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EC CARDIOLOGY
Case Report

# Tachycardiomyopathy: An Old Modality of Treatment Revisited!

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## Abstract

This case report is unusual case of severe dilated cardiomyopathy who presented to me in my clinic as an endstage case of dilated cardiomyopathy. However, from carefull history, examination and documented ECGs, I could realized that it may be caused by incessant SVT and thus it may be a case related to the entity of tachycardiomyopathy that can be cured by controlling rate.

***Keywords:*** *Tachycardiomyopathy; Modality; Echocardiography*

## Introduction

This case report is unusual case of severe dilated cardiomyopathy who presented to me in my clinic as an endstage case of dilated cardiomyopathy.

## Case Report

M. A.A. is 25 y-old male farmer from Sohag (south Egypt rural center). Four years before presentation, he started to suffer from frequent attacks of rapid palpitations. This was followed by progressive SOB on effort. He was hospitalized at the end of 2013 in ICU Assiut (Biggest City in south Egypt).

Clinical presentation was dyspnea at rest; BP 90/60, and signs of pulmonary congestion. ECG (Figure 1) revealed accelerated junctional tachycardia at a rate 180 /min. He received several antiarrhythmic drug trials (Amiodarone infusion; Adenosine iv, and lastly Digoxin iv), all failed to revert him into sinus. DC cardioversion reverted him into sinus for few minutes. Echocardiography (Figure 2) showed dilated LV with severe Global impairment of LV contractility (EF = 30%), with no evidence of any valvular disease. He was discharged on Digoxin 0.25 mg tablet plus carvedilol 6.25 mg PO twice daily, furosemide 40 mg PO and spironolactone 25 mg PO; and heart rate at discharge was 145/min.

When he was referred to me by his family, he was in the same condition of incessant junctional tachycardia with classic symptoms and signs of LVF and dilated cardiomyopathy. I considered the possibility of diagnosis of Tachycardiomyopathy (TCMP) because: A history of a diagnosis of LV dysfunction, preceded by Evidence of persistent tachycardia and the classic clinical presentation with symptoms and signs of dilated cardiomyopathy [1].

