

# Why is it reasonable to pace the Bachmann's bundle? Electrocardiographic and clinical considerations

## Dlaczego warto stymulować pęczek Bachmanna? Rozważania elektrokardiograficzne i kliniczne

Magdalena Sokołowska<sup>1</sup>, Jacek Zawadzki<sup>1</sup>, Aleksandra Gajek<sup>1</sup>, Jakub Adamowicz<sup>2</sup>,  
Jacek Gajek<sup>2</sup>, Magdalena Kliś<sup>3</sup>, Agnieszka Sławuta<sup>3</sup>

<sup>1</sup>Students' Scientific Association, Wrocław Medical University, Wrocław, Poland

<sup>2</sup>Department of Clinical Nursing, Wrocław Medical University, Wrocław, Poland

<sup>3</sup>Department of Cardiology, Kłodzko County Hospital, Kłodzko, Poland

### Abstract

Despite its widely-understood physiological mode, artificial atrial pacing can negatively influence heart performance in many respects. We present the case of an 80-year-old woman with the signs and symptoms of diastolic heart failure which had originated, according to the patient herself, due to the implantation of a dual chamber pacemaker inserted for the treatment of sinus node disease. The atrial electrode was located in the right atrial appendage. Electrocardiographic and echocardiographic assessment indicated the possibility of a deleterious relationship between the atrial and ventricular contraction, which could be responsible for patient's symptoms. Some changes in device programming could contribute to a clinical improvement. Yet the question remains: should pacing of the Bachmann's bundle be implemented in our patient?

Key words: atrial pacing, Bachmann's bundle pacing, diastolic heart failure

Folia Cardiologica 2018; 13, 5: 485–488

### Introduction

The physiological electrical activation of the atria starts from the sinoatrial node located in the right atrium in front of the superior vena cava ostium. The activation spreads anteriorly and inferiorly to the atrioventricular junction and simultaneously to the left atrium using the muscular fibres which form the Bachmann's bundle [1]. Physiological interatrial and atrioventricular conduction delays contribute to the normal filling of the ventricles by atrial contractions. Any pathological delays result in inappropriate atrioventricular mechanical coupling. This is of much greater importance on the left side, because of the higher filling pressure and possible pulmonary congestion [2].

The implantation of an artificial heart pacemaker changes the electrical and mechanical relations, both

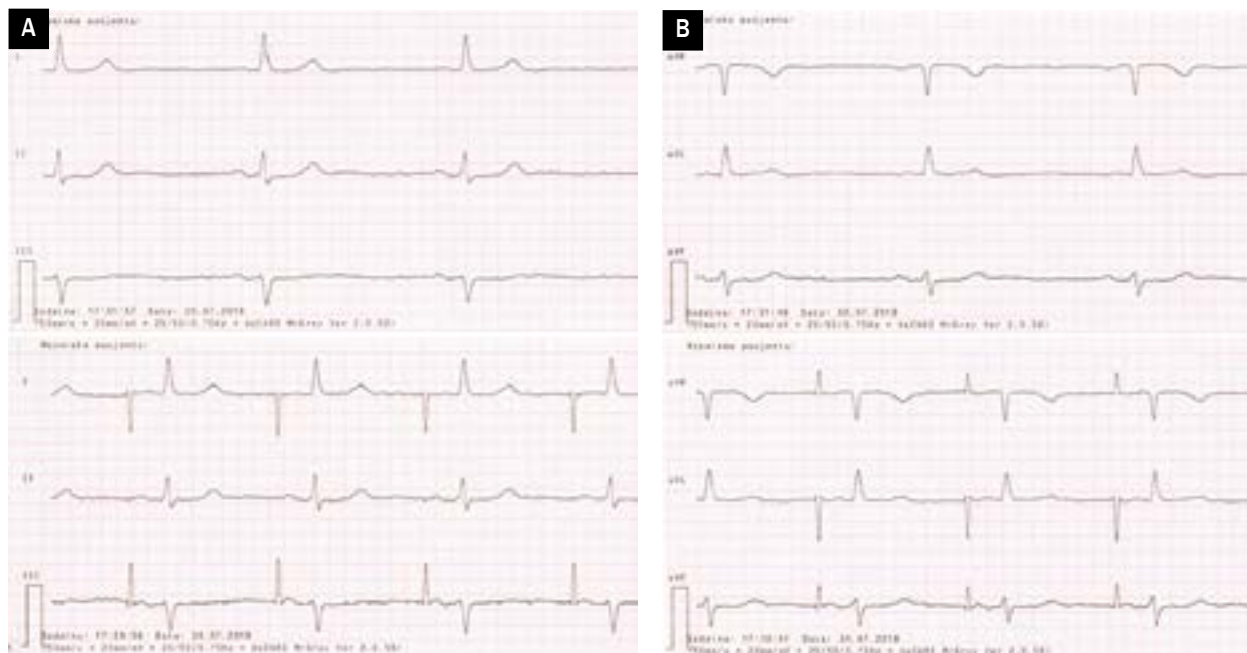
in the atria and the ventricles. Even if the programmed atrioventricular delay is sufficiently long and/or some algorithms to avoid ventricular pacing have been switched on, atrial pacing alone can negatively influence heart performance [3].

