



Caterina Chiara De Carlini<sup>1\*</sup>, Ester Meles<sup>1</sup>, Roberto Galbiati<sup>1</sup>, Marco Di Sabato<sup>1</sup>, Gaetano Gentile<sup>1</sup>, Andrea Farina<sup>2</sup> and Stefano Maggolini<sup>1</sup>

<sup>1</sup>Division of Cardiology, Ospedale S. Leopoldo Mandic, Merate (LC), Italy

<sup>2</sup>Division of Cardiology, Ospedale A. Manzoni, Lecco, Italy

**Dates:** Received: 22 February, 2017; Accepted: 15 March, 2017; Published: 16 March, 2017

**\*Corresponding author:** De Carlini Caterina Chiara, Cardiology Unit, SL Mandic Hospital, Largo Mandic 1, Merate (Lecco), Italy, Tel: 039-039-5916521; Fax: 039-039-5916471; E-mail: caterina.decarlini@libero.it

**Keywords:** Sinoatrial blocks, Hyperthyroidism, Beta-blockers

<https://www.peertechz.com>

## Case Report

# Sinoatrial Blocks in Hyperthyroidism Treated with Beta-Blockers: A Case of Paradoxical Treatment

## Abstract

We describe an interesting case of a 52 year-old patient affected by thyrotoxicosis crisis, in whom sinoatrial blocks were associated with syncope and treated efficiently with beta-blockers.

We hypothesized the Bezold-Jarisch reflex as the mechanism which leads to bradycardia and hypotension with syncope related to prolonged tachycardia.

This hypothesis has not been, to our knowledge, considered in previous studies and offers therapeutic methods, as the patient was not treated with permanent pacing.

## Introduction

Hyperthyroidism is commonly associated with sinus tachycardia and atrial fibrillation. A less known complication of thyrotoxicosis is impaired atrio-ventricular conduction. Complete atrio-ventricular block complicating hyperthyroidism has generally been described in patients with additional risk factors such as infections, drugs or electrolyte imbalance. In few of them, however, thyrotoxicosis was the only cause of atrio-ventricular block [1]. We describe the case of a patient with thyrotoxic crisis in whom sinoatrial blocks were associated with syncope.

## Case Report

A 52 year-old woman, without history of cardiovascular diseases, was referred to the emergency department for an episode of loss of consciousness.

Visited in Emergency Room she presented two other episodes of syncope, with pallor and marked cardiac sinus bradycardia, followed by vomit and sudden recovery of consciousness. In the second episode the patient was laying. The patient was then hospitalized in the Cardiology Division for cardiac monitoring.

Phases of sinus tachycardia followed by sinus bradycardia were recorded with telemetric surveillance.

An echocardiogram showed mild left ventricular hypertrophy and normal systolic function.

Physical examination revealed a smooth diffuse goitre.

Thyroid function tests showed suppressed thyrotropin (TSH) and high levels of FT<sub>3</sub> (7,1 pg/ml), FT<sub>4</sub> (4,1 ng/dl, normal value 0,8-2,7 ng/dl), and Ab antithyroperoxidase (309 U/ml). Methimazole was started immediately. During the following days she experienced several episodes of syncope with critical asystolic pauses (maximum 9 seconds) and critical sinus bradycardia demonstrated by electrocardiographic continuous monitoring. A temporary transvenous, cardiac pacemaker was then inserted.

Subsequently stable sinus tachycardia was registered.

Due to this fact, therapy with beta-blockers (propranolol), starting with a dose of 40 mg for three times per day, was introduced keeping the temporary pacing. In the following days the dose of beta-blockers, for the control of cardiac frequency, was gradually increased to a dose of 80 mg three times per day.

The heart blocks and the critical bradycardia were resolved. According to the clinical and rhythm stability, the patient was discharged with an endocrinological and cardiological follow up, without a permanent pacing.

During the follow-up she became clinically and biochemically euthyroid and serial ecg holter monitoring did not register any critical bradyarrhythmias. After a follow-up of two years, she was discharged without needing of permanent pacing.

## Discussion

Although cardiovascular manifestations are presenting features in hyperthyroidism, conduction disturbances are less

common, but potentially more serious [1]. A few cases have been described during thyrotoxicosis [2-4], and all the patients were treated with antithyroid treatment, but in a few cases permanent pacing was needed.

Several hypotheses have been proposed to explain these disturbances such as focal myocarditis of the cardiac conducting system, autoimmune reaction that affects the cardiac conducting tissue and metabolic effect that may also cause conduction disturbances since thyroid hormones directly influence cardiac electrophysiological function [5].

