SciMedCentral

Annals of Pediatrics & Child Health

Case Report

Echocardiographically-Assisted Pacemaker Optimization in an Infant with Severe Left Ventricular Dysfunction

Biagio Castaldi[1]*, Loira Leoni[2] and, Ornella Milanesi[1]

[1]*Department of Women's and Children's Health, University of Padua, Italy*

[2]*Department of Cardiac, Thoracic and Vascular Sciences, University of Padua, Italy*

***Corresponding author**

Castaldi Biagio, Pediatric Cardiology Unit, Department of Women's and Children's Health, University of Padua – Padua, Italy, via Giustiniani, 3 – 35128 Padova, Italy, Tel: 390498213558; Fax: 390498218089; Email: b.castaldi@yahoo.it

Submitted: 20 September 2016

Accepted: 14 November 2016

Published: 16 November 2016

Keywords

- Cardiac pacing
- Congenital heart disease
- Echocardiography
- Left ventricular dysfunction

Abstract

A male newborn with pre-natal diagnosis of complex congenital heart disease (Taussig-Bing heart) was referred to our Institution for surgical treatment. The procedure was complicated by permanent AV block and consequent implantation of epicardial PMK in DDD mode. Despite the absence of any residual stenosis the patient poorly tolerated oral feeding and LV function progressively worsened. The PMK setting was changed to VVI and the optimal heart rate was chosen by using the left ventricular outflow tract velocity time integral. Twelve days after, the echocardiographic evaluation showed a dramatic improvement of LV shape and function. This case has demonstrated that systolic dysfunction in paced ventricles may be due not only to LV dyssynchrony but also to PMK-induced diastolic dysfunction.

INTRODUCTION

Left ventricular (LV) dyssynchrony caused by permanent right ventricular free wall pacing has been associated with both reversible and irreversible LV dysfunction in children and adults. Biventricular pacing has been proposed to treat congestive heart failure in these patients. Atrioventricular (AV) delay optimization by means of echocardiography is part of the routine treatment of bi-ventricular pacing, however, it is poorly used in right ventricular pacing. Here, we report a case of pacemaker-induced LV dysfunction resolved after echo-guided optimization of the pacemaker (PMK) setting.

CASE PRESENTATION

A male newborn with pre-natal diagnosis of double outlet right ventricle, transposition of the great arteries, ventricular septal defect, sub-aortic obstruction and aortic coarctation (Taussig-Bing heart). His weight at birth was 3.58 kg. Fifteen day after birth he underwent surgical correction consisting in coarctectomy by end-to-end anastomosis, arterial switch procedure, ventricular septal defect closure (trans-atrial approach) and resection of sub-aortic stenosis. The procedure was complicated by permanent AV block and consequent implantation of epicardial PMK in DDD mode (AV delay 120 msec). The leads were implanted on the right atrial wall and on the apex of the left ventricle. The follow-up was characterized by recurrent aortic coarctation, requiring percutaneous balloon dilatation 60 days after surgery. Despite the absence of any residual stenosis the patient poorly tolerated oral feeding and LV function progressively worsened. Seventy days after surgery, LV ejection fraction (EF) calculated by echo using the biplane Simpson's method was of 27%. The daily atrial rate ranged from 110 to 170 (mean 130 bpm). Doppler interrogation revealed that the PMK current setting caused the diastolic time to be too short (27% of total cycle length). Global longitudinal strain (GLS) was severely impaired (-3.4%) and showed a dyskinetic motion of lateral, inferior and posterior walls of LV (Figure 1A and 1B).

RESULTS

Considering the short diastolic time, the PMK setting was changed to VVI and the optimal heart rate was chosen by using the left ventricular outflow tract velocity time integral (LVOT-VTI) by pulsed wave Doppler. The heart rate of 100 bpm was the best setting for the patient, having the highest LVOT-VTI. Even though the GLS was still heavily impaired (-5.4%), only one segment remained dyskinetic (Figure 1C and 1D). One mg/kg/die of Metoprolol was administered to decrease the atrial rate. Twelve days after, the echocardiographic evaluation showed a dramatic improvement of LV shape and function. Compared to the discharge, LVOT-VTI increased from 10.5 to 14.5 cm, GLS increased from -5.4% to -13.3% with no dyskinesia and EF increased from 27% to 60%. The heart failure signs clinically

