TACHYCARDIA CARDIOMYOPATHY

DR GEETHA SUBRAMANIAN

MD DM CARDIOLOGY
FISC,FCSI, FIAE, FISE, FMMC
PROFESSOR OF CARDIOLOGY
IMS, BHU, VARANASI, INDIA

- Dilated cardiomyopathy (DCM):
  Characterized by dilation and impaired contraction of one or both ventricles

- ≥ 50% of patients with DCM:
  - an etiologic basis will not be identified
  - **Idiopathic DCM**

- One series- 1278 patients with congestive heart failure
  - ✓ Idiopathic — 51 percent
  - ✓ Idiopathic myocarditis — 9 percent
  - ✓ Occult coronary disease — 8 percent
  - ✓ Other identifiable causes — 32 percent

- Felker, et al. The spectrum of dilated cardiomyopathy. The Johns Hopkins experience with 1,278 patients. Medicine (Baltimore) 1999; 78:270

Reversible causes of cardiomyopathy

- Peripartum cardiomyopathy
- ***Tachycardia-mediated cardiomyopathy***
- Takotsubo cardiomyopathy
- Alcoholic cardiomyopathy
- Cocaine
- Medications
- Ischemia
- Endocrine dysfunction
- SLE, Sarcoidosis
- Nutritional deficiencies
- Electrolyte abnormalities
- Obstructive sleep apnea

What is arrhythmia-induced cardiomyopathy ?

AIC is a condition characterized by
either a tachyarrhythmia
(tachycardia - induced cardiomyopathy)
or frequent ventricular ectopy
(PVC - induced cardiomyopathy)
that results in LV dysfunction and heart failure.

The hallmark of this condition is
partial or complete reversibility
once arrhythmia
control is achieved.

Summary of common reversible cardiomyopathies and proposed mechanisms.

CARDIOMYOPATHY TYPE	SUBTYPES EXAMPLES	KEY SPECULATED MECHANISM(S)
Tachyarrythmia-induced	*Sinus tachycardia* *Rapid AF/Aflutter* *Ventricular tachycardia* *Reentrant tachycardia*	1) Tissue adenosine triphosphate depletion due to sustained tachycardia 2) Impaired subendocardial flow and vasodilatory reserve
Autoimmune mediated	*Peripartum*	Cytokine/autoimmune mediated
Inflammatory/Infectious	*HIV cardiomyopathy* *Viral Myocarditis* *Sepsis/Septic shock induced*	1) T-cell autoimm 2) Direct
Sympathoexcitation	*Takotsubo* *Auto*	
		→protein receptor (Endocannabinoid, CO, NO)→ smic Ca^{+2} release →↓ contractility
Chronic		resistance→Glucotoxicity→Cardiomyocyte apoptosis 2) Chronic inflammatory state(IL-6) → Apoptosis via (Ca^{+2} influx) 3) Cardiac steatosis
		1) Cardiac hypertrophy caused by→Accumulation of cardiotonic steroids → Sustained ↑ volume and pressure 2) Cardiac fibrosis

Tachycardia-induced HF was first described in 1913 in a patient with atrial fibrillation

Gossage AM, Braxton Hicks JA. On auricular fibrillation. QJM 1913;6:435–440.

Fenelon et al proposed the following criteria:

- Dilatation of the heart or heart failure
- Chronic or very frequent cardiac arrhythmias, including incessant SVTs, AF or AFL and incessant VT

- If chronic tachycardia continued more than 10-15% of the day, with an atrial rate of more than 150% of that predicted for age, cardiomyopathy occurs

TWO CATEGORIES OF AIC

- **Type 1 AIC – (arrhythmia induced)** when arrhythmia is solely responsible for AIC and the LV function returns to normal upon successful treatment of the arrhythmia.

- **Type 2 AIC – (arrhythmia mediated)** Arrhythmia exacerbates the underlying cardiomyopathy and treatment of the arrhythmia results in partial resolution of the cardiomyopathy

A62-year-old man without significant past medical history presented with new onset HF symptoms. His ECG on presentation revealed a wide complex tachycardia and an echo demonstrated a LVEF of 10-15 % with normal LV wall thickness and a moderately dilated LV cavity.
An EPS was performed, which made the diagnosis of atrio ventricular reciprocating tachycardia (AVRT) with a concealed left lateral accessory pathway, which was successfully abated.
A follow-up echo one month later demonstrated an improved LVEF to 35-40 % and an echo performed one year later demonstrated normal LV wall thickness, cavity size and systolic function

