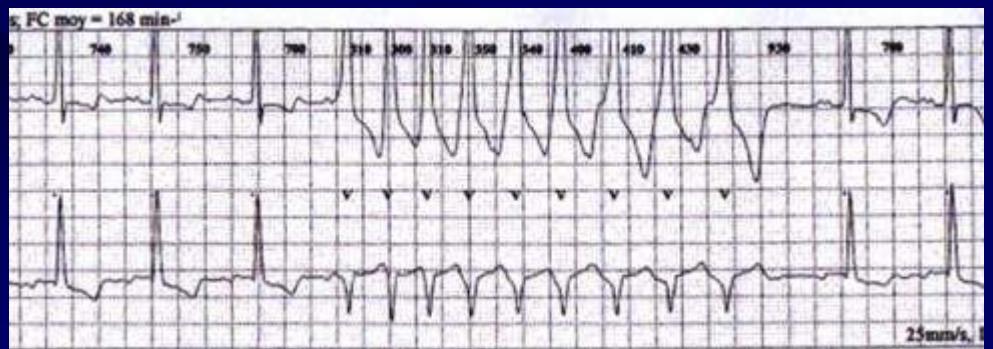
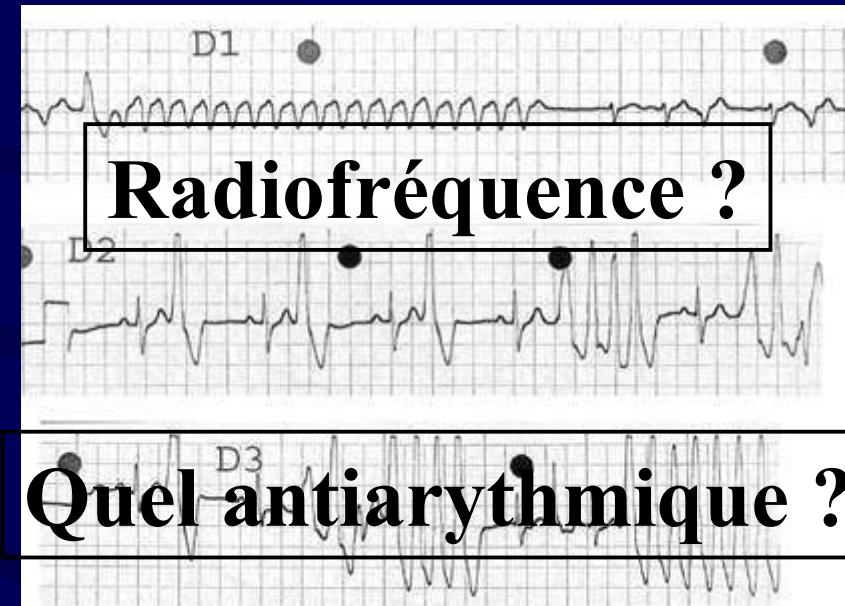
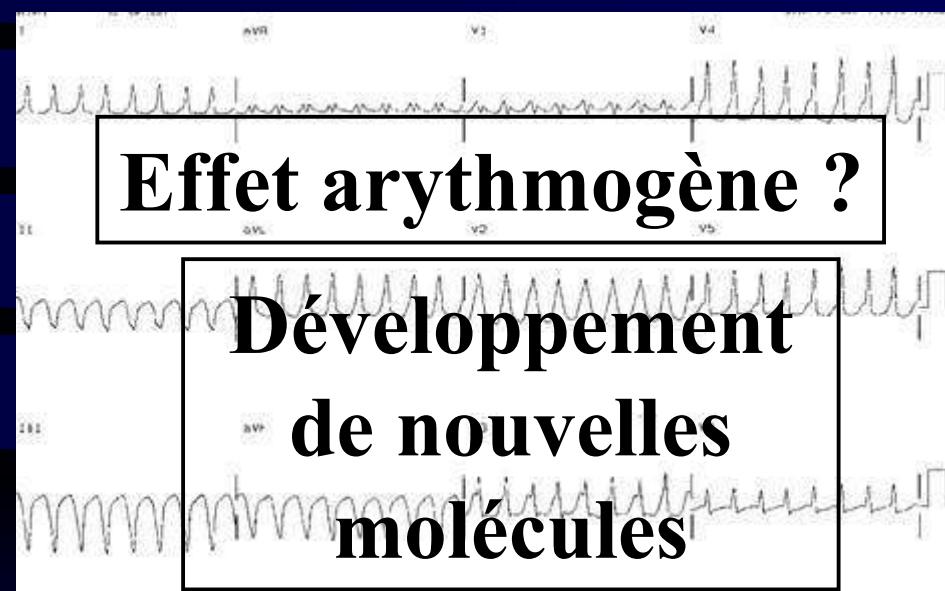
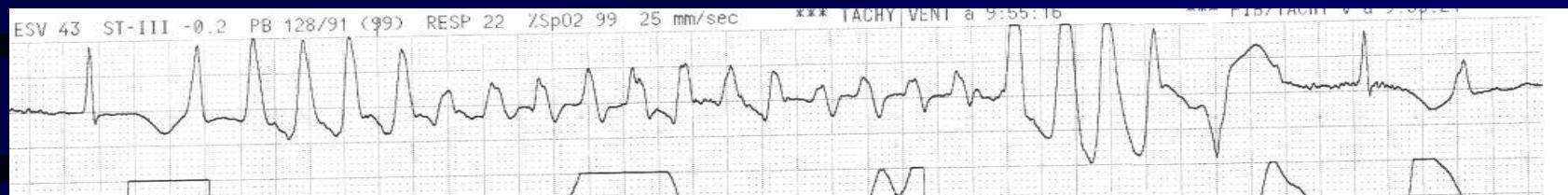


