

Induction of Sustained Ventricular Tachycardia by Programmed Stimulation through Permanent Pacemaker Generator

Programmed asynchronous stimulation of the heart to induce arrhythmias is a widely used prognostic and diagnostic tool in the invasive electrophysiology laboratory to define the approach in patients with arrhythmic substrate and suspected sustained ventricular or supraventricular arrhythmias. (1)

To perform a conventional electrophysiological study (EPS), it is necessary to take the patient to the electrophysiology or cardiac catheterization laboratory and, under local anesthesia, introduce at least one venous-access electrode-catheter connected to an external stimulator and a polygraph, with the subsequent, though minimal, risk and complications resulting from this type of puncture, in addition to the exposure of health care professionals to radiation during the procedure and the hospital system costs. (2)

Today, pacemaker (PM) generators, cardioverter defibrillators (CVD), and resynchronization devices (CRT) can follow, as EPS, different stimulation protocols, and can introduce up to three extrastimuli to induce arrhythmias and then abolish them with asynchronous burst stimulation. (3)

We report the case of an 82-year-old female patient with positive serology for Chagas, with history of permanent dual-chamber PM in February 2013 due to symptomatic 2:1 atrioventricular block (AVB) and syncope. The patient was under drug therapy with bisoprolol, amiodarone, and losartan.

In September 2015, the patient was admitted to the Department of Clinical Medicine due to syncope preceded by angina. During hospitalization, she underwent:

Electrocardiogram: It showed sinus rhythm with first-degree AV block and complete right bundle branch block, alternating with atrial regulation in AAI mode with long AV interval.

Echocardiography: It revealed mild left atrial enlargement and concentric hypertrophy, mid-anteroseptal hypokinesis, and preserved left ventricular systolic function.

24-hour Holter: It evidenced rare, isolated ventricular extrasystoles and frequent, isolated, polymorphic PEB. Frequent monomorphic and polymorphic couples and a single episode of 9 beats of nonsustained ventricular tachycardia (cycle length 300 msec) were also observed.

Coronary angiography: No angiographically significant lesions were found in LCA and the RCA could not be catheterized and targeted due to heterogeneity of coronary flow.

Due to a new syncopal episode during hospitalization, the patient was referred to the Coronary Care Unit to undergo an EPS with stimulation from the tip

of the RV for induction of ventricular arrhythmia.

Given that the patient had a dual-chamber PM that could perform asynchronous and/or programmed stimulation with up to three extrastimuli (Sensia DR [Medtronic Inc], also available in models such as Altrua 60 [Boston Scientific], and Accent DR [St Jude Medical]), it was decided not to perform conventional EPS.

An induction protocol was used, with stimulation from the RV apex (PM catheter) and basal train stimulation at 600 and 500 msec, and extrastimuli at sequential and programmed decreasing coupling intervals. (4)

On sinus rhythm, with a stimulation drive (S1-S2) of 500 msec, two extrastimuli (S2=300 msec and S3=260 msec) were introduced with induction of sustained, monomorphic, ventricular tachycardia (cycle length 350 msec), symptomatic for angina and trepidation (Figure 1), which was interrupted with burst stimulation of 290 msec (Figure 2).

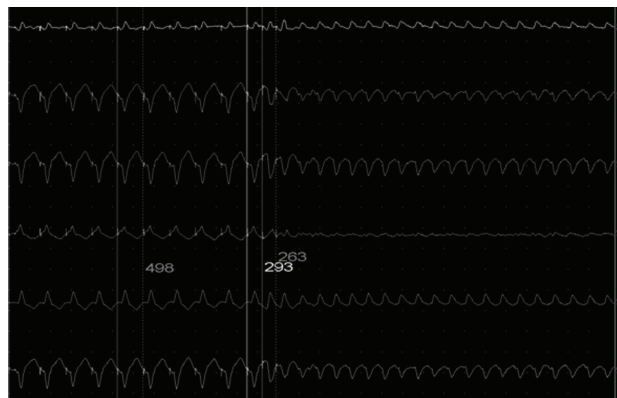


Fig. 1. Induction of sustained ventricular tachycardia with programmed stimulation using two extrastimuli.



Fig. 2. Termination of sustained ventricular tachycardia with burst stimulation of 290 msec.

