American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2019; 20: 810-815
DOI: 10.12659/AJCR.914480

Received: 2018.12.06
Accepted: 2019.01.31
Published: 2019.06.09

Biventricular Pacing Going Along with Acute Hemodynamic Response in a Patient with Huge Anterior Wall Aneurysm – Importance of Pacing Viable Myocardium

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

BDEF 1 Spyridon Liosis
AB 1 Evgeny Lyan
EF 1 Amr Abdin
BC 1 Ben Brüggemann
D 1 Stefan A. Lange
DE 1 Julia Vogler
D 1 Christian H. Heeger
EF 2 Kivanc Yalin
A 1 Roland R. Tilz
AB 1 Charlotte Eitel

1 Department of Cardiology, University Heart Center Lübeck, Lübeck, Germany
2 Department of Cardiology, Usak University, Faculty of Medicine, Usak, Turkey

Corresponding Author: Spyridon Liosis, e-mail: spyridon.liosis@uksh.de
Conflict of interest: None declared

Patient: Female, 85
Final Diagnosis: Ischemic cardiomyopathy with electrical storm
Symptoms: Dyspnea
Medication: —
Clinical Procedure: Ablation of ventricular tachycardia
Specialty: Cardiology

Objective: Unusual clinical course

Background: Response to cardiac resynchronization therapy (CRT) is variable among patients. Extensive scar tissue burden has been characterized as a negative predictor of significant response. Whereas mid-term and long-term response has been thoroughly investigated in randomized clinical trials; however, little is known about acute hemodynamic effects of biventricular pacing.

Case Report: We report a case of an elderly female patient with severe ischemic cardiomyopathy and a large anterior wall aneurysm, who received right ventricular and biventricular pacing during ablation of incessant pleomorphic ventricular tachycardia. During the procedure, biventricular pacing was associated with a 20% acute increase in systolic blood pressure compared to right ventricular pacing, although there was no acute or long-term effect on left ventricular function.

Conclusions: The acute hemodynamic effect of CRT in our patient suggests an effect of CRT even in patients with negative predictors of CRT response such as severe ischemic cardiomyopathy with a large aneurysm. Although no marked increase in left ventricular function might be observed, the acute effect of CRT might contribute to stabilization of heart failure in these patients.

MeSH Keywords: Cardiac Resynchronization Therapy • Heart Aneurysm • Myocardial Ischemia

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/914480

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

Background

Cardiac resynchronization therapy (CRT) is associated with an improved quality of life and reduction in hospitalization in patients with advanced heart failure with reduced left ventricular ejection fraction (LVEF) and increased QRS duration [1]. Moreover, it has been shown to induce reverse remodeling of the left ventricle [2] and to reduce long term mortality [3]. There is, however, a wide variability in the extent of LV remodeling and improvement in LV function after CRT. Some patients ("super responders") show an exceptional improvement resulting in a full recovery of LV function [4,5]. On the other hand, up to 30% of patients do not respond to CRT ("non-responders"). Several definition criteria have been used to define super response in studies [6,7], but there is still no universal consensus regarding the definition of a responder to CRT. Ischemic cardiomyopathy has been characterized as a negative predictor of response [4]. Especially extensive scar burden and lead position over transmural scar tissue may lead to an unfavorable clinical outcome and reduce reverse remodeling after CRT [8,9].

Case Report

An 85-year-old female was admitted to our hospital with acute heart failure due to recurrent slow ventricular tachycardia (Figure 1). She had a history of ischemic cardiomyopathy with a chronic total occlusion of the left anterior descending artery and an extended anterior wall aneurysm. A CRT-defibrillator (CRT-D) was implanted 4 years ago due to severely reduced LV function (30%), symptomatic heart failure, and left bundle branch block (LBBB). Two years later, atrioventricular node ablation was performed for refractory atrial fibrillation and reduced biventricular-pacing percentage. Furthermore, the patient underwent a percutaneous aortic valve implantation (TAVI) for symptomatic severe aortic valve stenosis 10 months prior to presentation.