We present the case of a female patient with sinus bradycardia implanted with a dual chamber pacemaker, with a complaint of resting and exertional dyspnoea related, in the patient's opinion, to the pacemaker implantation procedure.

### Case report

An 80-year-old woman with arterial hypertension and hypercholesterolemia was assessed in cardiology practice. Two years earlier, a diagnosis of sinus node disease in

Address for correspondence: Jacek Zawadzki, Studenckie Koło Naukowe, Zakład Pielęgniarstwa Internistycznego, Uniwersytet Medyczny we Wrocławiu, ul. Bartła 5, 51–618 Wrocław, Poland, e-mail: jacekzawadzki@gmail.com



**Figure 1.** Six-lead ECG: upper panel – sinus rhythm, lower panel – atrial pacing. Paper speed 50 mm/s, gain 20 mm/1 mV

the form of sinus bradycardia had been made, but the patient was actually asymptomatic. Now she presented with a dual chamber pacemaker which has been implanted eight months before. She complained of dyspnoea, even at rest, and severely limited exercise capacity. Physical examination was unremarkable. Her blood pressure on antihypertensive medication was 125/70 mm Hg, and heart rate was 63 bpm. Electrocardiogram showed unipolar atrial pacing; atrioventricular conduction delay of 250 ms with distorted P-wave indicated right atrial appendage pacing. Echocardiography revealed a relatively small left ventricle of 45 mm in diastole, normal systolic function, a left atrium of 37 mm, mild mitral regurgitation, and slight atrial wave termination by aortic ejection. The pacemaker basic rate was set to 60 bpm with AAI-DDD mode and rate-response mode with a maximal heart rate of 120 bpm. The pacemaker statistics indicated more than 90% of atrial pacing, but less than 1% of ventricular pacing. Slowing down the basic rate to 40 bpm allowed sinus rhythm of 44 bpm to be obtained. A six-lead ECG during sinus rhythm and 60 bpm atrial pacing is shown in Figure 1.

The echocardiographic assessment of the mitral filling at spontaneous rate compared to atrial pacing is shown in Figure 2.

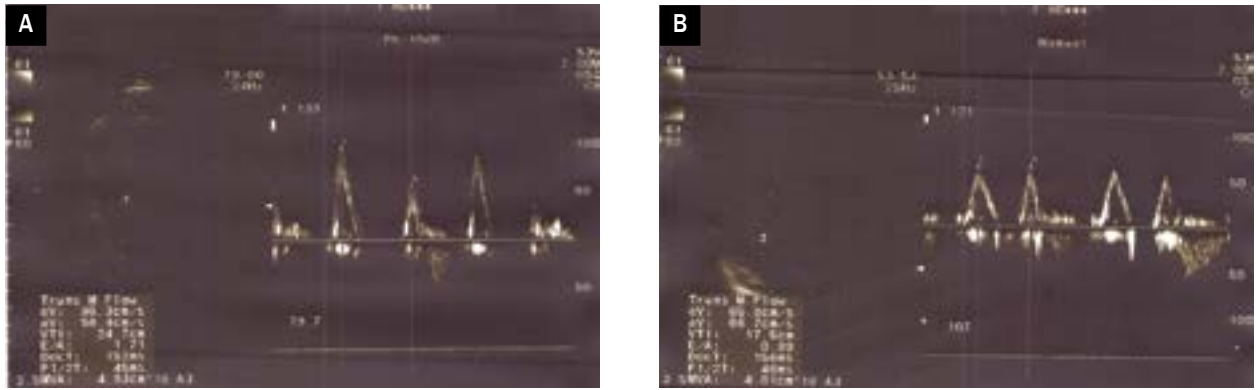
Even the rough estimation of cardiac output during sinus rhythm compared to 60 bpm atrial pacing showed a lower value during pacing, at a relatively good mitral valve area estimation of 4.8–4.9 cm<sup>2</sup>, which could have been responsible for the patient's symptoms.

The pacemaker parameters were changed: the basic rate was lowered to 55 bpm, and the maximal heart rate was set to 85 bpm. A follow-up visit scheduled for two months indicated substantial symptomatic improvement thus the repositioning of the atrial lead was not indicated.