Since the test was positive, upgrade to dual-chamber ICD (5) was successfully implanted on the following days.

The patient was asymptomatic at follow-up, and presented isolated episodes of monomorphic ventricular tachycardia during ICD control, which reversed with ATP.

In these cases, programmed stimulation can be performed from the site where the ventricular lead is implanted, so a conventional EPS with stimulation from the right ventricular outflow tract has to be performed in cases of non-induced arrhythmia from the RV apex.

The timely use of resources available in our daily practice will help us provide adequate follow-up to our patients, with the safest and most appropriate diagnostic and therapeutic options, always by experienced operators.

In addition to its safety and reliability, inductive telemetry completely rules out any complication associated with the puncture site, and avoids radiation exposure received during any invasive method.

Induction of complex and sustained arrhythmias should be performed in the coronary care unit with all the necessary resources to manage possible complications.

In addition to induction of sustained arrhythmias, asynchronous and/or programmed stimulation from implantable devices can be very useful as diagnostic tool, even for the evaluation of the permanent programming mode. Through this means, we can:

- Determine the Wenckebach point in those patients with preferential atrial stimulation (MVP, AAIsafeR, etc.).
- Determine sinus node recovery time to assess sinus function, and consider (or not) the frequency response sensor activation.
- Measure antegrade conduction refractory periods.

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REFERENCES

1. Zipes D, Miles W. Valoración de un paciente con arritmia cardíaca. En: Zipes D, Jalife J. Arritmias. Electrofisiología cardíaca. New York, USA: Marbán; 2006. p. 759-65.
2. Schuger C, Josephson M. Electrofisiología Clínica: Aspectos Técnicos y Conceptos Generales. En: González Zuelgaray J. Arritmias Cardíacas. Buenos Aires, Argentina: Inter-Médica; 2006. p. 19-52.
3. Novotný T, Dvůrák R, Lábrová R, Kozák M, Semrád B. Electric programmed ventricular stimulation using a permanent cardiac pacing system— A noninvasive method in arrhythmia. *Cas Lek Cesk* 1999;138:495-6.
4. Berne P, Tolosana JM. Estudio electrofisiológico: conceptos básicos. En: Mont L, Calvo N, Arbelo E, Berrueto A, Tolosana JM, Brugada J. Manual de Electrofisiología Clínica y Ablación. Barcelona, España: Marge; 2011. p. 27-37.
5. Chiale P, Acunzo R, Sánchez R. La prevención de la muerte súbita

cardíaca. El papel de los cardiodesfibriladores implantables. En: Chiale P, Garro H, Pastori J, Sánchez R, Selva H. Marcapasos, Resincronizadores y Cardiodesfibriladores Implantables. Buenos Aires: Total Graf; 2008. p. 597-613.

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High-Sensitivity Troponin for Prediction of Myocardial Infarct Size in Patients with ST Segment Elevation

The assessment of myocardial lesion size after acute ST-segment elevation myocardial infarction (STEMI) has significant prognostic and therapeutic implications. (1) Different imaging and lab techniques can be used, echocardiography being the most widely employed. High-sensitivity troponin T (hs-Tn) has recently been available in clinical practice, and to date, the information regarding its correlation with infarct size has been discordant. (2, 3)

The purpose of this report is to correlate the level of echocardiographic involvement, measured with the wall motion score index (WMSI), and ejection fraction (EF), with hs-Tn values obtained during the first hours of STEMI.

For this purpose, a retrospective analysis was conducted, including 67 patients admitted for STEMI from May 2012 to January 2013. Patients with previous infarction that could alter WMSI were excluded. STEMI definition was taken from the clinical practice guidelines of the European Society of Cardiology, in its third universal definition of infarction. (4) Infarction was confirmed with hs-TnI >14 ng measured by the Roche method with a 2010 Elecsys® analyzer. Blood was systematically withdrawn for lab tests on admission and between 6 and 12 hours. Spearman's test was used to correlate troponin with WMSI.

Mean age was 59±10.1 years, and 91% were male patients (Table 1). The mean pain-to-balloon time was 221 (110-311) minutes and, on admission, 91% were in Killip class A or B.

The main culprit vessel was the anterior descending artery in 44.6% of cases, but significant lesions in two or more vessels were found in 71% of patients.

