

Brunella Belli *, Antonio Michelucci **, Domenico Monizzi **,
Giuseppe Barletta **, Giandomenico Morace *,
Luigi Padeletti **, Giorgio Morace *

* *Istituto di Clinica Medica II,*

** *Cattedra di Malattie dell'Apparato Cardiovascolare, Università degli Studi, Firenze*

Reversible cardiomyopathy following chronic supraventricular tachycardia

Estratto da:

CARDIOLOGIA

Organo Ufficiale della Società Italiana di Cardiologia

Vol 33 - N 7 - Luglio 1988

Reversible cardiomyopathy following chronic supraventricular tachycardia

Brunella Belli*, Antonio Michelucci**, Domenico Monizzi**, Giuseppe Barletta**, Giandomenico Morace*, Luigi Padeletti**, Giorgio Morace*

*Istituto di Clinica Medica II, **Cattedra di Malattie dell'Apparato Cardiovascolare, Università degli Studi, Firenze

CARDIOMIOPATIA REVERSIBILE INDOTTA DA TACHICARDIA SOPRAVENTRICOLARE CRONICA. DESCRIZIONE DI UN CASO

Riassunto. Viene descritto un caso di tachicardia giunzionale reciprocante persistente con segni di cardiomiopatia congestizia. Subito dopo il ripristino del ritmo sinusale, in seguito a terapia farmacologica, la funzione cardiaca ritornava normale. La disfunzione del ventricolo sinistro potrebbe dipendere da alterazioni del metabolismo cardiaco indotte dalla lunga durata della tachicardia. (CARDIOLOGIA 1988; 33 (7): 709-711)

Key words: Tachicardia sopraventricolare cronica; Cardiomiopatia congestizia.

Introduction

It has been reported that the permanent form of junctional reciprocating tachycardia can be responsible for congestive heart failure¹⁻⁴.

This report describes 1 case of incessant junctional reciprocating tachycardia in which clinical and instrumental signs of congestive cardiomyopathy coexisted. When the arrhythmia could be terminated by antiarrhythmic drugs and sinus rhythm persisted, a regression of cardiomyopathy was evident.

Case report

The patient was a 22 year old man, who had in his infancy episodes of supraventricular tachycardia. During the previous 4 years he presented palpitations, dyspnea and at intervals, loss of consciousness on exertion. The patient had no evidence of valvular heart disease, hypertension, alcohol abuse nor any systemic illness known to be associated with cardiomyopathy. The pulse was tachycardic (heart rate 110/min).

On ECG the morphology of the QRS complex was normal, the P wave (P') was negative in II, III and aVF. The interval between the QRS complex and the subsequent P' wave (RP') was longer than the interval between the P' wave and the subsequent QRS complex (P'R).

It could be observed by Holter monitoring that the tachycardia was nearly incessant during the diurnal hours while it became less frequent during the night. The arrhythmia could be terminated temporarily by vagal maneuvers. The tachycardia began without an increase in the PR interval and was preceded by a critical reduction of sinus cycle length. Moreover at the beginning of tachycardia the interval between the QRS complex and the subsequent P' wave increased over the first few cycles. The P' wave of the first beat of the tachycardia was identical to the following P' waves, when the tachycardia ended with a P' wave, this was preceded by an increase of P'R interval (Fig 1). The AV conduction was constantly 1:1.

During the electrophysiologic testing the PA, AH and HV intervals were respectively 20, 110 and 55 ms. During programmed atrial stimulation performed in spontaneous rhythm, it was possible to induce episodes of tachycardia with characteristics identical to those of episodes which originated spontaneously (Fig 2). The induction was possible only when the AH interval of the extrastimulus was equal or longer than 190 ms.

On echocardiogram, a diffuse hypokinesis of left ventricle was present; the diastolic and systolic diameters were respectively 6.8 and 5.4 cm (Fig 3).

On gated angiocardioscintigraphy, ejection fraction of left ventricle was 47%.

