

**Extrasistolia**

# Parasistolie e ritmi idioventricolari

**Pasquale Crea**

# EXTRASISTOLI VENTRICOLARI

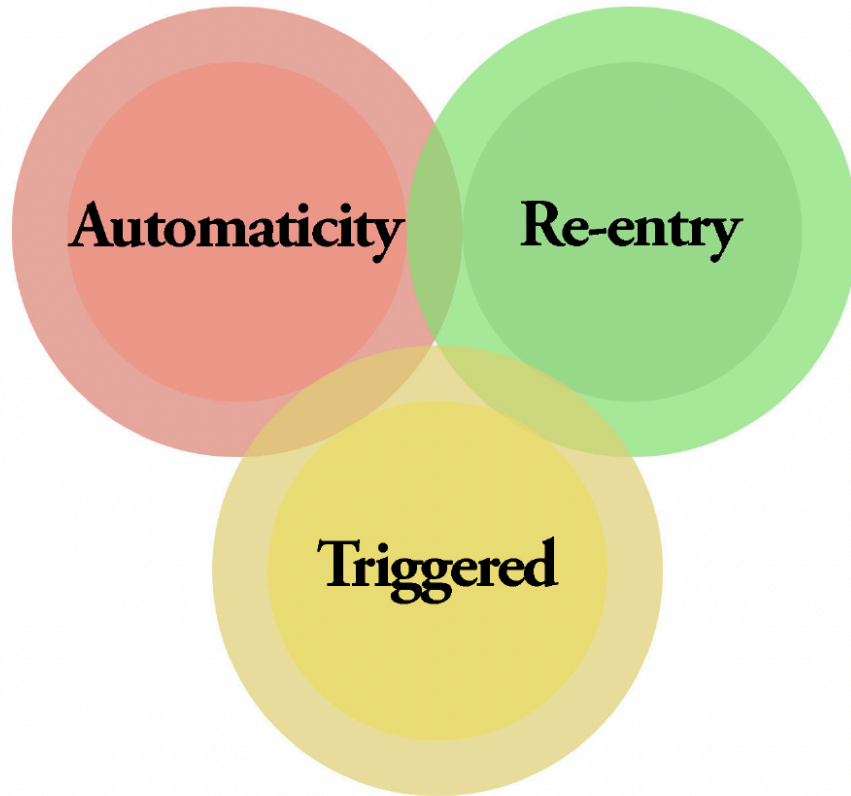
Le extrasistoli ventricolari sono **battiti prematuri di origine ventricolare**. L'extrasistole è legata al complesso precedente o dipendente da esso, contrariamente a quanto avviene nella **parasistolia**, nella quale la formazione dell'impulso ectopico è indipendente dal ritmo dominante. Nell'analisi delle extrasistoli ventricolari vanno presi in considerazione i seguenti punti:

- a) morfologia del complesso extrasistolico
- b) **copula dell'extrasistole**
- c) pausa postextrasistolica
- d) attivazione atriale, possibile retroconduzione agli atri
- e) distribuzione
- f) distinzione dalle extrasistoli sopraventricolari a conduzione aberrante
- g) aritmie che conseguono alle extrasistoli.

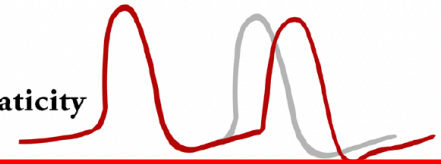


# EXTRASISTOLI VENTRICOLARI

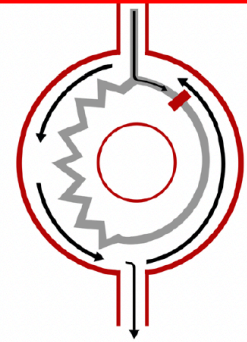
- Nelle extrasistoli ventricolari monomorfe l'intervallo di **copula** (intervallo che separa il battito prematuro dal precedente complesso sinusale o dominante) **è fisso o relativamente fisso**, cioè con variazioni non maggiori di 0.08 secondi; una copula marcatamente variabile deve invece far sospettare la presenza di una parasistolia.
- La copula fissa esprime la relazione causale esistente con il battito precedente, il quale induce, o quanto meno scatena, l'impulso ectopico. Tuttavia possono esservi extrasistoli ventricolari monomorfe a copula variabile; in questi casi la variazione di copula si accompagna spesso a una variazione della frequenza sinusale o della distribuzione dei battiti ectopici (per esempio dal bigeminismo al trigeminismo).



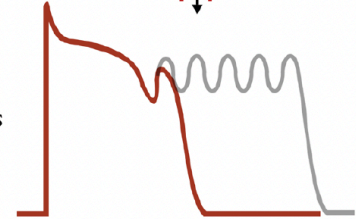
**Abnormal Automaticity**



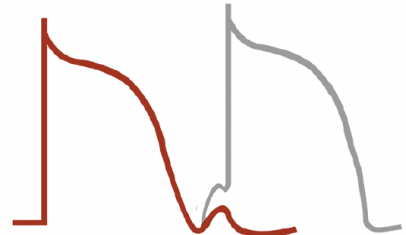
**Re-entry**



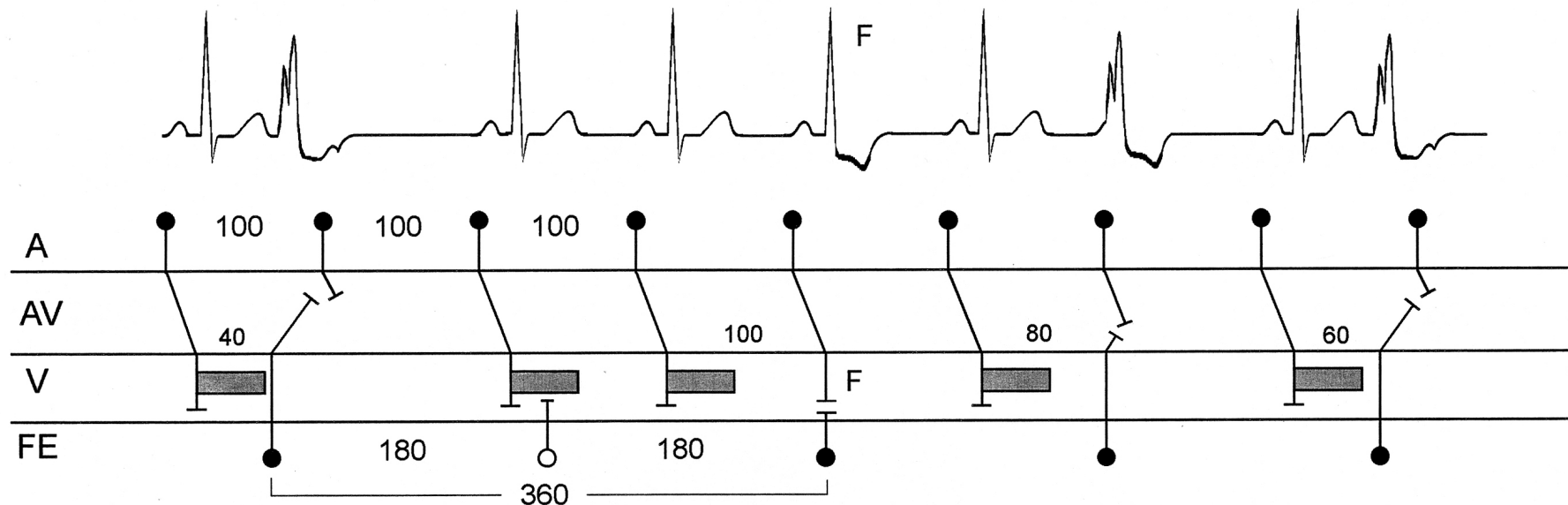
**Triggered activity**  
Early Afterdepolarizations



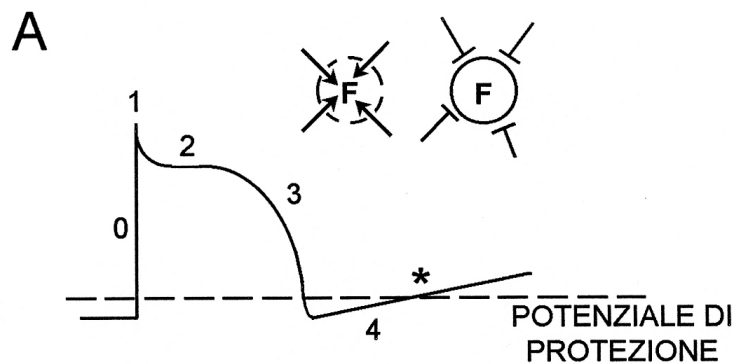
**Triggered activity**  
Delayed Afterdepolarizations



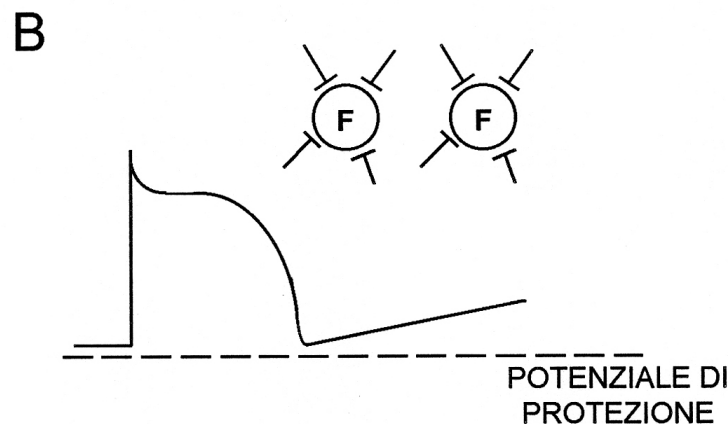
# PARASISTOLIA VENTRICOLARE



1. *Copula variabile dell'extrasistolia*
2. *Intervalli interectopici regolari o multipli*
3. *Battiti di fusione*



