Paula Anguita,[a,b] Juan C. Castillo,[a] José López-Aguilera,[a] Manuela Herrera,[b] Manuel Pan,[a] and Manuel Anguita[a,*]

[a]*Servicio de Cardiología, Hospital Universitario Reina Sofía, Córdoba, Spain*
[b]*Facultad de Odontología, Universidad de Sevilla, Sevilla, Spain*

*Corresponding author:
E-mail address: manuelanguita@secardiologia.es (M. Anguita).

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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

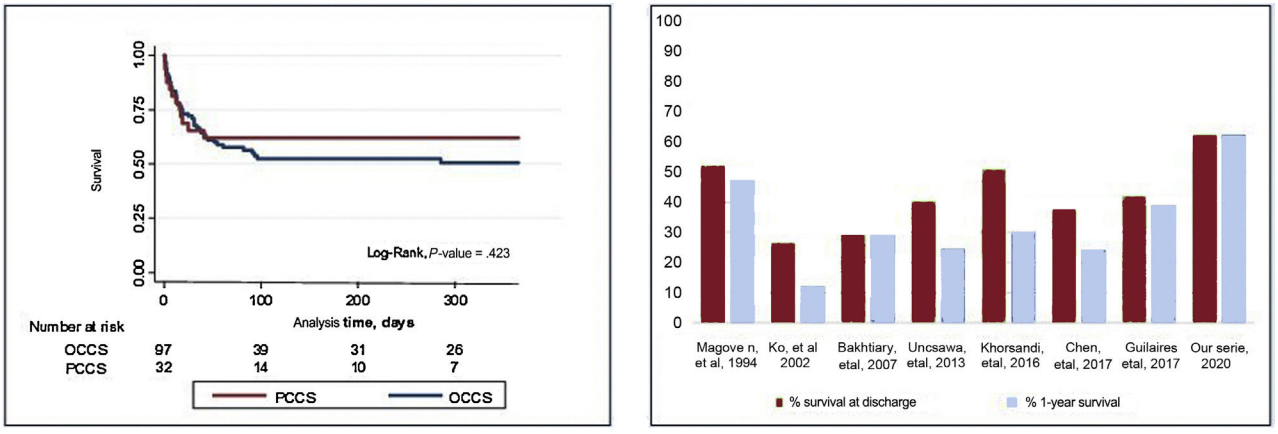

Figure 1. A: Kaplan-Meier analysis for 1-year survival estimates. Differences between postcardiotomy cardiogenic shock and other causes of cardiogenic shock in our series. B: comparison of survival at discharge and at 1 year in the main series collected recently by Lorusso et al., [1] including the results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

Table 1
Demographic features, clinical management, complications, outcome and destination in patients with postcardiotomy cardiogenic shock

