

## Should we opt at once for biventricular stimulation in case of atrial standstill?

## Faut-il opter d'emblée pour la stimulation biventriculaire en cas de paralysie auriculaire ?

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### Résumé

**Introduction:** La paralysie auriculaire permanente est une maladie cardiaque rare dont le diagnostic est basé des critères de présomption, principalement une bradycardie en absence d'ondes P à l'électrocardiogramme et un échec de stimulation à l'étage atrial. **Observation:** Nous rapportons le cas d'un jeune patient, admis dans notre service pour syncope. L'électrocardiogramme a montré une bradycardie à une vitesse de 45 cycles / min et aucune onde P. L'échocardiographie a révélé une fonction ventriculaire gauche normale. Nous avons conclu à un bloc sino-auriculaire de haut degré. Un pacemaker double chambre a été indiqué, mais nous n'avons pas réussi à stimuler l'oreillette. Nous avons conclu à une paralysie auriculaire et une stimulation ventriculaire droite a été réalisée. Quelques mois plus tard, le patient a été hospitalisé pour une insuffisance cardiaque globale. L'échocardiographie a montré une cardiomyopathie dilatée avec une dysfonction ventriculaire gauche estimé à 20% et une insuffisance mitrale grade II-III d'allure fonctionnelle. Bien qu'un traitement médical optimal a été instauré, le patient est resté symptomatique. Une stimulation bi-ventriculaire a été réalisée avec une amélioration clinique et échocardiographique notable.

**Conclusion:** Compte tenu du risque d'altération de la fonction ventriculaire gauche associée lors d'une stimulation ventriculaire apicale, pourrait-on indiquer immédiatement la stimulation bi-ventriculaire chez les patients avec une paralysie auriculaire permanente? D'autres études sont nécessaires.

### Mots-clés

Oreillette, paralysie, stimulation, dyspnée

### Summary

**Introduction:** The permanent atrial paralysis is a rare cardiac disease which diagnosis is based on many criteria, mainly a bradycardia with lack of P waves on electrocardiogram and a failure of atrium stimulating.

**Observation:** we report the case of young patient, admitted to our department for syncope. The electrocardiogram showed a bradycardia at a rate of 45 cycles/min and no P waves. The echocardiography revealed a normal left ventricular function. We concluded at a high-degree sinoatrial block. So a dual chamber pacing was indicated, but we failed to stimulate the atrium. We concluded at an atrial paralysis and a right ventricular pacing was achieved. Few months again, the patient was hospitalized for a global heart failure. The echocardiography showed a dilated cardiomyopathy with a left ventricular dysfunction estimated at 20% and a grade II-III mitral insufficiency. Although an optimal medical treatment, the patient remained symptomatic of dysnea (NYHA class 3). A biventricular pacing was performed with clinic and echocardiographic improvement.

**Conclusion:** Considering the risk of left ventricular impairment associated to right ventricular apical pacing, could we indicate straightaway biventricular stimulation in patients with permanent atrial standstill? More studies are needed.

### Keywords

Atrium, paralysis, pacing, dyspnea

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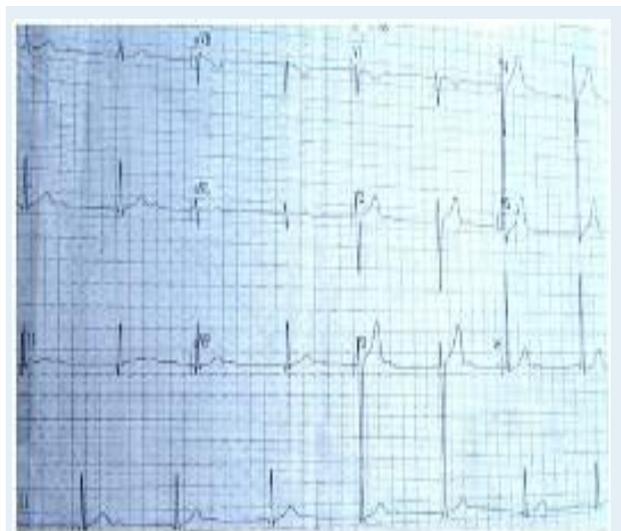
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## INTRODUCTION

The permanent atrial standstill is an uncommon arrhythmia with an incidence of 1/125000<sup>1</sup>. Its current diagnostic criteria include mainly a junctional bradycardia with absent P waves besides the lack of response of the atrium to direct electrical stimulation. That's why, in some cases the diagnosis is carried out only during pacing failure. The physician opts in many times to perform a right ventricular pacing, but there is growing body of literature suggesting that this mode of pacing could deteriorate the left ventricular (LV) performance and make the management of these patients more difficult. We report the observation of an adolescent with an atrial paralysis in whom the RV pacing was complicated of a dilated cardiomyopathy. The goal of this observation is to bring out the impact of biventricular stimulation in patients with atrial paralysis.