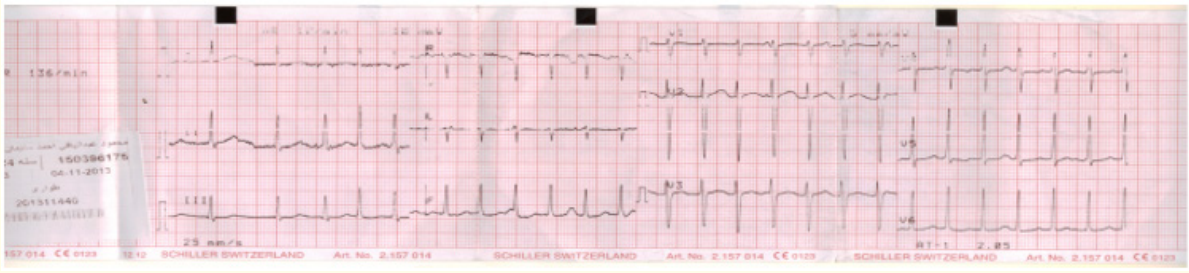

***Figure 1:*** *Accelerated junctional tachycardia at rate 175/min with AV dissociation.*

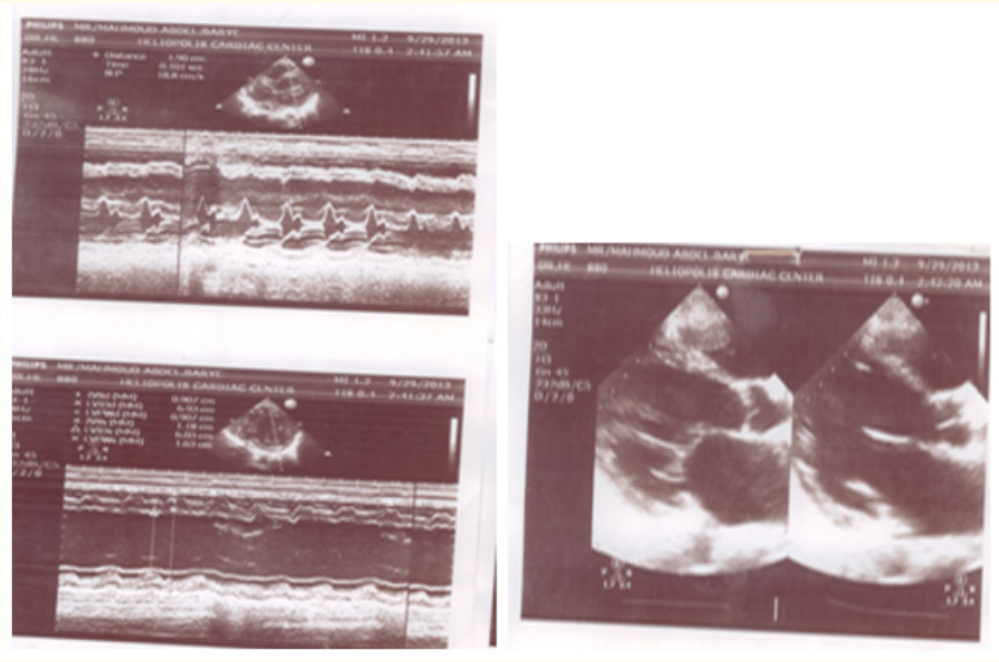

***Figure 2:*** *Echocardiography showing dilated LV with poor LV Global contractility EF = 30%.*

So, LV function may improve with any of a number of SVT treatment strategies, whether that be rhythm or rate control by drugs or Radiofrequency (RF) catheter ablation. A 'pace and ablate' procedure (AVN ablation and single chamber VVI pacing), can improves LV function [2,3]. However, this approach may result in LV dysynchrony and is usually a last resort reserved for elderly patients or intolerant of medications. So, I decided to go for catheter RF ablation [4,5].

At the EPS lab, I introduced 2 electrode catheters: one at the AV junction to elicit HIS bundle potential and one in the RV apex for backup pacing. A third catheter specialized for RF ablation (4 mm, non-irrigated cordis Biosense) was positioned at AVN Kock triangle for trial of slow pathway ablation (Figure 3). The details of EPS (including intracardiac ECG) and catheter RF ablation is beyond the scope of this manuscript. In short, successful ablation of junctional tachycardia was achieved after 30-watt RF catheter ablation, and he was in sinus rhythm at end of the RF ablation (Figure 4 ECG after ablation).