Biochemical tests at presentation showed elevated creatinine (177 µmol/L) and markedly elevated NT-pro brain natriuretic peptide (BNP) (24 497 ng/L) values as a result of decompensated heart failure. Moreover, hematological tests showed a known normochromic, normocytic anemia (probably renal anemia, due to chronic renal failure) with a hemoglobin of 8.7 g/dL. Leukocytes and C-reactive protein were only marginally elevated. Serum sodium, potassium, and calcium were normal. Echocardiography at presentation showed a severely reduced LVEF (30%) in the presence of a large anterior wall aneurysm and a normal function of the aortic bio-prosthesis. Chest x-ray revealed a cardiomegaly with bilateral moderate pleural effusions and signs of pulmonary congestion.

Device interrogation at admission revealed recurrent episodes of pleomorphic slow ventricular tachycardia, which was

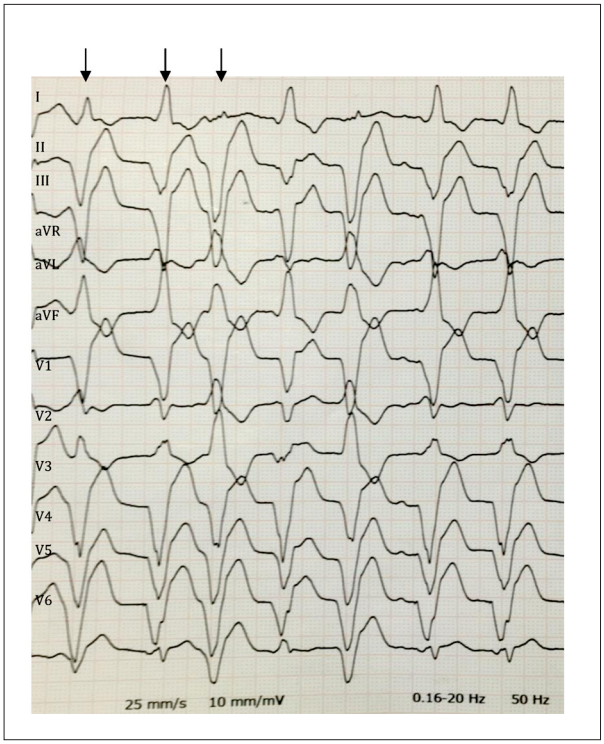

Figure 1. Electrocardiogram of the clinical tachycardia: pleomorphic slow ventricular tachycardia with 3 distinct morphologies, indicated with arrows (probably 3 exit sites of 1 circuit or 3 different circuits).

identified as the apparent cause of heart failure decompensation. The patient refused any kind of interventional therapy and was therefore started on amiodarone. However, ventricular tachycardia recurred, and the patient now presented with incessant ventricular tachycardia resulting in several implantable cardioverter defibrillator (ICD) interventions (>30 ATP therapies and 2 ICD shocks). Despite the high risk for periprocedural complications including LV perforation due to the large aneurysm, we again recommended catheter ablation, given the extremely unfavorable short-term prognosis of an electrical storm with multiple shocks and severe heart failure, not responding to antiarrhythmic medication. We discussed thoroughly the risks and benefits with the patient and proceeded to the intervention after written informed consent was given.

Prior to the ablation procedure tachycardia detection was deactivated and the device was programmed to a VVI 40 bpm back-up mode. After single transseptal puncture, electroanatomical mapping of the LV, using CARTO 3 (Biosense Webster, USA) was performed. Bipolar electrograms were recorded on the EP-recording system (Lab System Pro, Boston Scientific). Mapping revealed a huge anteroseptal LV (left ventricular) aneurysm with low voltage and fragmented potentials, extending from the apex to the basis, covering 71% of the LV surface. A relatively small area of healthy myocardium was found

