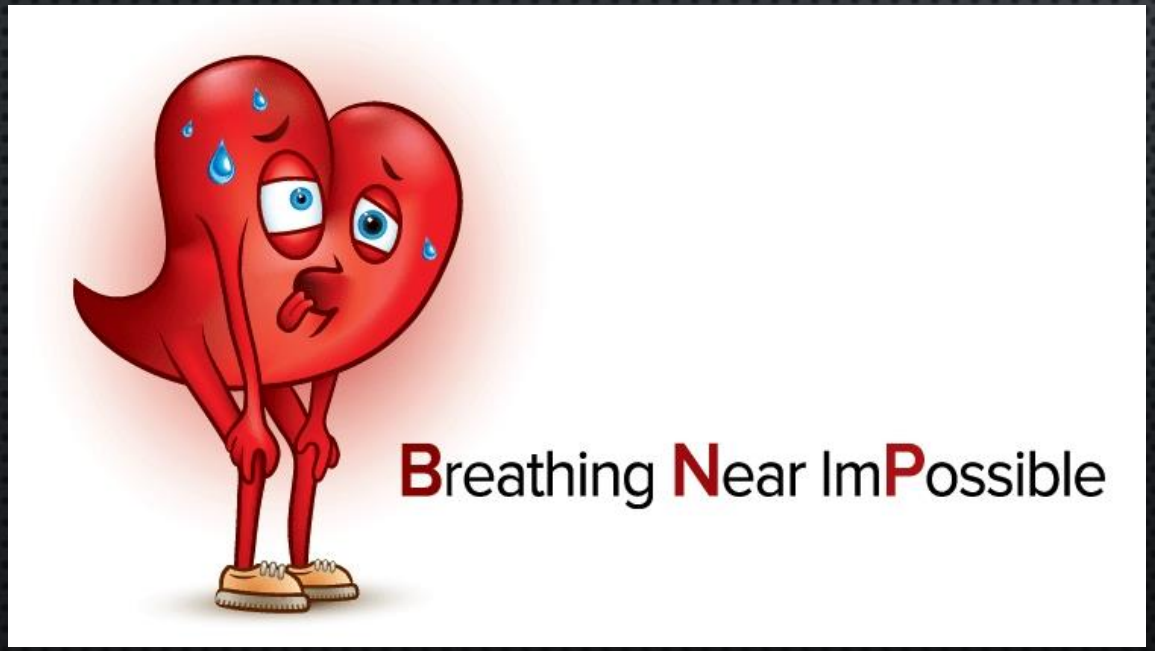
TACHYCARDIA-INDUCED CARDIOMYOPATHY



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DEFINITION

Tachycardia-induced cardiomyopathy (TCM), or more accurately arrhythmia-induced cardiomyopathy, is a reversible cause of impaired LV function due to persistent tachycardia or very frequent ventricular premature beats that can lead to HF and death. The incidence of TCM is unknown but has been reported in all age groups, from foetuses to the elderly.

CAUSES

permanent junctional reciprocating tachycardia

The syndrome was initially described with PJRT, but we know now that any chronic cardiac arrhythmia may cause TCM: incessant AVRT due to septal APs, rapid AF, idiopathic VT, AT, and persistent ectopic beats are most well described. In patients <18 years of age, focal AT is the com-monest cause.

MECHANISM

Rapid pacing in animal models induces cytoskeletal changes and remodelling of the extracellular matrix attributed to abnormal calcium cycling, increased catecholamines, decreased beta-1 adrenergic receptor density, oxidative stress, depletion of myocardial energy stores, and myocardial ischaemia due to increased heart rate.[559,564]

Endomyocardial biopsy specimens from patients with TCM exhibit features distinct from those of other types of cardiomyopathy, including deranged cardiomyocyte and mitochondrial morphology, and macrophage-dominated cardiac inflammation.[565] However, it has not been fully established how the majority of patients with frequent premature ventricular contractions have a benign course, whereas ≤30% of them may develop cardiomyopathy.[566]

DIAGNOSIS

TCM is one of the very few reversible causes of HF and dilated cardiomyopathy, and should be considered in any patient with new onset of LV dysfunction. In the presence of persistent or frequent tachycardia, or frequent premature ventricular contractions, a high index of suspicion should be maintained. The diagnosis is established by excluding other causes of cardiomyopathy, and demonstrating recovery of LV function after eradication of the arrhythmia or control of the ventricular rate. Typically in TCM, LV ejection fraction is <30%,

the ventricular rate. Typically in TCM, LV ejection fraction is <30%, LV end-diastolic diameter is <65 mm, and LV end-systolic diameter is <50 mm.[557] More dilated ventricular volumes suggest underlying dilated cardiomyopathy, although some overlapping of the two conditions can occur. In patients with suspected TCM, cardiac magnetic resonance (CMR) is advisable to exclude intrinsic structural change. Serial assessment of N-terminal pro-B-type natriuretic peptide (NT-proBNP) and estimation of the ratio of NT-proBNP at baseline to NT-proBNP during follow-up can help differentiate TCM from irreversible idiopathic dilated cardiomyopathy.