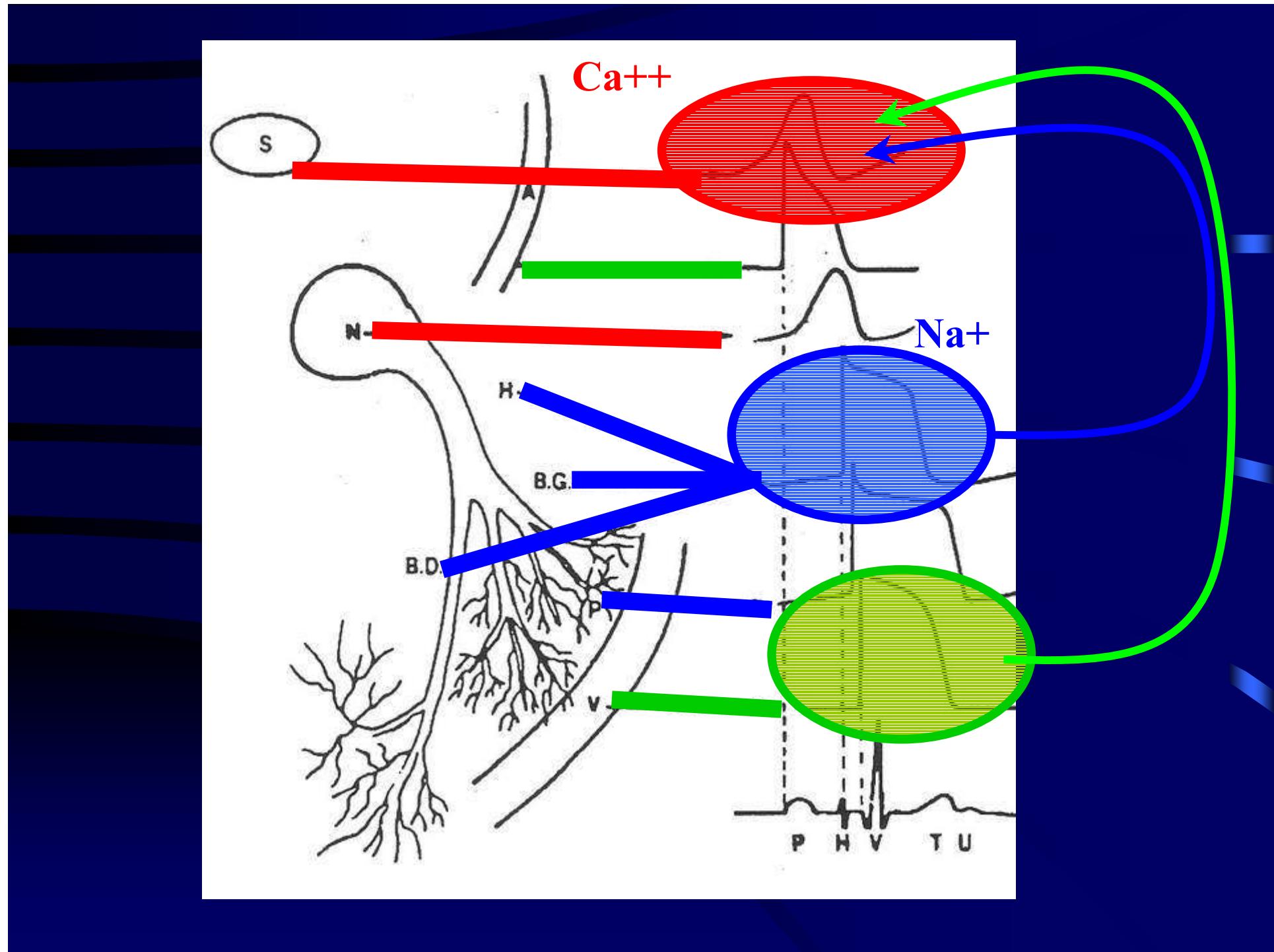
Physiopathologie des Arythmies Ventriculaires