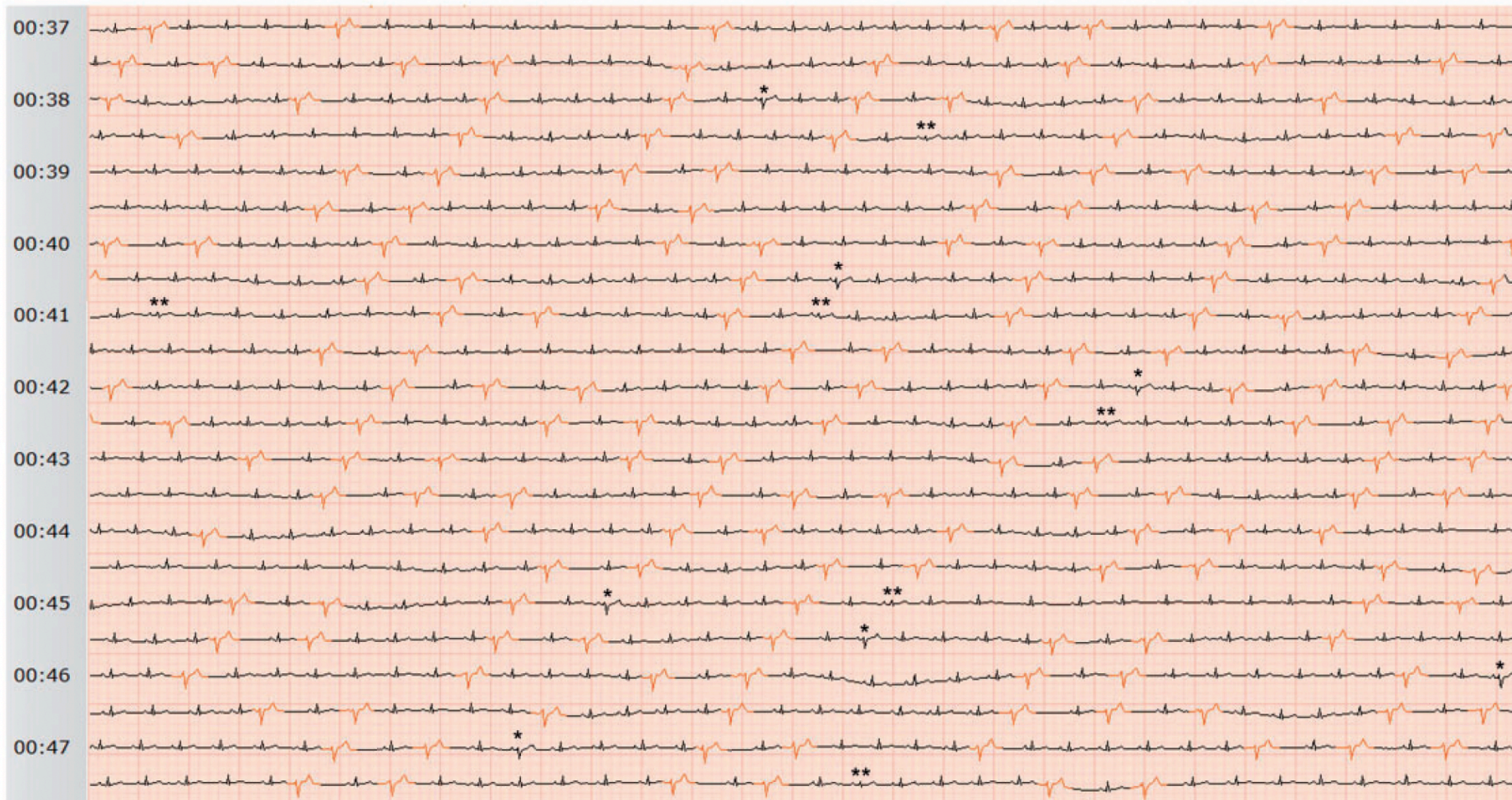
**Figura 37-2.** Lo schema A raffigura il potenziale d'azione transmembrana di una cellula miocardica. Durante la fase 4 il livello di polarizzazione si riduce, e il potenziale diviene meno negativo. A un determinato valore (asterisco) viene raggiunto il "potenziale di protezione", in corrispondenza del quale la cellula non è più eccitabile in modo normale. Perciò un impulso estraneo può penetrare nel focus solo se lo raggiunge prima che il potenziale intracellulare si porti al livello di protezione. Ciò è rappresentato dalle frecce che penetrano nel focus (F). Al contrario, impulsi estranei che giungono al focus più tardi risultano bloccati e non vi penetrano. Nello schema B il livello del potenziale intracellulare diastolico all'inizio della fase 4 è già meno negativo del potenziale di protezione; il focus è perciò "protetto" per tutta la durata del ciclo.