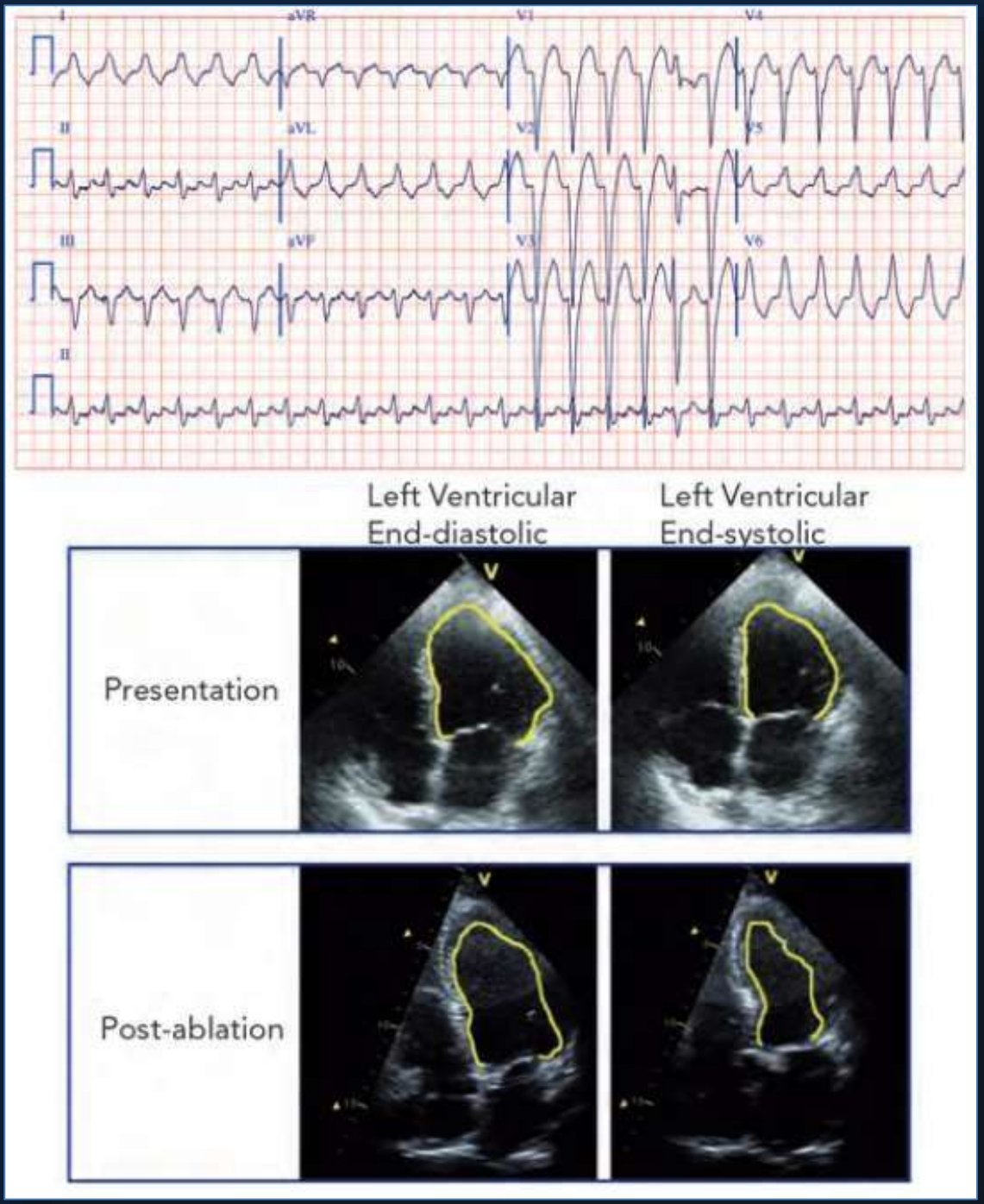


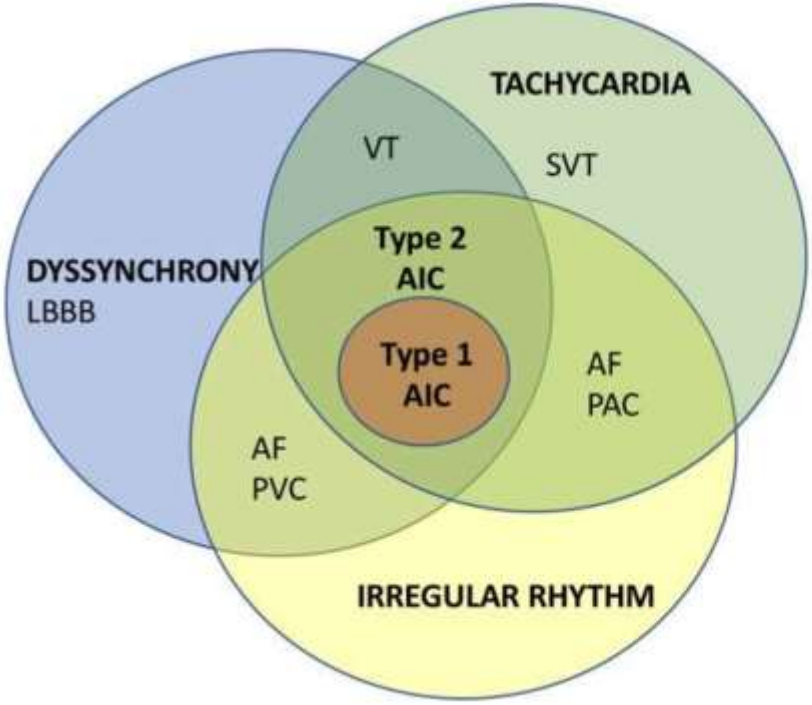
TABLE 1 A wide range of atrial and ventricular arrhythmias have been reported in association with AIC has been listed

Causes of tachycardia-mediated AIC	
Supraventricular	Atrial fibrillation[2,7]
	Ectopic atrial tachycardia[25,26]
	Atrial flutter
	Atrioventricular nodal re-entry tachycardia
	Atrioventricular tachycardia
	Permanent junctional reciprocating tachycardia
Ventricular	Premature ventricular contraction (PVC)[14,54]
	Ventricular tachycardia (high burden)

PATHOPHYSIOLOGY OF AIC

- three mechanisms with considerable overlap between 3 factors

1. Tachycardia
2. Irregular rhythm
3. Dys-synchrony



- In animal models ,LV dysfunction is relatively reproducible with rapid pacing resulting in LV dysfunction within weeks of tachycardia onset. Three phases have been reported

- **Phase 1**

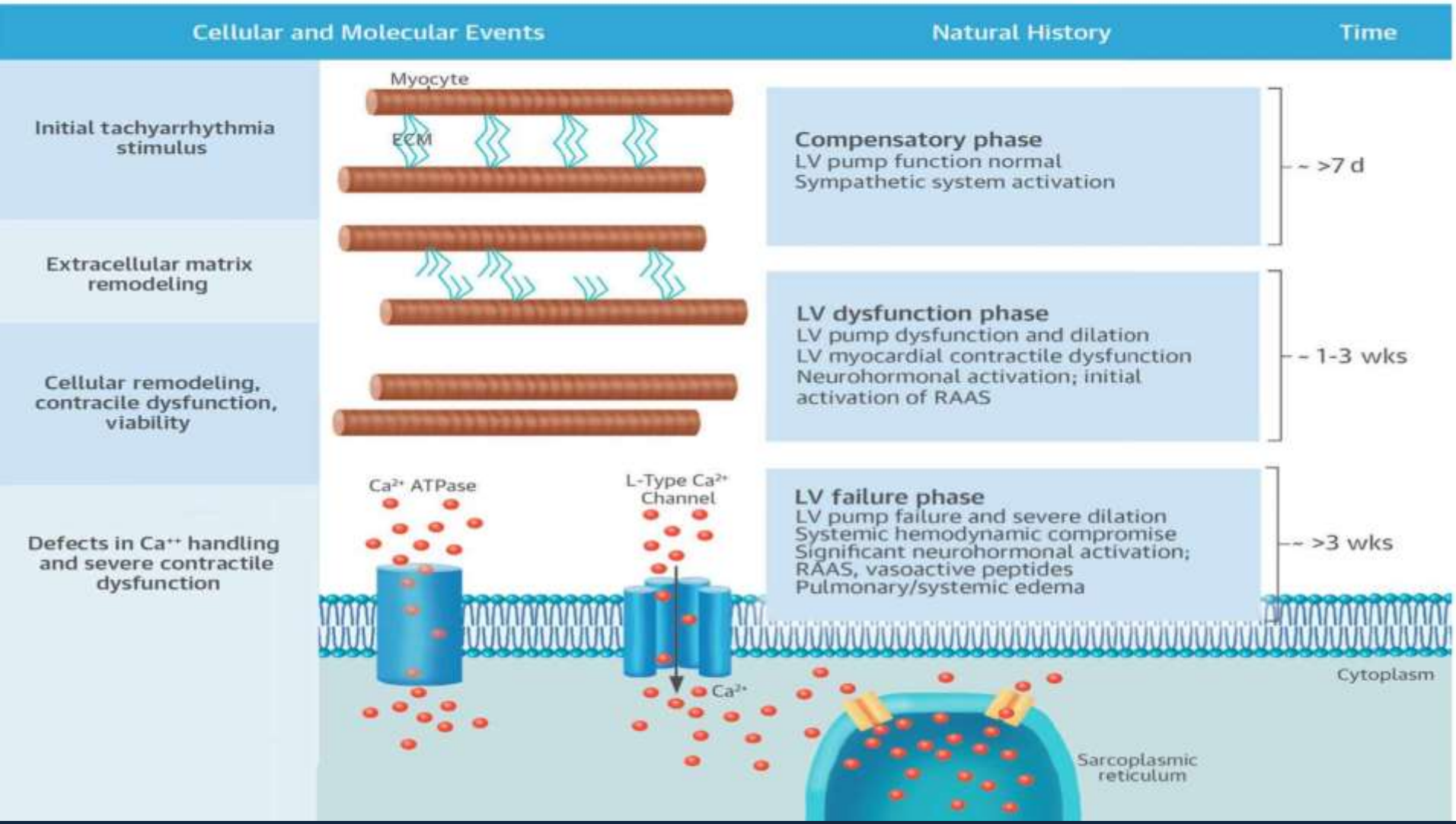
Compensatory phase (>7 days). During this phase, there is ***increased neurohormonal activation with early changes to the extra cellular matrix and preserved LV systolic function***.

- **Phase 2-** LV dysfunction phase (1-3 weeks).

Continued neurohormonal activation and ***upregulation of the renin angiotensin system.*** There is cellular remodeling, contractile dysfunction with ***LV systolic dysfunction and dilatation***.

- **Phase 3-** ***LV failure phase*** (>3 weeks).

Further adverse LV remodeling with pump failure, severe dilatation, and ***abnormal intracellular calcium handling***.