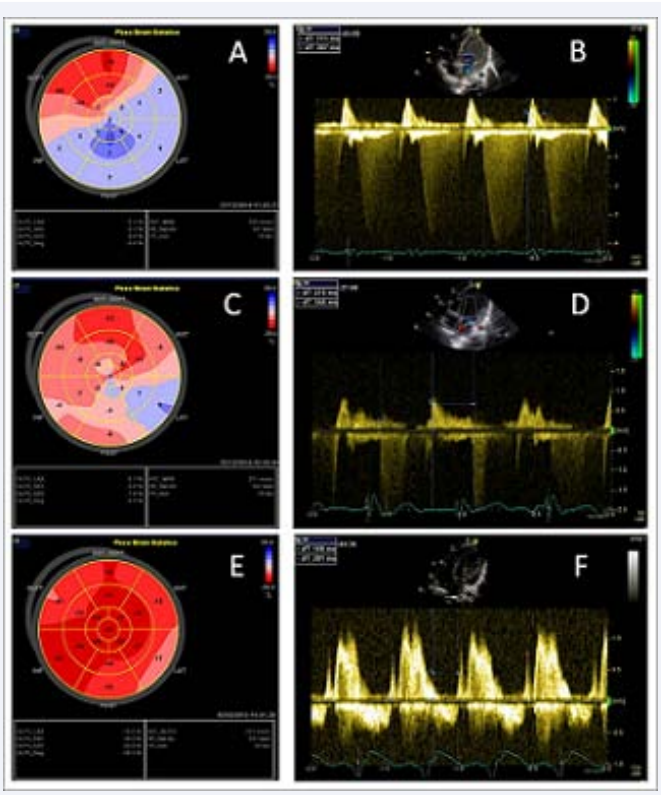

Figure 1 A) In DDD mode, the global longitudinal strain showed a dyskinetic motion of lateral, inferior and posterior walls. B) The duration of diastole was 111 ms, the 27% of the total cycle length. C) In VVI mode, left ventricular function immediately improved. Only the lateral wall was still dyskinetic. D) Diastolic time was 245 ms, the 46% of the cycle length. E) Seventy days after, the patient had a complete recovery of systolic longitudinal function with no dyskinetic segment. F) The duration of diastole was normal (63% of the total cycle length).

disappeared, the patient started to feed orally. Forty days after discharge the body weight was 4.8 kg (+1.0kg), GLS was -17% and EF was 68%. The PMK setting was switched again in DDD mode, the dose of Metoprolol was adjusted for the weight. Seventy days after discharge the weight was 5.85 kg, heart rate was 120 bpm, GLS was - 17.6% and EF was 70% (Figure 1E and 1F). The patient was 20 months old at the last clinical evaluation. His weight was 11.5 kg, he was asymptomatic, the LV-EF was normal, there was a mild residual isthmic stenosis and a mild distortion of both the pulmonary branches due to the Lecompte maneuver. He took Metoprolol 3 mg/kg/daily. The PMK was still in DDD mode, the mean heart rate was 90 bpm, the stimulation was AS-VP in 85% of beats and AP-VP in 15% of beats.

CONCLUSION

Left ventricular dyssynchrony may occur in 5-30% of infants after PMK implantation. The rate of LV dysfunction is higher when the ventricular lead is implanted on the right ventricular free wall, however a LV dyssynchrony can be found by echo in 5 to 15% of patients despite LV epicardial stimulation. The biventricular pacing is the standard care in case of PMK-induced LV dyssynchrony and EF impairment [1,2].

This case has demonstrated that systolic dysfunction in paced ventricles may be due not only to LV dyssynchrony but also to PMK-induced diastolic dysfunction. In our patient, a too short diastolic time was responsible for high LV wall stress and subsequent myocardial ischemia, which contributed itself to the prolongation of systolic time and to the LV dyssynchrony. The reverse remodeling obtained by the PMK setting optimization in spite of the loss of atrial systole contribution clearly demonstrated this issue.

REFERENCES

1. Janousek J, Tomek V, Chaloupecky V, Gebauer RA. Dilated cardiomyopathy associated with dual-chamber pacing in infants: improvement through either left ventricular cardiac resynchronization or programming the pacemaker off allowing intrinsic normal conduction. J Cardiovasc Electrophysiol. 2004; 15: 470-474.

2. Moak JP, Barron KS, Hougen TJ, Wiles HB, Balaji S, Sreeram N, et al. Congenital heart block: development of late-onset cardiomyopathy, a previously underappreciated sequela. J Am Coll Cardiol. 2001; 37: 238-242.

Cite this article

Castaldi B, Leoni L, Milanesi O (2016) Echocardiographically-Assisted Pacemaker Optimization in an Infant with Severe Left Ventricular Dysfunction. Ann Pediatr Child Health 4(4): 1114.