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Definitions and classification

- Supraventricular Tachycardia (SVT): atrial rates **>100 bpm at rest**,
- The mechanism involves tissue from the **His bundle or above.**
- Traditionally, SVT describe **all kinds of tachycardias apart from ventricular tachycardias (VTs) and AF** (included atrioventricular (AV) re-entry tachycardias due to accessory connections)
- The term '**narrow** QR tachycardia' indicates those with a QRS duration **<_120 ms.**
- A wide QRS tachycardia refers to one with a QRS duration >120 ms
- In clinical practice, SVT may present as narrow or wide QRS tachycardias, **most** of which, although not invariably, manifest as **regular** rhythms

Conventional classification of supraventricular tachycardias (1)

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Atrial tachycardias

Sinus tachycardia
- Physiological sinus tachycardia
- Inappropriate sinus tachycardia
- Sinus nodal re-entrant tachycardia

Focal atrial tachycardia

Multifocal atrial tachycardia

Macro-re-entrant atrial tachycardia (MRAT)

Conventional classification of supraventricular tachycardias (2)

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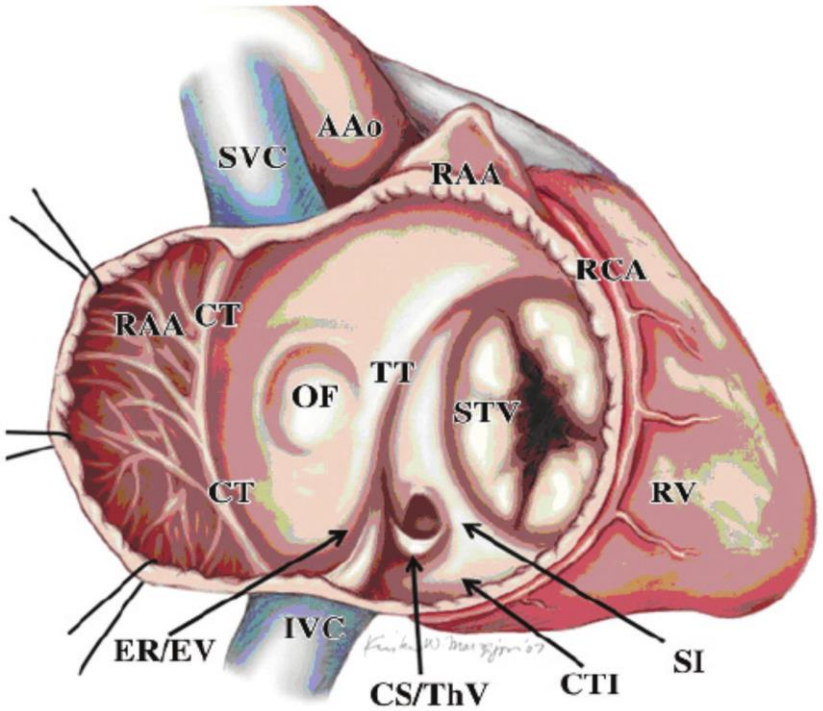
Atrial tachycardias

- Cavotricuspid isthmus-dependent MRAT
  - -Typical atrial flutter, counter-clockwise (common) or clockwise (reverse)
  - -Other cavotricuspid isthmus-dependent MRAT
- Non-cavotricuspid isthmus-dependent MRAT
  - -Right atrial
  - -Left atrial

Atrial fibrillation



(European Heart Journal; 2019)

Conventional classification of supraventricular tachycardias (3)

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Atrioventricular junctional tachycardias
Atrioventricular nodal re-entrant tachycardia •Typical •Atypical
Non-re-entrant junctional tachycardia •Junctional ectopic tachycardia (focal junctional tachycardia) •Other non-re-entrant variants
Atrioventricular re-entrant tachycardias
•Orthodromic (including permanent junctional reciprocating tachycardia) •Antidromic (with retrograde conduction through the atrioventricular node or, rarely, over another pathway)

ESC Guidelines for the management of patients with supraventricular Tachycardia (European Heart Journal; 2019)

Initial evaluation of the patient with SVT

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Standard

- History, physical examination, and 12-lead ECG
- Full blood count, biochemistry profile, and thyroid function
- An ECG during tachycardia should be sought
- Transthoracic echocardiography

Optional

- Exercise tolerance testing
- 24 h ECG monitoring, trans-telephonic monitoring, or an implantable loop recorder
- Myocardial ischaemia testing in patients with coronary artery disease risk factors (including men >40 years and post-menopausal women)
- An EPS should be considered for a definitive diagnosis and when catheter ablation is anticipated