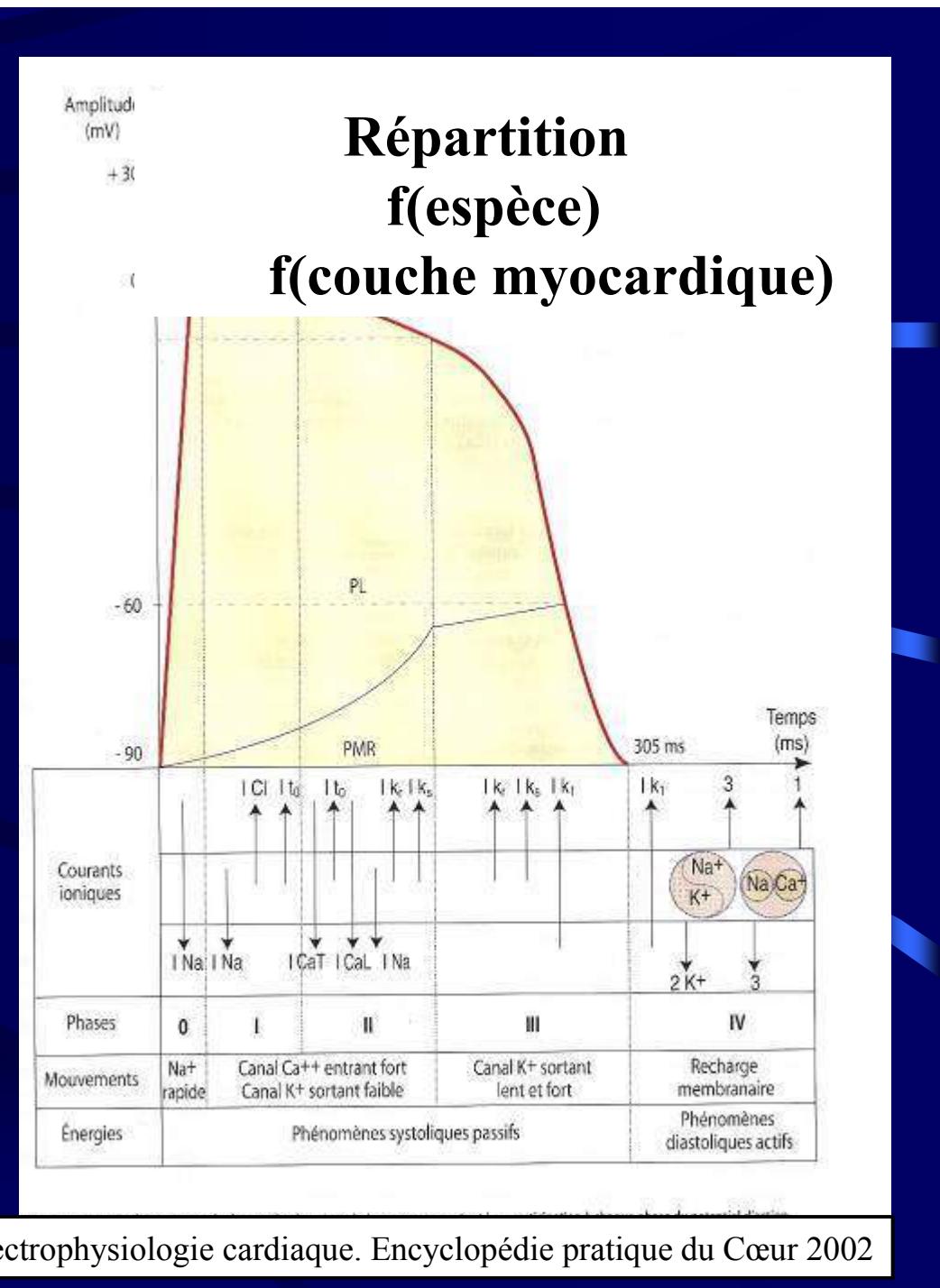
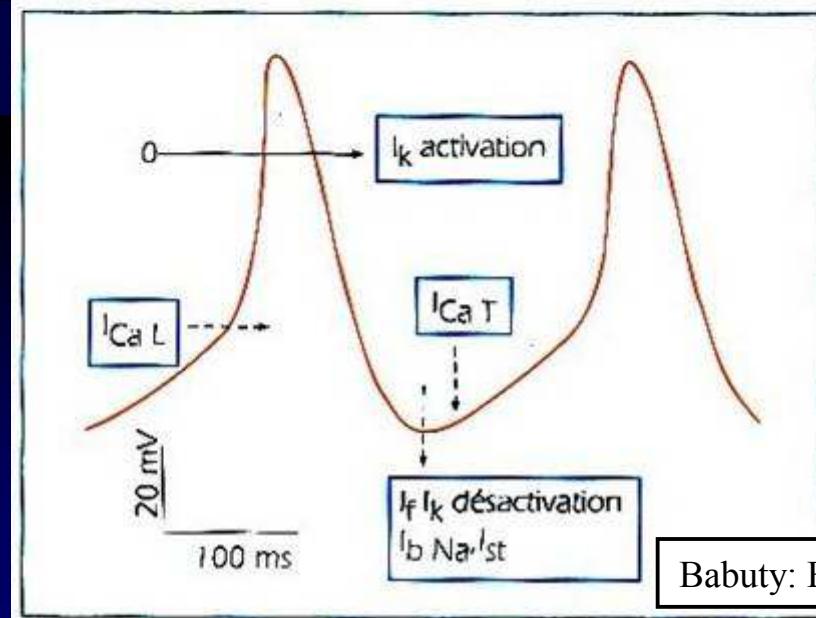