Variable	Global PCCS n = 32	Group A PCCS[a] survivors n = 20	Group B PCCS[a] nonsurvivors n = 12	P
Demographic				
Age, y	59 ± 17	55 ± 18	65 ± 12	.118
Male sex	21 (66)	13 (65)	8 (67)	.923
BMI, kg/m^2	28 ± 8	28 ± 9	29 ± 4	.103
Clinical history				
Hypertension	17 (53)	10 (50)	7 (58)	.647
Diabetes	7 (22)	1 (5)	6 (50)	.003
History of stroke	6 (19)	1 (5)	5 (42)	.010
Cardiac surgery in our center	26 (81)	15 (75)	11 (92)	.242
Emergency surgery	6 (19)	4 (20)	2 (17)	.815
Type of surgery				.088
CABG	2 (6)	0 (0)	2 (17)	
Valve surgery	16 (50)	9 (45)	7 (58)	
CABG + valve surgery	12 (38)	10 (50)	2 (17)	
CABG + others	1 (3)	1 (5)	0 (0)	
Others	1 (3)	0 (0)	1 (8)	
Clinical variables				
ECC time, min	190 (165-268)	197 (173-272)	175 (157-268)	.408
MAP[b], mmHg	70 (58-82)	71 (58-82)	68 (58-81)	.533
HR,[b] bpm	95 (85-105)	93 (82-100)	98 (90-103)	.266
PaO_2/FiO_2[b]	247 (165-323)	266 (229-321)	198 (165-323)	.311
VIS[c] 24 h[d]	34 (12-53)	32.2 (7.7-52.5)	33.9 (25-90)	.454
VIS[c] 48 h[f]	12 (4-43)	9.6 (2.4-33.8)	30 (9-55)	.220
SOFA 24 h[d]	11 (10-12)	10 (9-11)	11 (11-12)	.08
SAPS II 24 h[d]	41 (32-45)	36 (30-41)	46 (41-54)	.003
APACHE II 24 h[d]	18 (15-24)	18 (14-22)	20 (17-25)	.182
Laboratory results				
Lactate,[b] mmol/L	11 (5-16)	7 (4-14)	14 (11-20)	.010
Lactate 24 h,[d] mmol/L	2.5 (1.4-4.5)	1.7 (1.15-2.9)	5.2 (2.7-9.8)	.022
Peak[e] lactate, mmol/L	11.3 (5.3-17.6)	5.6 (3.9-14.4)	16.1 (12.1-20)	.005
Creatinine,[b] mg/dL	1.3 (0.8-1.5)	1.3 (0.9-1.5)	1.6 (1-1)	.267
Creatinine 24 h,[d] mmol/L	1.4 (1.1-2.13)	1.2 (0.8-1.7)	2.1 (1.1-2.1)	.024
Peak creatinine,[e] mg/dL	1.6 (1.3-2.5)	1.5 (1-1.8)	2.5 (1.8-3)	.005
Blood glucose,[b] mg/dL	232 (172-274)	199 (159-255)	260 (232-294)	.047
Total bilirrubin, mg/dL	1 (0.7-1.6)	1 (0.7-1.9)	1 (0.8-1.4)	.799
Peak[e] bilirrubin, mg/dL	1.9 (1.4-3.2)	2 (1.5-4)	1.7 (1.3-3.2)	.838
ALT,[b] mg/dL	37 (20-318)	34 (19-190)	147 (20-1957)	.302
AST,[b] mg/dL	104 (65-325)	104 (65-331)	104 (65-240)	.901
PaO_2/FiO_2[b]	247 (165-323)	266 (229-321)	198 (165-323)	.311
WBC,[b] $\times 10^9$/L	13.7 (9.4-18.4)	13.9 (10-17)	12 (9-20)	.800
Hemoglobin[b], g/dL	9.2 (8.4-10.4)	9.1 (8.4-10.5)	9.5 (8.4-10.1)	.922
Peak procalcitonine,[e] ng/mL	10.1 (1.3-24.2)	5.8 (1-15.7)	29.3 (9.7-69.1)	.019
Clinical managment				
Use of IABP	24 (75)	16 (80)	8 (67)	.399
Use of TMCS	31 (97)	19 (95)	12 (100)	.431
Implant during surgery[g]	21 (68)	12(63)	9 (75)	.492
TMCS[g] device				.621
VA ECMO	24 (75)	15 (75)	9 (75)	
Central VA ECMO	20 (63)	13 (65)	7 (58)	
Peripheral VA ECMO	4 (13)	2 (10)	2 (17)	
Centrimag Levitronix	6 (19)	4 (20)	2 (17)	
Impella CP	1 (3)	0 (0)	1 (8)	
Support time, d[g]	6 (5-14)	9 (5-14)	6 (2-14)	.501

Table 1 *(Continued)*
Demographic features, clinical management, complications, outcome and destination in patients with postcardiotomy cardiogenic shock

Variable	Global PCCS n = 32	Group A PCCS[a] survivors n = 20	Group B PCCS[a] nonsurvivors n = 12	P
TMCS[g] complications				
> 1 complications[h]	23 (74)	14 (70)	9 (75)	.935
Neurological events				.048
Ischemic stroke	2 (6)	0	2 (17)	
Hemorrhagic stroke	1 (3)	0	1 (8)	
Encephalopathy	4 (13)	1 (5)	3 (25)	
Others	1 (3)	1 (5)	0	
Tracheostomy	10 (31)	8 (40)	2 (17)	.134
Use of RRT	14 (44)	6 (30)	8 (67)	.043
ICU admission time, d	18 (11-31)	27 (15-40)	10 (2-18)	.009
Hospital admission time, days	30 (14-51)	45 (29-64)	10 (2-18)	< .001
In-hospital mortality, causes				
Multiorgan dysfunction	9 (28)	-	9 (75)	
Stroke	2 (6)	-	2 (17)	
Bleeding	1 (3)	-	1 (8)	
Patient destination				
Death with device	7 (22)	0 (0)	7 (58)	
Weaning from device	20 (62)	15 (75)	5 (42)	
Transplant	5 (16)	5 (25)	0 (0)	