Cellular and Molecular Events
Natural History
Time
Initial tachyarrhythmia stimulus
Extracellular matrix remodeling
Cellular remodeling, contracile dysfunction, viability
Defects in Ca++ handling and severe contractile dysfunction
Myocyte
ECM
Compensatory phase
LV pump function normal
Sympathetic system activation
~ >7 d
LV dysfunction phase
LV pump dysfunction and dilation
LV myocardial contractile dysfunction
Neurohormonal activation; initial activation of RAAS
~ 1-3 wks
LV failure phase
LV pump failure and severe dilation
Systemic hemodynamic compromise
Significant neurohormonal activation; RAAS, vasoactive peptides
Pulmonary/systemic edema
~ >3 wks
Ca2+ ATPase
L-Type Ca2+ Channel
Ca2+
Cytoplasm
Sarcoplasmic reticulum

Clinical features

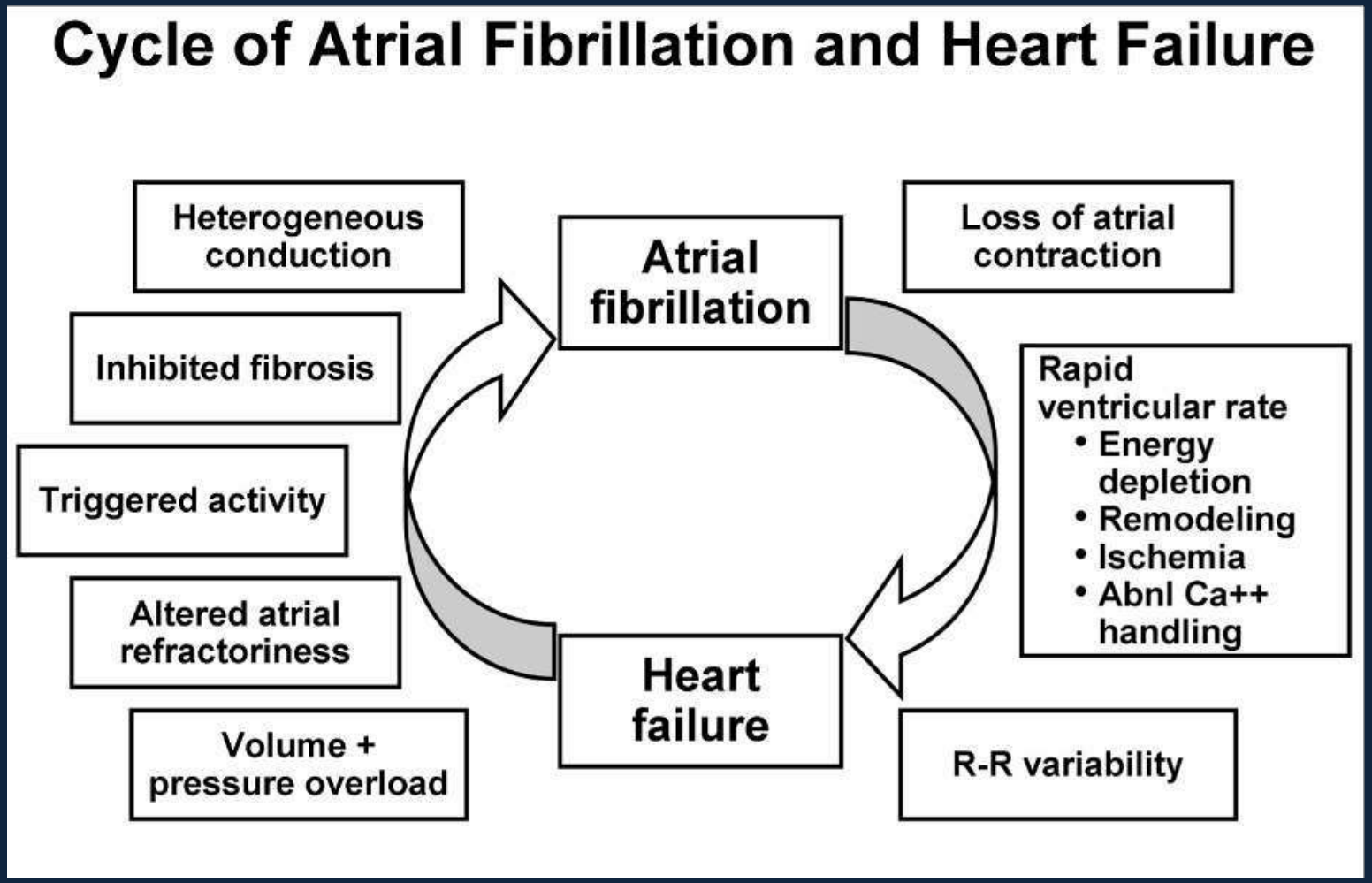
- AGE:
  - can occur at any age
  - reported in infants, children, adolescents, and adults
- Follows any type of chronic or frequently recurring paroxysmal tachyarrhythmias
- Atrial fibrillation, atrial flutter, ectopic atrial tachycardia, atrioventricular tachycardia, and ventricular tachycardia have all been reported to cause

- Unclear why some patients with chronic tachyarrhythmia develop ventricular dysfunction whereas others tolerate high rates and maintain normal systolic function

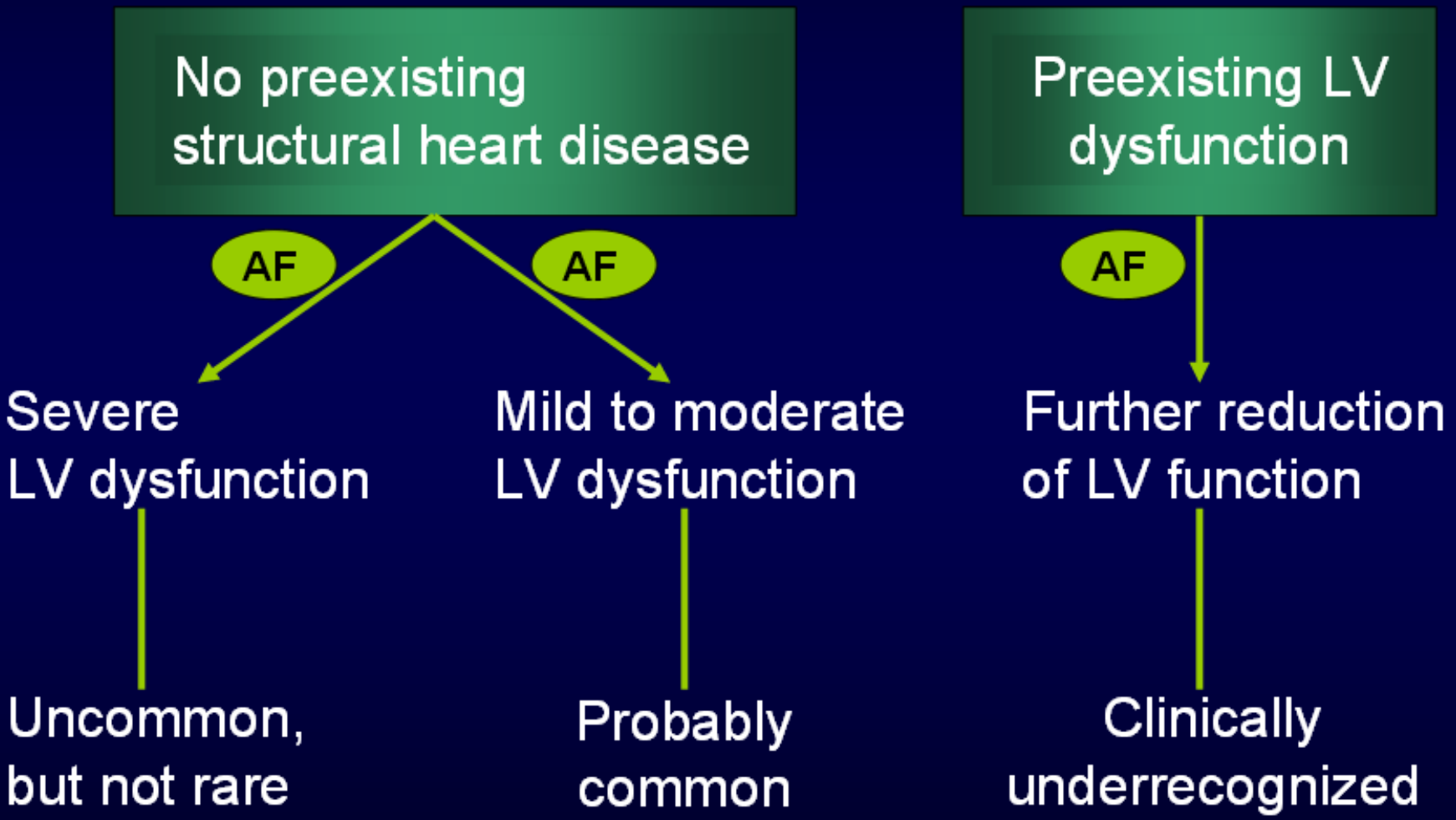
- Presumed risk factors include:
  - Type, rate and duration of tachyarrhythmia
  - Patient’s age
  - Underlying heart disease
  - Drugs
  - Coexisting medical conditions