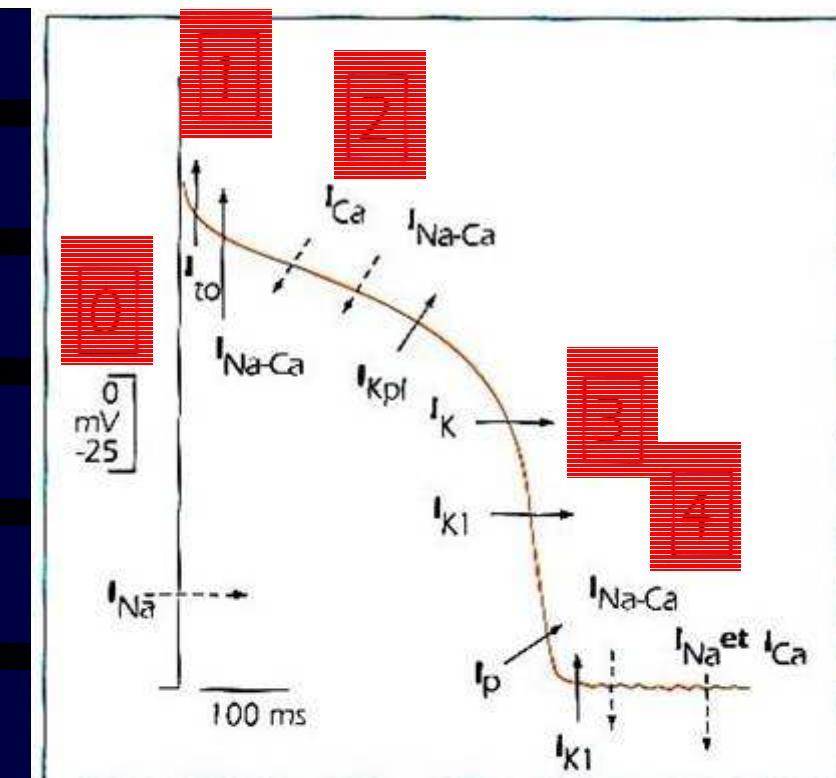
Philip Aouate
Hôpital Laënnec
Creil