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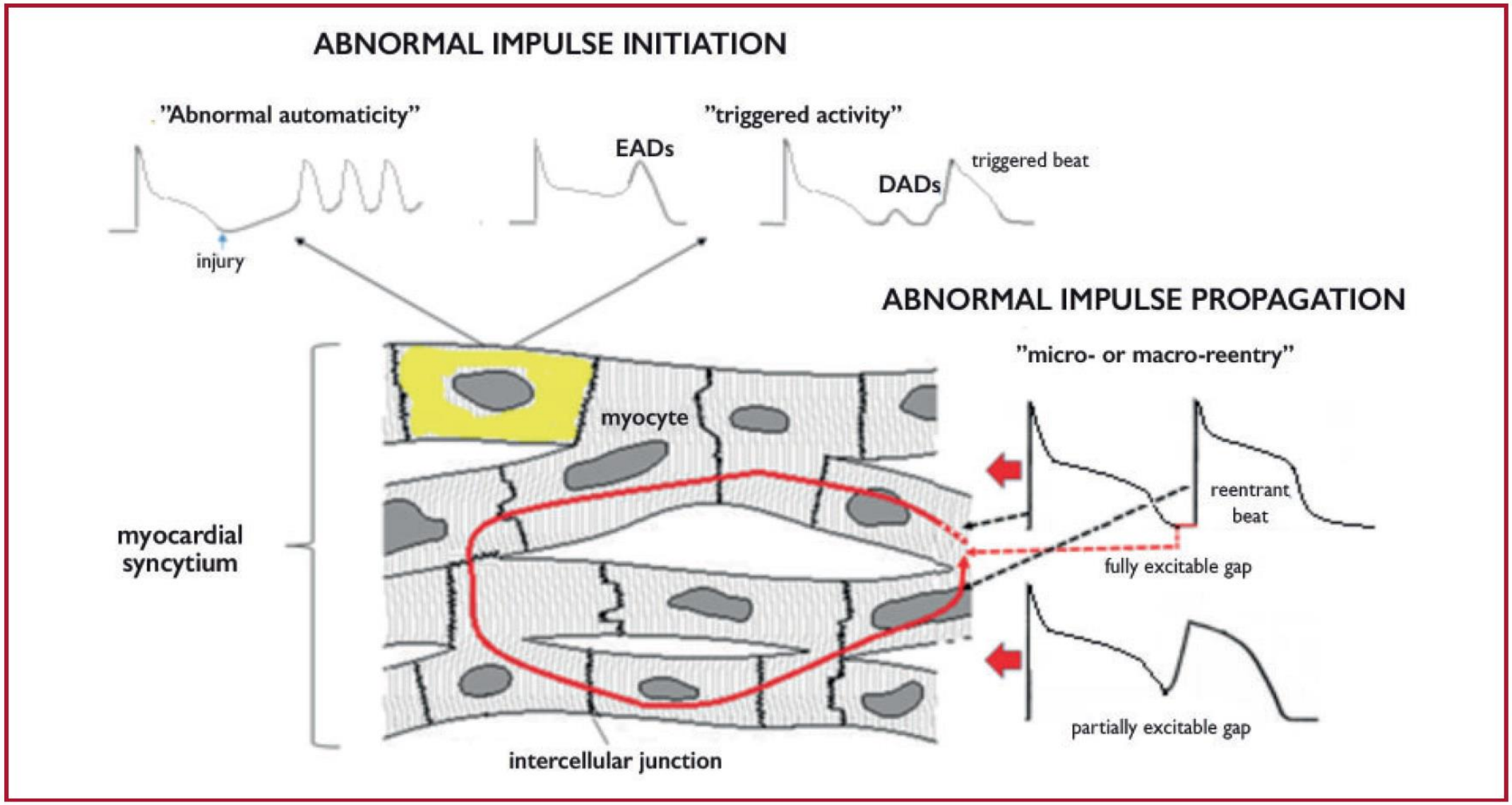