AF is the commonest cause of AIC

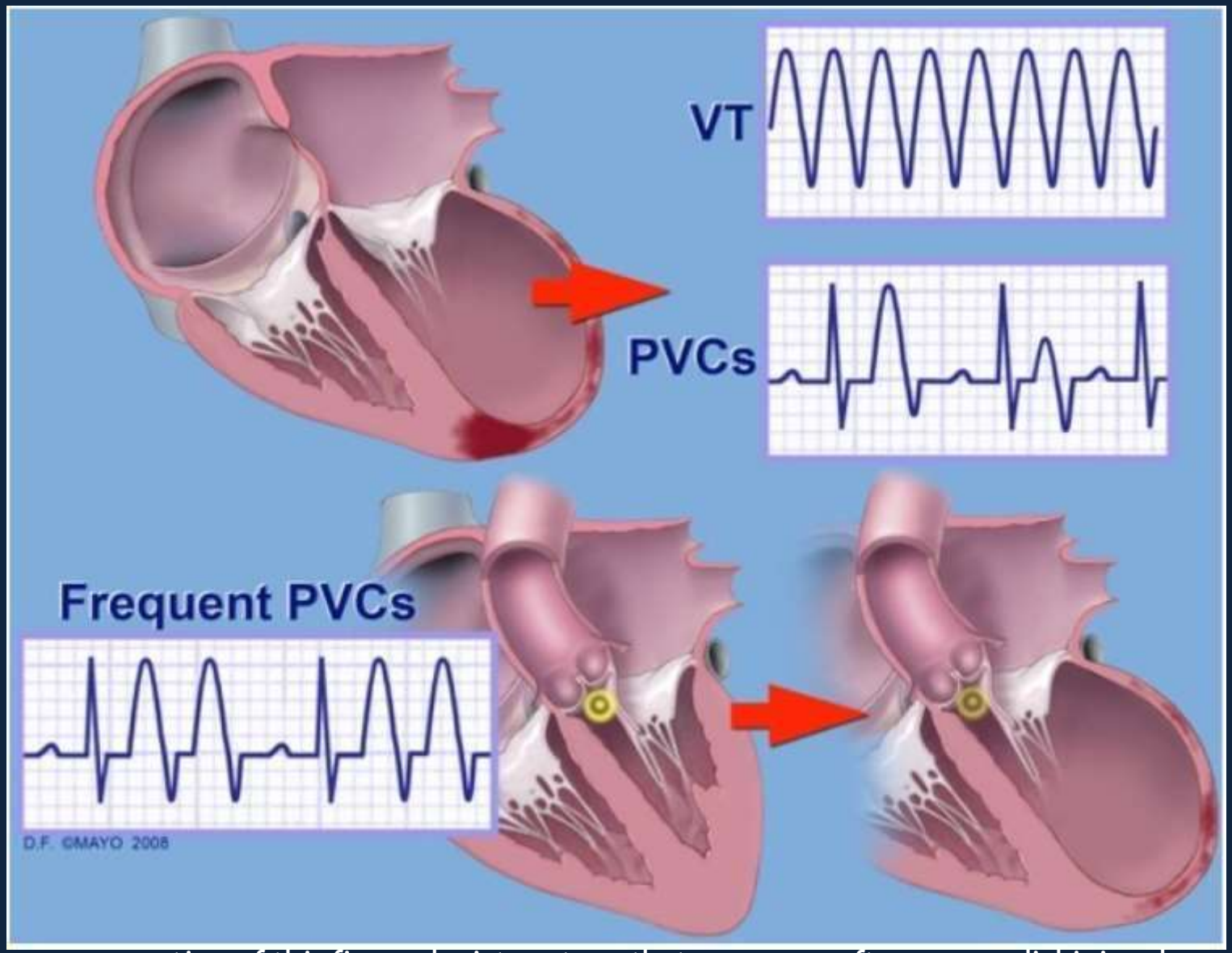
and is reported to be present in 10-50% of patients with heart failure



AF-induced LV dysfunction frequency



The PVC and Cardiomyopathy : Which Came First ?

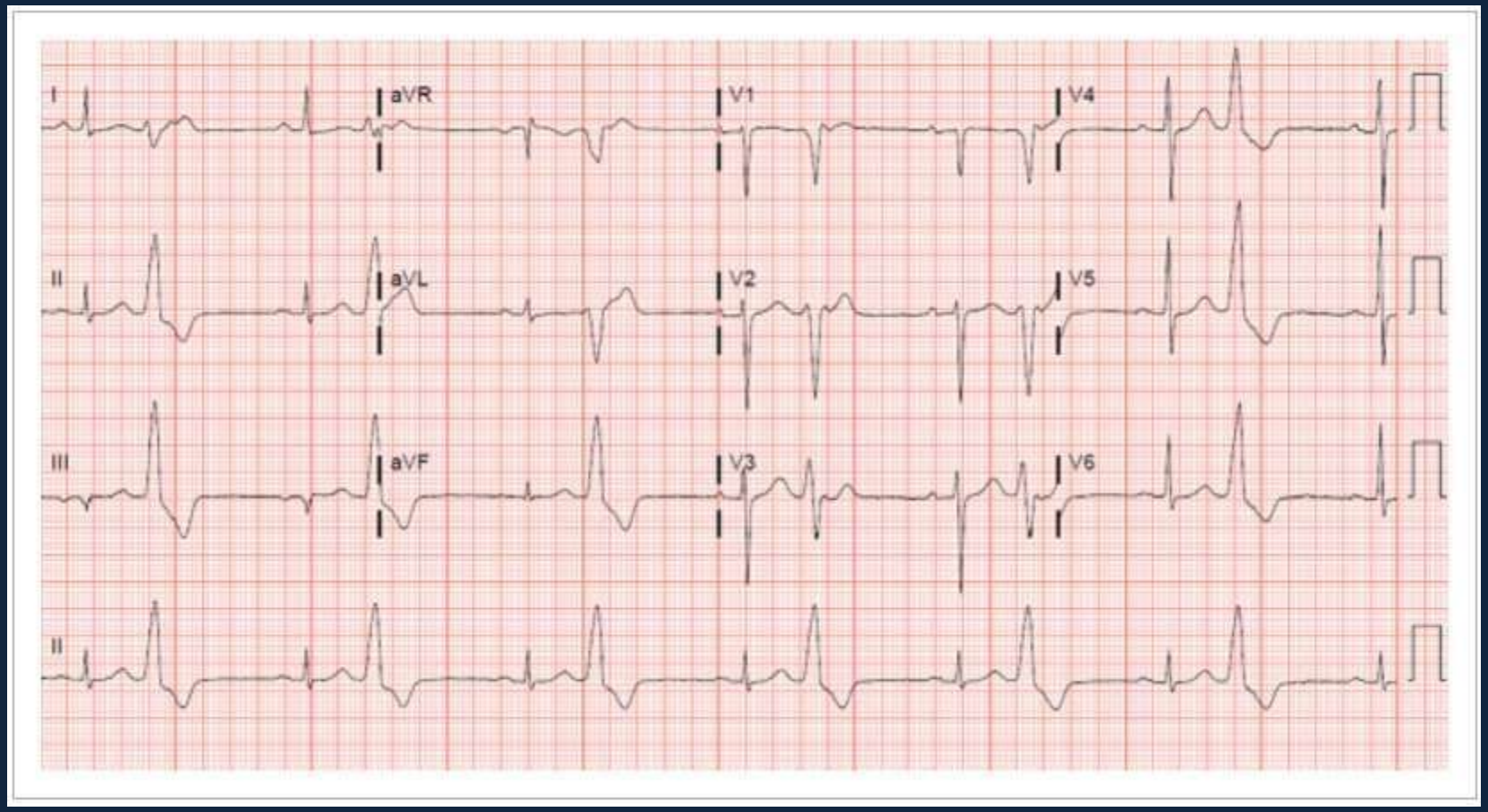


The upper portion of this figure depicts ectopy that can occur after myocardial injury has caused ventricular dysfunction.Frequent PVC's may also cause ventricular remodeling as seen in the lower portion of the figure.