Mécanisme ?







Babuty: Electrophysiologie cardiaque. Encyclopédie pratique du Cœur 2002

Mécanismes

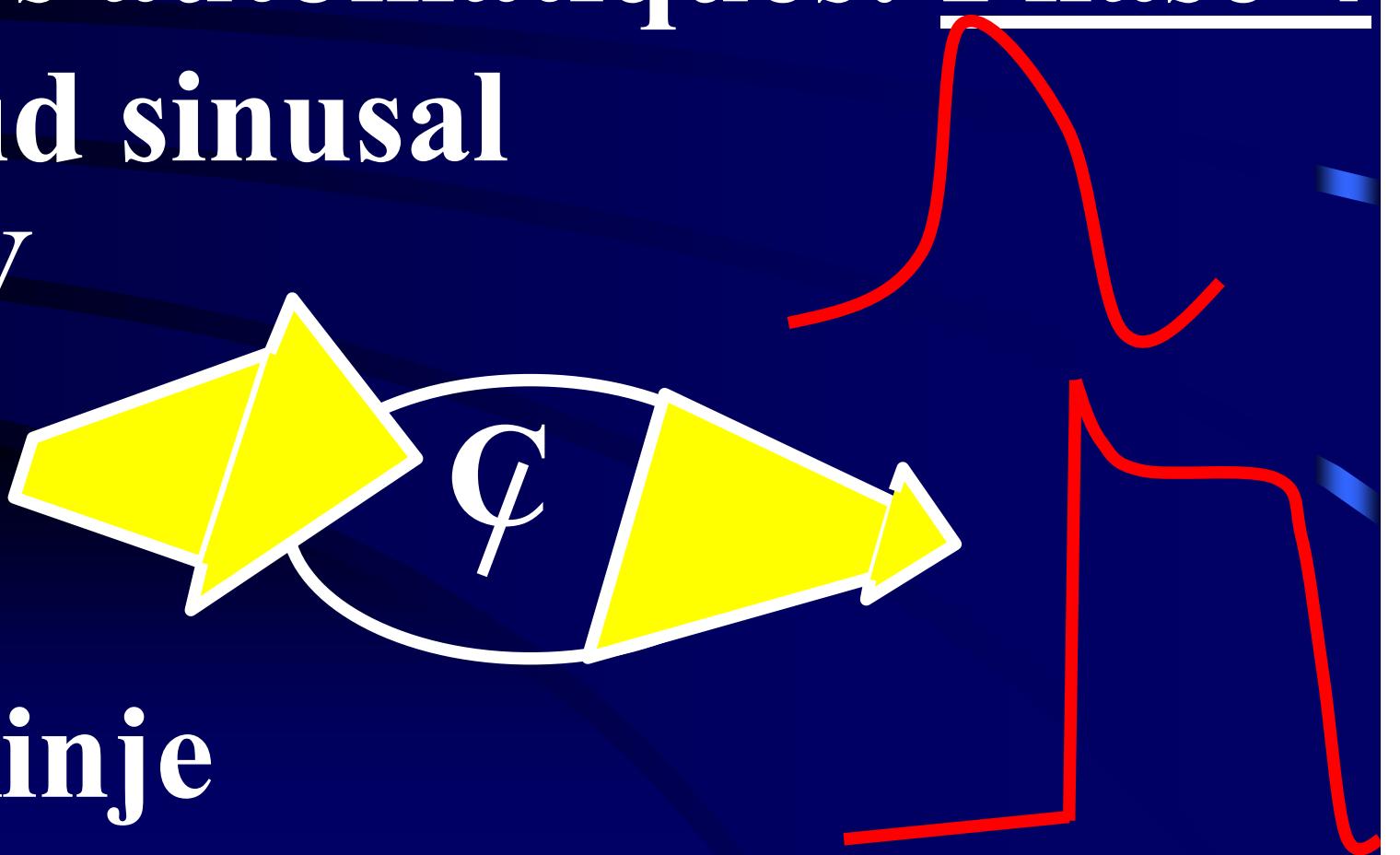
- Hyperautomatismes
- Automatismes anormaux
- Activités déclenchées
- Réentrées