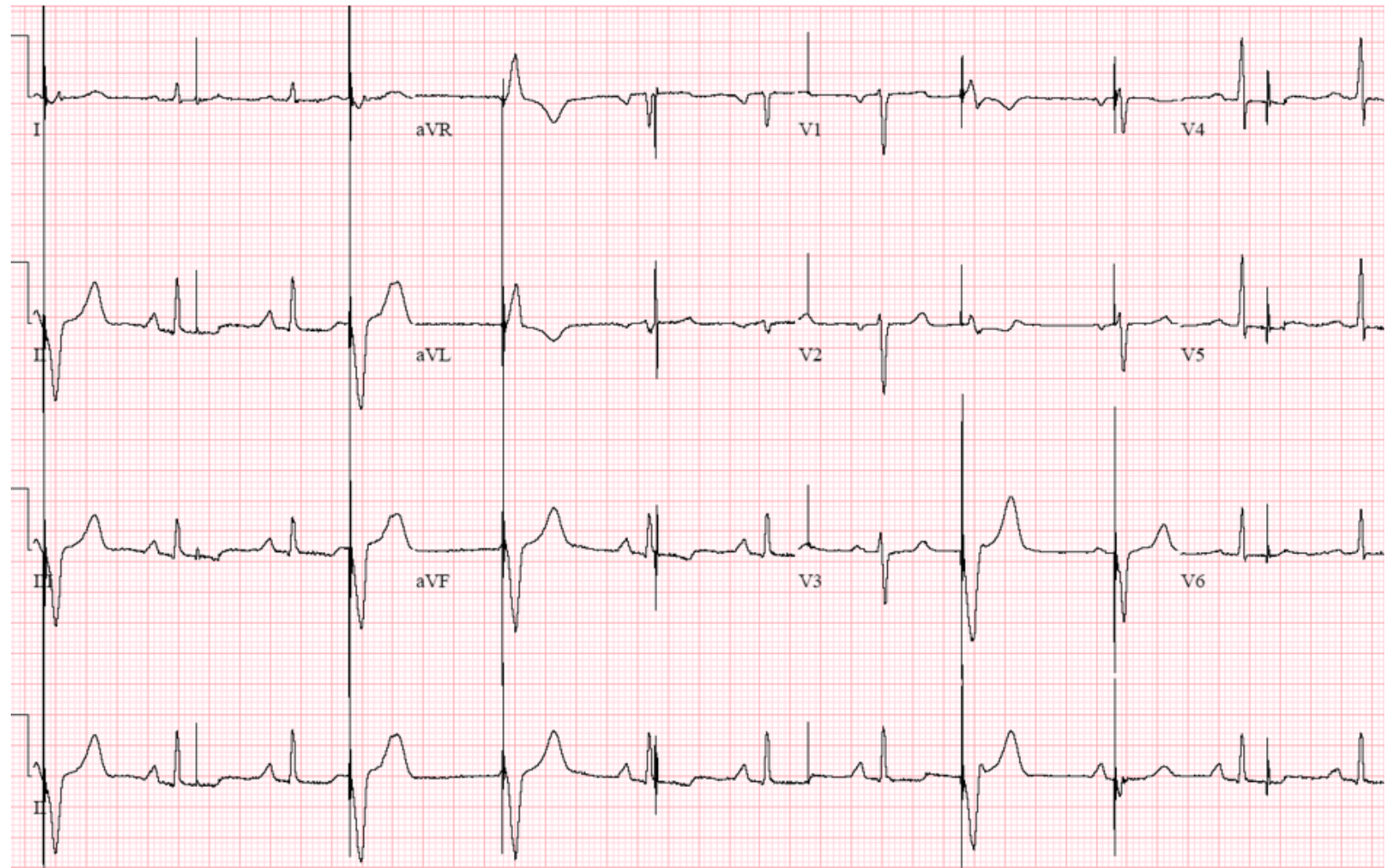




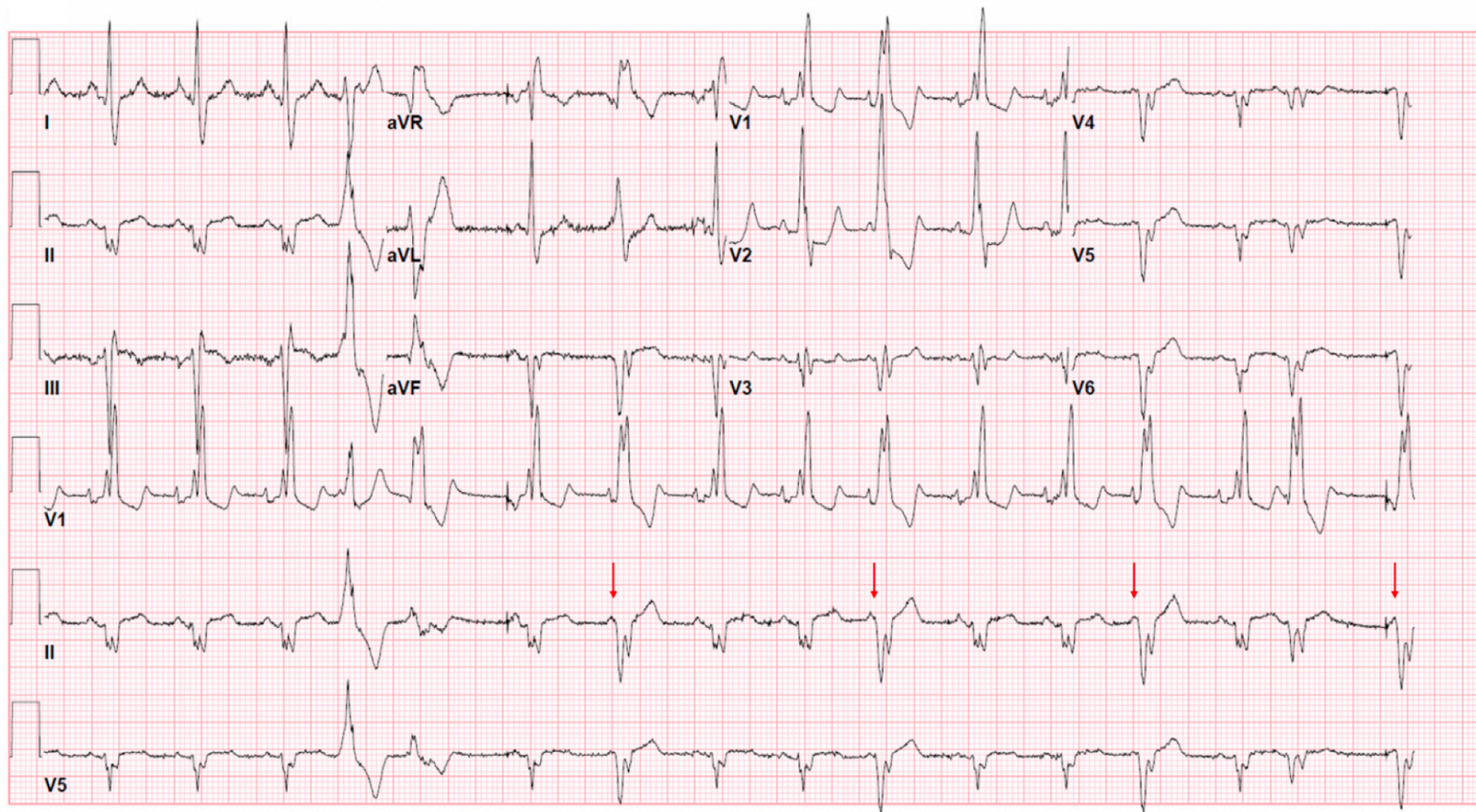






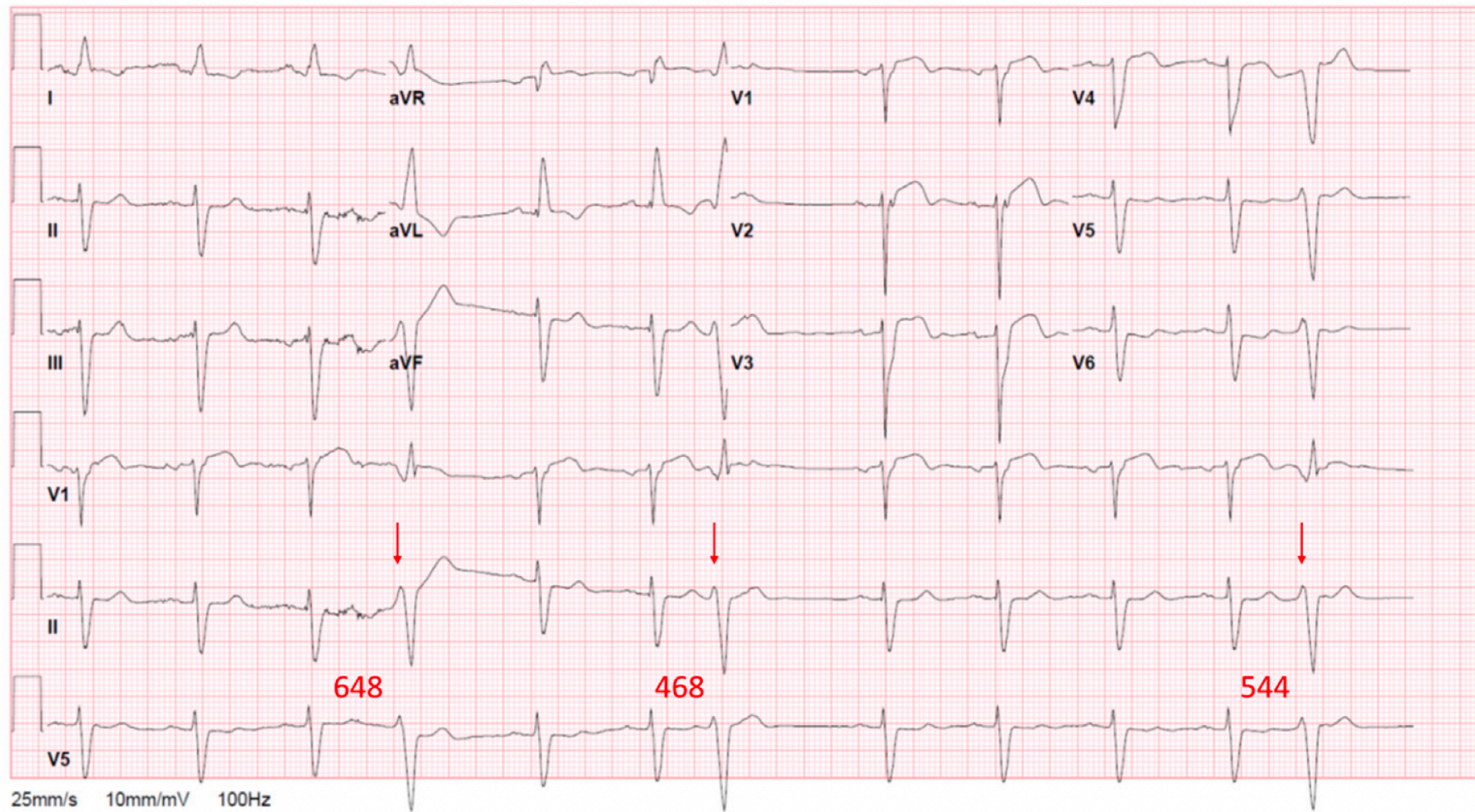




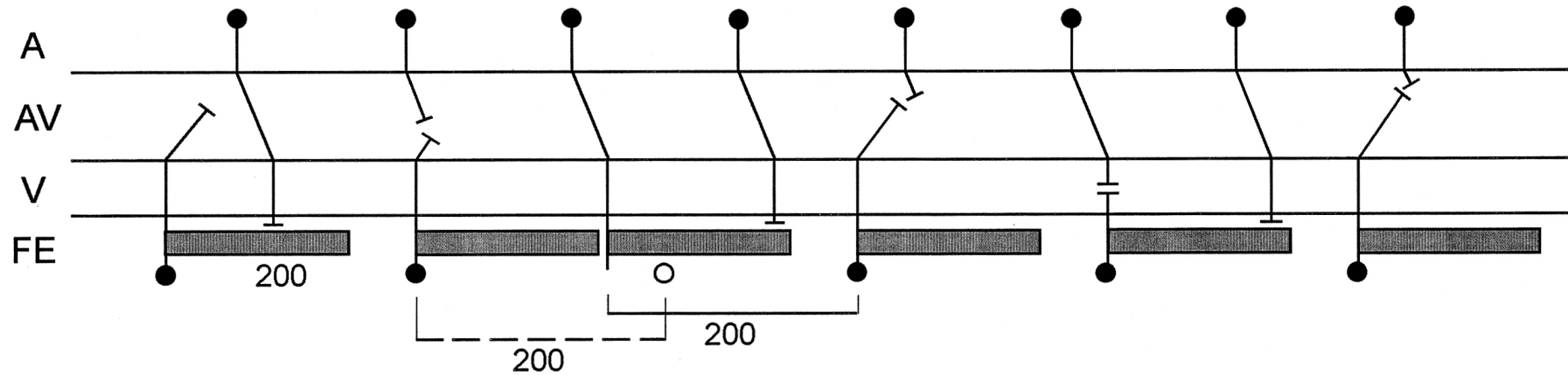
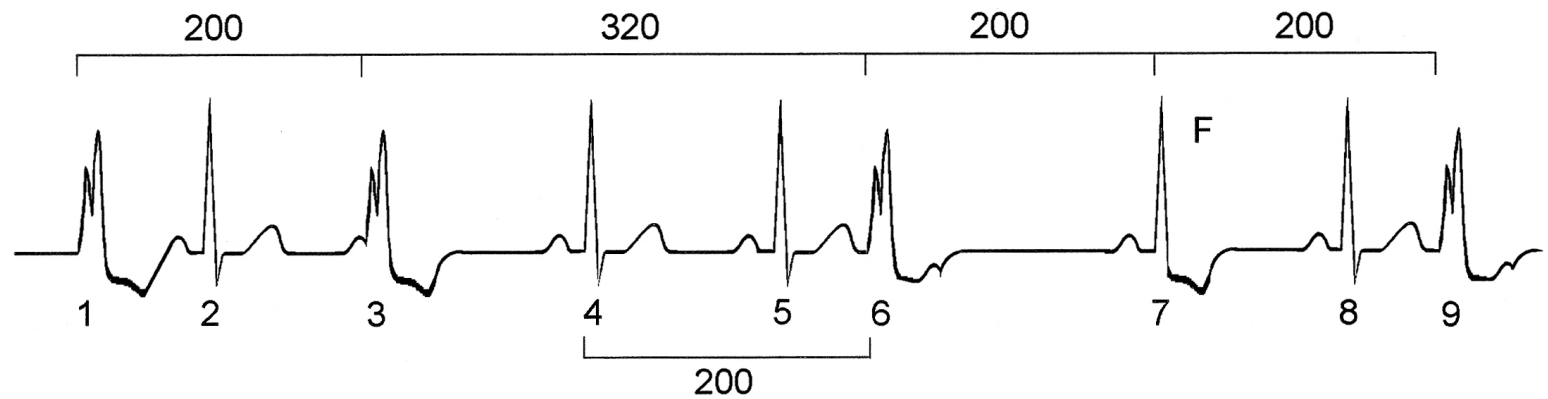