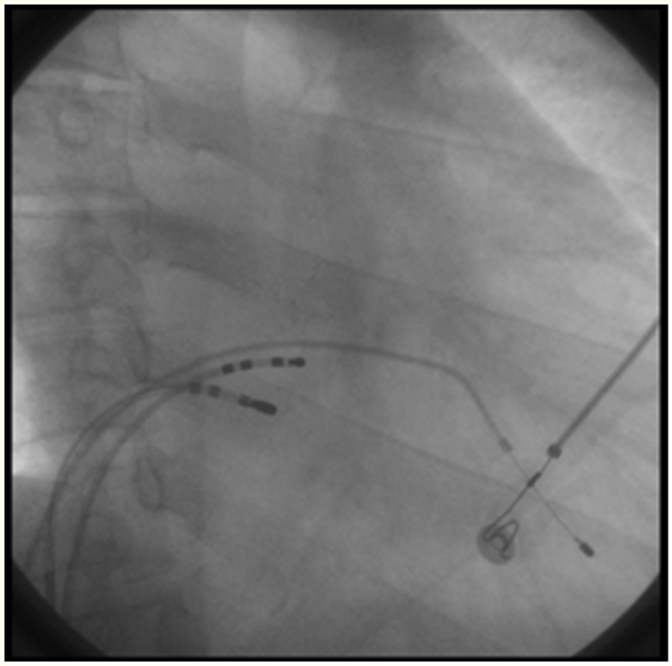

***Figure 3:*** *RAO view showing RV apex, His Bundle and RF ablation catheter.*

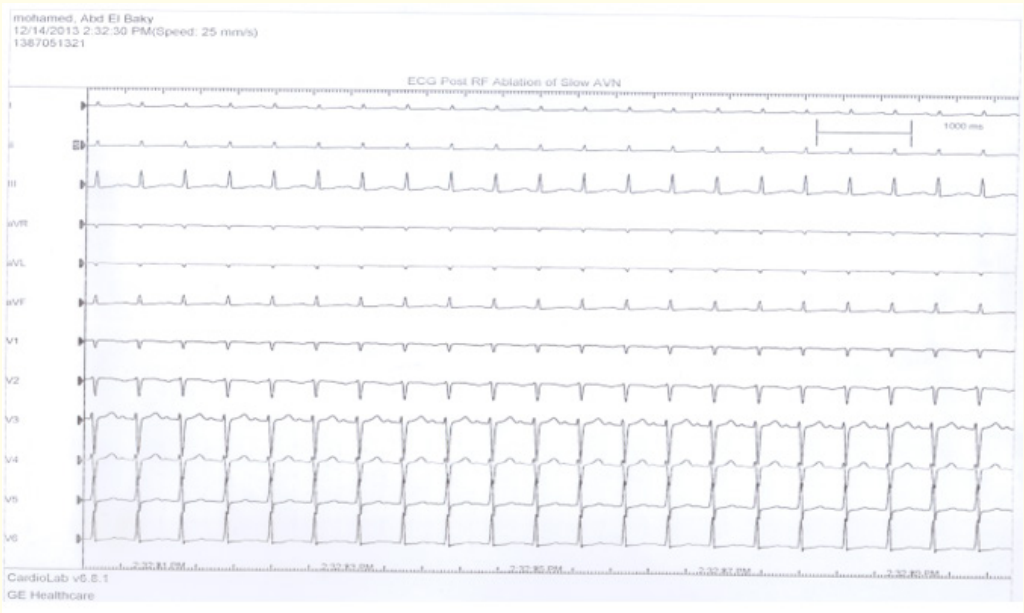


***Figure 4:*** *ECG after RF ablation of AV junction slow pathway.*

Patient markedly improved symptomatically and could resumes his manual hard work. However, after few months he started to suffer back of same dyspnea on mild effort and that was associated with recurrence of incessant junctional tachycardia documented by ECG that showed junctional tachycardia with AV dissociation (Figure 5). Echo showed dilated LV (LVEDD = 7.3 cm) and EF = 37 % (Figure 6).

