Journal of Cardiology Cases 9 (2014) 170–171

Contents lists available at www.sciencedirect.com

Journal of Cardiology Cases

journal homepage: www.elsevier.com/locate/jccase

Editorial

Editorial: Reappraisal of increasing heart rate for cardiac performance

Keywords:
Cardiac resynchronization therapy
Functional mitral regurgitation
Atrial pacing

Functional mitral regurgitation which deteriorates hemodynamics often develops in patients with systolic heart failure. The functional mitral regurgitation is associated with poor prognosis and is tried to be treated with surgical interventions or trans-catheter interventions [1]. On the other hand, cardiac resynchronization therapy (CRT) is well established for the standard treatment of heart failure [2]. In addition, there are studies showing that CRT may improve functional mitral regurgitation. Although the guidelines [3] suggest the most suitable candidates for CRT are patients with electrocardiogram of "wide QRS complex more than 150 ms and left bundle branch block morphology", a considerable number of patients showed no response to the therapy. Thus, the individual prediction of the effect of CRT is still under discussion.

The study concerning the relation between heart rate and prognosis of heart failure [4] suggested that lowering the heart rate may improve the prognosis of heart failure. Although lowering heart rate is not a sole mechanism of beta-blocker therapy because carvedilol which has a less negative chronotropic effect than bisoprolol [5] is more effective in improvement of prognosis of heart failure with beta-blocker therapy [6,7], the effect of beta-blocker treatment for heart failure relates to the decrease in heart rate [8]. The sub-analysis of the SHIFT study [9] showed that the most effective group was heart failure with tachycardia. The more the heart rate decreases during treatment for acute heart failure, the better the prognosis of heart failure [10]. These studies created the myth of heart rate: "the lower the better".

We should keep in mind that the increase in heart rate in heart failure is a compensatory mechanism for maintaining the hemodynamics, since heart rate is an important factor for increasing cardiac output.

Chikata et al. [11] presented an interesting case of heart failure that showed hemodynamic improvement by atrial pacing in this issue of the journal. According to the previous studies concerning heart failure treatment as mentioned above, this case might have been expected to be improved by CRT, because CRT might have caused reverse remodeling and decrease in functional mitral regurgitation [12]. However, atrial pacing rather than CRT improved the hemodynamics unexpectedly. Although CRT is a standard therapy for heart failure, its effect is not completely predictable because the effect may vary depending on the position of the lead and pattern of dyssynchrony – electrical or contractile. In general, CRT is less effective in ischemic cardiomyopathy than idiopathic dilated cardiomyopathy [13]. In addition, the mechanism of functional mitral regurgitation may vary depending on the global and regional remodeling and distortion of the components of the mitral valve [14].

The increase in heart rate by atrial pacing caused the dramatic improvement in hemodynamics in this case. It was noteworthy that the patient did not show tachycardia, as this report described that the heart rate at admission was 60 bpm, although the patient was admitted because of decompensated acute heart failure. Inappropriate bradycardia may worsen heart failure in the depressed left ventricle, although bradycardia alone does not cause heart failure as long as the left ventricular function is normal. To maintain cardiac output in systolic dysfunction, there are two major compensatory mechanisms, that is, increase in heart rate and increase in stroke volume. To maintain stroke volume, left ventricular volume should be increased in the face of diminished ejection fraction. In this case, left ventricular end-diastolic dimension was 60 mm, which might not be large enough and heart rate was only 60 bpm in congestive heart failure, suggesting inadequate compensatory mechanisms. The akinetic inferior wall might produce deformity of left ventricular geometry causing tethering of mitral valve complex.

The electrocardiogram in this case showed that the duration of QRS complex was 120 ms and the morphology of QRS complex was not typical left bundle branch block where there was tall R′ wave instead of S wave in V_1 lead. The effect of CRT was far less remarkable in patients with QRS duration between 120 ms and 150 ms than those with QRS duration more than 150 ms [15]. This may suggest the ineffectiveness of CRT in this patient. The CRT could improve the electrical dyssynchrony but not the contractile dyssynchrony in a left ventricle composed of a mixture of normal and infarcted or ischemic myocardium. The ventricular pacing may worsen the propagation of the electrical impulse rather than the orthodromic atrio-ventricular conduction.

The authors showed that the atrial pacing alone decreased V wave of pulmonary capillary wedge pressure more than the biventricular pacing. The improvement in functional mitral regurgitation was probably caused by the increase in heart rate with atrial pacing. The increase in heart rate by atrial pacing resulted in increase in cardiac output causing the decrease in left atrial pressure and the decrease in left ventricular size or mitral annulus, which in turn resulted in the reduction of mitral regurgitation. As the authors

DOI of original article: http://dx.doi.org/10.1016/j.jccase.2013.09.009.

http://dx.doi.org/10.1016/j.jccase.2013.11.003

mentioned, the optimal heart rate may vary among individual patients depending on left ventricular systolic function, geometry, and diastolic function. We have to keep in mind that we cannot apply directly the result of a large-scale study to an individual case without questioning whether there are some variant characteristics, although a large-scale study is useful to clarify the scientific and statistical effects of the intervention. The clinician may be required to be able to detect and observe such variant clinical findings from each individual patient.

