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Incessant narrow-complex tachycardia in a patient with heart failure – what is the mechanism?

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We present the case of a 68-year-old woman without any relevant prior medical history who was admitted to our emergency department because of worsening dyspnoea and hypotension.

She reported having intermittent palpitations with a sudden onset for years. On clinical examination, she had signs of decompensated heart failure. Echocardiography showed a dilated left ventricle with severely depressed left ventricular ejection fraction of 15% with diffuse hypokinesis. N-terminal-pro-brain natriuretic peptide (NT-proBNP) levels were markedly elevated (11,440 ng/l). The initial 12-lead ECG showed narrow-complex tachycardia (fig. 1).

Questions:

1. What is the most likely diagnosis shown in figure 1?
2. What diagnostic test/therapy would be the most appropriate in this clinical scenario?

The differential diagnosis includes:

T-wave alternans

Dual atrioventricular non-re-entrant tachycardia (DAVN-NT)

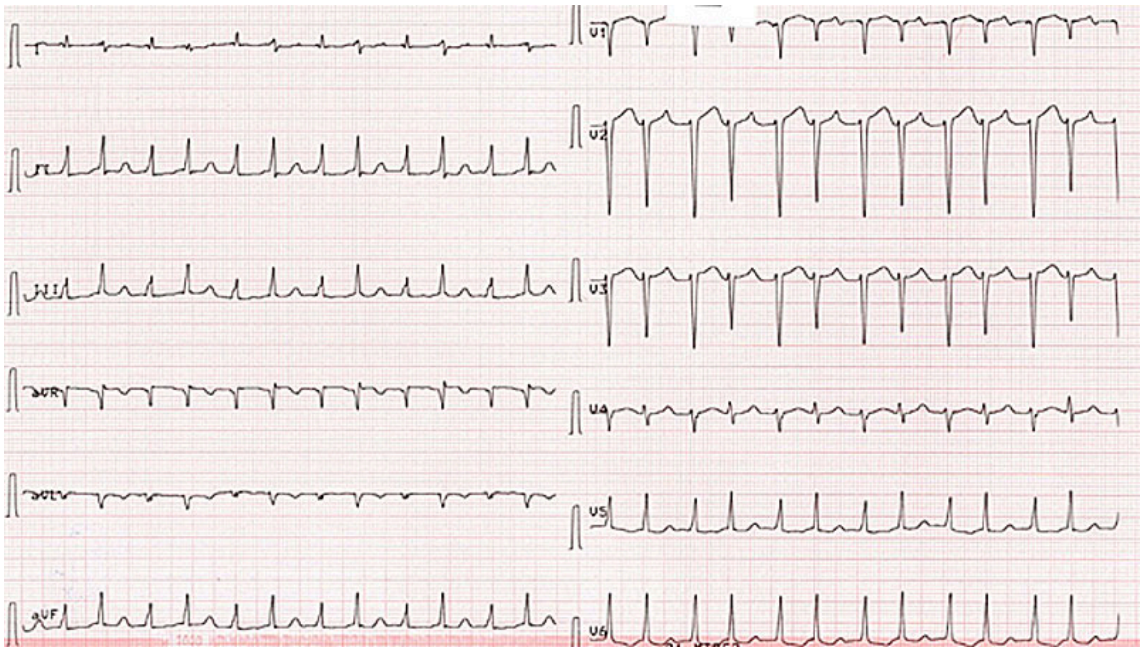
Atrial or junctional bigeminy

Atrioventricular nodal re-entrant tachycardia (AVNRT) with 2:1 retrograde block

Atrial fibrillation

The ECG shows an irregular narrow-complex tachycardia at a heart rate of 150 beats/min. However, the irregularity has a regularity, with two alternating recurring RR intervals of 366 ms and 454 ms. Therefore, atrial fibrillation can be ruled out. A deflection resembling a T wave or a P wave occurs in the ST segment of each second QRS complex. Although there could be marked T wave changes

Figure 1: Initial 12-lead ECG recorded in the emergency department.



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("macroscopic" T wave alternans), a T wave phenomenon was ruled out because there has to be a discernible T wave after every QRS complex and none was visible in this case.

In atrioventricular nodal re-entrant tachycardia with 2:1 retrograde block (upper common pathway block), a P wave (if visible) would result from a low–high activation of the atria and produce a negative P wave in the inferior leads. The positive P wave seen in the inferior leads in Figure 1 therefore rules out AVNRT. Ectopic beats originating from the atrioventricular nodal or His region occurring in a bigeminal pattern cannot be ruled out but the undisturbed regularity of the phenomenon would be uncommon – Atropine may have helped in the differential diagnosis by suppressing dual atrioventricular node conduction and enhance junctional activity.

On the assumption that the deflection between every second QRS complex was a P wave, most likely originating from the superior part of the atria (positive in II, III, aVF), and typical biphasic morphology in lead V1, the ECG can be described as showing sinus P waves, each followed by two QRS complexes.