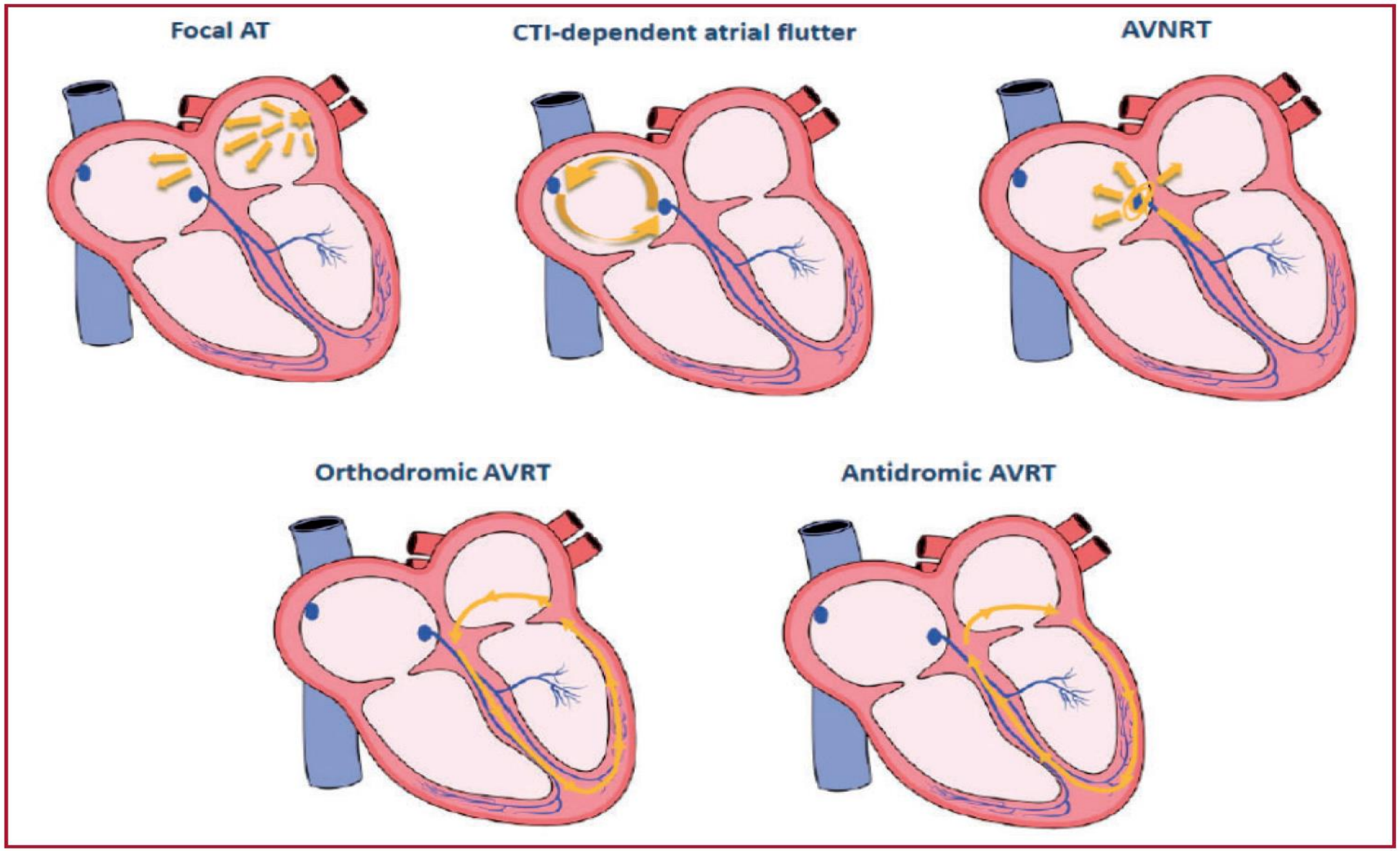
Mechanisms of arrhythmogenesis.