Characteristics of primary cardiomyopathy versus tachycardia-induced cardiomyopathy

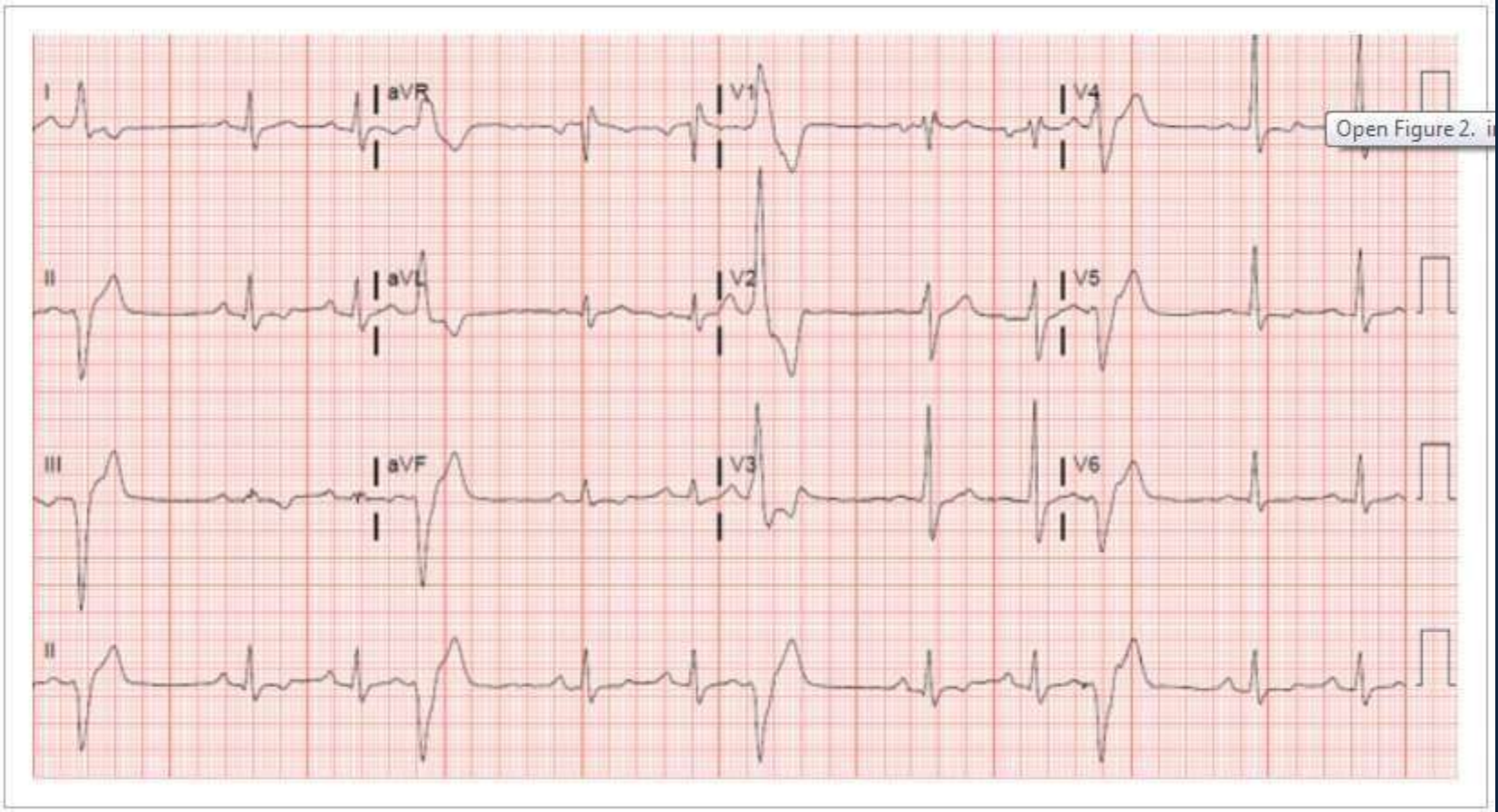
	PVCs resulting from cardiomyopathy	PVCs causing cardiomyopathy
Patient Characteristics	**Older patients with known cardiovascular disease**	**Otherwise healthy individuals**
Comorbidity	**Hypertension, ischemic heart disease, myocarditis, ventricular dysplasia, family history of myocardial disease**	**Often no prior cardiac or family history of relevance**
Ejection fraction	**Depressed**	
Frequency of PVCs	**Less than 5000/24 hours**	**More than 10,000/24 hours; often more than 20,000/24 hours**
Pattern of PVCs	**Multi-morphic**	**Monomorphic**
QRS Morphology	**Nonspecific**	**- Outflow tract (right of left bundle-branch block with strong inferior access** **- Fascicular (atypical right bundle-branch block pattern with superior access)**
Response of Temporary Antiarrhythmic Therapy (amiodarone)	**Despite PVCs being suppressed, there is no improvement in myocardial function**	**If PVCs suppressed, myocardial function improves**
Response to Radiofrequency Ablation	**Only required if associated with ventricular tachycardia that has been triggering frequent ICD shocks, no effect on ventricular function**	**Normalization of ventricular function frequently seen**

(PVC-LBBB)



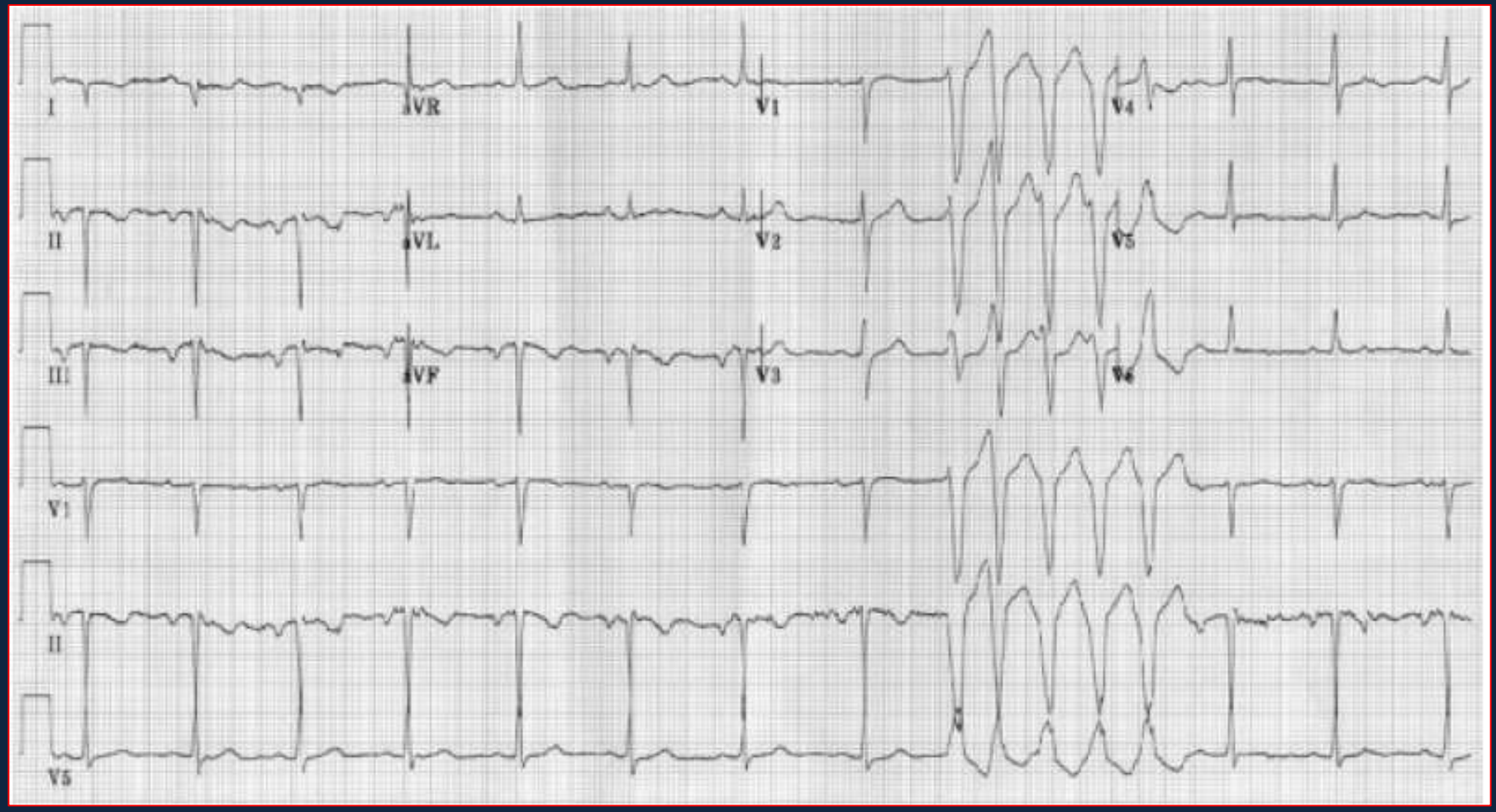
A 12-lead ECG shows LBBB morphology PVCs with inferior axis in bigeminy pattern. (RVOT origin)

(PVC-RBBB)



A 12-lead ECG shows RBBB morphology PVCs with superior axis in trigeminy pattern.