In our case it is important to underline the rapid regression of sinoatrial blocks with the use of beta-blockers and with the consequent reduction of the heart rate. This observation is not in favour of the hypothesis of a direct effect of hyperthyroidism on sinoatrial node in consideration of the rapid regression of blocks after the introduction of beta-blocker and also considering the number of days necessary to normalize blood levels of TSH through thyrotoxicosis treatment. The onset of the action of thionamide therapy is indeed slow because it blocks the biosynthesis rather than the release of thyroid hormones [6]. We hypothesized that the mechanism that causes sinoatrial blocks in our case could be the Bezold-Jarisch reflex (BJR), exalted by the likely long duration of tachycardia. This reflex originates in cardiac sensory receptors sited in the left ventricle, particularly in the inferoposterior wall, with nonmyelinated vagal afferent pathways. In hyperthyroid women or in patients with sub-clinical hyperthyroidism, structural cardiac changes on the left ventricle were demonstrated: a reduction in end diastolic ventricular volume and a concentric hypertrophy, with an increased left ventricular mass index. These data suggest the presence of left ventricular diastolic dysfunction. In 1996 [7], Biondi et al. demonstrated that in patients with acclimated hyperthyroidism there is an increased left ventricular mass index caused by the growth of interventricular septum's and posterior wall thickness [7]. The stimulation of these receptors promotes bradycardia, vasodilation and hypotension reflexes. Previous studies observed that a pre-treatment with oral beta-blockers prevents the Bezold-Jarisch reflex, preventing tachycardia that precedes reflex stimulation and consequently

bradycardia and hypotension up to syncope [8]. On the other side there is a study demonstrating that the absence of tachycardia during head-up tilt test is predictive of beta-blockers therapy failure in patients with neurocardiogenic syncope [9]. The hypothesis that in thyrotoxicosis with sustained tachycardia the BJR is the cause of sinoatrial blocks is confirmed by their rapid and permanent regression after treatment with beta-blockers. This hypothesis has not been, to our knowledge, considered in previous studies and it offers not only interesting pathophysiological, but also therapeutic suggestions. Moreover the patient was not treated with permanent pacing.

## References

1. Atri SK, Chugh SN, Goya S, Chugh K (2011) Reversible atrioventricular blocks in thyroid storm. *J Assoc Physicians India* 59: 178-179. [Link: https://goo.gl/81Mv97](https://goo.gl/81Mv97)
2. Kramer MR, Shilo S, Hershko C (1985) Atrioventricular and sinoatrial block in thyrotoxic crisis. *Heart J* 54: 600-602. [Link: https://goo.gl/Z2nXVk](https://goo.gl/Z2nXVk)
3. Ho SC, Eng PHK, Ding ZP, Fok ACK, Khoo DHC (1998) Thyroid Storm Presenting as Jaundice and Complete Heart Block. *Ann Acad Med Singapore* 27: 748-751. [Link: https://goo.gl/dAjaR](https://goo.gl/dAjaR)
4. Muggia AL, Stjernholm M, Houle T (1970) Complete Heart Block with Thyrotoxic Myocarditis-Report of a Case. *N Engl J Med* 283: 1099-1100. [Link: https://goo.gl/Dx01IB](https://goo.gl/Dx01IB)
5. Yusoff K, Khalid BA (1993) Conduction abnormalities in thyrotoxicosis-a report of three cases. *Ann Acad Med of Singapore* 22: 609-612. [Link: https://goo.gl/WBY38w](https://goo.gl/WBY38w)
6. (2002) *Endocrine Pharmacotherapy Module: thyroid Section*, Spring. [Link: https://goo.gl/a41FFk](https://goo.gl/a41FFk)
7. Biondi B, Calmieri EA, Lombardi G, Fazio S (2002) Effects of subclinical thyroid dysfunction on the heart. *Ann Intern Med* 137: 904-914. [Link: https://goo.gl/avENEU](https://goo.gl/avENEU)
8. Cox MM1, Perlman BA, Mayor MR, Silberstein TA, Levin E, et al. (1995) Acute and Long-term beta-adrenergic blockade for patients with neurocardiogenic syncope. *J Am Coll of Cardiol* 26: 1293-1298. [Link: https://goo.gl/XCOr0S](https://goo.gl/XCOr0S)
9. Leor J, Rotsteinz Z, Vered Z, Kaplinsky E, Truman S, et al. (1994) Absence of tachycardia during tilt test predicts failure of beta-blocker therapy in patients with neurocardiogenic syncope. *Am Heart J* 127: 1539-1543. [Link: https://goo.gl/bqT2Bt](https://goo.gl/bqT2Bt)