After some unsuccessful attempts with various antiarrhythmic drugs (quinidine-like drugs, amiodarone, propafenone, sotalol), it was possible to stop permanently the tachycardia by oral daily administration of combination of verapamil (480 mg) and digoxin (0.5 mg). The maintenance of sinus rhythm was confirmed by

Address:

Prof Giorgio Morace
Via Luigi Castaldi, 2/6 - 50139 Firenze

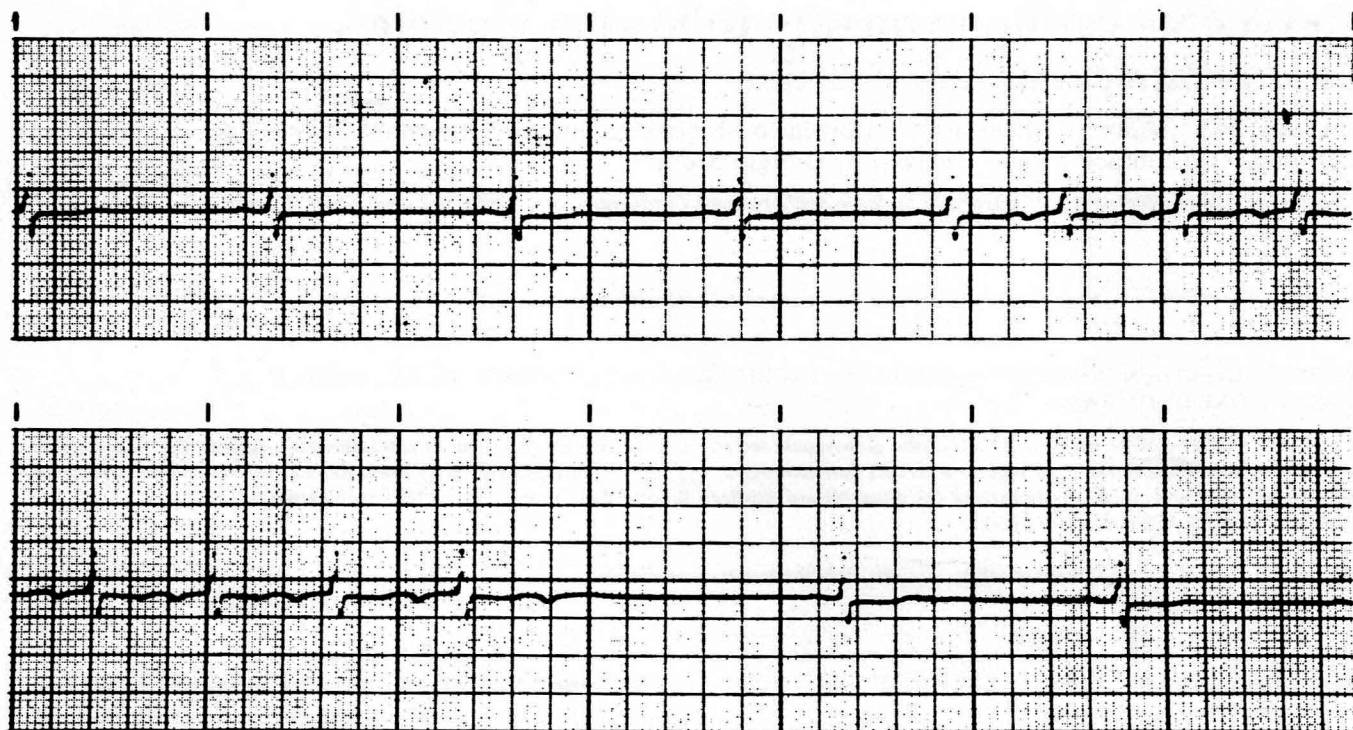


Figure 1. - ECG showing a decrease in sinus cycle length before the beginning of tachycardia, an increase of the RP' interval over the first few cycles and the cessation of the tachycardia with a P' wave preceded by a slight increase of the P'R interval.