ALT, alanine transaminase; APACHE II, acute physiology and chronic health evaluation II; AST, aspartate transaminase; BMI, body mass index; CABG, coronary artery bypass surgery; ECC, extracorporeal circulation; ECMO, extracorporeal membrane oxygenation; FiO_2, fraction of inspired oxygen; HR, heart rate; IABP, intra-aortic balloon pump; LVAD, left ventricular assist device; MAP, mean arterial pressure; PaO_2, arterial oxygen pressure; PCCS, postcardiotomy cardiogenic shock; RRT, renal replacement therapies; SAPS II, simplified acute physiology score; SOFA, sequential-related organ failure assesment score; TMCS, temporary mechanical circulatory support; VA, veno-arterial; VIS, vasoactive inotropic score; WBC, white blood cell count.
Continuous variables are expressed as mean ± standard deviation or, if variables were not normally distributed, as median (interquartile range). Categorical variables are presented as frequency and percentage
[a] Indicated at hospital discharge.
[b] Indicated value gathered upon ICU admission.
[c] VIS. Calculated as "dopamine dose (μg/kg/min) + dobutamine dose (μg/kg/min) + 100 x adrenaline dose (μg/kg/min) + 10 x milrinone dose (μg/kg/min) + 10.000 x vasopressin dose (UI/kg/min) + 100 x noradrenaline dose (μg/kg/min)".
[d] Worst value registered within first 24 hours of ICU admission.
[e] Highest value (peak) during ICU stay.
[f] Value gathered 48 hours after ICU admission.
[g] Only in patients in whom TMCS were used.
[h] More than 1 complication (bleeding, reoperation, device malfunction, infection/sepsis).

recovery, and in the remaining 5 (16%) a heart transplant was performed.

The survival rate at discharge of all treated PCCS patients was 63% (20 patients). There were no deaths during the first year after discharge. Survival at discharge and at 1-year of follow-up did not differ from that in patients with other causes of CS in our series (figure 1A). On univariate analysis, the main factors associated with in-hospital mortality were as follows: a history of diabetes or stroke, lactate levels, creatinine value 24 hours after ICU admission, peak creatinine value, glucose levels, highest value of procalcitonine during ICU stay, and acute neurologic complications (table 1). Most of these findings agree with those of previous series.[1,3,4] On average, patients who died were 10 years older, but this finding did not reach stadistical significance probably due to the sample size.[4]

In recent years, due to an increased and generalized use of TSCM devices, the range of therapeutic possibilities available in PCCS has expanded. However, this development does not seem to have translated into a clear benefit in terms of hospital survival.[1,2]

This study shows some distinctive features of the experience of an organized CS unit, which reflect the contemporary management of PCCS in dedicated multidisciplinary teams. Despite the limited number of patients, which is common in CS series, the study shows one of the highest survival rates at discharge and at 1 year published to date (figure 1B). This experience could indicate the potential benefit of trained specialized teams operating within an organized structure,[3] resulting in an immediate and probably more efficient response.[5]

Patients in our series showed tissue hypoperfusion and failure of other organs on ICU admission. Both conditions seem to improve by decreasing time to effective myocardial support with prompt use of an appropriate circulatory support system. Increases in hypoperfusion biomarkers were more significant in CS patients who died. However, the ranges that determine the prognosis and potential degree of reversibility of this damage are not yet well known.

Although ECMO seems to have become the first-line treatment as a TMCS, in our opinion, the use of other centrifugal central-access pumps should not be undervalued when uni- or biventricular failure is observed and respiratory support is not needed, especially when central access is available. The use of a peripheral access support in this context,[1] which has the advantage of permitting sternal closure, did not seem to provide any further chances of survival in our series (table 1).

Another differential characteristic is the use of heart transplant as the final destination in 5 (16%) of the patients. This option has been less used in other series,[1] and may suggest an easier access to emergency transplant in Spain, as opposed to the use of long-term assist devices.

Finally, this series confirms the excellent prognosis of CS patients who survive hospitalization. Thus, PCCS is a serious disorder with a high probability of early death, but it is treatable and, if appropriately addressed, can result in full recovery.