Recommendations for the therapy of SVT in patients with suspected or established heart failure due to tachycardiomyopathy		
Recommendations	**Class[a]**	**Level[b]**
Catheter ablation is recommended for tachycardiomyopathy due to SVT.	I	B
Beta-blockers (from the list with proven mortality and morbidity benefits in HFrEF) are recommended for tachycardiomyopathy due to SVT, when catheter ablation fails or is not applicable.	I	A
It is recommended to consider tachycardiomyopathy in patient with reduced LVEF with an elevated heart rate (>100 bpm).	I	B

24 h (or multi-day) ambulatory ECG monitoring should be considered for diagnosis of tachycardiomyopathy by identifying subclinical or intermittent arrhythmias.	IIa	B
AV nodal ablation with subsequent pacing ("ablate and pace") either biventricular or His-bundle pacing, is recommended if the tachycardia responsible for the tachycardiomyopathy cannot be ablated or controlled by drugs.	I	C

SVT AND SPORTS

Athletes with frequent supraventricular arrhythmias should be assessed to exclude the presence of an underlying cardiac disease, electrolyte imbalances, thyroid dysfunction and the use of stimulants or performance-enhancing drugs.

WPW

Ventricular pre-excitation (WPW syndrome) is a rare cause of sudden cardiac death in young athletes.[575] Although many individuals with ventricular pre-excitation remain asymptomatic throughout their lives, symptomatic AVRT may occur. Patients with WPW may also develop other arrhythmias, such as AF, which could degenerate into ventricular fibrillation and sudden cardiac

ventricular pre-excitation. Asymptomatic athletes with intermittent pre-excitation (at rest or during exercise) or abrupt disappearance of pre-excitation during stress testing may be considered to be at low risk (see section 11.3.11 for reservations), but should be further evaluated as they may be eligible for competitive sports activity. In asymptomatic athletes with ventricular pre-excitation, invasive risk stratification should be conducted as described in *section 11.3.11* and catheter ablation should be performed in those with high-risk features.[577] Asymptomatic patients stratified at low risk are allowed to practice competitive sports.

Table 10 Recommendations for sports participation in athletes with ventricular pre-excitation and supraventricular arrhythmias

	Criteria for eligibility	Eligibility
Premature atrial beats.	No symptoms, no cardiac disease.	All sports.
AVRT or atrial fibrillation in the context of WPW syndrome.	Ablation is mandatory. Sports are allowed one month after ablation if there are no recurrences.	All sports.

AVNRT, ORTHODROMIC AVRT, AT

AVNRT, orthodromic AVRT over a concealed AP, and AT are not listed among the causes of sudden cardiac death during exercise in patients with a structurally normal heart. However, their occurrence during sports activity may be associated with very high heart rate because of sympathetic stimulation and may cause haemodynamic impairment even in patients with structurally normal hearts. Accordingly, catheter ablation should generally be recommended for all athletes with a history of paroxysmal SVT. Athletes

NO ABLATION !!