Median hs-Tn on admission was 241 ng/L (27.5-1,350) and 1,965 ng/L (655.2-6,770) at 6-12 hours, with a median EF of 50% (41.5-57.5). When the relationship with WMSI was analyzed, we found that hs-Tn at 6-12 hours had a moderate but significant correlation (r=0.54, p=0.005) (Figure 1). In turn, correlation between hs-Tn on admission and 6-12 h and EF was not significant (p=0.545 and p=0.253, respectively).

Lack of correlation with EF could be explained because several factors are involved in its assessment after STEMI: compensatory mechanism of normal tissue, affected myocardial mass, location, level

Table 1. Baseline population characteristics

Variable	n = 67
Age, years	59 ± 10.1
Male subjects, %	91
DM, %	11.9
SMK/EXSMK, %	73.1
HFH, %	14.9
HTN, %	59.7
Dyslipidemia, %	64.2
Physical inactivity, %	13.4
Obesity, %	16.4
Systolic blood pressure, mm Hg	127.8 ± 21.7
Diastolic blood pressure, mm Hg	72.7 ± 13.1
Pain-to-balloon time, min	221 (110-311)
Heart rate on admission, bpm	74.8 ± 16.5
Troponin on admission, ng/L	241 (27.5-1350)
Troponin 6-12 hours, ng/L	1965 (655.2-6770)
Creatinine on admission, mg/dl	1.1 ± 0.22
Killip & Kimball on admission, %	
A	68
B	23
C	0
D	9
VT/VF, %	6
Aspiration thrombectomy, %	32.8
IIb/IIIa inhibitors, %	29.9
Culprit vessel, %	
ADA	44.6
RCA	26.2
Cx	29.2
Number of vessels, %	
1	29
2	35.8
3	35.8
Emergency surgery, %	3
Ejection fraction, %	50 (41.5-57.5)
In-hospital mortality, %	3
Hospital stay, days	4 (3-5)

DM: Diabetes mellitus. SMK: Smoker. EXSMK: Ex-smoker. HFH: Hereditary family history. HTN: Hypertension. VT/VF: Ventricular tachycardia/Ventricular fibrillation ADA: Anterior descending artery. RCA: Right coronary artery. Cx: Circumflex artery.

of involvement of myocardial tissue surrounding the necrotic area, previous ventricular hypertrophy, and associated multivessel disease, among other factors.

It is possible that as a result of this factors Chia Et al. found poor correlation between fourth generation troponin T at 12 hours and EF measured by SPECT five days after the event ($r=0.39$). (5) In turn, Steen et al. evaluated the correlation of AMI size with magnetic resonance imaging and fourth-generation troponin

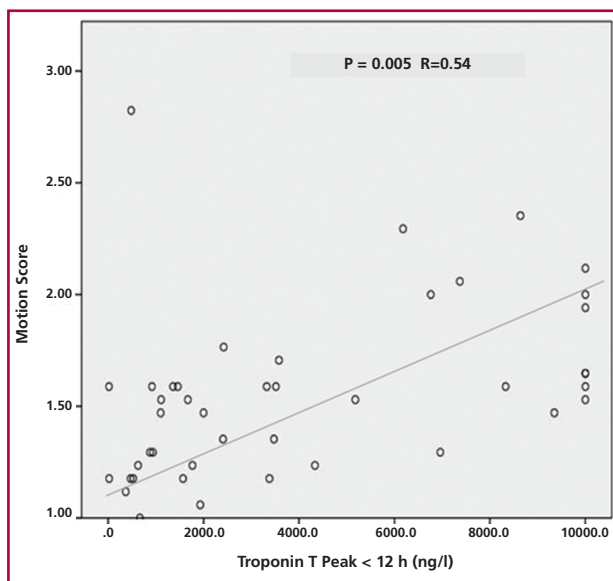


Fig. 1. Correlation between troponin T within 12 hours and echocardiography motion score.

T, measured 96 hours since symptom onset, and found an excellent association for STEMI ($r=0.91$). (6) Apparently, late troponin values improve correlation with infarct size, since baseline values are influenced not only by the affected myocardium but also by the level of reperfusion and time of ischemia.