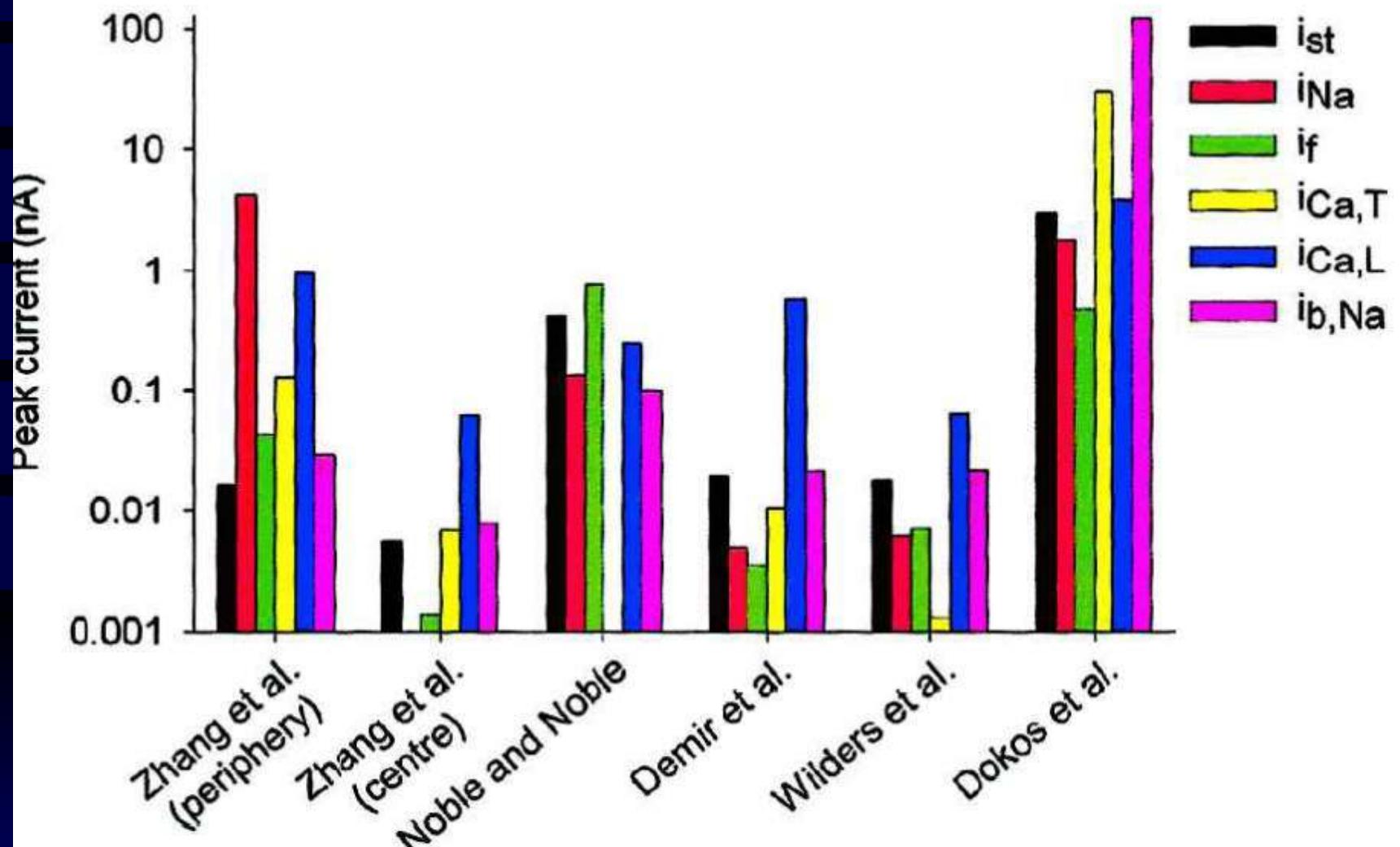
Hyperautomatisme

Cellules automatiques: Phase 4

- Nœud sinusal
- NAV

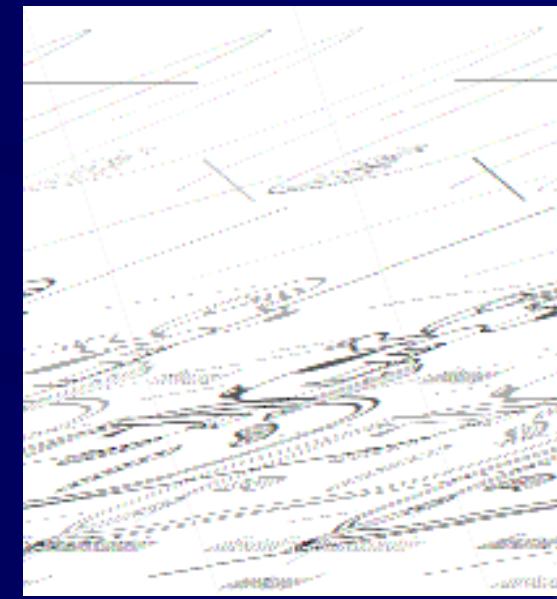
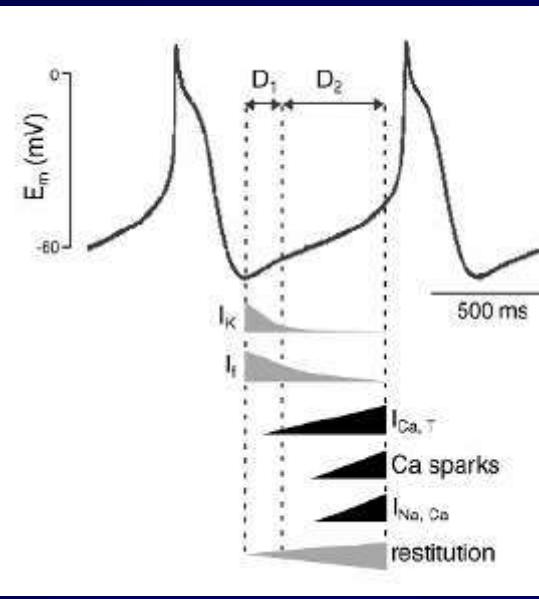
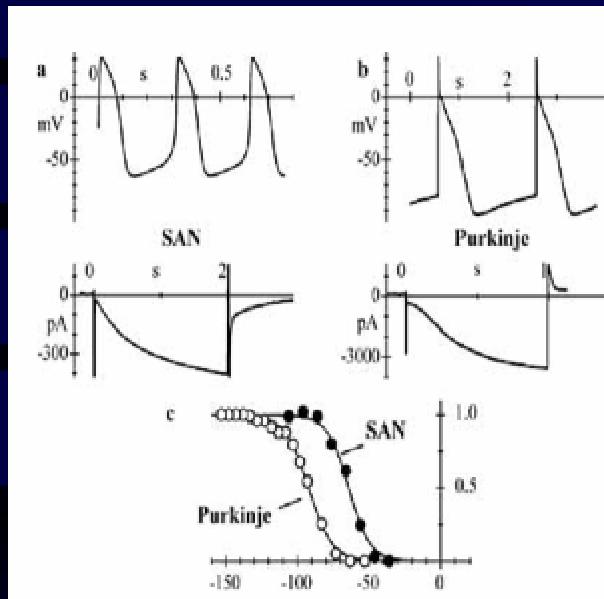
-Purkinje