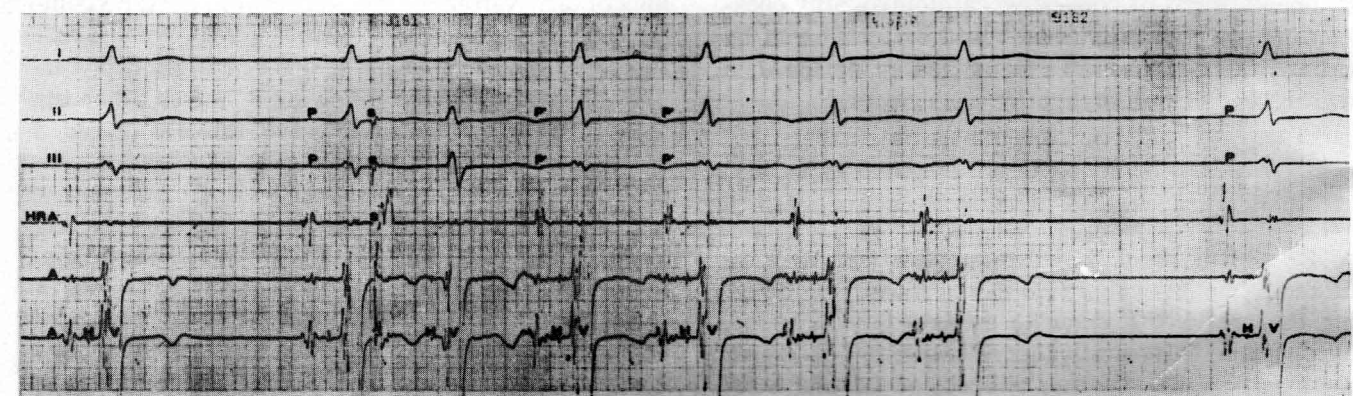


Figure 2. - Electrophysiologic testing. Induction of reciprocating tachycardia by atrial extrastimulus. The tachycardia has the same characteristics of the spontaneous one. The VA interval was longer than the AV interval and heart rate was 107/min. HRA: atrial electrogram obtained near the atrioventricular node.

various Holter monitorings. The restoration of sinus rhythm was followed, after about 7 days, by a normalization of left ventricular diameters on echocardiography (diastolic diameter = 5.3 cm, systolic diameter = 3.6 cm) and by an increase of ejection fraction on radioisotopic angiocardigraphy (54%).

Discussion

The absence of increase of PR interval at the beginning of the tachycardia, the constant P' wave morphology during the tachycardia, the initial increase of the interval between the QRS complex and the subse-

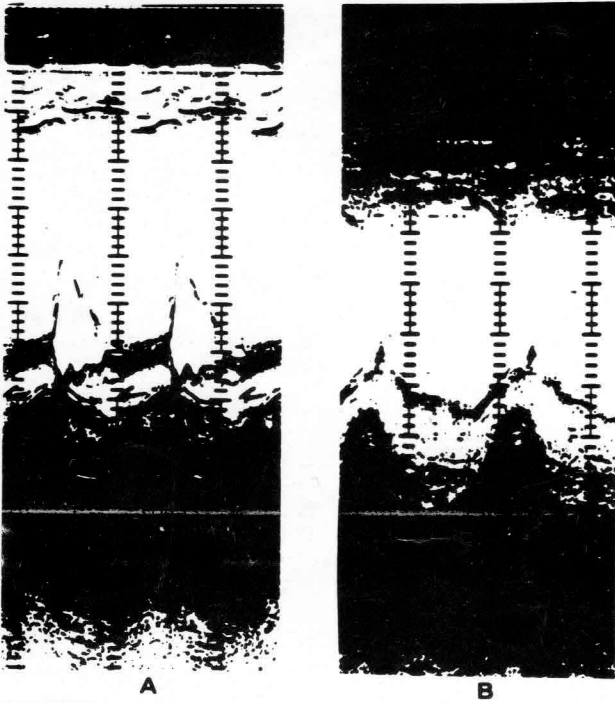


Figure 3. - M-Mode echocardiogram during tachycardia (A) and during sinus rhythm (B). Normalization of the ventricular dimensions and improvement of the wall motion after restoration of the sinus rhythm is observed.