The wall motion score index is simple to measure and is less dependent of the injured territory because the amounts of points for each segment is equal regardless of the arterial territory. In this case, hs-Tn improved its predictive ability, supporting the hypothesis that its concentration is associated with the affected myocardial tissue.

Ours is a retrospective study with the risk of bias involved in data collection. The echocardiographic assessment of infarct size was performed with portable equipment at the patient bedside, by operators who were not blind to the patient's condition.

Based on these results, we can conclude that hs-Tn values within 12 hours in patients with STEMI and no history of previous AMI present a regular predictive ability of infarct size assessed by WMSI.

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REFERENCES

1. Carrick D, Berry C. Prognostic importance of myocardial infarct characteristics. *Eur Heart J Cardiovasc Imaging* 2013;14:313-5. <http://doi.org/bcwh>

2. Lebeau R, Serri K, Morice M-C, Hovasse T, Untersee T, Piéchaud J-F, et al. Assessment of left ventricular ejection fraction using the wall motion score index in cardiac magnetic resonance imaging. *Arch Cardiovasc Dis* 2012;105:91-8. <http://doi.org/bewj>
3. Licka M, Zimmermann R, Zehlein J, Dengler TJ, Katus H a, Kübler W. Troponin T concentrations 72 hours after myocardial infarction as a serological estimate of infarct size. *Heart* 2002;87:520-4. <http://doi.org/dwp6br>
4. Steg PG, James SK, Atar D, Badano LP, Lundqvist CB, Borger MA, et al. ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology (ESC). *Eur Heart J* 2012;33:2569-619. <http://doi.org/zkn>
5. Chia S, Senatore F, Raffel OC, Lee H, Wackers FJT, Jang IK. Utility of cardiac biomarkers in predicting infarct size, left ventricular function, and clinical outcome after primary percutaneous coronary intervention for ST-segment elevation myocardial infarction. *JACC Cardiovasc Interv* 2008;1:415-23. <http://doi.org/d3bz3w>
6. Steen H, Giannitsis E, Futterer S, Merten C, Juenger C, Katus H. Cardiac troponin T at 96 hours after acute myocardial infarction correlates with infarct size and cardiac function. *J Am Coll Cardiol* 2006;48:2192-4. <http://doi.org/djkbks>

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Simultaneous Percutaneous Closure of Paravalvular Aortic Leak and Aorto-Atrial Fistula Guided by Two-Dimensional Transesophageal Echocardiography

Paravalvular prosthetic leak is a complication of valve surgery caused by degeneration of annular tissue, affecting about 6% to 15% of surgically implanted prosthetic valves and annuloplasty rings. (1)

We report the case of a 75-year-old male patient, who underwent aortic valve replacement with a bioprosthesis (Biocor 27; St Jude®) and myocardial revascularization surgery (mammery artery bypass graft to the anterior descending artery and venous bypass graft to the first diagonal artery). The patient was discharged on the sixth day after uneventful post-operative course. Two months after the procedure, a control transthoracic echocardiography revealed a moderate anterior paravalvular leak (PVL), 5-6 hour of surgical view, and a fistula between the aorta and the right atrium (AO-RA), 8-9 hour of surgical view (Figure 1). There was no clinical, analytic, or echocardiographic evidence of endocarditis. During the 2-year follow-up, the patient had chronic hemolytic anemia and progressive heart failure despite optimal medical treatment; therefore, percutaneous leak closure was suggested.

Under two-dimensional transesophageal echocardiography (TEE) and angiographic guidance, simultaneous paravalvular leak closure was performed via retrograde aorta with 8 mm Amplatzer Vascular Plug II (St Jude®) device, with immediate minimal residual shunt, and AO-RA fistula was closed with 6 mm Amplatzer Vascular Plug III (St Jude®) (Figure 2).

Today, three years after the percutaneous treat-

ment, the patient is asymptomatic, with no evidence of hemolysis. Control echocardiography showed mild paravalvular leak, with mild increase in transvalvular aortic gradient (mean gradient 24 mm Hg) and preserved left ventricular ejection fraction.