B**Zhang PACE 2002**

Hyperautomatisme

- Phase 4 = If Na et K : $\sum - p \sum$ via AMPc
- Diminution d'un courant K⁺ sortant (cellules calciques)
- Courants calciques (L – T)/Na



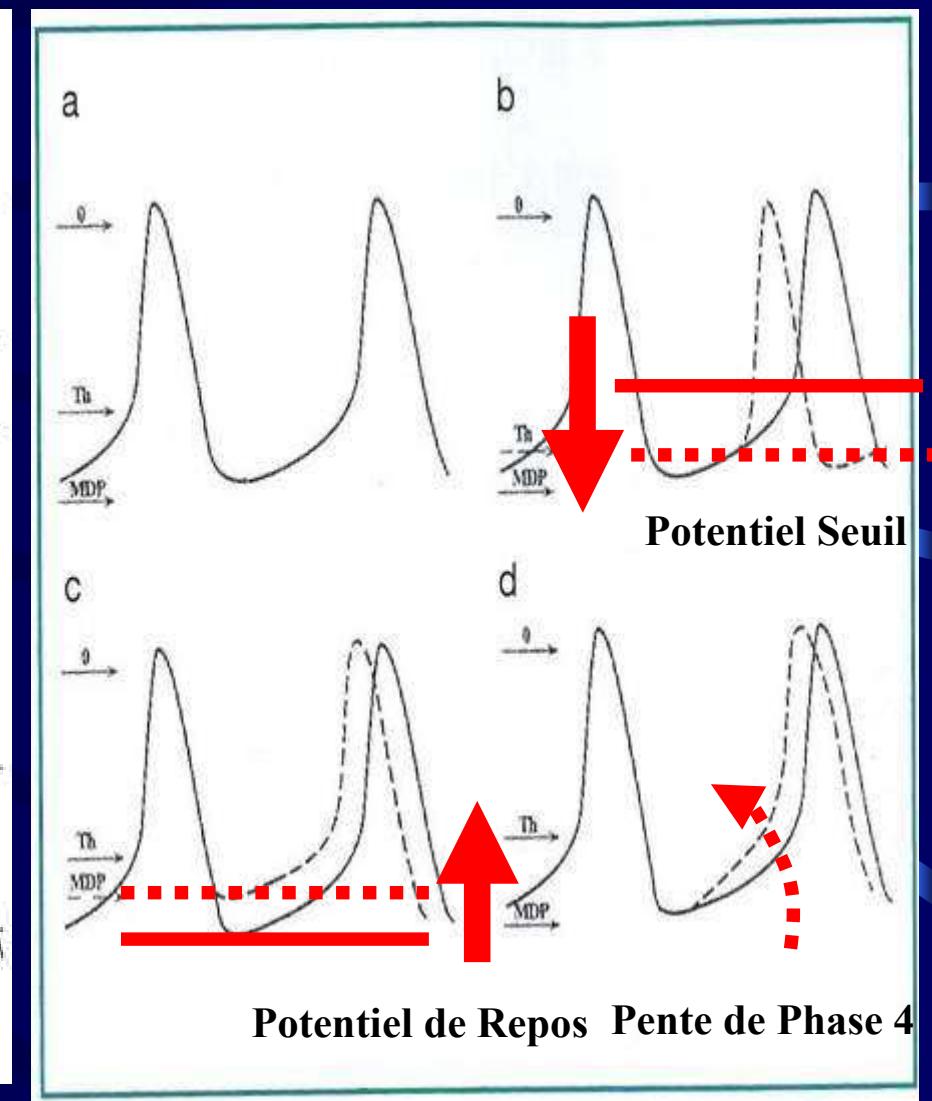
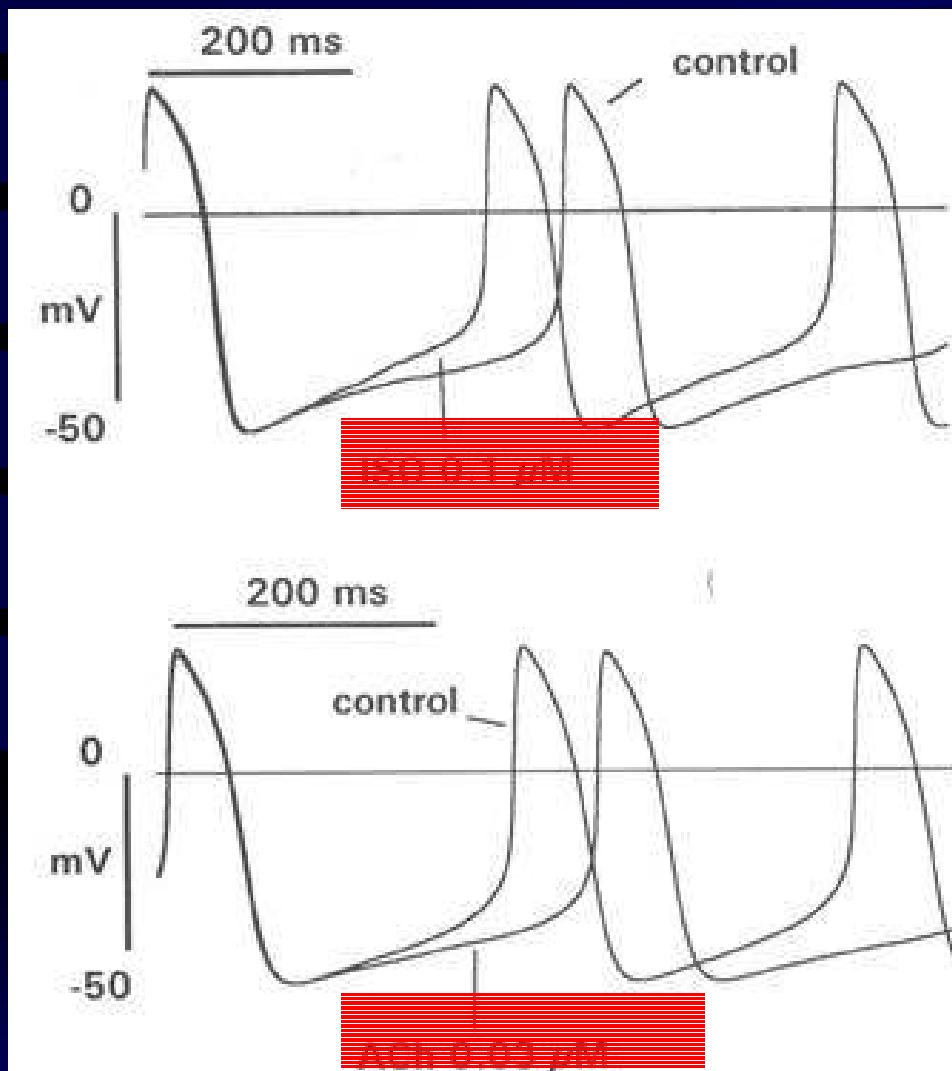
The Pacemaker Current: From Basics to the Clinics