25mm/s 10mm/mV 150Hz

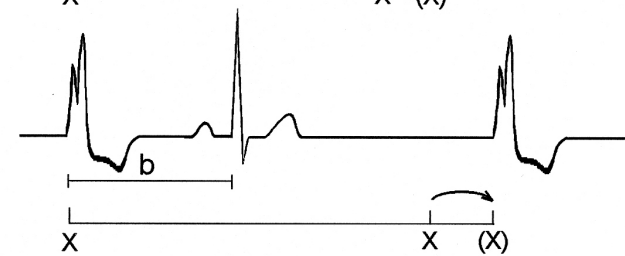
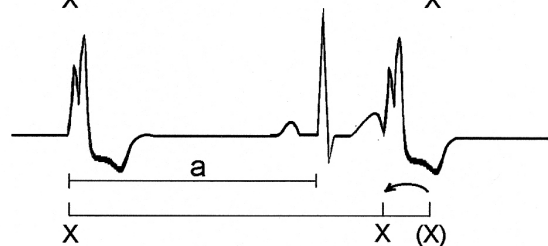
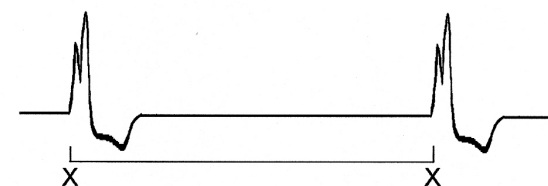
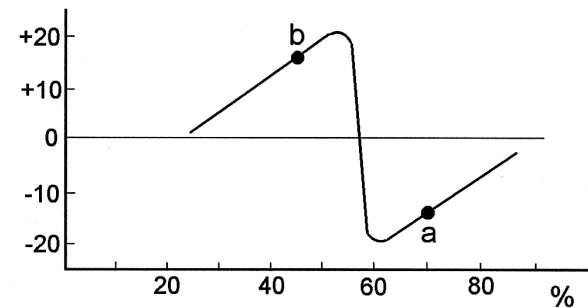
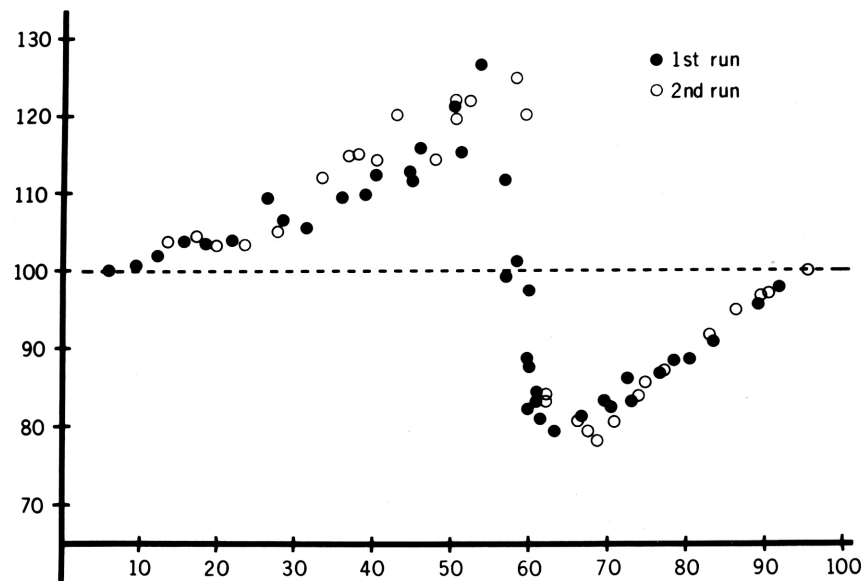












# **Modulated Ventricular Parasystole as a Mechanism for Concealed Bigeminy**

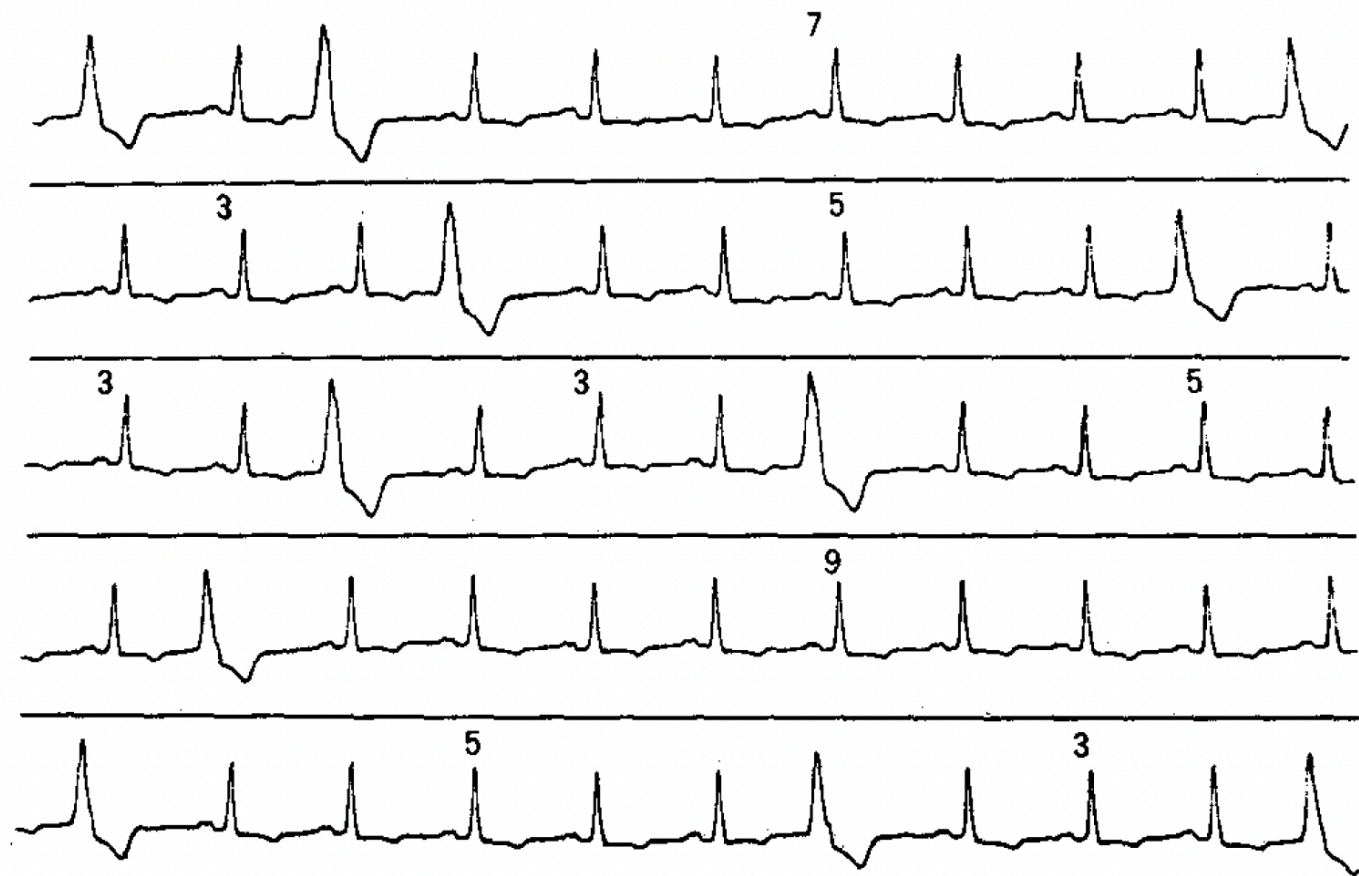
**GIUSEPPE ORETO, MD, FRANCESCO LUZZA, MD, GAETANO SATULLO, MD,  
and LEO SCHAMROTH, MD, DSc**

**Concealed extrasystolic ventricular bigeminy reflects a distributional pattern of ventricular extrasystoles where intervening sinus beats are always in odd numbers. This has been explained on bigeminal rhythm associated with exit block. This presentation reflects a modification of this concept in that**

**the distributional pattern may be explained on the basis of complex concealed modulation of ventricular parasystole, the concealment being due to impulses falling within the refractory period rather than suffering true exit block.**

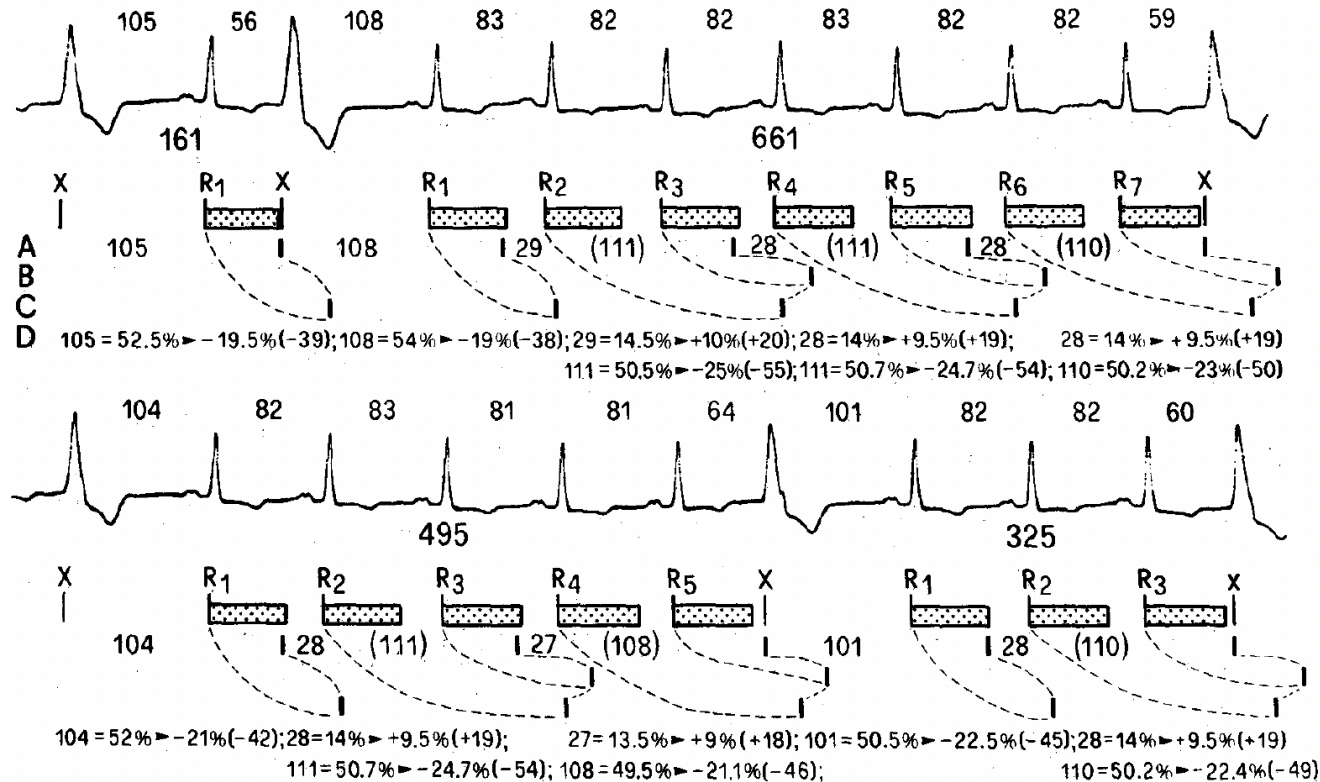
**(Am J Cardiol 1986;58:954-958)**

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**FIGURE 1. Continuous recording of standard lead II. Figures below the tracing indicate the numbers of intervening sinus beats.**