## Discussion

Couple of years ago Bąkowski and Wożakowska-Kapłon [4] have described the case of a patient with compromised systolic function and benign atrioventricular conduction abnormalities in the form of 1<sup>st</sup> degree atrioventricular block implanted with a dual chamber ICD. Our case represents another example of the possibly deleterious effects of the right atrial appendage pacing, and expands the issue to include patients with normal left ventricular function. In our patient, the initial indication for pacemaker implantation was sinus bradycardia. The ejection fraction was totally normal, and there was no problem with the ventricular pacing as the atrioventricular conduction was normal as well. And, even in this case, the historical, non-physiological location of the atrial pacing electrode was able to induce the signs and symptoms of left ventricle heart failure.

Recently, Eicher et al. [5] introduced the concept of atrial dyssynchrony syndrome as inappropriate atrioventricular mechanical coupling. This phenomenon was related to the prolonged interatrial activation which resulted in making the left atrium contract at the same time as the mitral valve closed. The subsequent increase of atrial pressure



**Figure 2.** Mitral filling profile: **A.** Sinus rhythm; **B.** Atrial pacing

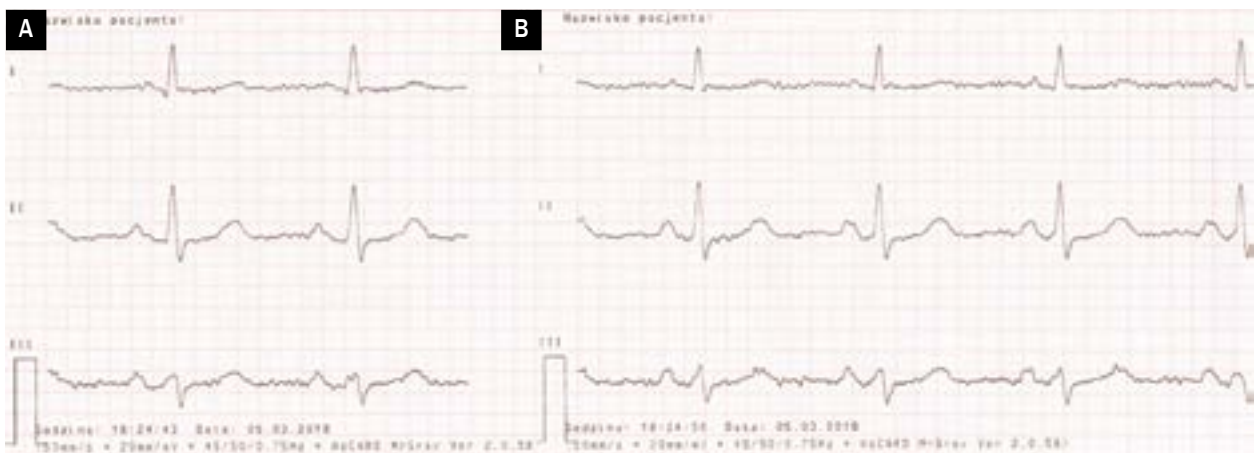
contributed to a reversed flow of the blood and pulmonary congestion, which resulted in dyspnoea. The described mechanism is less frequent than the one originating from the 1<sup>st</sup> degree atrioventricular block haemodynamics, but the result is more or less the same.

Even if atrial pacing has been considered for a long time on physiological grounds, the appropriate location of the atrial electrode can be of crucial importance [6]. The inferior and more lateral positions of the initial atrial electrical activation contribute to prolongation of the P-wave in the electrocardiogram and to inappropriate mechanical function of the atrial pump [7]. Both these factors constitute a well-established mechanical and electrical cause of atrial fibrillation. The introduction of active-fixation atrial pacing has enabled unrestricted atrial lead positioning. There have been a series of studies looking at this issue, indicating the Bachmann's bundle pacing to be the most physiological location of the atrial electrode [8, 9]. The

electrical activation of the right atrium roof, and the high interatrial septum area, are the best mimic for physiological activation spreading from the sinus node.

The implantation of a pacing lead into the right atrial appendage causes prolongation of interatrial and atrioventricular conduction. If both delays express the same degree/percentages, left atrium contraction occurs at more or less the same time of the ventricular mechanical cycle, contributing properly to the left ventricular filling. If the atrioventricular conduction delay predominates, the mitral filling time shortens. If the interatrial conduction delay predominates, the atrial systole collides with the mitral valve closure. Those both last circumstances result in inappropriate left ventricular filling, and could negatively influence the patient's status.

Despite some programming changes in our patient in order to correct the obvious fault, and the maximal rate of 120 bpm in an 80-year-old patient, the question remains:



**Figure 3.** Three-lead ECG. Example of sinus rhythm (A) and slightly faster Bachmann's bundle bipolar pacing (B). Note the shortening of P-wave duration and atrioventricular conduction

should the position of the atrial electrode be changed to the more physiological one if the symptoms persist?