References

[1] Rossi A, Dini FL, Faggiano P, Agricola E, Cicoira M, Frattini S, Simioniuc A, Gullace M, Ghio S, Enriquez-Sarano M, Temporelli PL. Independent prognostic value of functional mitral regurgitation in patients with heart failure. A quantitative analysis of 1256 patients with ischaemic and non-ischaemic dilated cardiomyopathy. Heart 2011;97:1675–80.

[2] Stevenson WG, Hernandez AF, Carson PE, Fang JC, Katz SD, Spertus JA, Sweitzer NK, Tang WH, Albert NM, Butler J, Westlake Canary CA, Collins SP, Colvin-Adams M, Ezekowitz JA, Givertz MM, et al. Indications for cardiac resynchronization therapy: 2011 update from the Heart Failure Society of America Guideline Committee. J Card Fail 2012;18:94–106.

[3] Brignole M, Auricchio A, Baron-Esquivias G, Bordachar P, Boriani G, Breithardt OA, Cleland J, Deharo JC, Delgado V, Elliott PM, Gorenek B, Israel CW, Leclercq C, Linde C, Mont L, et al. 2013 ESC guidelines on cardiac pacing and cardiac resynchronization therapy: the Task Force on cardiac pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA). Eur Heart J 2013;34:2281–329.

[4] Swedberg K, Komajda M, Bohm M, Borer JS, Ford I, Dubost-Brama A, Lerebours G, Tavazzi L, SHIFT Investigators. Ivabradine and outcomes in chronic heart failure (SHIFT): a randomised placebo-controlled study. Lancet 2010;376:875–85.

[5] Dungen HD, Apostolovic S, Inkrot S, Tahirovic E, Töpper A, Mehrhof F, Prettin C, Putnikovic B, Neskovic AN, Krotin M, Sakac D, Lainscak M, Edelmann F, Wachter R, Rau T, et al. Titration to target dose of bisoprolol vs. carvedilol in elderly patients with heart failure: the CIBISELD trial. Eur J Heart Fail 2011;13:670–80.

[6] Packer M, Bristow MR, Cohn JN, Colucci WS, Fowler MB, Gilbert EM, Shusterman NH. The effect of carvedilol on morbidity and mortality in patients with chronic heart failure. U.S. Carvedilol Heart Failure Study Group. N Engl J Med 1996;334:1349–55.

[7] The Cardiac Insufficiency Bisoprolol Study II (CIBIS-II): a randomised trial. Lancet 1999;353:9–13.

[8] McAlister FA, Wiebe N, Ezekowitz JA, Leung AA, Armstrong PW. Meta-analysis: beta-blocker dose, heart rate reduction, and death in patients with heart failure. Ann Intern Med 2009;150:784–94.

[9] Bohm M, Borer J, Ford I, Gonzalez-Juanatey JR, Komajda M, Lopez-Sendon J, Reil JC, Swedberg K, Tavazzi L. Heart rate at baseline influences the effect of ivabradine on cardiovascular outcomes in chronic heart failure: analysis from the SHIFT study. Clin Res Cardiol 2013;102:11–22.

[10] Takahama H, Yokoyama H, Kada A, Sekiguchi K, Fujino M, Funada A, Amaki M, Hasegawa T, Asakura M, Kanzaki H, Anzai T, Kitakaze M. Extent of heart rate reduction during hospitalization using beta-blockers, not the achieved heart rate itself at discharge, predicts the clinical outcome in patients with acute heart failure syndromes. J Cardiol 2013;61:58–64.

[11] Chikata A, Murai H, Usui S. Successful treatment of functional mitral regurgitation in severe heart failure with atrial pacing. J Cardiol Cases 2014;9:50–3.

[12] Breithardt OA, Sinha AM, Schwammenthal E, Bidaoui N, Markus KU, Franke A, Stellbrink C. Acute effects of cardiac resynchronization therapy on functional mitral regurgitation in advanced systolic heart failure. J Am Coll Cardiol 2003;42:486–94.

[13] Barsheshet A, Goldenberg I, Moss AJ, Eldar M, Huang DT, McNitt S, Klein HU, Hall WJ, Brown MW, Goldberger JJ, Goldstein RE, Schuger C, Zareba W, Daubert JP. Response to preventive cardiac resynchronization therapy in patients with ischaemic and nonischaemic cardiomyopathy in MADIT-CRT. Eur Heart J 2011;32:1622–30.

[14] Magne J, Senechal M, Dumesnil JG, Pibarot P. Ischemic mitral regurgitation: a complex multifaceted disease. Cardiology 2009;112:244–59.

[15] Stavrakis S, Lazzara R, Thadani U. The benefit of cardiac resynchronization therapy and QRS duration: a meta-analysis. J Cardiovasc Electrophysiol 2012;23:163–8.

Makoto Akaishi (MD, PhD, FJCC)*
Department of Cardiology, Kitasato University Kitasato Institute Hospital, Tokyo, Japan

*Correspondence to: Department of Cardiology, Kitasato University Kitasato Institute Hospital, 5-9-1 Shirokane, Minato-ku, Tokyo 108-8642, Japan.
Tel.: +81 03 3444 6161;
fax: +81 03 3448 0553.
E-mail address: akaishim@insti.kitasato-u.ac.jp

14 November 2013