**FIGURE 3.** Sections of the electrocardiogram shown in Figure 1 (*top and bottom strip*) diagrammed to show the effects of modulation. *Vertical bars* reflect the ectopic discharges. *Line C* = scheduled discharges; *Line B* = intermediate discharges; *Line A* = actual discharges. Numbers in line A reflect the time intervals between the delivery of the parasystolic impulse and the following sinus beats. The time intervals between an ectopic discharge and the second consecutive modulated sinus beat are shown in parentheses. Detailed calculation of each modulation is reflected in line D. *Dotted areas* represent the refractory period of the ventricle.

ORIGINAL RESEARCH

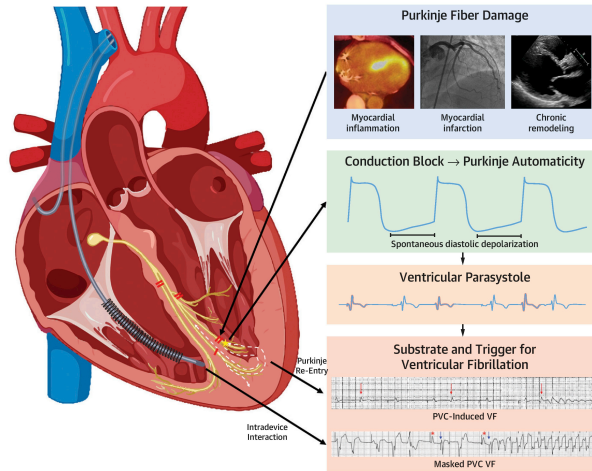
## Ventricular Parasystole in Cardiomyopathy Patients

### A Link Between His-Purkinje System Damage and Ventricular Fibrillation

Duc H. Do, MD, MS,<sup>3</sup> Kyle O'Meara, MD,<sup>3</sup> Jiyoung Lee, BS,<sup>3</sup> Scott Meyer, BS,<sup>3</sup> Peter Hanna, MD, PhD,<sup>3</sup> Shumpei Mori, MD, PhD,<sup>4</sup> Michael C. Fishbein, MD,<sup>5</sup> Noel G. Boyle, MD, PhD,<sup>4</sup> Marcelo V. Elizari, MD,<sup>6</sup> Jason S. Bradfield, MD,<sup>4</sup> Kalyanam Shivkumar, MD, PhD<sup>3</sup>



#### CENTRAL ILLUSTRATION: Parasystole as the Link Between Conduction System Damage and Ventricular Fibrillation



Do DH, et al. J Am Coll Cardiol EP. 2023;9(7):936-948.

#### ABSTRACT

**BACKGROUND** The clinical relevance and prognostic implications of ventricular parasystole are unknown.

**OBJECTIVES** This study sought to assess the prevalence of ventricular parasystole in patients with implantable cardioverter-defibrillators (ICDs) and ventricular parasystole's association with ventricular arrhythmias and conduction system abnormalities.

**METHODS** This study retrospectively evaluated patients who underwent ICD interrogation at a single center between June 1, 2019, and August 31, 2020, and reviewed all available ICD and electrocardiogram data. This study identified patients with ventricular parasystole and compared the prevalence of ventricular fibrillation (VF), ventricular tachycardia (VT), and new conduction system abnormalities in those with  $\geq 5$  years of intrinsic QRS-complex electrocardiograms to those without parasystole.

**RESULTS** This study included 374 patients (age  $57 \pm 21$  years, 72% male, 45% nonischemic, 32% ischemic cardiomyopathy), of which, 104 (28%) had VT only, 39 (10%) VF only, and 10 (3%) both VT/VF. Ventricular parasystole was identified in 33 patients (9%); parasystolic foci were predominantly from the His-Purkinje system. Compared with those without parasystole, patients with parasystole had a significantly higher rate of VF (36% vs 11%;  $P < 0.01$ ), but not VT (42% vs 29%;  $P = 0.12$ ). Patients with parasystole, compared with those without parasystole, had a higher prevalence of new conduction abnormalities, particularly progressive intraventricular conduction delay (11 of 18 [61%] vs 12 of 83 [14%];  $P < 0.01$ ) and new right bundle branch block (4 of 18 [22%] vs 1 of 83 [1%];  $P < 0.01$ ).

**CONCLUSIONS** Ventricular parasystole was strongly associated with new conduction system abnormalities and VF in patients who have cardiomyopathy with ICDs, suggesting a potential link between VF and His-Purkinje damage in this patient population. (J Am Coll Cardiol EP 2023;9:936-948) © 2023 by the American College of Cardiology Foundation.



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21.04.2016

25.02.2015

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## VISITA CARDIOLOGICA

Paziente che assume da anni piccole (sub-terapeutiche) dosi di farmaci antiaritmici dopo il riscontro di aritmie ventricolari.

All'esame obiettivo del cuore non sono presenti elementi patologici.

L'elettrocardiogramma mostra ritmo sinusale frequenza 60; tracciato normale.

Nella documentazione in possesso della signora si osservano, soprattutto in un Holter, battiti ectopici ventricolari espressione di parasistolia, come dimostra la variabilità della copula e i rapporti matematici fra gli intervalli interectopici.

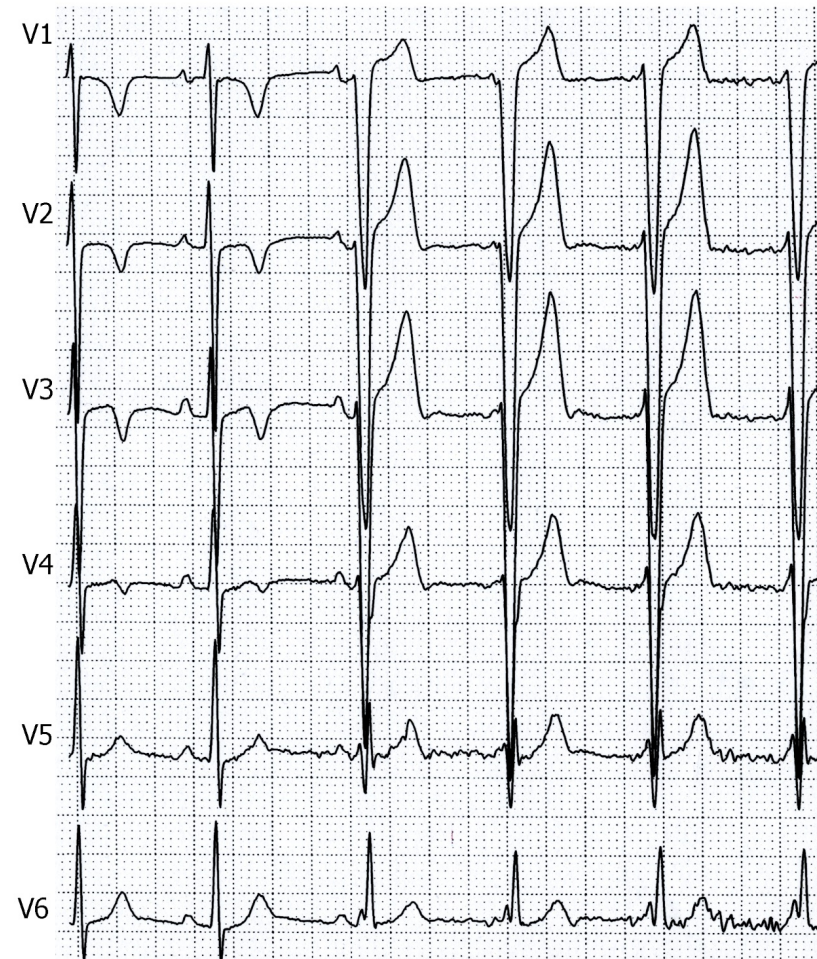
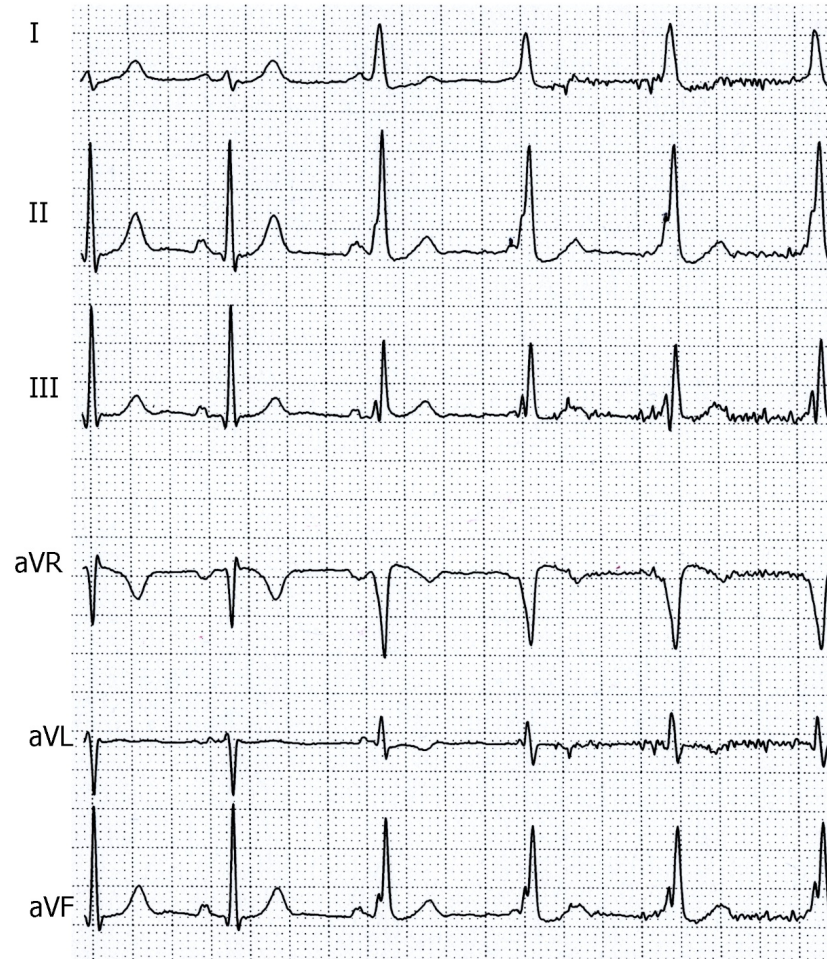
Questo genere di aritmia è del tutto benigna, e non va curata anche perché refrattaria alla terapia.