NSVT



ECG demonstrating sinus rhythm with a five beat run of non sustained ventricular tachycardia with LBBB morphology (negative in V1).

How do I recognize the patient with AIC?

We are frequently faced with patients in clinical practice who have the combination of arrhythmia and heart failure, however teasing out which came first (the old chicken or the egg question) is not easy.

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Europace (2012) **14**, 466–473
doi:10.1093/europace/eur348

REVIEW

Arrhythmia-induced cardiomyopathies: the riddle of the chicken and the egg still unanswered?

Emmanuel N. Simantirakis, Emmanuel P. Koutalas, and Panos E. Vardas*

Cardiology Department, Heraklion University Hospital, PO Box 1352, 71110 Heraklion, Crete, Greece

A high index of suspicion is therefore required for AIC, particularly given the reversibility of the condition.

There are no established diagnostic criteria for AIC.

However, in a patient presenting with new onset LV dysfunction and a chronic or recurrent tachycardia with heart rate over 100 beats per minute, the diagnosis of AIC may be suggested by the following **once ischemic cardiomyopathy is ruled out:**

1. No other cause of non-ischemic cardiomyopathy found (eg. hypertension, alcohol or drug use, stress etc.)

2. Absence of LVH

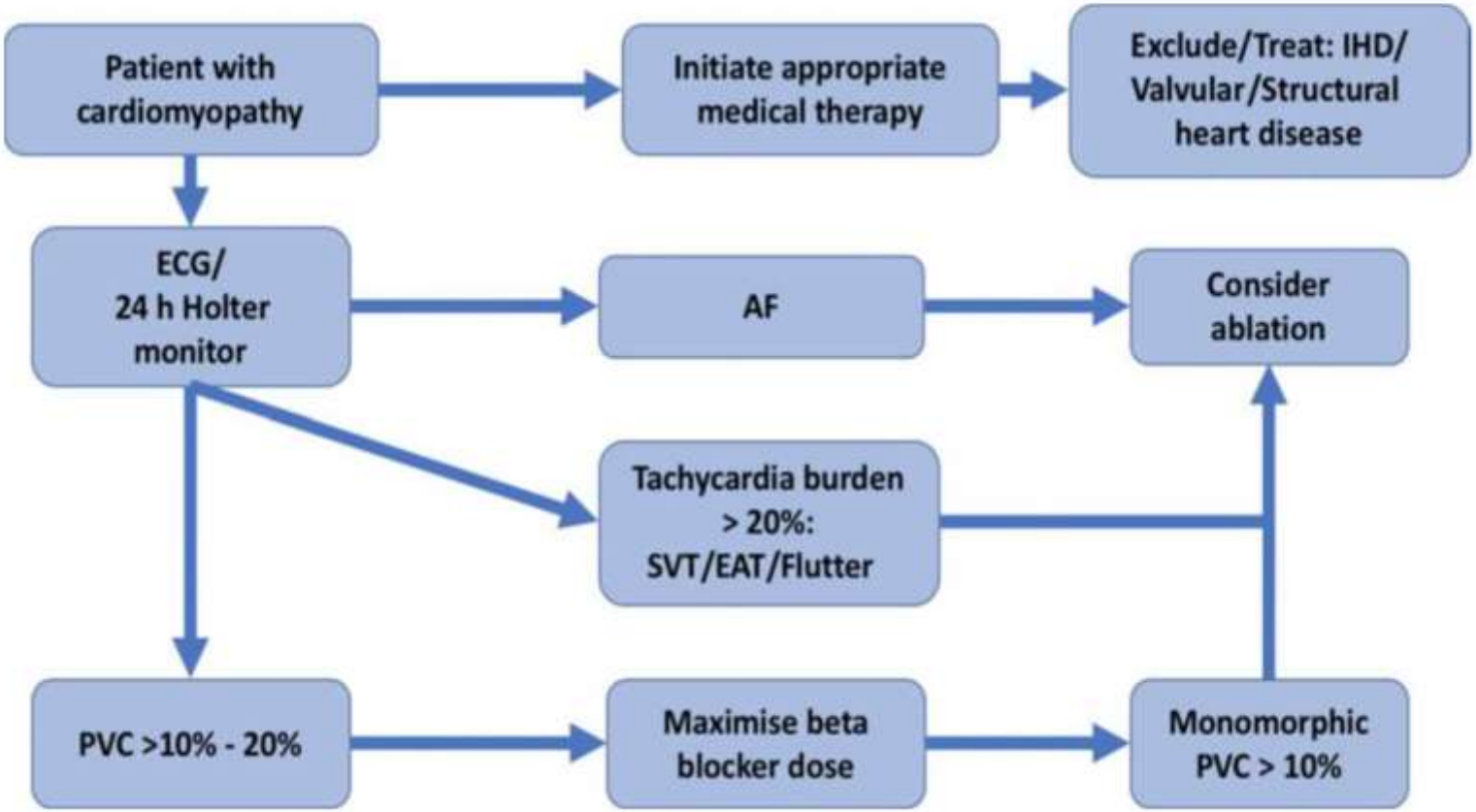
3. Relatively normal LV dimensions (LV end-diastolic dimension< 5.5 cm)

4. Recovery of LV function after control of tachycardia (by rate control, cardioversion or radiofrequency ablation) within one to six months.

5. Rapid decline in LVEF following recurrence of tachycardia in a patient with recovered LV function after control of tachycardia previously.

cMRI may help differentiate AIC from dilated cardiomyopathy. (evidence for LGE, suggesting underlying scar)

MANAGEMENT OF AIC



How do I manage AIC?

PRINCIPLES OF MANAGEMENT

AIC management should focus on concerted attempts to **eliminate or control the arrhythmia**, with the goal of improving symptoms, reversing LV dysfunction , and preventing arrhythmia recurrence

AIC Associated with specific Arrhythmias in adult

Atrial fibrillation

AF is the most common cause of AIC in adults

Management of AF consists of rate and/or rhythm control

European Heart Journal (2016) **37**, 2893–2962
doi:10.1093/eurheartj/ehw210

ESC GUIDELINES

2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS

The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC)

Developed with the special contribution of the European Heart Rhythm Association (EHRA) of the ESC

Endorsed by the European Stroke Organisation (ESO)

Atrial flutter

Atrial flutter is more difficult to rate control than AF

Catheter ablation to eliminate atrial flutter is recommended when AIC is suspected.

For those in whom catheter ablation is not feasible or desired, cardioversion with antiarrhythmic therapy or aggressive rate control should be used.

Supraventricular tachycardias

A curative strategy by catheter ablation should be pursued whenever possible as first-line therapy for SVT-mediated AIC.