## OBSERVATION

A 19 years old adolescent without personal or family history of cardiac diseases, was admitted to our department for a recurrent syncope (3 episodes) occurring at rest. He had never complained of any symptom, beforehand and he didn't take any medication. The physical examination revealed a bradycardia at 45 beats/min ; the blood pressure was measured to be 140/80mmHg. No anomalies were noted on cardiac or pulmonary auscultation. The neurologic exam was normal. The electrocardiogram showed a marked bradycardia at a rate of 29cycles/min, a regular rhythm and no P waves, the QRS duration was about 110ms (figure 1).



**Figure 1:** atrial standstill regular junctional rhythm without P wave

The biologic tests were correct especially the blood potassium level. We noted on echocardiography, a good left ventricular function (ejection fraction=60%) but atriums were few moving. Thus we concluded at a sinoatrial block complicated of syncope and we decided to implant a dual chamber pacemaker. But during the procedure, the atrial stimulation had failed among all the atrial area.

Considering the electric features and the lack of response to atrial stimulation we suggested a permanent atrial standstill in our patient. Thus, a right ventricular pacing was achieved in July2003.

The patient had being asymptomatic during two years. In October 2005, he was hospitalized for dyspnea. The exam revealed symptoms of global heart failure. The electrocardiogram was stimulated. There was an important cardiomegaly with a biventricular dilatation on chest radiography (figure 2).



**Figure 2:** Biventricular dilatation on chest ray

We noted at the echocardiography a dilated cardiomyopathy (LV end diastolic diameter=70mm), the ejection fraction was estimated at 20% and an enlarged right cavity too (RV diameter/LV diameter>1).The septum had a paradoxal motion and there was a grade II-III mitral regurgitation. A biventricular pacing was performed: one electrode was implanted into the coronary sinus (left lateral vein), the other on the right ventricular apex. A spectacular improvement was so obtained. The patient became asymptomatic 2 months later. In July 2006, the echocardiography showed a left ventricle fraction about 40%, a LV end diastolic diameter =61mm and in December 2009 a LVEF=50% with a grade I mitral regurgitation.

## DISCUSSION

The atrial paralysis is a rare clinical entity, described in the literature as cases reports or small groups. This arrhythmia is classified into two types according to the duration: temporary and persistent. The last one is a rather rare event.

The temporary form is caused by marked sinus arrhythmia, drug or Carbon dioxide toxicity, Hyperpotassaemia, Anoxia, myocardial infarction ...while persistent form is known to happen in patients with longstanding Cardiac disease, diabetes mellitus, amyloidosis, or Ebstein's anomaly and in 30% of patients with Emery- Dreifuss muscular dystrophy (EDMD)<sup>2</sup>.

The diagnostic criteria of this last disorder includes: 1) Absence of "P" waves on electrocardiograms; 2) Absence of "A" waves on jugular venous pulse and right atrial pressure tracings; 3) Supraventricular QRS complex; 4) Immobility of the atria on fluoroscopy and/or angiography; 5) Inability to electrically stimulate the atria.

Theoretically, it would appear that in permanent atrial paralysis, the stimulation is always unsuccessful, because the abnormality was generalized throughout the atria and was explained by a fibro-fatty degeneration of the whole atrial muscle<sup>3</sup>. This mechanism apparently differs from that of transient atrial standstill which is results from depolarization of atria and subsequent lack of responsiveness to electrical stimulation could be corrected by reversing the underlying cause or by the administration of small amounts of isoproterenol<sup>4</sup>.

The failure of the atrial stimulation in these patients obliges the physician to implant in most cases a right ventricular pacemaker when the left ventricular function is preserved. But many studies have demonstrated that this pacing mode is deleterious for left ventricular function, as it had happen in our patient.

In the CTOPP (Canadian trial of physiologic pacing)<sup>5</sup>, the annual rate of heart failure hospitalization was 3.5% in patients with ventricular pacing. In that patient population, approximately 70% of patients had normal ventricular function and 65% were in NYHA class 1 at baseline. In a study performed by Thakray et al.<sup>6</sup> including 225 patients randomized to either single chamber atrial pacing or single chamber ventricular pacing, the left ventricular shortening decreased significantly in the ventricular group but not in the atrial group. There was also an increase in the dilatation of the left atrium in the ventricular paced patients. Kachboura et al.<sup>7</sup> showed also in a prospective study enrolled on 43 patients a significant decrease of LVEF (60±6 % versus 51±13 %, p = 0,0002) and 25% of patients developed a significant left ventricular dysfunction (LVEF<40%).