La signora può condurre vita normale, senza assumere farmaci cardiaci, e non necessita di controlli.

Prof. Giuseppe Oreto

Prof. Giuseppe Oreto

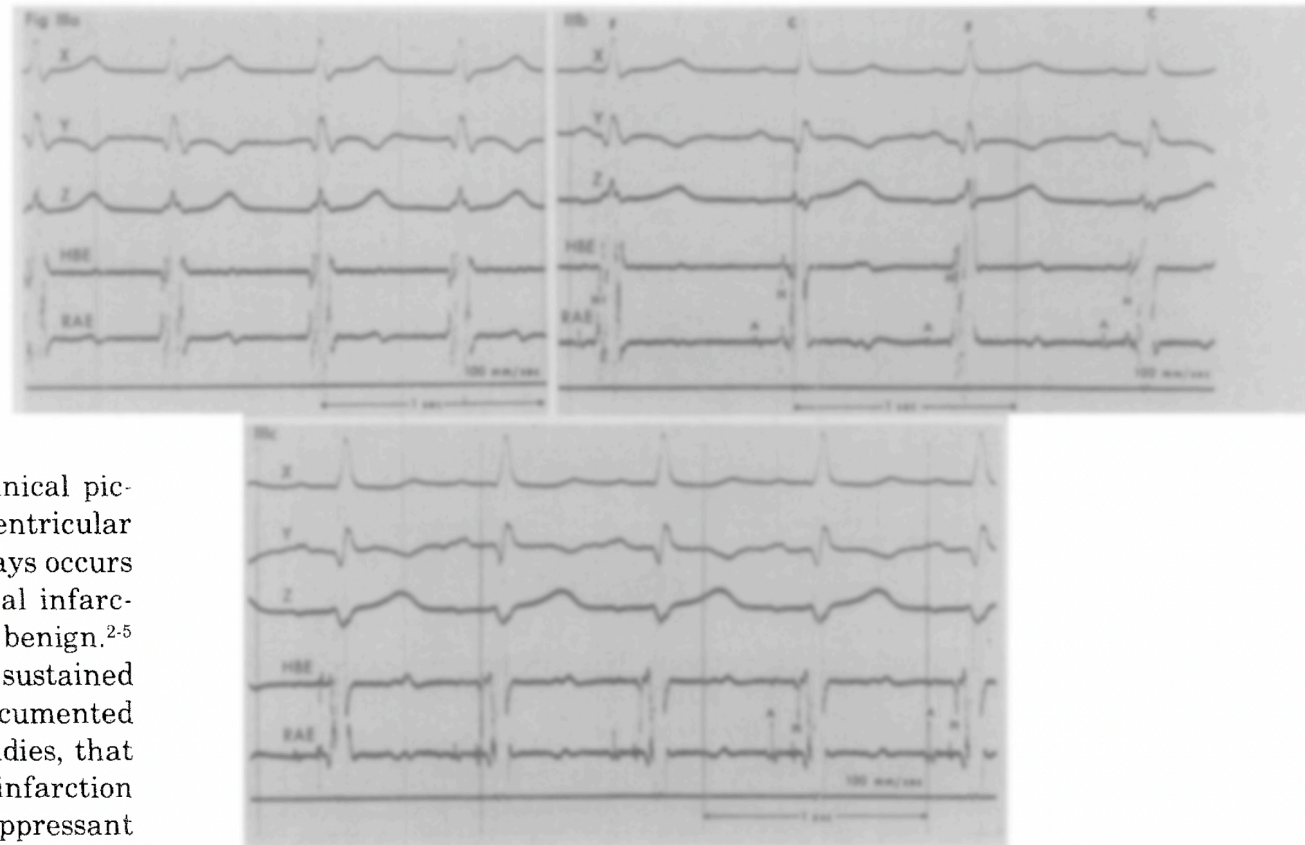




## Case reports

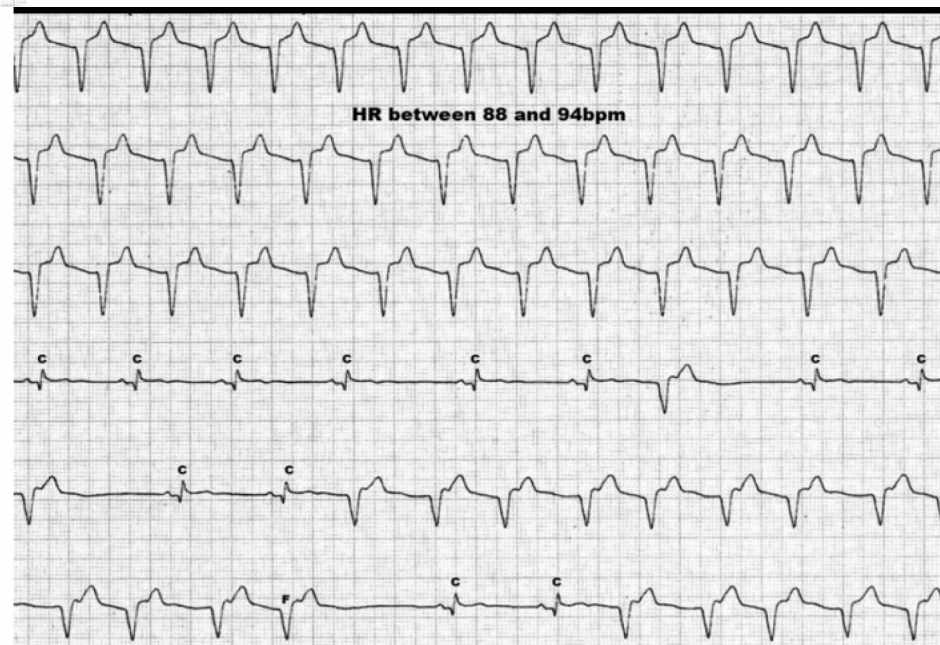
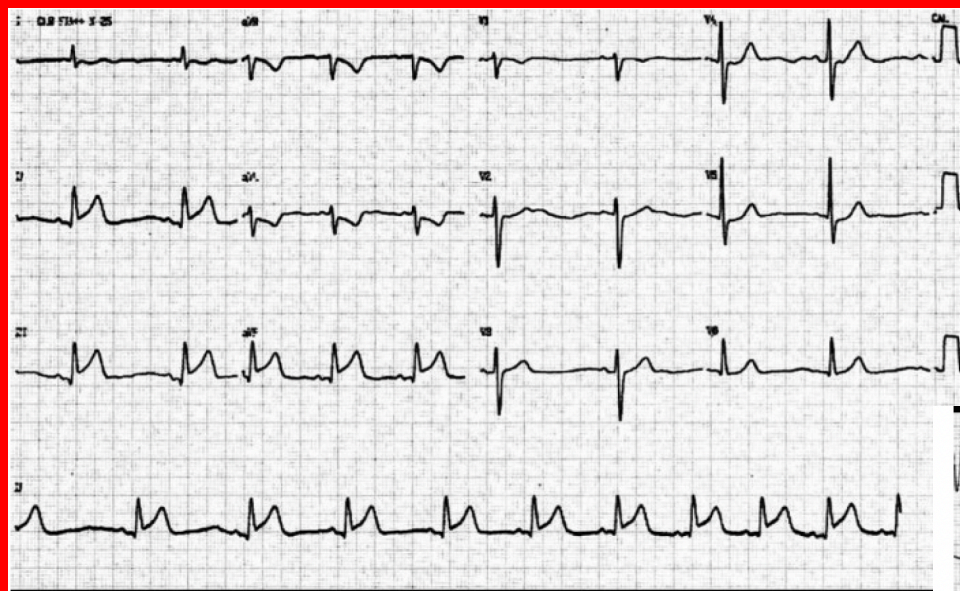
### Sustained accelerated idioventricular rhythm

Debal Basu, M.D.  
Melvin Scheinman, M.D.\*  
San Francisco, Calif.



Recent studies have elucidated the clinical picture of patients with accelerated idioventricular rhythm.<sup>1-5</sup> This arrhythmia almost always occurs within 48 hours of an acute myocardial infarction, is intermittent, and is generally benign.<sup>2-5</sup> Reported herein is an unusual case of sustained accelerated idioventricular rhythm, documented by His bundle electrocardiographic studies, that appeared six weeks after myocardial infarction and was successfully treated by a suppressant anti-arrhythmic drug.