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

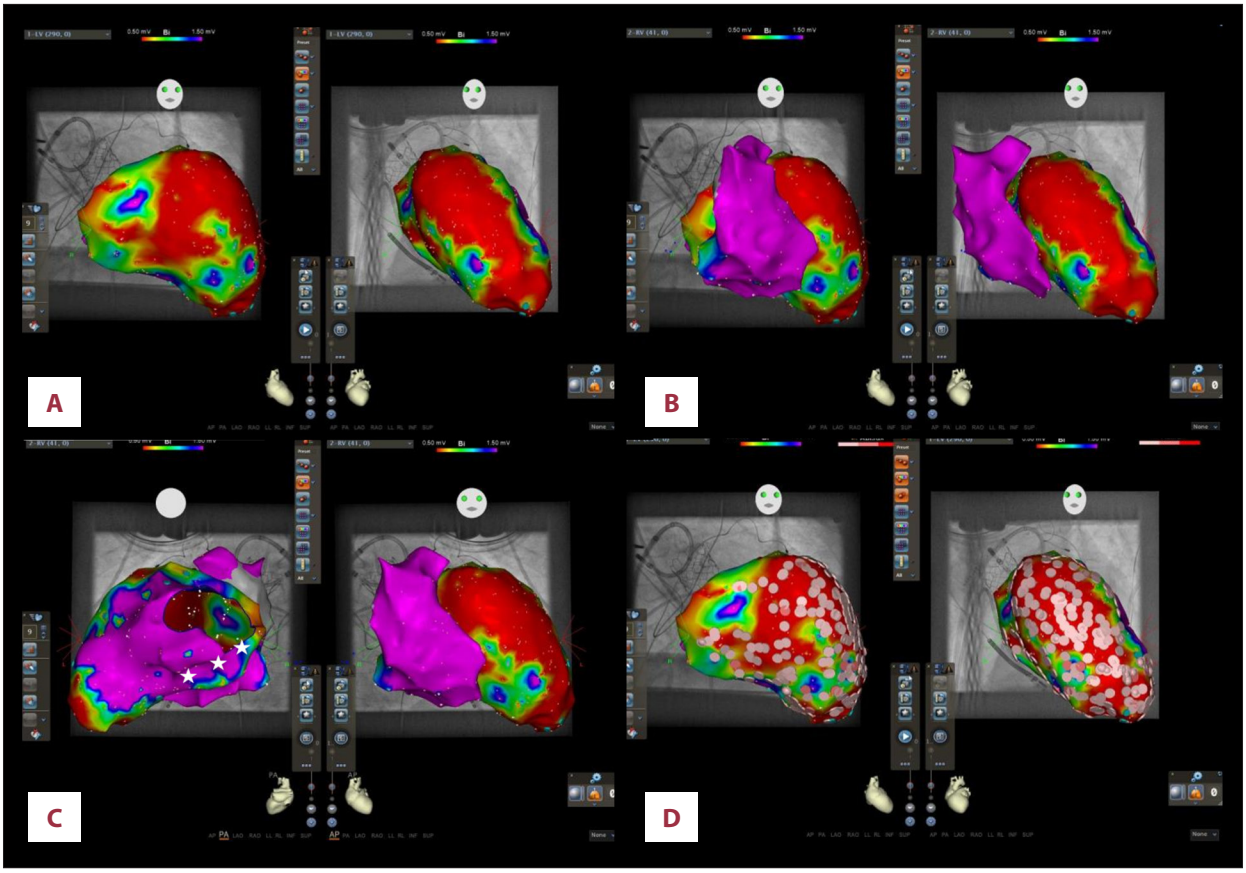

Figure 2. (**A**) 3-dimensional electroanatomical mapping of the left ventricle showing extensive low voltage area (red color) in the anterior, apical, and septal segments (scar tissue, aneurysm). (**B**) Right ventricle without signs of scar tissue (violet color = no low voltage areas). (**C**) Lateral part of the left ventricle (left image) with viable myocardium (violet color). The position of the left ventricular lead is marked with white stars. (**D**) Substrate modification with visualization of ablation points (white points).

at the lateral LV, just where the LV lead was placed (Figure 2). The right ventricle (RV) showed no low-voltage areas as a correlate for scar tissue. Two of 3 morphologies of the clinical pleomorphic slow ventricular tachycardia were induced through programmed ventricular stimulation. Due to hemodynamic intolerance, direct current cardioversion of both was performed after unsuccessful overdrive pacing. Very good pace mapping (93%) of the ventricular tachycardia morphologies was shown in the apical and basolateral segments of the aneurysm. Extensive substrate modification was performed, aiming at ablation of all late potentials and fragmented potentials in these areas. After ablation, ventricular tachycardia was no longer inducible through programmed ventricular stimulation.

At the end of the procedure, the patient's blood pressure dropped. Echocardiography was performed to exclude pericardial effusion. Reprogramming of the device in a biventricular VVI 60 bpm mode resulted in an immediate increase of blood pressure from 65/40 mmHg to 120/80 mmHg. Furthermore, programming to a DDD mode was performed as the patient was now in sinus rhythm, after having received an ICD-shock for accelerated slow ventricular tachycardia before the procedure.