A: diastolic and systolic diameters are 6.8 cm and 5.4 cm respectively.
B: diastolic and systolic diameters are 5.3 cm and 3.6 cm respectively.

quent P' wave over the first few cycles (suggestive of decremental properties of ventriculoatrial conduction), the constant 1:1 AV conduction suggest an incessant form of junctional reciprocating tachycardia. This can be included in the group of "long RP" tachycardias⁵ which are persistent and generally refractory to medical therapy.

The rapid normalization of cardiac function allows us to conclude that the observed signs of cardiomyopathy were caused by the arrhythmia. The increase of the ejection fraction on radioisotopic angiocardiology and the ventricular diameters normalization on echocardiography were significant according to the reproducibility limits of these methods⁶⁻⁸. The duration of tachycardia, instead of its frequency, seems to be of crucial importance. In this regard it should be considered that neither supraventricular paroxysmic tachycardia⁹ nor brief period of atrial pacing¹⁰ are able to induce signs of heart failure in normal subjects. On the contrary, heart failure has been observed in dogs after a long period of rapid pacing¹¹. In such condi-

tions the dysfunction of the left ventricle has been related to the depletion of myocardial stores of creatine, creatine phosphate and adenosine triphosphate. The reversibility of cardiac failure in patients with persistent tachycardia could depend, as previously indicated¹¹, by re-establishment of such high-energy substrates.

Summary

We report a case of persistent junctional reciprocating tachycardia with signs of congestive cardiomyopathy. The disappearance of tachycardia after successful medical therapy produced a concomitant complete regression of signs of cardiac failure. The left ventricular dysfunction could depend upon reversible changes of cardiac metabolism due to the length of tachycardia.

Key words: Chronic supraventricular tachycardia; Congestive cardiomyopathy.

References

- Engel TR, Bush CA, Schaal SF: Tachycardia aggravated heart disease. *Ann Intern Med* 1974; 80: 384-388.
- Keane YF, Plauth WN, Nadas AS: Chronic ectopic tachycardia of infancy and childhood. *Am Heart J* 1972; 84: 748-757.
- Shachnow N, Spellman S, Rubin I: Persistent supraventricular tachycardia: case report with review of the literature. *Circulation* 1954; 10: 232-236.
- Packer DL, Bardy GM, Worley SJ, Smith MS, Cobb FR, Coleman RE, Gallagher JY, German LD: Tachycardia-induced cardiomyopathy: a reversible form of left ventricular dysfunction. *Am J Cardiol* 1986; 57: 563-570.
- Guarnieri T, German LD, Gallagher JY: The long R-P' tachycardias. *Pace* 1987; 10: 103-117.
- Samn OJ, De Maria A, Kisslo J, Wayman A: The committee on M-mode standardization of the American Society of Echocardiography: recommendations regarding quantitation in M-mode echocardiography; results of a survey of echocardiographic measurements. *Circulation* 1978; 58: 1072-1083.
- Crawford NH, Grant D, O'Rourke RA, Starling MR, Groves BN: Accuracy and reproducibility of new M-Mode echocardiographic: recommendations for measuring left ventricular dimensions. *Circulation* 1980; 61: 137-143.
- Bisi G, Gallini C, Barletta GA, Malfanti PL: Assessment of systolic and diastolic ventricular function by gated radionuclide angiocardiology. A study of variability at rest. *J Nucl Med All Sci* 1983; 27: 74-75.
- Swiryns S, Pavel D, Byron E, Wyndham C, Pietras R, Banerfeind R, Rosen K: Assessment of left ventricular function by radionuclide angiography during induced supraventricular tachycardia. *Am J Cardiol* 1981; 47: 555-561.
- Rozenmann Y, Weiss AT, Atlan H, Gosman MS: Left ventricular function during atrial pacing: a radionuclide angiographic study. *Clin Cardiol* 1984; 7: 349-355.
- Coleman HN, Taylor RR, Pool PE, Whipple GH, Covell JW, Ross JR, Braunwald E: Congestive heart failure following chronic tachycardia. *Am Heart J* 1971; 81: 790-798.