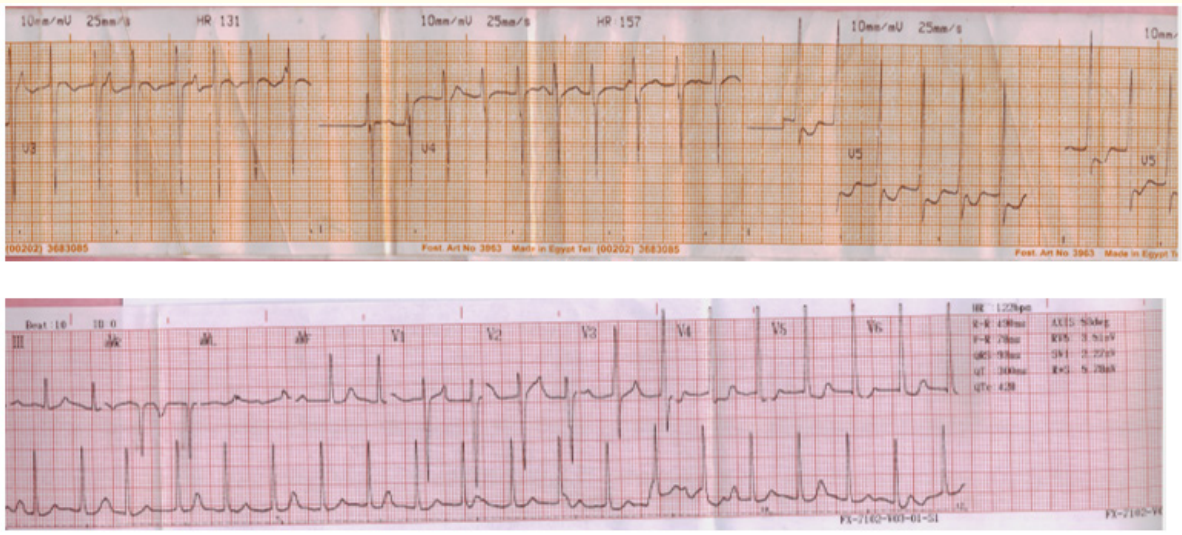

***Figure 5:*** *2 ECGs showing accelerated junctional tachycardia with AV dissociation.*

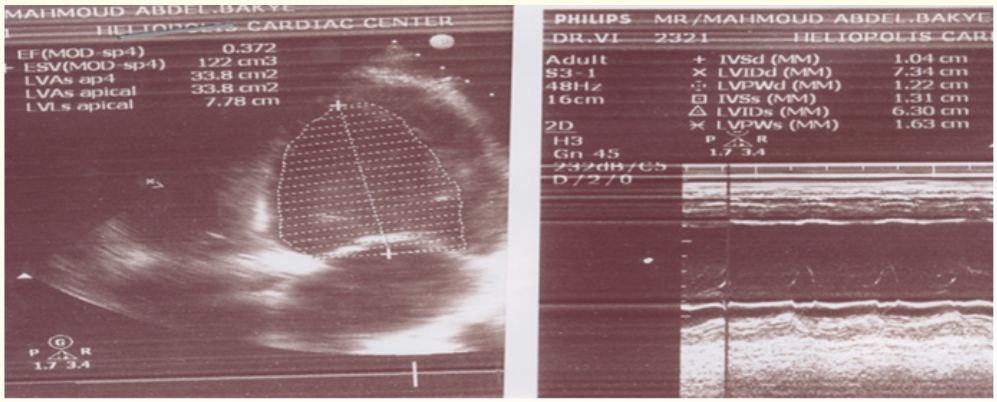


***Figure 6:*** *2 D and M mode Echo showing dilated LV with low EF of 37%.*

So, I decided to apply the " Ablate and Pace " technique, specially I had a past personal experience of few cases who improved on this approach. In the EPS lab, I introduced only 2 catheters: a Bibolar RV apical catheter for backup pacing and a RF ablation catheter at HIS Bundle position aiming at inducing complete AV block by applying radiofrequency current of 40 watt for 30 seconds. After achieving the target, I implanted VVI permanent pacemaker. Notice that sinus rhythm was achieved during RF ablation, but because of possible recurrence, I contined RF current at 45 watts until definite infrahisian block. ECG post procedure showed sinus rhythm and VVI pacing at 60 BPM (Figure 7).