During the course of the hospital stay the patient showed no recurrence of ventricular tachycardia. Four hours after the procedure, device interrogation revealed a marked decrease in invasively measured systolic blood pressure with RV pacing, which again increased from 75/50 mmHg to 90/50 mmHg under biventricular stimulation (Figure 3). Programming the device to VVI 60 bpm with RV pacing only did not result in differing hemodynamic effects compared to DDD 60 bpm with RV pacing only. Despite the prominent change in invasive systolic blood pressure, LVEF (biplane Simpson's method) showed no difference between RV pacing and biventricular pacing (30% versus 28%).

The post-interventional course was uncomplicated, and the patient was discharged back home. She was free from any arrhythmia recurrence until her death 2 months after the procedure due to an acute, gangrenous cholecystitis with septic shock.

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

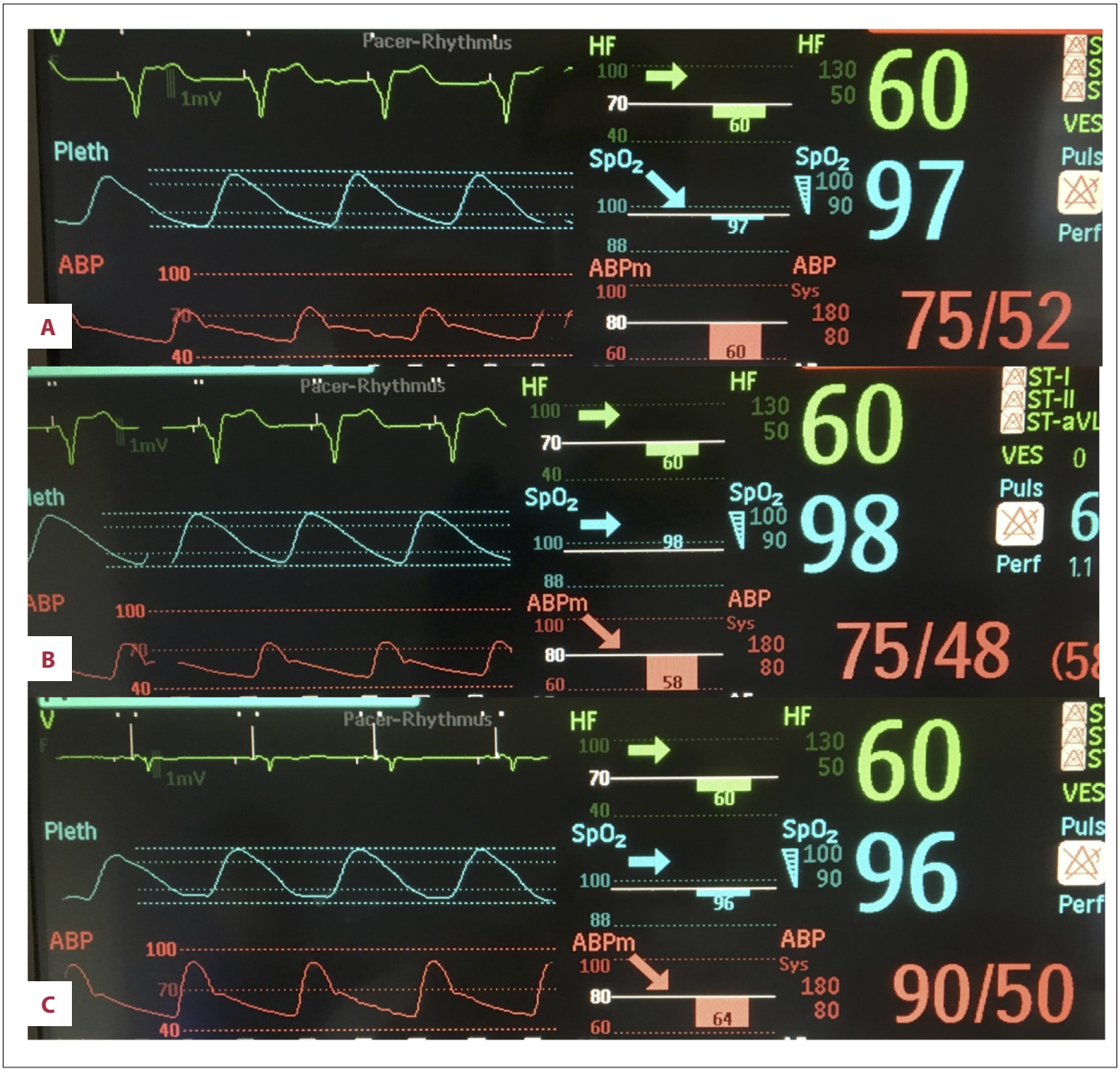

Figure 3. Invasive arterial blood pressure measurement: (**A**) under VVI 60 bpm right ventricle (RV) pacing, (**B**) under DDD 60 bpm RV pacing, (**C**) under DDD 60 bpm biventricular pacing.