The Bachmann's bundle pacing provides another advantage, as described by our team recently. If the activation of the Bachmann's bundle occurs slightly anteriorly, it facilitates atrioventricular conduction [10]. In the setting of a longer atrioventricular conduction, even the small shortening of atrioventricular conduction can be advantageous in many respects. It comes as a surprise to us that nobody has raised this problem to date...

The appropriate ECG example of sinus rhythm and bipolar Bachmann's bundle pacing is shown in Figure 3.

## Conclusions

The true physiological atrial pacing should be defined as the Bachmann's bundle pacing. Right atrial appendage pacing can negatively influence left atrioventricular mechanical coupling. This could be the explanation for our patient's decreased exercise capacity, and the symptoms of left-sided heart failure.

## Conflict of interest

The authors declare no conflict of interest.

## Streszczenie

Stymulacja przedsionkowa może wpływać negatywnie na pracę serca mimo jej pierwotnie fizjologicznego charakteru. Zaprezentowano przypadek 80-letniej kobiety z objawami rozkurczowej niewydolności serca, które – według samej pacjentki – mają związek czasowy z implantacją z powodu choroby węzła zatokowego dwujamowego stymulatora serca. Elektroda przedsionkowa została umiejscowiona typowo w uszku prawego przedsionka. Elektro- oraz echokardiograficzna ocena pacjentki wykazały nieprawidłową relację czasową skurczu przedsionka i komory, co mogło być przyczyną objawów klinicznych. Optymalizacja programowania urządzenia może częściowo poprawić stan kliniczny pacjentki, jednak wciąż pozostaje pytanie, czy należy dążyć do zmiany lokalizacji elektrody przedsionkowej i zastosować stymulację pęczka Bachmanna w opisanym przypadku?

Słowa kluczowe: stymulacja przedsionkowa, stymulacja pęczka Bachmanna, niewydolność serca rozkurczowa

Folia Cardiologica 2018; 13, 5: 485–494

## References

1. Braunwald E, Bonow RO. Braunwald's heart disease: a textbook of cardiovascular medicine. Elsevier Saunders, Philadelphia 2012.
2. Rein AJ, O'Donnell CP, Colan SD, et al. Tissue velocity Doppler assessment of atrial and ventricular electromechanical coupling and atrioventricular time intervals in normal subjects. *Am J Cardiol.* 2003; 92(11): 1347–1350, indexed in Pubmed: [14636920](#).
3. Wang K, Xiao HB, Fujimoto S, et al. Atrial electromechanical sequence in normal subjects and patients with DDD pacemakers. *Br Heart J.* 1995; 74(4): 403–407, indexed in Pubmed: [7488455](#).
4. Bąkowski D, Woźakowska-Kapton B. Blok przedsionkowo-komorowy I stopnia u chorego z dwujamowym kardiowerterem-defibrylatorem – pozornie błahy problem. *Folia Cardiol.* 2016; 11(1): 75–79, doi: [10.5603/fc.2016.0010](#).
5. Eicher JC, Laurent G, Mathé A, et al. Atrial dyssynchrony syndrome: an overlooked phenomenon and a potential cause of 'diastolic' heart failure. *Eur J Heart Fail.* 2012; 14(3): 248–258, doi: [10.1093/eurjhf/hfr169](#), indexed in Pubmed: [22291437](#).
6. Kliś M, Sławuta A, Gajek J. Antiarrhythmic properties of atrial pacing. *Adv Clin Exp Med.* 2017; 26(2): 351–357, doi: [10.17219/acem/61429](#), indexed in Pubmed: [28791857](#).
7. Miyazaki H, Noma K, Date T, et al. Atrial septal pacing to resynchronize atrial contraction and improve atrial transport function. *J Cardiol.* 2005; 45(6): 239–246, indexed in Pubmed: [15991607](#).
8. Bailin SJ, Adler S, Giudici M. Prevention of chronic atrial fibrillation by pacing in the region of Bachmann's bundle: results of a multicenter randomized trial. *J Cardiovasc Electrophysiol.* 2001; 12(8): 912–917, indexed in Pubmed: [11513442](#).
9. Kristensen L, Nielsen JC, Mortensen PT, et al. Sinus and paced P wave duration and dispersion as predictors of atrial fibrillation after pacemaker implantation in patients with isolated sick sinus syndrome. *Pacing Clin Electrophysiol.* 2004; 27(5): 606–614, doi: [10.1111/j.1540-8159.2004.00494.x](#), indexed in Pubmed: [15125716](#).
10. Sławuta A, Kliś M, Skoczyński P, et al. Bachmann's bundle pacing not only improves interatrial conduction but also reduces the need for ventricular pacing. *Adv Clin Exp Med.* 2016; 25(5): 845–850, doi: [10.17219/acem/63351](#), indexed in Pubmed: [28028946](#).