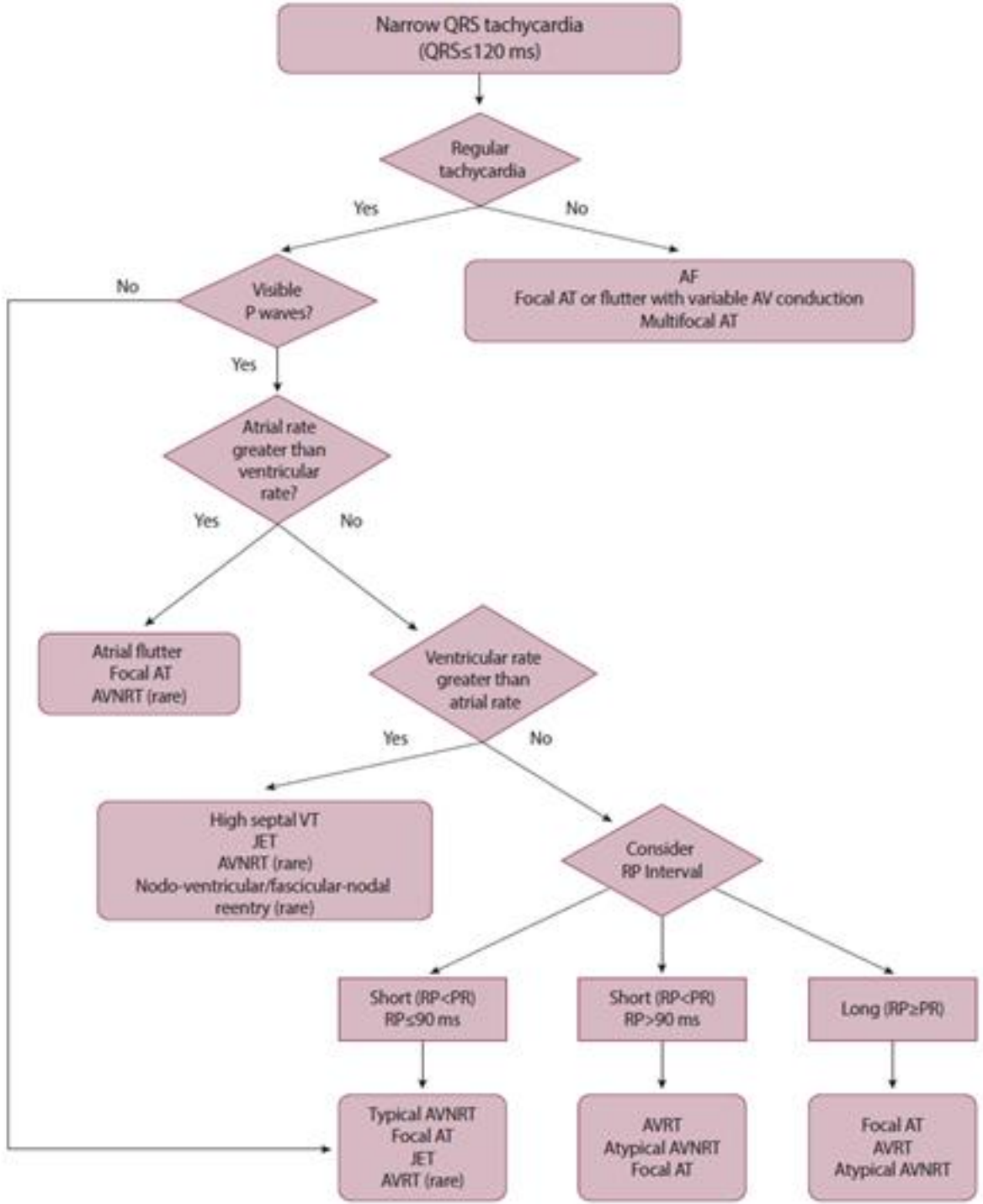


Tachycardia circuit in different types of narrow QRS tachycardia



Differential diagnosis of tachycardia

1. **Narrow QRS (=120ms) tachycardia**
    1. Electrocardiographic differential diagnosis
    2. Vagal manoeuvres and adenosine
    3. Electrophysiology study
2. **Wide QRS (>120ms) tachycardia**
    1. Electrocardiographic differential diagnosis
    2. Electrophysiology study
3. **Irregular tachycardia**



Differential diagnosis of narrow QRS tachycardia

ESC Guidelines for the management of patients with supraventricular Tachycardia
(European Heart Journal; 2019)

Epidemiology of supraventricular tachycardia

In the general population:

- The SVT prevalence is 2.25/1000 persons and the incidence is 35/100 000 person-years.
- Women have a risk of developing SVT 2 times greater than that of men
- Persons aged ≥ 65 years have more than 5 times the risk of developing SVT than younger individuals[1]
- the prevalence of a WPW pattern on surface electrocardiogram (ECG) ranges from 0.15-0.25%[2]
- the incidence of atrial flutter in men (125/100 000) is >2.5 times that of women (59/100 000) and increases exponentially with age[3]
- The prevalence of AF before flutter ablation ranges from 24-62%, and after ablation could be 30-70%[3].

1. Orejarena LA, Vidaillet H, DeStefano F, Nordstrom DL, Vierkant RA, Smith PN, Hayes JJ. Paroxysmal supraventricular tachycardia in the general population. J Am Coll Cardiol 1998;31:150-157.
2. Krahn AD, Manfreda J, Tate RB, Mathewson FL, Cuddy T. The natural history of electrocardiographic preexcitation in men: the Manitoba Follow-up Study. Ann Intern Med 1992;116:456-460.
3. Granada J, Uribe W, Chyou P-H, Maassen K, Vierkant R, Smith PN, Hayes J, Eaker E, Vidaillet H. Incidence and predictors of atrial flutter in the general population. J Am Coll Cardiol 2000;36:2242-2246.