Paravalvular leak after cardiac valve replacement involves abnormal flow through the native tissue and prosthetic valve, due to incomplete apposition of the sewing ring to the native tissue. This is generally a consequence of suture dehiscence. It may develop more commonly in patients with heavy annular calcification, localized infection, or due to technical considerations. (2)

Paravalvular leaks are usually small and asymptomatic, and have a benign course. Larger PVLs -with greater clinical involvement occur in about 1-5% of the patients with prosthetic valves. (3) Biological valves are more commonly involved than mechanical valves.

Patients with significant PVLs present symptoms because blood flow through the valve and subsequent volume overload are associated with reduction of effective cardiac output and congestive heart failure, resulting in decreased exercise tolerance and dyspnea. Symptoms can also be associated with hemolytic anemia, which is caused by red cell fragmentation in the elevated shear stress of the regurgitant jet. Infectious endocarditis can be cause or consequence of PVLs.

Diagnosis is made based on clinical and echocardiographic findings, the latter being very difficult at times. Three-dimensional TEE allows for the visualization of the entire prosthetic valve, increasing the definition and characterization of PVLs. (4)

Reoperation is the treatment of choice in PVLs, either repairing the defect or, most commonly, replacing the valve. It is generally performed in very symptomatic patients due to severe anemia or progressive heart failure, and is associated with elevated morbidity and mortality. It also has the risk of recurrent paravalvular failure. (5)

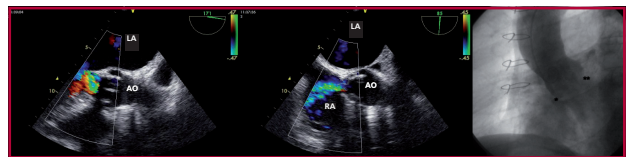


Fig. 1. Aortic (AO) paravalvular leak (left) and fistula to the right atrium (RA) (middle) in transesophageal echocardiography. Paravalvular leak (***) and fistula (*) in angiography (right).



Fig. 2. Post-procedural transesophageal echocardiography and angiography. Paravalvular residual shunt (left).

In this scenario, the use of percutaneous closure as a less invasive technique is a valid therapeutic option for the treatment of these patients, with good results in terms of morbidity and mortality during follow-up. (6) It should be noted that the oval or half-moon morphology in most PVLs hampers finding a specific device for each defect. For this reason, a variety of devices not specifically designed for the treatment of PVLs have been used.

Finally, we would like to point out that three-dimensional TEE is the ideal monitoring during the procedure, particularly of posterior structures such as the mitral valve, because it provides improved spatial resolution to channel the regurgitation, choosing the approach, deciding on the device to use, and assessing complications after the occlusion (interference with prosthetic valve motion, residual regurgitation, etc.). (4) However, a strategy based on two-dimensional TEE and angiography, as in our case, can be used even in complex cases.

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REFERENCES

1. Eleid MF, Cabalka AK, Malouf JF, Sanon S, Hagler DJ, Rihal CS. Techniques and outcomes for the treatment of paravalvular leak. *Circ Cardiovasc Interv* 2015;8:e001945. <http://doi.org/bcwk>
2. Pate GE, Al Zubaidi A, Chandavimol M, Thompson CR, Munt BI, Webb JG. Percutaneous closure of prosthetic paravalvular leaks: case series and review. *Catheter Cardiovasc Interv* 2006;68:528-33. <http://doi.org/d3sqvb>
3. Rallidis LS, Moyssakis IE, Ikonomidis I, Nihoyannopoulos P. Natural history of early aortic paraprosthesis regurgitation: A five-year follow-up. *Am Heart J* 1999;138:351-7. <http://doi.org/d4kp6h>
4. Tsang W, Weinert L, Kronzon I, Lang RM. Ecocardiografía tridimensional en la evaluación de las válvulas protésicas. *Rev Esp Cardiol* 2011;64:1-7. <http://doi.org/fmz2kq>
5. Jones JM, O'Kane H, Gladstone DJ, Sarsam MA, Campalani G, MacGowan SW, et al. Repeat heart valve surgery: risk factors for operative mortality. *J Thorac Cardiovasc Surg* 2001;122:913-8. <http://doi.org/b7xg3d>
6. Millan X, Skaf S, Joseph L, Ruiz C, Garcia E, Smolka G, et al. Transcatheter reduction of paravalvular leaks: A systematic review and meta-analysis. *Can J Cardiol* 2015;31:260-9. <http://doi.org/bcwm>

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