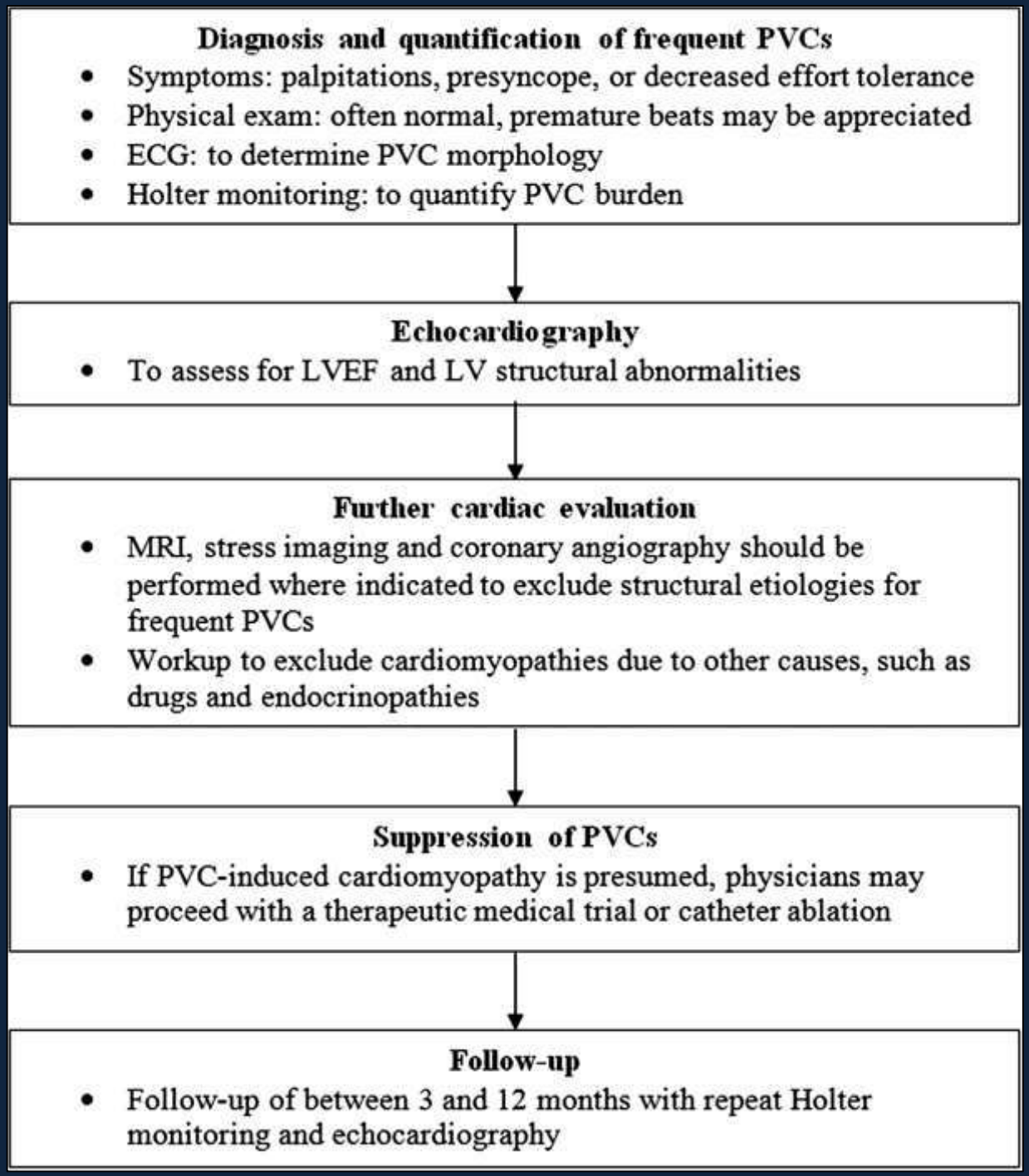
PVC-induced cardiomyopathy.

How many PVC's are too much? **PVC burden**

- *The most prominent predictor of cardiomyopathy in patients with frequent PVCs appears to be the daily burden of PVCs.*
- *There appears to be a threshold burden of approximately 10,000 PVCs/day for developing AIC.*
- *Ventricular function can improve if the PVC burden is reduced to <5,000/day*

- ✓ Therapy for PVC-mediated AIC should be targeted at suppressing or eliminating the PVCs and should include antiarrhythmic therapy and catheter ablation.
- ✓ Catheter ablation has emerged as the definitive therapy for PVC-mediated AIC, with success rates ranging from

Frequent PVCs in the office setting: What do you do?



Yong-Mei Cha et al. Circ Arrhythm Electrophysiol. 2012;5:229-236

Recovery , prognosis, and impact of recurrent arrhythmia on AIC

•Clinical and animal studies have documented the resolution of signs and symptoms of HF and recovery of LV dysfunction with termination of culprit arrhythmia.

- **Although AIC was originally**

Resolution of HF and recovery of LVEF may not imply normalization of LV structure and function.

Another major factor affecting recovery and outcomes is the effect of recurrent arrhythmia.

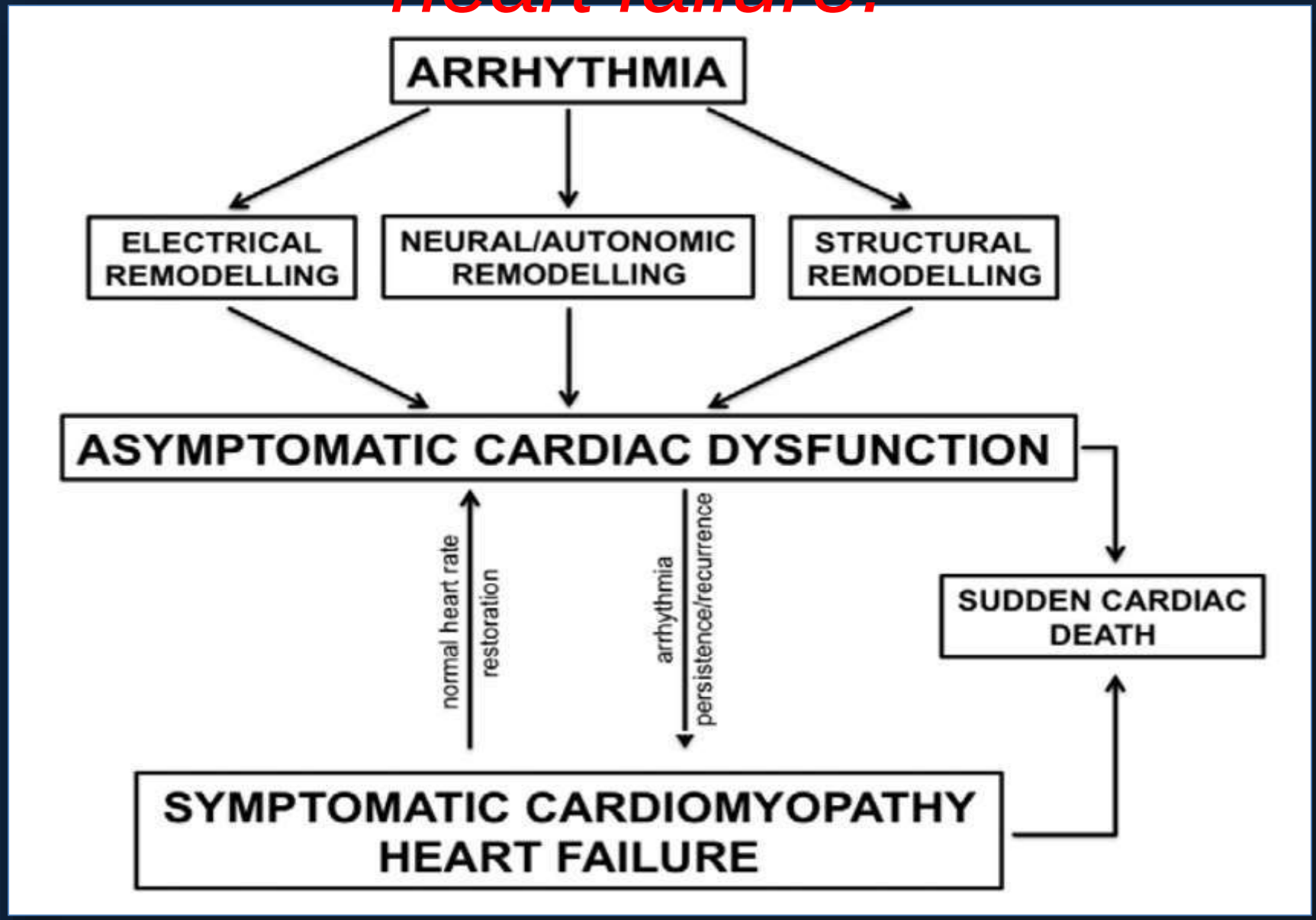
AIC really means "cure."

Risk of sudden death

- Long-term survival of patients with AIC following arrhythmia resolution is likely; however, concerns remain.

- Sudden cardiac death has been reported in patients with AIC following symptom recovery and LVEF normalization

From arrhythmia occurrence to heart failure.