Epidemiology of supraventricular tachycardia

In specialized centres:

- AVNRT is the most frequently treated substrate after AF, followed by atrial flutter and AVRT, in patients referred for catheter ablation.
- Women are more likely to be affected by AVNRT than men (ratio 70:30), while the converse is true for AVRT (ratio 45:55).[1-3]

1. Porter MJ, Morton JB, Denman R, Lin AC, Tierney S, Santucci PA, Cai JJ, Madsen N, Wilber DJ. Influence of age and gender on the mechanism of supraventricular tachycardia. Heart Rhythm 2004;1:393396.
2. Gonzalez-Torrecilla E, Almendral J, Arenal A, Atienza F, Atea LF, del Castillo S, Fernandez-Aviles F. Combined evaluation of bedside clinical variables and the electrocardiogram for the differential diagnosis of paroxysmal atrioventricular reciprocating tachycardias in patients without pre-excitation. J Am Coll Cardiol 2009;53:23532358.
3. Liuba I, Jo¨nsson A, S€afstro¨m K, Walfridsson H. Gender-related differences in patients with atrioventricular nodal reentry tachycardia. Am J Cardiol 2006;97:384388.

Differential diagnosis of tachycardia

1. **Narrow QRS (=120ms) tachycardia**
    1. Electrocardiographic differential diagnosis
    2. Vagal manoeuvres and adenosine
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    1. Electrocardiographic differential diagnosis
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3. **Irregular tachycardia**

Differential diagnosis of tachycardia

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Tachycardia induced cardiomyopathy (TIC)

- TIC: systolic and/or diastolic ventricular **dysfunction resulting from a prolonged elevated heart rate** which is reversible upon control of the arrhythmia or the heart rate..
- The sustained heart rate limit above which TIC will appear is not well established. However, a prolonged heart rate above **100 beats per minute** may be relevant and deserves attention[1].
- TIC may **manifest months to years after the onset** of the responsible tachycardia, but because TIC is a rate dependent cardiomyopathy, those patients with **higher tachycardia rates develop TIC earlier**[2,3].
- Time to onset of ventricular dysfunction is also dependent on the presence of an **underlying structural heart disease**. Other factors include the **type** and **duration** of tachyarrhythmia, the patient's **age**, **drugs**, and **coexisting medical conditions**[4].

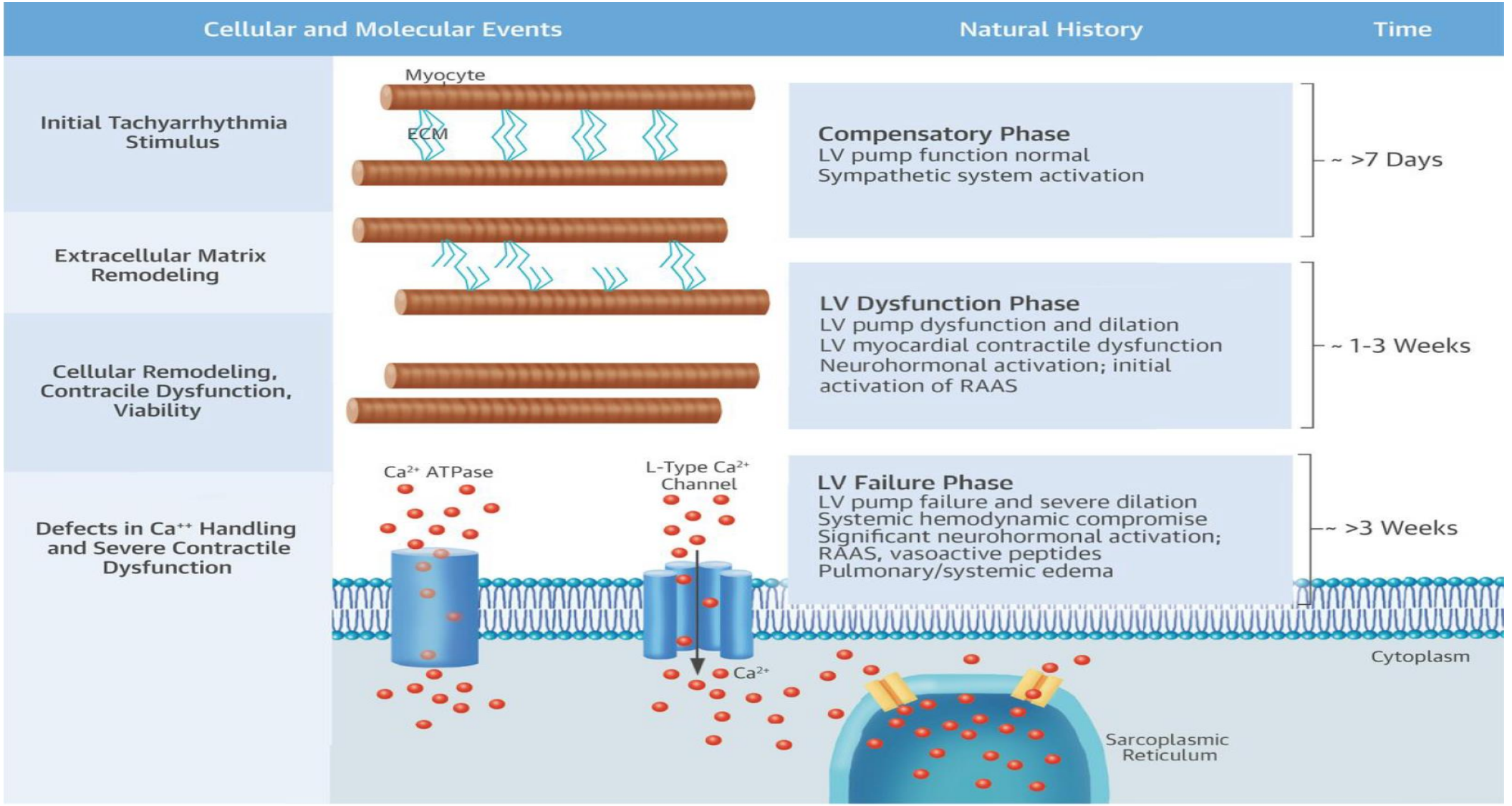
1. Ernesto Umana, MD, C. Arturo Solares, MD, Martin A. Alpert, MD. Tachycardia-Induced Cardiomyopathy. Am J Med. 2003; 114:51–55.
2. Quiniou G, Chevalier JM, Barbou F, Bire F, Clementy J. Tachycardia-induced cardiomyopathy, unusual and reversible cause of left ventricular dysfunction: Report of 9 cases. Ann Cardiol Angeiol 2000; 49(5):301–308.
3. Shinbane JS,Wood MA, Jensen DN, Ellenbogen KA, Fitzpatrick AP, Scheinman MM. Tachycardia-induced cardiomyopathy: A review of animal models and clinical studies. J Am Coll Cardiol 1997; 29(4):709–715.
4. Fenelon G,Wijns W,Andries E, Brugada P. Tachycardiomyopathy: mechanisms and clinical applications. Pacing Clin Electrophysiol: 1996; 19:95-106.

