



Pathophysiology of Tachycardia and its Diagnosis

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Description

Tachycardiomyopathies (TCMPs) are an important cause of left ventricular (LV) dysfunction that clinicians should recognize because they are potentially reversible and have a significant impact on morbidity and prognosis. They are classically defined as reversible impairment of ventricular function induced by persistent arrhythmia. However, it is becoming increasingly clear that they can be induced by atrial and ventricular ectopy promoting dyssynchrony, and indeed the term “arrhythmia-induced cardiomyopathy” is emerging to describe this phenomenon. A more current proposed definition emphasizes the etiology: ‘Atrial and/or ventricular dysfunction secondary to rapid and/or asynchronous/irregular myocardial contraction, partially or completely reversed after treatment of the causative arrhythmia’. There are two categories of this condition: the arrhythmia is the sole reason for the ventricular dysfunction (arrhythmia-induced) and another reason is, where arrhythmia worsens the ventricular dysfunction and/or worsens heart failure (HF) in a patient with existing heart disease (arrhythmia-mediated), exclusion of underlying structural heart disease may be challenging because current imaging techniques such as MRI cannot readily identify diffuse fibrosis, which itself may be primary or secondary to the effects of the arrhythmia promoting ventricular wall dyskinesia and valvular stretch or regurgitation [1]. Tachycardia, also called tachyarrhythmia, is a heart rate that exceeds normal resting heart rate. In general, a resting heart rate above 100 beats per minute is accepted as tachycardia in adults. A heart rate above the resting rate can be normal (such as during exercise) or abnormal (such as electrical problems in the heart).

Pathophysiology

The mechanisms of Tachycardiomyopathies are not fully defined, but include subclinical ischemia, abnormalities in energy metabolism, redox stress, and calcium overload [2]. In animal models of persistent high-frequency atrial or ventricular pacing, ventricular injury is also associated with changes in myocardial electrophysiology, including action potential prolongation and spontaneous ventricular arrhythmias [3]. Persistent left bundle branch block leads to lateralization of gap junctions promoting functional anisotropy and apoptosis [4]. This can be reversed by left ventricular stimulation in HF models. These molecular and cellular changes lead to abnormalities in ventricular geometry and negative ventricular patterning. It is this reversibility of ventricular function in these disorders that can be corrected by treating the primary tachycardia, which is why it is important to quickly identify and treat Tachycardiomyopathies [5].

Diagnosis

An electrocardiogram (ECG) is used to classify the type of tachycardia. Based on the QRS complex, they can be divided into narrow and wide complexes [6]. Equal to or less than 0.1 s for a narrow complex. They are listed in order from most common to least common:

Narrow complex :

- Sinus tachycardia, which originates in the sinoatrial (SA) node, near the base of the superior vena cava
- Atrial fibrillation
- Atrial flutter
- AV nodal reentrant tachycardia
- Accessory pathway mediated tachycardia

- Atrial tachycardia
- Multifocal atrial tachycardia
- Cardiac tamponade
- Junctional tachycardia (rare in adults)

A wide complex :

- Ventricular tachycardia, any tachycardia that originates in the ventricles
- Any narrow complex tachycardia combined with a problem with the heart's conduction system, often called "supraventricular tachycardia with aberration"
- Narrow complex tachycardia with an accessory pathway, often called "supraventricular tachycardia with preexcitation" (eg Wolff–Parkinson–White syndrome)
- Pacemaker-mediated or pacemaker-mediated tachycardia

Tachycardias can be classified as narrow complex tachycardia (supraventricular tachycardia) or wide complex tachycardia [7]. Narrow and wide indicate the width of the QRS complex on the EKG. Narrow complex tachycardias tend to originate from the atria, while wide complex tachycardias tend to originate from the ventricles. Tachycardias can be further classified as regular or irregular [8].

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