	3 (3.2)	2 (1.7)	5 (3.9)	.712
Enterococci	12 (12.7)	25 (21.2)	24 (19.0)	.267
Other microorganisms	16 (17.0)	8 (6.7)	13 (10.2)	.047[d]
Not identified	11 (12.2)	19 (16.1)	14 (11.1)	.484
Early-onset prosthetic infective endocarditis	1987-1997 (n = 20)	1998-2008 (n = 21)	2009-2019 (n = 23)	P[a]
Staphylococci	5 (25.0)	15 (68.2)	15 (65.2)	.005[d]
S aureus	2 (10.0)	4 (19.0)	3 (13.0)	.474
Coagulase-negative staphylococci	3 (15.0)	11 (49.2)	12 (52.2)	.019[d]
Oral streptococci	1 (5.0)	0	2 (8.7)	.746
S viridans	1 (5.0)	0	2 (8.7)	.746
Other	0	0	0	-
S gallolyticus (S bovis)	2 (10.0)	1 (4.7)	0	.641
Enterococci	3 (15.0)	0	3 (13.0)	.676
Other microorganisms	5 (20.0)	1 (4.7)	1 (4.3)	.095
Unknown	4 (20.0)	4 (19.0)	2 (8.7)	.520
Late-onset prosthetic infective endocarditis	1987-1997 (n = 22)	1998-2008 (n = 29)	2009-2019 (n = 32)	P[a]
Staphylococci	4 (18.2)	11 (37.9)	12 (37.5)	.083
S aureus	1 (4.5)	8 (27.6)	4 (12.5)	.088
Coagulase-negative staphylococci	3 (13.7)	3 (10.3)	8 (25.0)	.436
Oral streptococci	8 (36.4)	6 (20.7)	4 (12.5)	.110
S viridans	8 (36.4)	6 (20.7)	4 (12.5)	.110
Other	0	0	0	—
S gallolyticus (S bovis)	1 (4.5)	3 (10.3)	2 (6.2)	.369
Enterococci	0	4 (13.8)	7 (21.9)	.048[d]
Other microorganisms	8 (34.1)	1 (4.7)	2 (6.2)	< .001[d]
Unknown	1 (4.5)	4 (13.8)	5 (15.6)	.485

Values are expressed as No. (%).

[a] Statistical comparisons were made using the chi-square test or, for categories with frequencies < 5, the Fisher-Freeman-Halton test.

[b] Other oral streptococci: *Abiotrophia* (3 cases), *Granulicatella* (1 case).

[c] Other microorganisms: *Corynebacterium* spp (12 cases), *Coxiella burnetii* (15 cases), *Brucella* spp (4 cases), fungi (11 cases), *Listeria monocytogenes* (1 case), *Lactobacillus* spp (1 case), *Propionibacterium* spp (1 case), enterobacteria (2 cases); HACEK microorganisms (*Haemophilus*, *Aggregatibacter*, *Cardiobacterium*, *Eikenella*, and *Kingella* (5 cases), and multiple pathogens (polymicrobial infection) (2 cases).

[d] Statistically significant *P* values.

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REFERENCES

1. Pruitt RD. William Osler and his Gulstonian Lectures on Malignant Endocarditis. *Mayo Clin Proc.* 1982;57:4–9.
2. Castillo JC, Anguita MP, Ruiz M, et al. Changing epidemiology of native valve infective endocarditis. *Rev Esp Cardiol.* 2011;64:594–598.
3. Fernández-Hidalgo N, Tornos P. Epidemiología de la endocarditis infecciosa en España en los últimos 20 años. *Rev Esp Cardiol.* 2013;66:728–733.
4. Olmos C, Vilacosta I, Fernández-Pérez C, et al. The evolving nature of infective endocarditis in Spain. A population-based study (2003 to 2014). *J Am Coll Cardiol.* 2017;70:2795–2804.
5. Habib G, Erba PA, Iung B, et al. Clinical presentation, aetiology, and outcome of infective endocarditis. Results of the ESC-EORP EURO-ENDO (European infective endocarditis) registry: a prospective cohort study. *Eur Heart J.* 2019;40:32–33.
6. García-Granja PE, López J, Vilacosta I, et al. Predictive model of in-hospital mortality in left-sided infective endocarditis. *Rev Esp Cardiol.* 2020;73:902–909.