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THE PRESENT AND FUTURE

COUNCIL PERSPECTIVES

Arrhythmia-Induced Cardiomyopathies

Mechanisms, Recognition, and Management

Rakesh Gopinathannair, MD, MA,* Susan P. Etheridge, MD,† Francis E. Marchlinski, MD,‡
Francis G. Spinale, MD, PhD,§ Dhanunjaya Lakkireddy, MD,‖ Brian Olshansky, MD¶

Overview of the Current Understanding of AIC, From Mechanisms to Management and Prognosis

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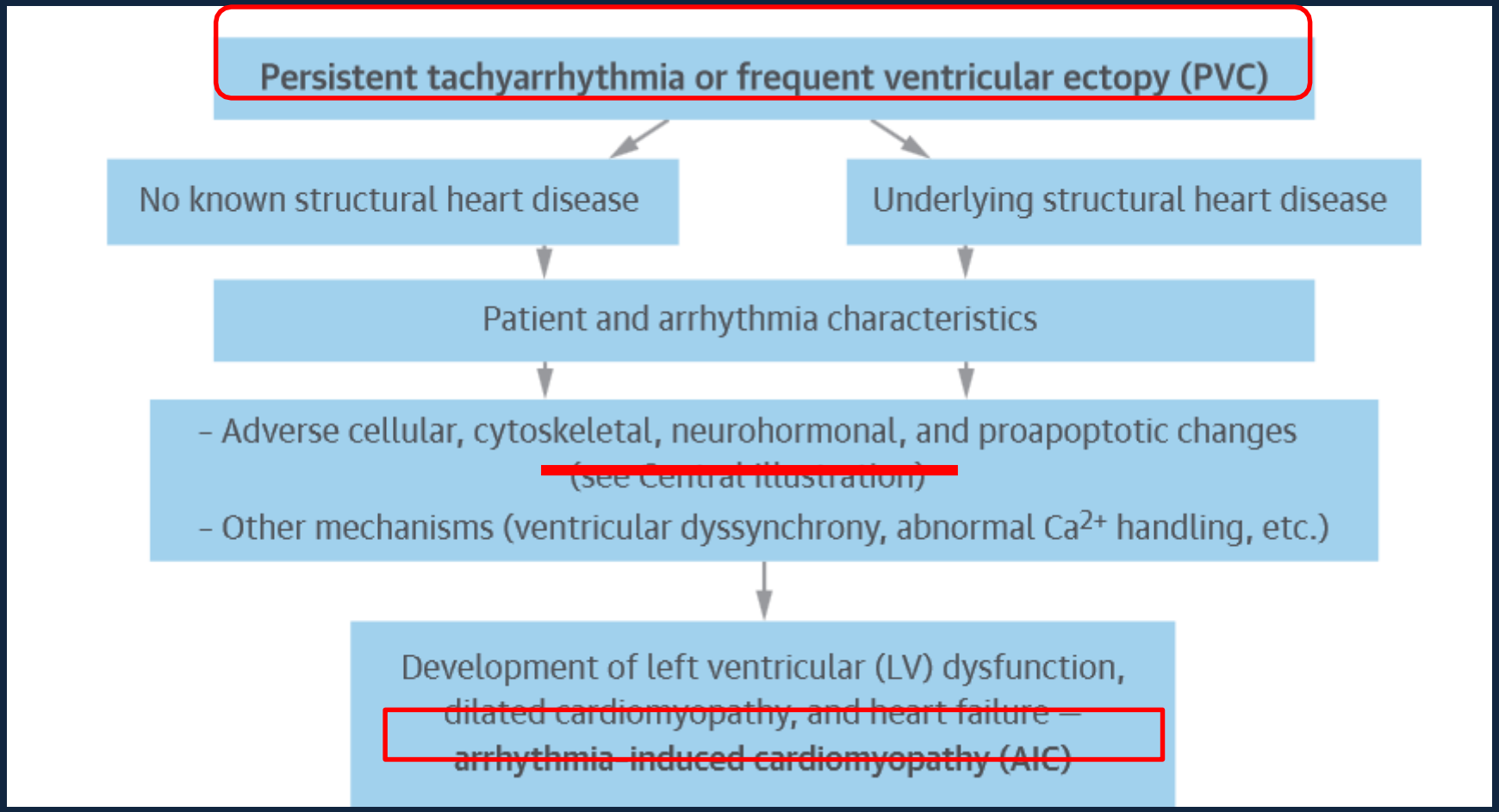
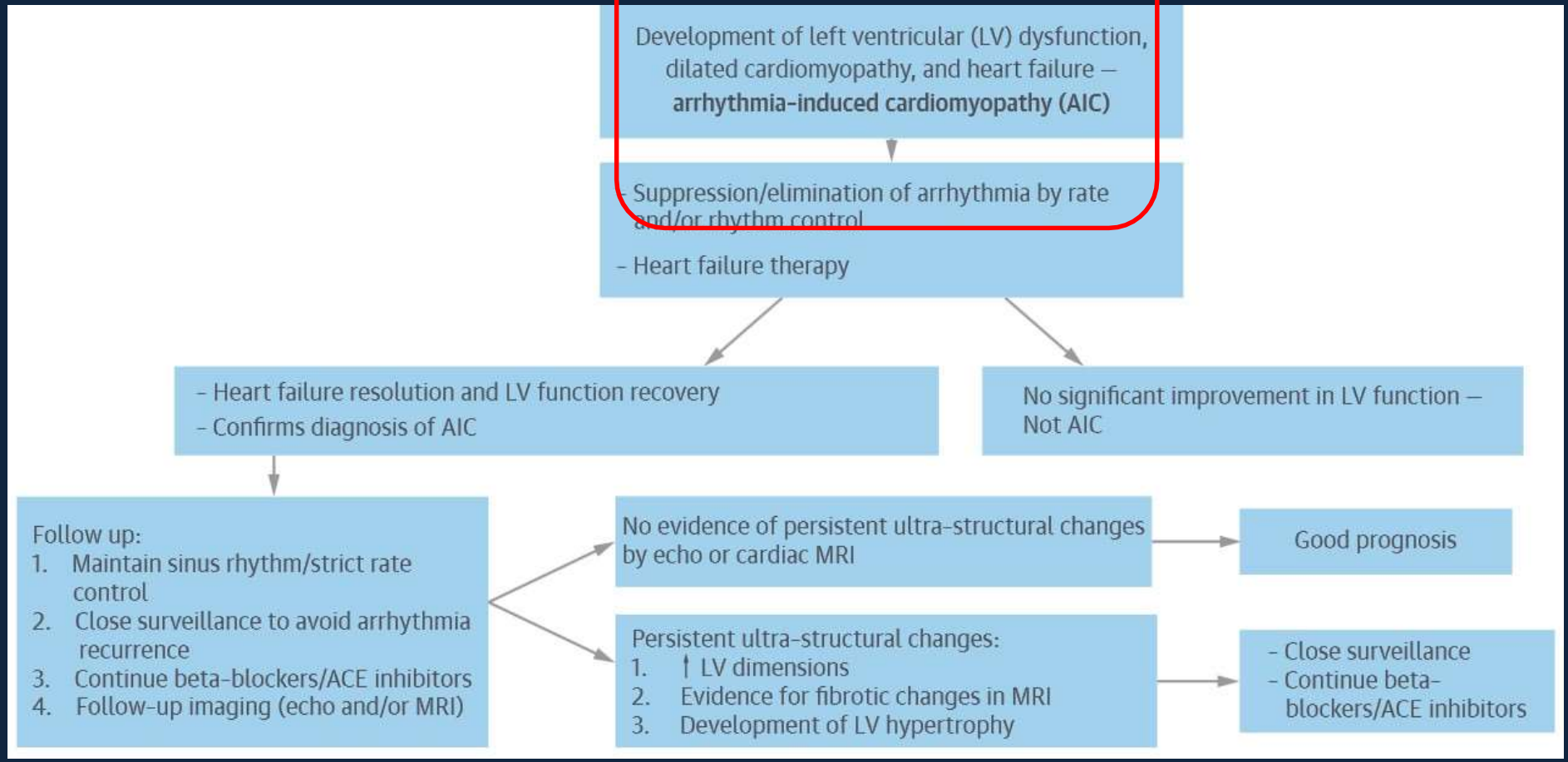


Overview of the Current Understanding of AIC, From Mechanisms to Management and Prognosis



Conclusion

When confronting a patient with heart failure and any kind of tachyarrhythmias:

- ***AF and uncontrolled ventricular rates***
- ***Frequent PVCs***

Arrhythmia-induced Cardiomyopathy
or
Arrhythmia-aggravated Cardiomyopathy

Clinicians should focus on eliminating the arrhythymia with catheter ablation and "attempt careful and aggressive control of rate and rhythm"

Thank you!