Discussion

We report on a patient with ischemic cardiomyopathy and large LV aneurysm who underwent ablation for incessant ventricular tachycardia. We observed a marked acute increase (20%) in systolic blood pressure under biventricular compared to RV pacing. It was surprising how the resynchronization of such a small part of viable myocardium improved acute hemodynamics. We could not find a similar acute or long-term effect on LV function though.

The presented case is, to the best of our knowledge, the first one to show a significant acute increase in blood pressure under CRT in a patient with a very large left ventricular aneurysm, a well characterized negative predictor of therapy response. Moreover, the acute hemodynamic improvement in our patient was not related to a simultaneous increase in LV function, implicating the presence of other underlying pathophysiological mechanisms. Future research addressing these mechanisms could lead to a better understanding of CRT.

Several studies have shown that reverse remodeling due to biventricular stimulation can lead to a mid-term and long-term increase in LVEF and functional status in responders. Knappe et al. [10] showed an acute deterioration in LVEF and dyssynchrony after switching off biventricular function in

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

responders 12 months after CRT implantation. LV function remained better than baseline (pre-implantation) though. This observation implicates a separate acute and chronic effect of resynchronization therapy.

A few studies with very small patient enrollment evaluating acute hemodynamic response under biventricular pacing have been conducted in the past years. Marked increase in systolic blood pressure was only rarely described. Leclerque et al. studied 18 patients with end-stage systolic heart failure and compared RV with biventricular pacing [11]. They found no difference in systolic blood pressure, but a slight significant improvement in cardiac index. Hamdan et al. [12] observed only a marginal increase in mean systolic blood pressure during biventricular pacing (146 mmHg versus 141 mmHg) in 15 male patients with severely reduced systolic LV function, but only 6 of the participants had a LBBB. Gademan et al. studied 32 patients with CRT, reduced LVEF, and prolonged QRS duration before and after switching off biventricular function [13]. They observed a mean increase of 5% in LVEF but no significant increase in systolic blood pressure. Pieragnoli et al. did not find any difference in systolic blood pressure in 37 heart failure patients between switching CRT on and off [14].

To the best of our knowledge, the only hemodynamic study showing a direct increase in systolic blood pressure under biventricular versus RV pacing in ischemic cardiomyopathy was conducted from Blanc et al., who evaluated 27 patients with severe heart failure including 9 patients with ischemic cardiomyopathy and LBBB [15]. Seven of the 9 patients demonstrated an increase in systolic blood pressure under biventricular pacing with a maximum of 14% in 1 patient (from 91 mmHg to 104 mmHg), whereas the rest of the study patients showed only a marginal increase of 5% to 10%. Data about LV function were not documented.

It is unclear why some patients show a substantial systolic blood pressure increase to biventricular pacing whereas others do not. Randomized trials on CRT did not investigate whether an acute improvement in contractility as measured by systolic blood pressure can predict long-term outcome. A non-randomized study by deVecchi et al. that included 96 patients with systolic heart failure and CRT demonstrated that acute contractile improvement under biventricular versus RV pacing is not associated with 3 years prognosis [16].

Considering the big scar area in our patient (71% of the LV) and the small viable myocardium where the LV lead was placed, we were surprised about the acute hemodynamic effect of biventricular pacing compared to RV pacing. The patient had been classified as non-responder to CRT because the LV function had not improved since CRT-D implantation. However, given the natural course of patients with congestive heart failure, stabilization of the disease indicates some response, whereas true non-responders continue their downward course. Moreover, in this patient, long-term response to CRT in terms of LV function improvement may have been withheld because of the reduced biventricular-pacing percentage due to recurrent ventricular tachycardia and atrial fibrillation (which lead to AV node ablation 2 years ago), as well as due to the severe aortic stenosis treated with TAVI 10 months ago.