The limitations of our study include its observational nature and the limited number of patients involved. The applicability of our conclusions should be restricted to the clinical context described. Comparison between series remains challenging.[3]

We conclude that early detection of PCCS and rapid response by means of a dedicated, multidisciplinary and adequately organized shock team could improve management and survival in post-cardiotomy shock patients. This conclusion should be confirmed in future series and lines of research.

José Manuel Álvarez Avello,[a,b,*] Francisco José Hernández Pérez,[c,d] Reyes Iranzo Valero,[a] Carlos Esteban Martín,[e] Alberto Forteza Gil,[e] and Javier Segovia Cubero[c,d]

[a]*Departamento de Anestesiología y Reanimación, Hospital Universitario Puerta de Hierro Majadahonda, Madrid, Spain*
[b]*Departamento de Anestesiología y Cuidados Intensivos, Clínica Universidad de Navarra, Madrid, Spain*
[c]*Departamento de Cardiología, Hospital Universitario Puerta de Hierro, Majadahonda, Madrid, Spain*
[d]*Centro de Investigación en Red de Enfermedades Cardiovasculares (CIBERCV), Spain*
[e]*Departamento de Cirugía Cardíaca, Hospital Universitario Puerta de Hierro, Majadahonda, Madrid, Spain*

[*] Corresponding author:
E-mail address: jomaavello@hotmail.com (J.M. Álvarez Avello).

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Evolution of electrical and hemodynamic parameters after permanent left bundle branch pacing

Evolución de parámetros eléctricos y hemodinámicos tras estimulación ventricular permanente en el área de la rama izquierda

To the Editor,

Right ventricular pacing has a deleterious effect on ventricular contraction that can lead to the development of pacing-induced cardiomyopathy.[1]

His bundle pacing is the most physiological method of permanent ventricular pacing. His bundle pacing has been demonstrated to reduce adverse events (cardiomyopathy, heart failure and mortality) compared with pacing of the right ventricular apex.[2] There are several factors that limit the widespread use of His bundle pacing: *a)* progression of the block to distal zones, *b)* the rate of successful implantation, *c)* late threshold increase due to microdislocation,[3] and *d)* patients whose block occurs in the most distal portion of the bundle of His.

Huang et al.[4] recently demonstrated the feasibility of a physiological left bundle branch pacing (LBBP); this allows capture of the His-Purkinje system distal to the bundle of His with lower thresholds and better stability and detection. LBBP has been used successfully for ventricular pacing, as well as for the correction of left bundle branch block, as an alternative to cardiac resynchronization.[5] However, the number of patients included in the publications was low, and there are no randomized trials.

In this article we report the effect of LBBP on electrocardiographic and echocardiographic variables in a consecutive series of patients with indication for conventional pacing or cardiac resynchronization therapy.

We included consecutive patients referred to our unit for implantation of a permanent cardiac pacing device. We excluded patients whose percentage of ventricular pacing was predicted to be low.

The pacing lead was implanted in the left bundle branch following the technique previously described by Huang et al.[4] The lead used was the 3830-69 Select-Secure (Medtronic Inc, USA) and the catheter used was the C315His (Medtronic Inc, USA).

Lead position was checked on a left anterior oblique projection, and interventricular septum penetration was confirmed using iodinated contrast. The criteria described by Chen et al.[6] were used to determine left bundle branch capture.

One operator, who was blinded, assessed LV function on echocardiography. This was performed once before implantation and repeated after at least 4 weeks. Left ventricular ejection fraction (LVEF) was calculated according to the Simpson method. Electrocardiograms performed with the multichannel recording system Cardiolab Prucka (GE Inc, USA) were collected. QRS duration (QRSd) was obtained before and after implantation. In all patients, the first follow-up was performed at 3 months after device implantation.

We included 24 consecutive patients who underwent implantation of an LBBP lead. Successful implantation was achieved in all patients (n = 24). The characteristics of the patients and the details of the procedure are given in table 1.

Analysis of the acute electrical parameters at the first follow-up showed no differences in threshold (0.58 ± 0.2 vs 0.57 ± 0.1 V in 0.4 ms; P = .988) or ventricular detection (13.6 ± 7 vs 13.5 ± 5 mV; P = .978). No patient showed a sudden increase in threshold or change in impedance that would require lead revision or replacement.