Pathophysiology

- High ventricular rates initially result in **cardiac dilatation** and **mitral regurgitation**, which are typically associated with **elevated ventricular filling pressures**, **decreased contractility**, **right and left ventricular wall thinning**, and, eventually, **heart failure** with neurohormonal activation. Cardiac output is usually reduced, and systemic vascular resistances are typically elevated[1].
- In experimental models, some of these changes can be seen **as early as 24-48 hours** after rapid cardiac pacing with continuing deterioration in ventricular function for **up to 3-5 weeks**. **Abnormal calcium handling**, **reduced cellular energy storing**, and **abnormal energy use** have been proposed as the underlying mechanisms responsible for this syndrome. These mechanisms determine myocardial remodeling. Cellular changes include **loss of myocytes**, **cellular elongation**, **myofibril misalignment**, and **loss of sarcomere register**, which may be due to **derangement of the extracellular matrix[2]**.

1. Tanaka R, Spinale FG, Crawford FA, Zile MR. Effect of chronic supraventricular tachycardia on left ventricular function and structure in newborn pigs. J Am Coll Cardiol 1992; 20:1650-60.
2. Wilson J, Douglas P, Hickey W, et al. Experimental congestive heartfailure produced by rapid ventricular pacing in the dog: cardiac effects. Circulation. 1987;75:857–867.