Conclusions

Our case suggests that CRT could lead to a significant hemodynamic improvement even in patients with large left ventricular aneurysms, provided the LV lead is placed in a region of viable myocardium. This treatment option should not be withheld in such patients. Even in the absence of improvement of LV systolic function, slowing down remodeling-related decline of EF is important.

References:

1. Bristow MR, Saxon LA, Boehmer J et al: Cardiac-resynchronization therapy with or without an implantable defibrillator in advanced chronic heart failure. N Engl J Med, 2004; 350(21): 2140–50
2. St John Sutton M, Cerkvenik J, Borlaug BA et al: Effects of cardiac resynchronization therapy on cardiac remodeling and contractile function: Results from Resynchronization Reverses Remodeling in Systolic Left Ventricular Dysfunction (REVERSE). J Am Heart Assoc, 2015; 4(9): e002054
3. Cleland JG, Daubert JC, Erdmann E et al: The effect of cardiac resynchronization on morbidity and mortality in heart failure. N Engl J Med, 2005; 352(15): 1539–49
4. Hsu JC, Solomon SD, Bourgoun M et al: Predictors of super-response to cardiac resynchronization therapy and associated improvement in clinical outcome: The MADIT-CRT (multicenter automatic defibrillator implantation trial with cardiac resynchronization therapy) study. J Am Coll Cardiol, 2012; 59(25): 2366–73
5. Franke J, Keppler J, Abadei AK et al: Long-term outcome of patients with and without super-response to CRT-D. Clin Res Cardiol, 2016; 105(4): 341–48
6. Cay S, Ozeke O, Ozcan F et al: Mid-term clinical and echocardiographic evaluation of super responders with and without pacing: The preliminary results of a prospective, randomized, single-centre study. Europace, 2016; 18(6): 842–50
7. van der Heijden AC, Hoke U, Thijssen J et al: Super-responders to cardiac resynchronization therapy remain at risk for ventricular arrhythmias and benefit from defibrillator treatment. Eur J Heart Fail, 2014; 16(10): 1104–11
8. Adelstein EC, Tanaka H, Soman P et al: Impact of scar burden by single-photon emission computed tomography myocardial perfusion imaging on patient outcomes following cardiac resynchronization therapy. Eur Heart J, 2011; 32(1): 93–103
9. Mele D, Agricola E, Monte AD et al: Pacing transmural scar tissue reduces left ventricle reverse remodeling after cardiac resynchronization therapy. Int J Cardiol, 2013; 167(1): 94–101
10. Knappe D, Pouleur AC, Shah AM et al: Acute effects of withdrawal of cardiac resynchronization therapy on left and right ventricular function, dyssynchrony, and contractile function in patients with New York Heart Association functional class I/II heart failure: MADIT-CRT. J Card Fail, 2013; 19(3): 149–55

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

11. Leclercq C, Cazeau S, Le Breton H et al: Acute hemodynamic effects of biventricular DDD pacing in patients with end-stage heart failure. J Am Coll Cardiol, 1998; 32(7): 1825–31
12. Hamdan MH, Barbera S, Kowal RC et al: Effects of resynchronization therapy on sympathetic activity in patients with depressed ejection fraction and intraventricular conduction delay due to ischemic or idiopathic dilated cardiomyopathy. Am J Cardiol, 2002; 89(9): 1047–51
13. Gademan MG, van Bommel RJ, Ypenburg C et al: Biventricular pacing in chronic heart failure acutely facilitates the arterial baroreflex. Am J Physiol Heart Circ Physiol, 2008; 295(2): H755–60
14. Pieragnoli P, Perego GB, Ricciardi G et al: Cardiac resynchronization therapy acutely improves ventricular-arterial coupling by reducing the arterial load: Assessment by pressure-volume loops. Pacing Clin Electrophysiol, 2015; 38(4): 431–37
15. Blanc JJ, Etienne Y, Gilard M et al: Evaluation of different ventricular pacing sites in patients with severe heart failure: Results of an acute hemodynamic study. Circulation, 1997; 96(10): 3273–77
16. DeVecchi F, Facchini E, Degiovanni A et al: Acute contractile recovery extent during biventricular pacing is not associated with follow-up in patients undergoing resynchronization. Int J Cardiol Heart Vasc, 2016; 11: 66–73

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]