# Electrophysiologic Studies during Accelerated Idioventricular Rhythms

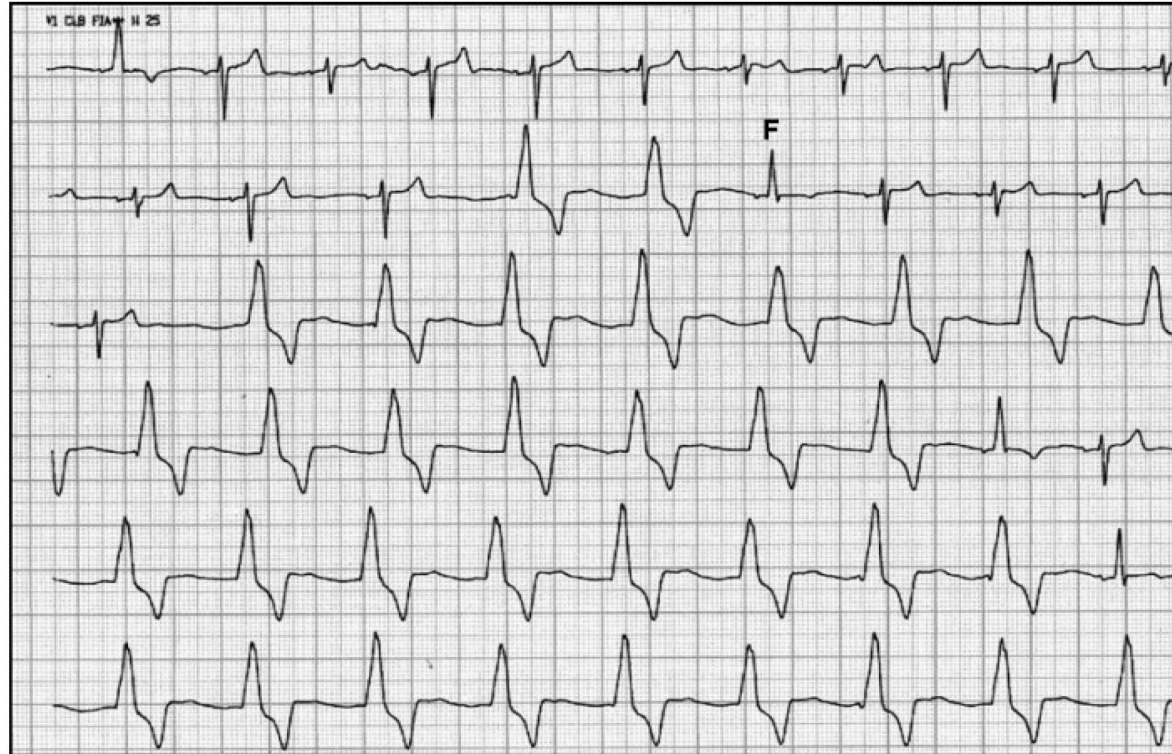
By JOHN J. GALLAGHER, M.D., ANTHONY N. DAMATO, M.D.,  
AND SUN H. LAU, M.D.

## SUMMARY

Accelerated idioventricular rhythms (AIVR) are ectopic ventricular rhythms with rates intermediate between idioventricular escape rhythms (30 to 40/min) and ventricular tachycardia (120 to 180/min). Differentiation of AIVR from supraventricular arrhythmias rests primarily on demonstration of their ventricular origin. His bundle electrograms (HBE) were recorded in four patients during AIVR. HBE verified the idioventricular nature of the ectopic rhythm and excluded supraventricular rhythm with aberration as a cause. In addition, they permitted the recognition of normally conducted sinus beats, fusion beats, and idioventricular beats. The pacemaker site for the AIVR was below the bundle of His. AIVR became manifest when the heart rate was slowed by increasing vagal tone, premature atrial stimulation, and high degree atrioventricular (A-V) block. AIVR could be suppressed and 1:1 A-V conduction established by increasing the atrial rate with atropine or by atrial pacing.

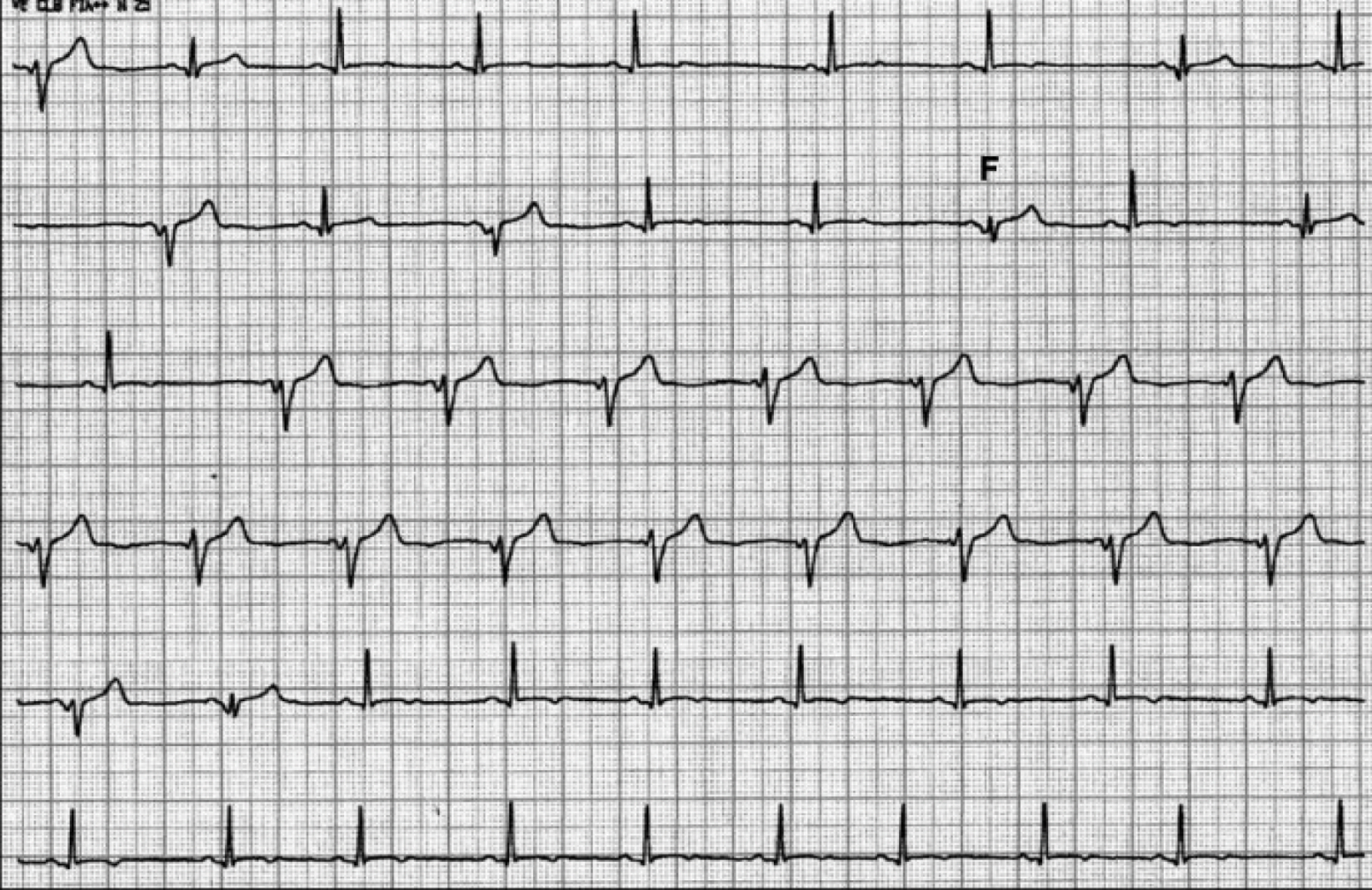
AIVR has been noted in association with acute myocardial infarction,<sup>3, 6-9, 13, 16, 19-22, 30, 31</sup> digitalis toxicity,<sup>2, 5</sup> and in a pregnant woman with no other evidence of cardiovascular disease.<sup>2</sup> In this study, three cases appeared related to digitalis toxicity and the remaining patient had no associated findings. The clinical course in all was benign.

The main electrophysiological mechanism involved in AIVR is an abnormal calcium-dependent automatism (ectopic automaticity) that affect phase 4 of action potential (diastolic depolarization) [19]. When AIVR is associated with digitalis intoxication, the main arrhythmogenic mechanism involved is trigger activity [20].