Solution

The ECG suggests the presence of dual atrioventricular nodal physiology with antegrade conduction down a fast pathway to create a QRS complex followed by simultaneous antegrade conduction down a slow pathway to create a second QRS complex (fig. 2). This results in double ventricular activation for a single sinus beat ("two for one") leading to a dual atrioventricular non-re-entrant tachycardia (DAVNNT). Lack of retrograde conduction into the slow pathway by antegrade conduction down the fast pathway is mandatory for this phenomenon to happen [1]. DAVNNT – also termed "atrioventricular nodal double firing" – was first reported in 1975 [2]; however, the occurrence of incessant DAVNNT leading to tachycardiomyopathy, as in this case, is extremely rare [2].

When double firing is not regular, as in this case, due to conduction changes from the slow to the fast pathway and intermittent double firing leading to irregular RR intervals it might be misdiagnosed as atrial fibrillation.

The patient underwent an electrophysiology study which confirmed the presence of dual atrioventricular physiology during sinus rhythm (fig. 2). Radiofrequency ablation of the slow pathway resulted in interruption of the tachycardia and restoration of 1:1 AV conduction (fig. 3). In addition to the ablation, the patient received medical treatment for heart failure. Three months after the ablation, she reported no symptoms of tachycardia and had no signs or symptoms of heart failure. Echocardiography revealed normalisation of the left ventricular ejection fraction.

Dual atrioventricular nodal non-re-entrant tachycardia is a rare cause of tachycardiomyopathy and may be misdiagnosed [3]. Ablation can eliminate the palpitations and cure heart failure.

Disclosure statement
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Figure 2: Intracardiac recordings during DAVNNT. The underlying rhythm is sinus rhythm and the high–low atrial activation sequence can be seen. Owing to consecutive antegrade conduction via the fast (FP) and slow pathway (SP) from the atrioventricular node, a 1:2 ventricular depolarisation occurs. HRA = high right atrium; HIS = His bundle recordings; RV = right ventricle.

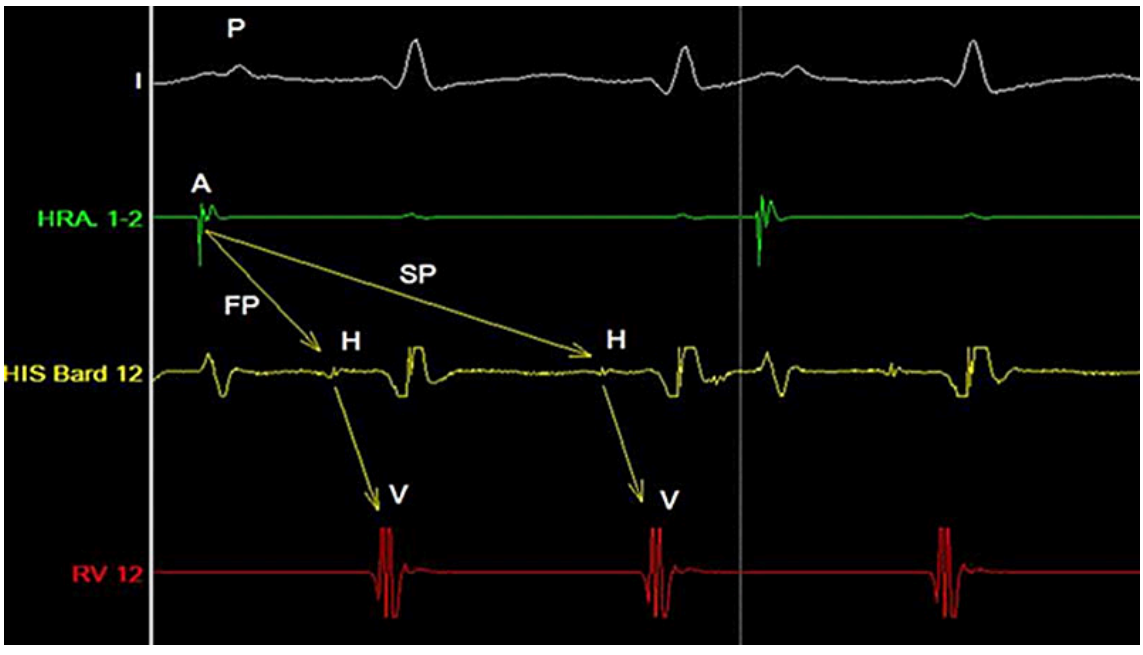

Figure 3: At 5.8 seconds after starting radiofrequency ablation in the slow pathway position, the tachycardia was interrupted and followed by sinus rhythm with 1:1 atrioventricular conduction or junctional beats, for example where the PR interval is short. HRA = high right atrium, HIS = his bundle recordings, RV = right ventricle. ABL = ablation catheter.