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Contemporary management of postcardiotomy cardiogenic shock: results of a specialized care team

Abordaje contemporáneo del shock *cardiogénico tras la cardiotomía: resultados desde la instauración de una unidad de atención especializada*

To the Editor,

Postcardiotomy cardiogenic shock (PCCS) continues to be linked to high morbidity and mortality.[1] Despite advances in the development of biotechnological resources, mortality figures have not shown a clear improvement during the last decade.[1] Furthermore, survival rates of PCCS continue to be significantly lower than those observed in other types of cardiogenic shock (CS).[2] This could potentially change with the implementation of dedicated structures specifically designed for CS treatment.[3]

We performed an observational analysis of a series of adult patients with PCCS treated after the establishment of an organized interdisciplinary shock-team. All consecutive patients were prospectively included, whether from our own center or referred to from other hospitals. Clinical follow-up covered a time period from September 2014 through to June 2019.

Bivariate analysis was performed of factors associated with in-hospital mortality. The Mann-Whitney test was used for numeric variables, and the chi-square test for categorical variables. Actuarial survival analysis used Kaplan-Meier curves and the log-rank test for comparison. The baseline shock variables used were those taken on admission in our intensive care unit (ICU). A value of $P < .05$ was considered statistically significant. The program used for the analysis was STATA IC/15.

The most representative results are displayed in table 1. A total of 32 PCCS patients were analyzed. Twenty-six cases (81%) occurred in our hospital, while 6 (19%) were referred from other institutions. In 31 patients (97%), a temporary mechanical circulatory support (TMCS) was used. Extracorporeal membrane oxygenation (ECMO) was chosen in 24 (75%), with central cannulation in 20 patients (83% of ECMOs used). The TMCS was implanted during the surgery itself in 68% of the patients, and on the same day in 87%. The median [range] time on circulatory mechanical support was 6 [5-14] days.

Weaning from TMCS was achieved in 24 patients (77%). In 19 patients (61%), weaning followed myocardial function

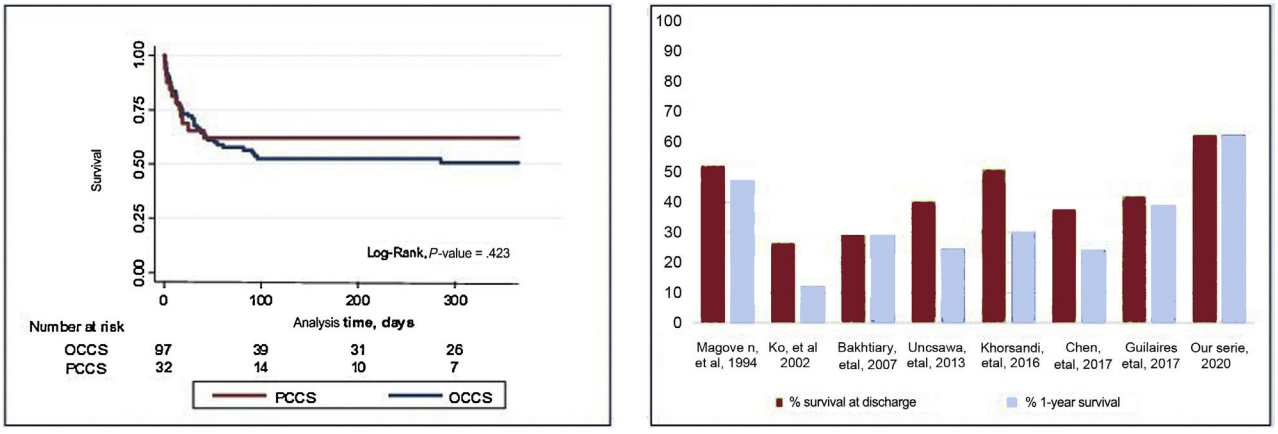

Figure 1. A: Kaplan-Meier analysis for 1-year survival estimates. Differences between postcardiotomy cardiogenic shock and other causes of cardiogenic shock in our series. B: comparison of survival at discharge and at 1 year in the main series collected recently by Lorusso et al., [1] including the results of our series. OCCS, other causes of cardiogenic shock; PCCS, postcardiotomy cardiogenic shock.