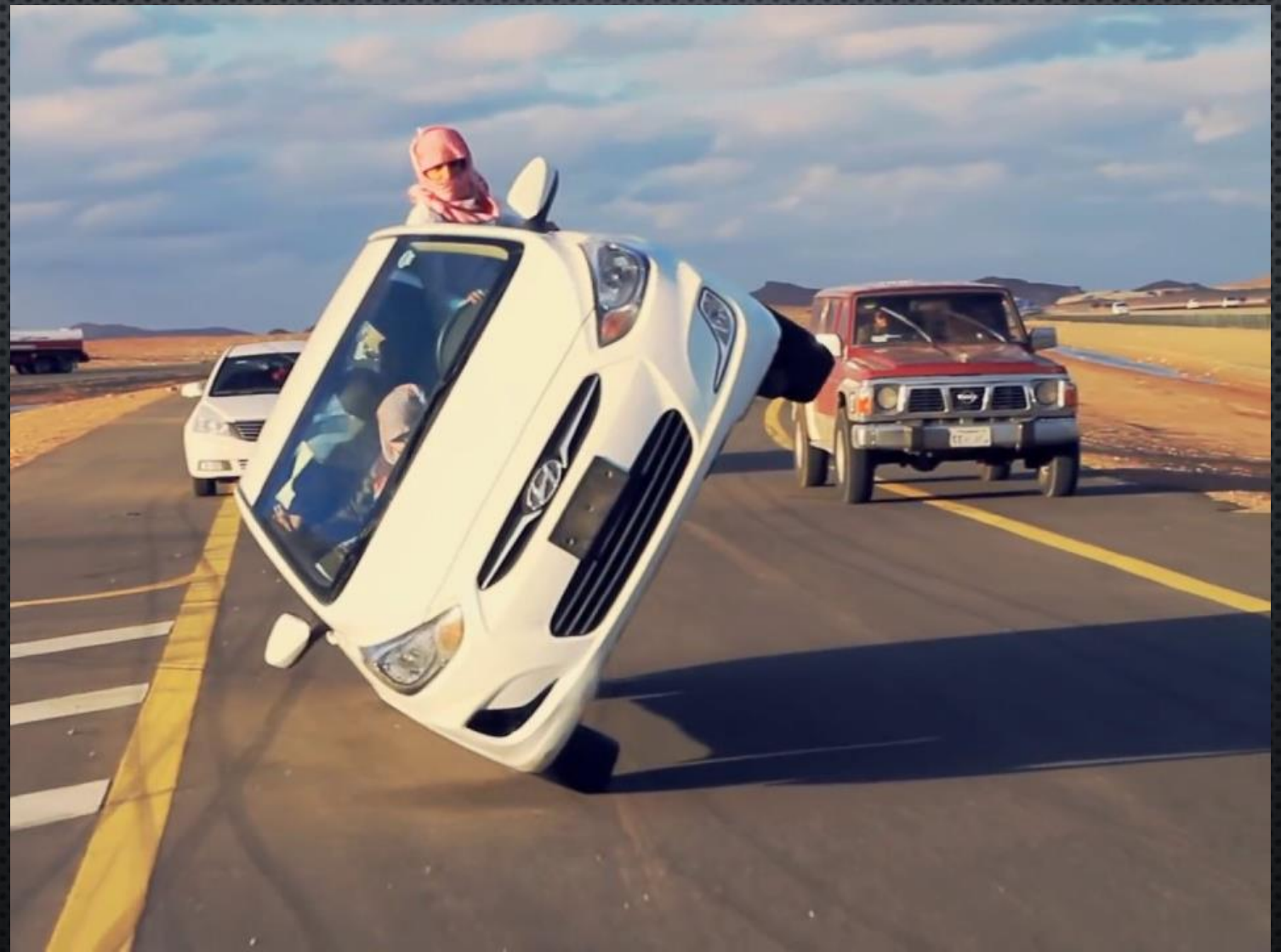
with SVT who do not wish to undergo catheter ablation, or in whom the procedure has been unsuccessful, may be considered eligible for competitive sports activity if the arrhythmia is sporadic, unrelated to cardiac disease, well tolerated, and unrelated to exercise, and when the sports activity does not have a high intrinsic risk of loss of consciousness (such as divers, pilots, horse riders, etc.).[578]

Asymptomatic ventricular pre-excitation.	In patients at high-risk ablation is mandatory. Sports are allowed one month after ablation if there are no recurrences.	All sports.
Paroxysmal supraventricular tachycardia (AVNRT, AVRT over a concealed accessory pathway and atrial tachycardia).	Ablation is recommended. Sports are allowed one month after ablation if there are no recurrences. Ablation undesirable or not feasible.	All sports. All sports, except those with high intrinsic risk of loss of consciousness.

B-BLOCKERS NA-CHANAL BLOCERS

Treatment of paroxysmal SVT with beta-blockers or sodium channel blockers is discouraged in athletes, because these drugs may reduce performance during sports and have limited ability to prevent arrhythmia recurrence during sports activity. Moreover, beta-blockers are listed by the World Anti-Doping Agency as prohibited drugs in particular sports.

SVT AND DRIVING



Two groups of drivers are considered. Group 1 comprises drivers of motorcycles, cars and other small vehicles with and without a trailer. Group 2 includes drivers of vehicles over 3500 kg or passenger carrying vehicles exceeding eight seats excluding the driver. Drivers of taxicabs, small ambulances and other vehicles form an intermediate category between the ordinary private driver and the vocational driver.

Table 11 Recommendations on driving restriction in patients with SVT

Conduction Disorder/ Arrhythmia	Group 1	Group 2
Atrial fibrillation/atrial flutter/focal atrial tachycardia.	Driving may continue provided no history of syncope. If history of syncope, driving must cease until the condition has been satisfactorily controlled / treated.	Driving may continue provided no history of syncope and anticoagulation guidelines are adhered to. If history of syncope, driving must cease unless the underlying cause is treated, and the risk of recurrence is low. Rate control during tachycardia should be adequate. Driving can only be resumed after medical assessment.
AVNRT, AVRT, and WPW.	If history of syncope, driving must cease until the condition has been satisfactorily controlled /treated.	Driving may continue provided no history of syncope or other significant symptoms (e.g. palpitations with dizziness). If so, driving must cease until the underlying cause is treated so that the risk of recurrence is low. In case of pre-excitation, driving may only be allowed after specialist assessment.

THANK YOU