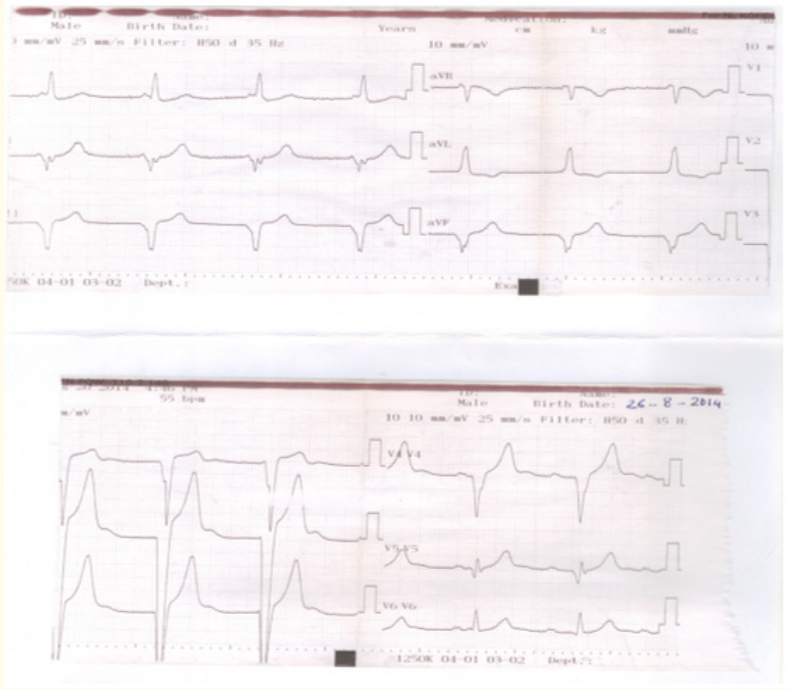

***Figure 7:*** *ECG showing VVI pacing Post induction of 3 AVB by RF ablation of AVN.*

In few weeks, patient markedly improved and continued his normal daily work in few months. Echo 9 months later showed normalization of LV size and LV global contractility (LVEDD= 5.9 cm, EF = 58%, Figure 8). At long term follow-up (5 years), his LV function remained normal on no medications (Figure 9).

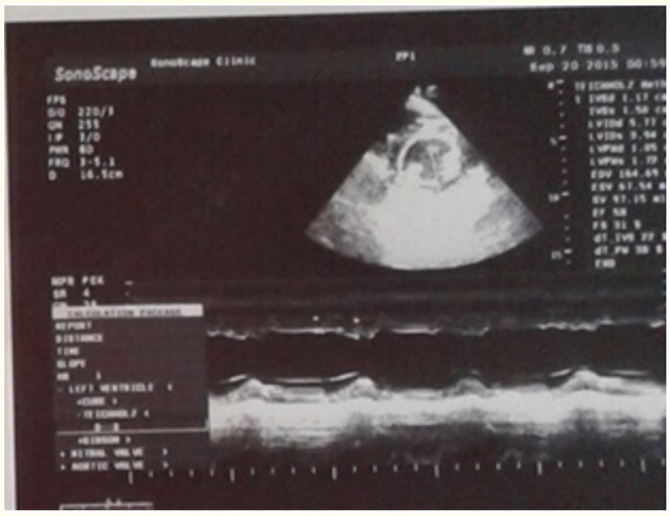


***Figure 8:*** *M mode Echo 9 month after AVN RF ablation and VVI permanent pacing showing normal LV size (LVED = 5.9 cm) and normal LVEF = 58%.*