The ventricular asynchrony is the main cause of LV impairment. This inhomogeneity of ventricular activation

could affect the normal functioning of the mitral apparatus potentially causing mitral insufficiency, too. It also disorders diastolic function and diastolic filling times<sup>8</sup>. Furthermore, there are metabolic effects of RV apical pacing: first it could decrease regional myocardial flow and perfusion; second, it could increase the serum catecholamine levels with deleterious long-term effects on functional status and survival. In some studies we have even demonstrated the histological myocardial changes in left ventricular explaining the failure of this cavity after pacing<sup>7</sup>.

Formerly, RV outflow tract pacing has been suggested to preserve cardiac function but comparative studies of the two modes failed to show improvements in functional status or in ventricular performance. Recent studies<sup>9</sup> demonstrated that biventricular pacing is able to correct pathologic mechanical ventricular asynchrony, and so it could be an alternative to prevent and to treat iatrogenic heart failure after pacing. Prospective trials evaluating biventricular pacing in this setting are ongoing.

The Post AV Nodal Ablation Evaluation (PAVE) trial<sup>10</sup> concluded that biventricular pacing in patients having chronic AF who underwent atrioventricular nodal ablation preserved better LVEF compared to RV pacing. In fact, the biventricular stimulation was successful in our patient, he became asymptomatic and the left ventricular systolic function was clearly improved. That proves the benefit of this mode of pacing.

## CONCLUSION

Atrial paralysis is an uncommon cardiac disease. The permanent entity usually need pacing but considering the risk of left ventricular dysfunction associated to RV apical pacing, could we indicate straightaway a biventricular stimulation? Therefore, that postulation needs many studies comparing the two modes of pacing in such patients.

### Competing interests

The authors declare no competing interests.

### Author's contribution

All the authors have contributed to the manuscript.

## REFERENCES

1. Combs.D, Bellaci.H, Shively.H And Gregoratos. G. Persistent atrial standstill. *Am J Med* 1974;56:231-3.
2. Wożakowska-Kapłon B, Bąkowski D. Atrial paralysis due to progression of cardiac disease in a patient with Emery-Dreifuss muscular dystrophy. *Cardiol J.* 2011;18(2):189-93.
3. Bensaid J. Persistent atrial standstill documented over a 22-year period *Am Heart J* February 1996; 131:404-7
4. Marini M, Arbustini E, Disertori M. Atrial standstill: a paralysis of cardiological relevance. *Ital Heart J Suppl.* 2004 Sep;5(9):681-6.
5. Skanes AC, Krahn AD, Yee R, et al. Canadian Trial of Physiologic Pacing. Progression to chronic atrial fibrillation after pacing: the Canadian Trial of Physiologic Pacing. CTOPP Investigators. *J Am Coll Cardiol* 2001;38(1):167-72.
6. Thackray S, Witte K, Nikitin N, et al. The prevalence of heart failure and asymptomatic left ventricular systolic dysfunction in a typical regional pacemaker population. *Eur Heart J* 2003;24:1143-52.
7. S. Kachboura , A. Ben halima, I. Fersi, S. Marrakchi, W. Zouaoui, I. Kammoun Assessment of heart failure and left ventricular systolic dysfunction after cardiac pacing in patients with preserved left ventricular systolic function *Ann de Cardiol et d'Ang* 57 2008: 29-36
8. Betocchi S, Piscione F, Villari B, et al. Effects of induced asynchrony on left ventricular diastolic function in patients with coronary artery disease. *J Am Coll Cardiol* 1993;21:1124-31 Giudici MC, Thornburg GA, Buck DL, et al. Comparison of right ventricular outflow tract and apical lead permanent pacing on cardiac output. *Am J Cardiol* 1997;79:209.
9. Nielson JC, Bottcher M, Nielson TT, et al. Regional myocardial blood flow in patients with sick sinus syndrome randomised to long-term single chamber atrial or dual chamber pacing effect of pacing mode and rate. *J Am Coll Cardiol* 2000;(35):1453-61.
10. Daoud E, Doshi R, Fellows C, et al. Ablate and Pace with cardiac resynchronization therapy for patients with reduced ejection fraction: Sub analysis of PAVE study. *Heart Rhythm* 2004;1:5-59..