Natural History and Pathophysiology of Tachycardia-Induced Cardiomyopathy



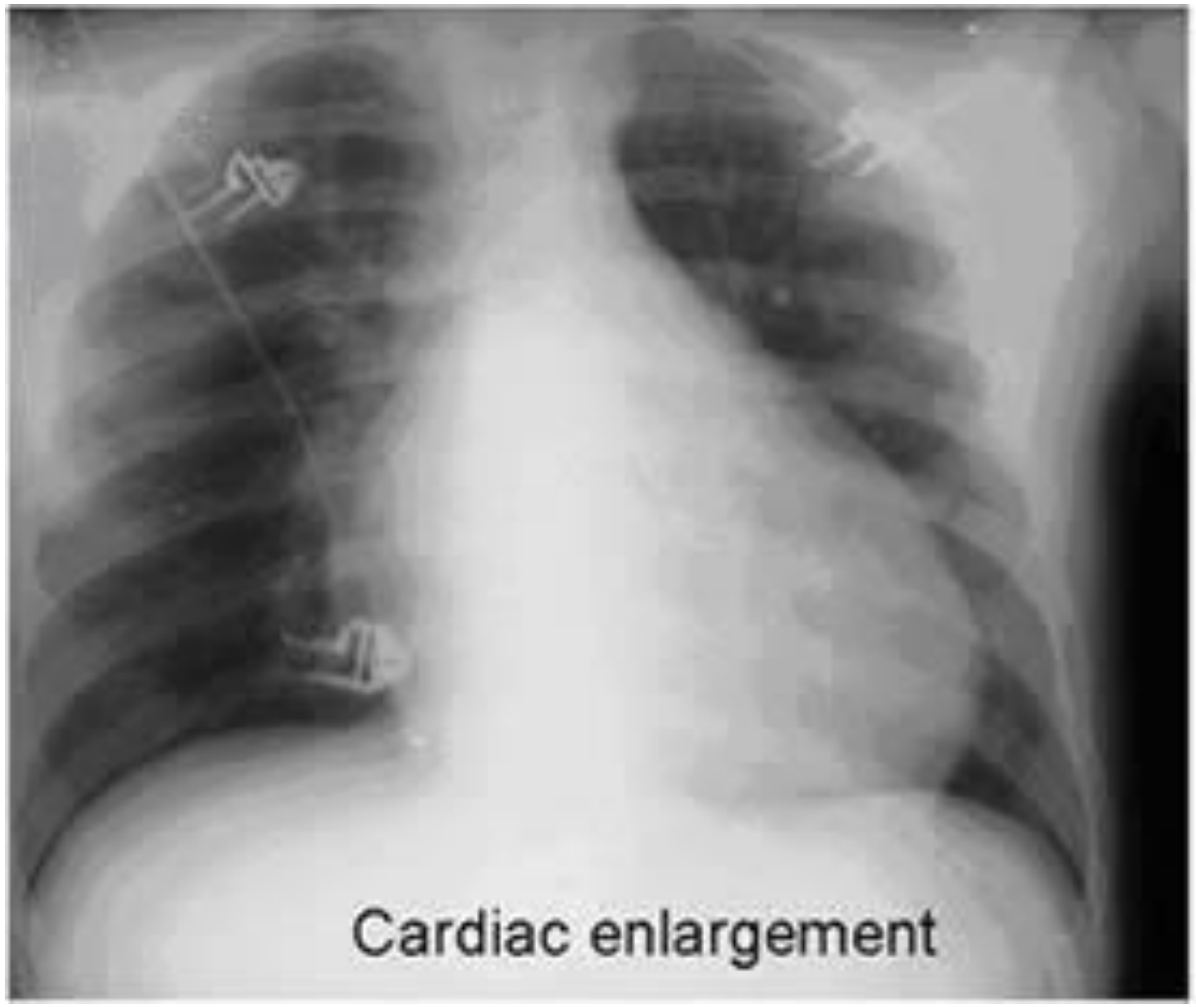
Albakri A (2018) Tachycardia-induced cardiomyopathy: a review of literature on clinical status and meta-analysis of diagnosis and clinical management method. Integr Mol Med, 2018 Volume 5(1): 1-13. doi: 10.15761/IMM.1000324