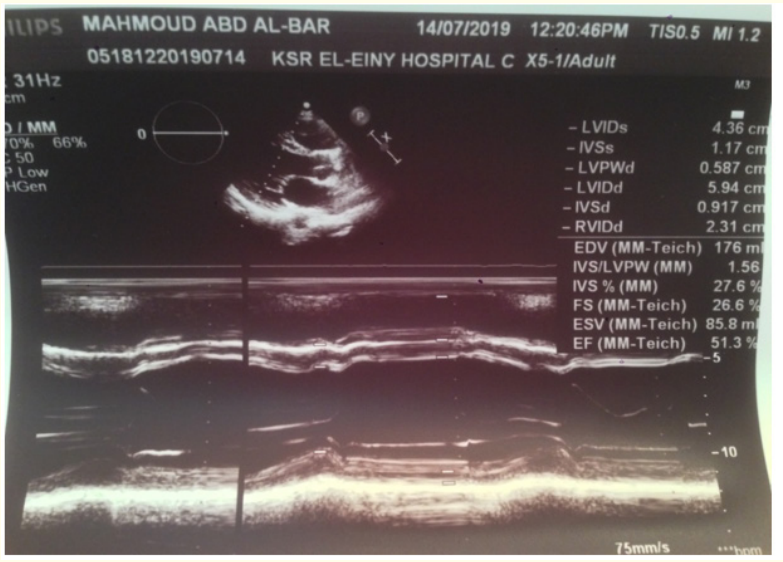


***Figure 9:*** *Same comment as figure 8, but Echo done 5 years after VVI pacing.*

## Discussion

Numerous studies have demonstrated that multiple forms of tachycardia may result in TCMP; these include AF, atrial flutter, incessant SVT, VT and PVCs [6-9]. Re-entrant SVTs including atrioventricular nodal reentrant tachycardia (AVNRT) and atrioventricular reentrant tachycardia (AVRT) are usually paroxysmal and are therefore a rare cause of TCMP. One well-known example, however, is persistent junctional reciprocating tachycardia, which is a form of incessant AVRT, most common in paediatric cases Incessant junctional tachycardia is one of the forms that can result in dilated cardiomyopathy or what is called Tachycardiomyopathy (TCMP) [6,7]. It is characterized by ventricular systolic dysfunction and dilatation and clinical manifestation of heart failure that are reversible with normalization of heart rate. This can be achieved by restoring sinus rhythm or controlling ventricular rate by medications or ablation of origin or pathway of ectopic tachycardia resulting in normalization of LV function and relief of symptoms of HF [10,11].

Whipple., *et al*. first described experimental TCMP in 1962, where Sustained rapid atrial or ventricular pacing in experimental models produced a markedly cardiac dilatation resulting right and left ventricular wall thinning, markedly elevated ventricular filling pressures and severe impairment of systolic function [12]. Rapid ventricular rate significantly decrease ventricular filling time resulting low cardiac output which lead to Neurohormonal activation, resulting marked elevations of plasma catecholamines, atrial natriuretic peptide, rennin and aldosterone levels; further worsening LV function.

The precise mechanism of TCMP is not fully understood, but myocardial energy depletion, impaired energy utilization and myocardial ischemia have been proposed as possible mechanisms for myocardial dysfunction. (paper) Also, chronic SVT caused significant disruption of the sarcolemma basement membrane interface which could reduce mechanical pump performance and impair ventricular function [13]. Abnormalities in calcium handling, transport, and availability to myocytes may reduce contractility; and can be responsible for TCMP [14].

Clinically, Tachycardia-induced cardiomyopathy may follow any type of chronic or frequently recurring paroxysmal tachyarrhythmias: AF, Atrial flutter, Ectopic AT, Atrioventricular Junctional tachycardia, and VT. TCMP can occur at any age. No specific tests or markers available to diagnose TCMP. A high index of suspicion derived from history and clinical features remains the only available tool to diagnose

this entity. Therefore, the diagnosis of tachycardiomyopathy should be considered in any patient with LV systolic dysfunction and chronic or frequently recurring Tachyarrhythmias.

Once the diagnosis of TCMP has been made, a therapeutic strategy employed can be selected dependent on the causative arrhythmia, where either a rate or rhythm control strategy maybe more appropriate [2]. Potentially curative ablation is often the treatment of choice, especially for SVTs, and also AF. A 'pace and ablate' procedure (AVN ablation and single chamber VVI pacing) is used for rate control in drug resistant cases of AF or SVT. A 'pace and ablate' procedure improves LV function in AF even if the ventricular response was well controlled prior, showing that regularity is likely to be important as well as rate [15].

Our patient is a clear example of a case of TCMP due to incessant junctional tachycardia which was resistant to drug therapy and induced severe LVF that was reversed by Radiofrequency catheter AV junctional ablation plus VVI pacing. The patient rapidly improved clinically and by Echocardiography in few weeks and LV size and function was completely reversed. Now, after 4 years follow up, he is fine doing hard manual work, and have good marital life on no drug treatment of any kind!

## Conclusion

Tachycardiomyopathy is a rare but potentially curable form of dilated cardiomyopathy. A tachycardiomyopathy should be always considered in patients with Idiopathic" dilated Cardiomyopathy and heart failure who suffer from chronic or frequently recurring tachyarrhythmias. Heart rate normalization, either by rate or rhythm control, is the cornerstone of therapy. The best means to achieve heart rate control vary depending on the type of arrhythmia. These may include antiarrhythmic drug therapy, external DC cardioversion, radiofrequency catheter ablation, or pacemaker therapy.

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