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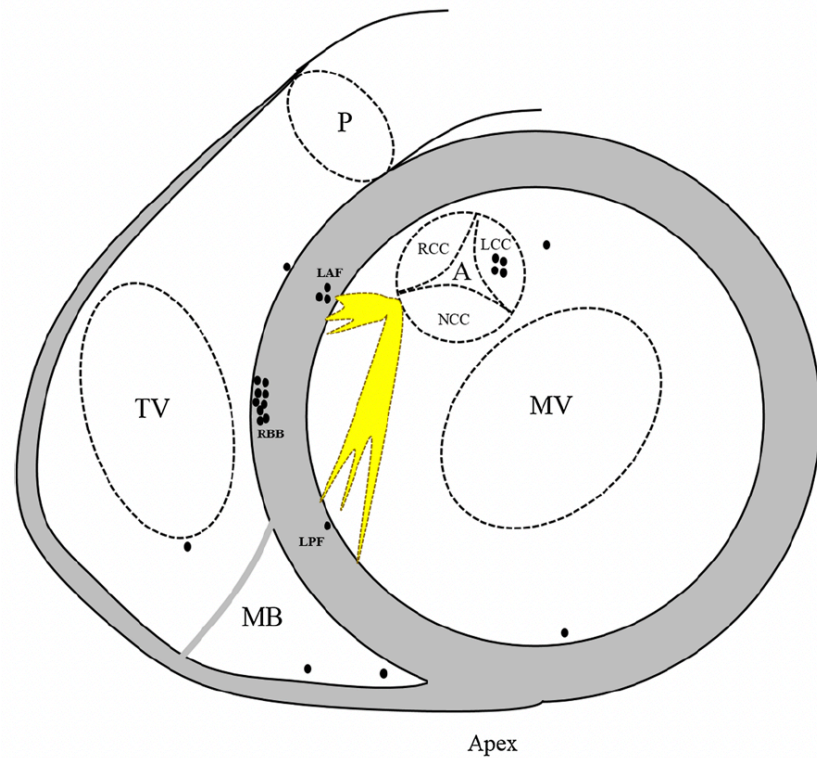
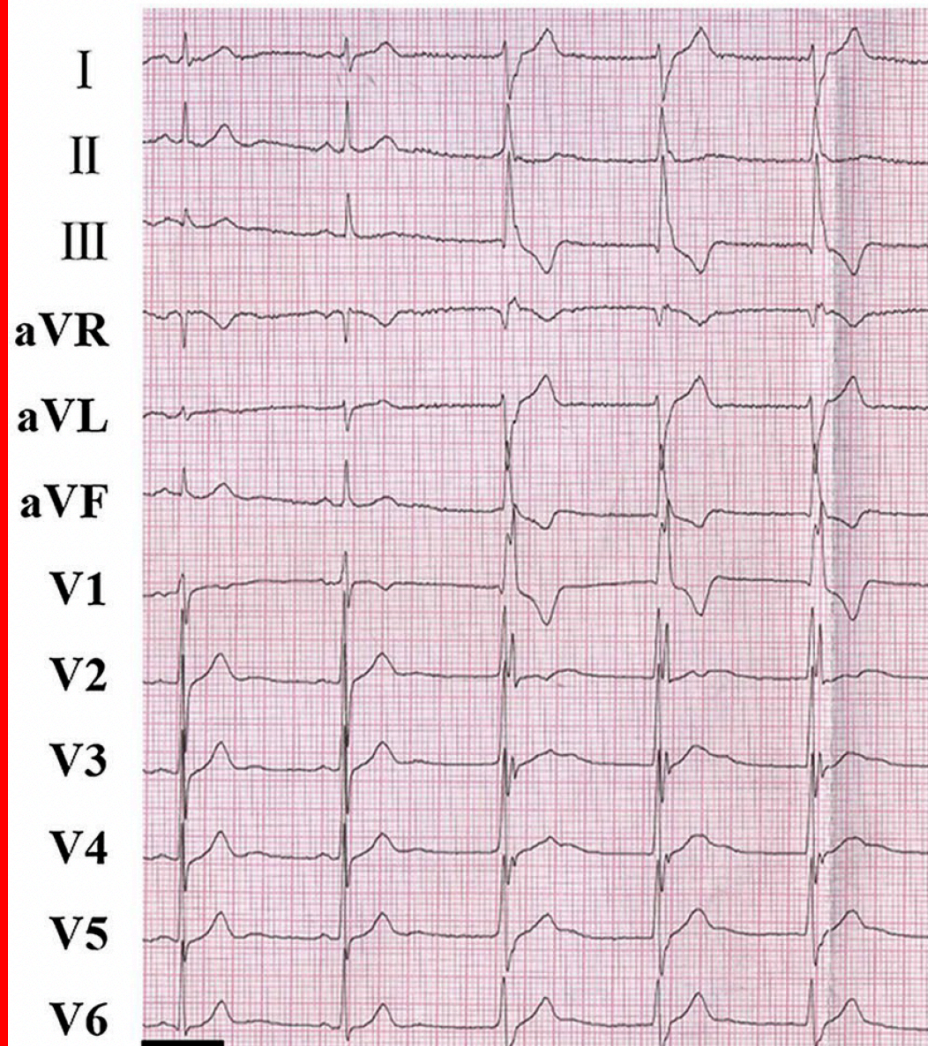




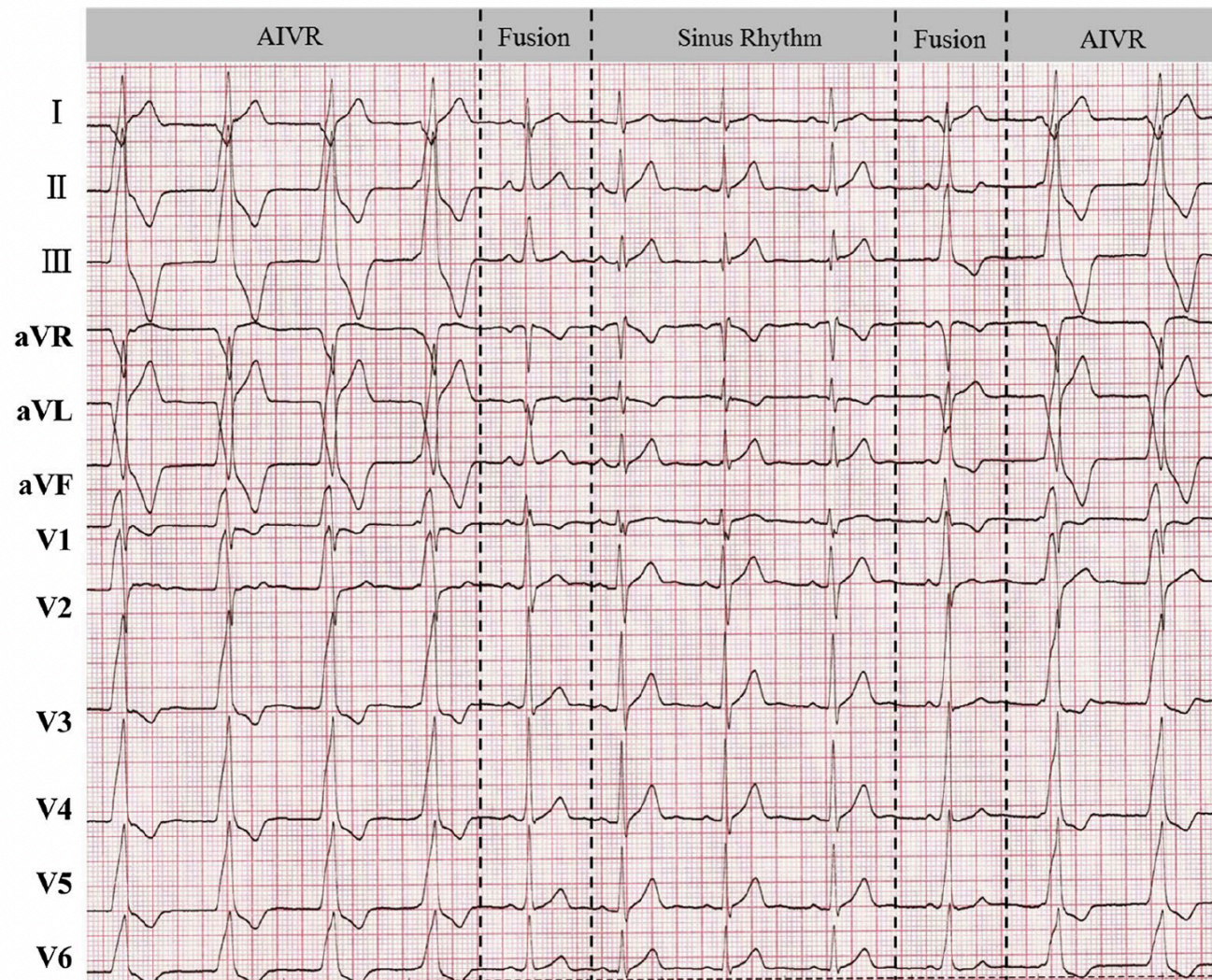
Rarely, AIVR can be detected in the youth and, in this subgroup, is usually benign [1] and requires no specific treatment.

AIVR occurs infrequently in patients without demonstrable heart disease and, in this case, it has a good prognosis. When AIVR presents in individuals with no structural heart disease, the mechanism involved is usually an increased vagal tone and decreased sympathetic tone [2], as observed in athletes. The acceleration of an independent focus during exercise strongly suggests that this ectopic focus is under autonomic influence, a phenomenon also seen during central nervous system (CNS) stimulation in dogs [3]. A similar mechanism has also been described for pregnant women in the antenatal period [4].

AIVR is frequently observed during the reperfusion phase that follows an acute myocardial infarction (AMI) [5]. It remains controversial whether or not, AIVR implies complete reperfusion of the culprit lesion or only some degree of reperfusion. AIVR has been also described associated with several drug intoxications such as halothane [6], aconitine [7], desflurane [8], cocaine [9], and digitalis [10-12]. Electrolyte imbalance has also been associated with AIVR in an experimental study [13]. There are also case reports published on the association of AIVR with the post-resuscitation period [14] (as an initial resolving rhythm), in chronic ischemic and non ischemic dilated cardiomyopathy [15], and less frequently in cardiomyopathies such as hypertrophic cardiomyopathy [16], arrhythmogenic right ventricular dysplasia (ARVD) [17], and in newborn infants with different congenital heart diseases [18].











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09.05.2016

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anni 15

### VISITA CARDIOLOGICA

Atleta asintomatico (Karate), in allenamento trisettimanale (2 ore per seduta). Controlli cardiologici precedenti hanno messo in evidenza fasi di ritmo idioventricolare accelerato con frequenza variabile fra 50 e 65.

L'esame obiettivo del cuore non mette in evidenza nulla di anormale.

All'elettrocardiogramma si osserva bradicardia sinusale a frequenza 56; tracciato normale.

Un test ergometrico massimale eseguito il 05/02/2016 non presenta alcuna aritmia. L'ECG dinamico secondo Holter mostra poche extrasistoli ventricolari isolate e qualche fase notturna di ritmo idioventricolare accelerato a frequenza 50. L'ecocardiogramma eseguito l'1/2/2016 è normale.

In conclusione, si tratta di un soggetto esente da cardiopatie. L'aritmia rilevata è da considerare di nessuna rilevanza clinica.

Prof. Giuseppe Oreto



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### VISITA CARDIOLOGICA

Dall'età di 9 anni sono state ripetutamente notati episodi, anche prolungati ma asintomatici, di ritmo idioventricolare accelerato, documentati con Holter.

È stata sospettata, in base a una prima RM cardiaca, una cardiomiopatia aritmogena ventricolare destra, successivamente esclusa da ulteriori RM.

I numerosi reperti ECG ed Holter permettono di mettere in evidenza un ritmo idioventricolare automatico a frequenza intorno a 70/m', originato da un focus posto nelle parete infero-posteriore del ventricolo sinistro.

Tale aritmia, oggi non presente, è assolutamente sprovvista di significato patologico, e non richiede trattamento né ulteriore approfondimento diagnostico. Oggi l'obiettività cardiaca è negativa, e l'ECG mostra un blocco incompleto di branca destra.

Non sono necessari ulteriori controlli approfonditi, ma è consigliabile un Holter ogni 2-3 anni.

Prof. Giuseppe Oreto

*Non tutto ciò che è strano è pericoloso!*