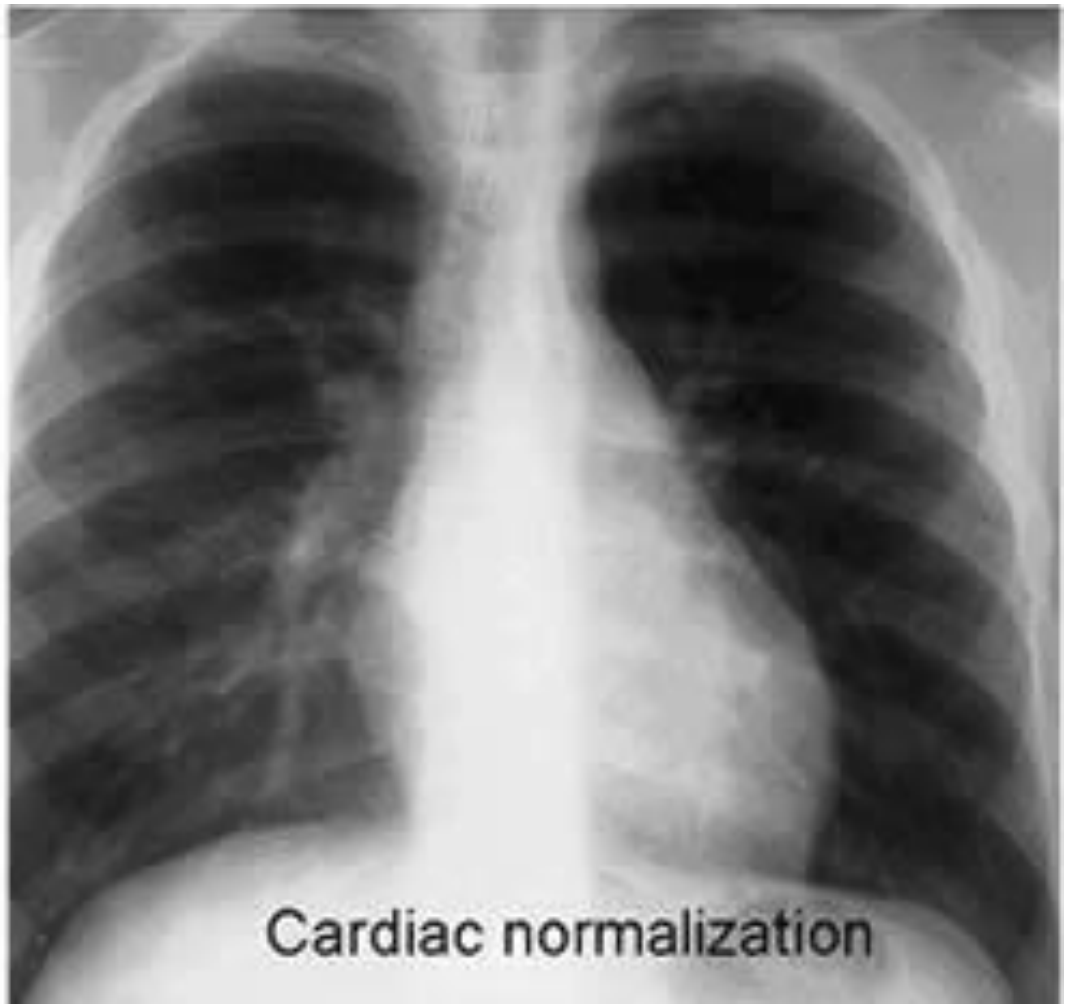
Diagnostic Clues Leading to the Detection of TIC

Step	Clinical Description or Outcomes
1.	**No** other **determinable cause of non-ischemic cardiomyopathy** such as hypertension, alcohol, drug use and stress
2.	The **absence of LV hypertrophy**
3.	Relatively **normal LV dimensions** such as LV-end diastolic dimension **< 5.5cm**
4.	**Restoration of LV function following control of tachycardia** using rate control,radio-frequency ablation or cardioversion between one and six months
5.	**Rapid decline in LV ejection fraction after recurrence of tachycardia** in patients with restored LV function following control of tachycardia.

Gupta S, Figueredo VM (2014) Tachycardia mediated cardiomyopathy: pathophysiology, mechanisms, clinical features and management. International Journal of Cardiology 172: 40-46.

Chest xray images taken on admission and 3 months after successful ablation of the accessory pathway





Recommendations on driving restriction in patients with SVT (1)

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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.

ESC Guidelines for the management of patients with supraventricular Tachycardia (European Heart Journal; 2019)

Recommendations on driving restriction in patients with SVT (2)

Conduction Disorder/ Arrhythmia	Group 1	Group 2
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.

ESC Guidelines for the management of patients with supraventricular Tachycardia (European Heart Journal; 2019)

Recommendations for sports participation in athletes with supraventricular arrhythmias (1)

	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

ESC Guidelines for the management of patients with supraventricular Tachycardia (European Heart Journal; 2019)

Recommendations for sports participation in athletes with supraventricular arrhythmias (2)

	Criteria for eligibility	Eligibility
Asymptomatic ventricular preexcitation	In patients at high-risk ablation is mandatory.	All sports
	Sports are allowed one month after ablation if there are no recurrences.	

ESC Guidelines for the management of patients with supraventricular Tachycardia
(European Heart Journal; 2019)

Recommendations for sports participation in athletes with supraventricular arrhythmias (3)

	Criteria for eligibility	Eligibility
Paroxysmal supraventricular tachycardia (AVNRT, AVRT over a concealed accessory pathway and atrial tachycardia)	Ablation is recommended.	All sports
	Sports are allowed one month after ablation if there are no recurrences.	
	Ablation undesirable or not feasible.	All sports, except those with high intrinsic risk of loss of consciousness.

ESC Guidelines for the management of patients with supraventricular Tachycardia (European Heart Journal; 2019)