ANDREA BARBUTI, PH.D., MIRKO BARUSCOTTI, PH.D., and DARIO DIFRANCESCO, PH.D.

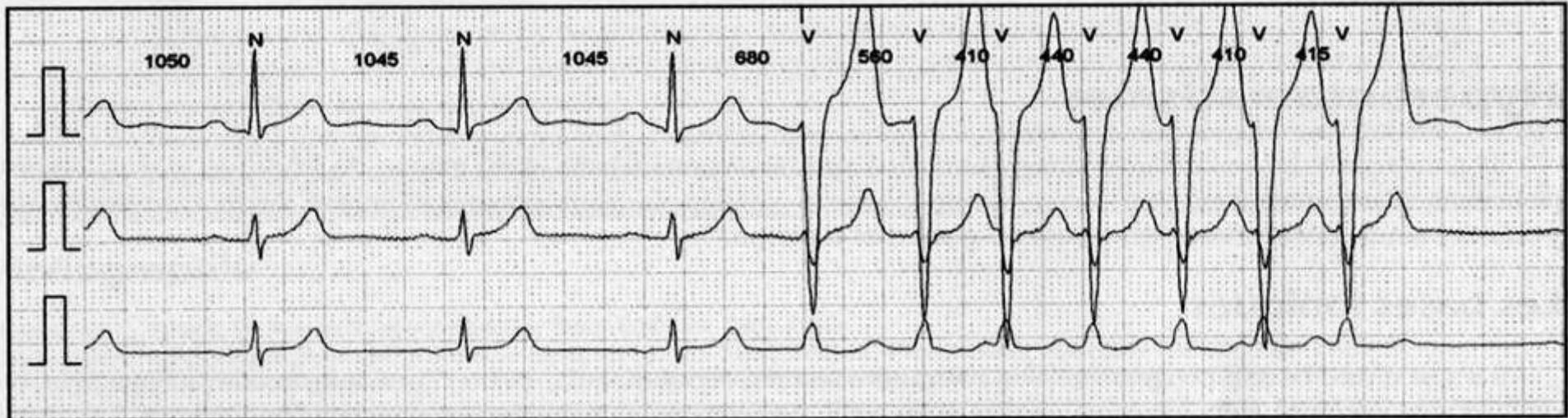
applications of the concept of I_f -based pacemaking. (*J Cardiovasc Electrophysiol*, Vol. 18, pp. 342-347, March 2007)

Overdrive suppression et Echauffement

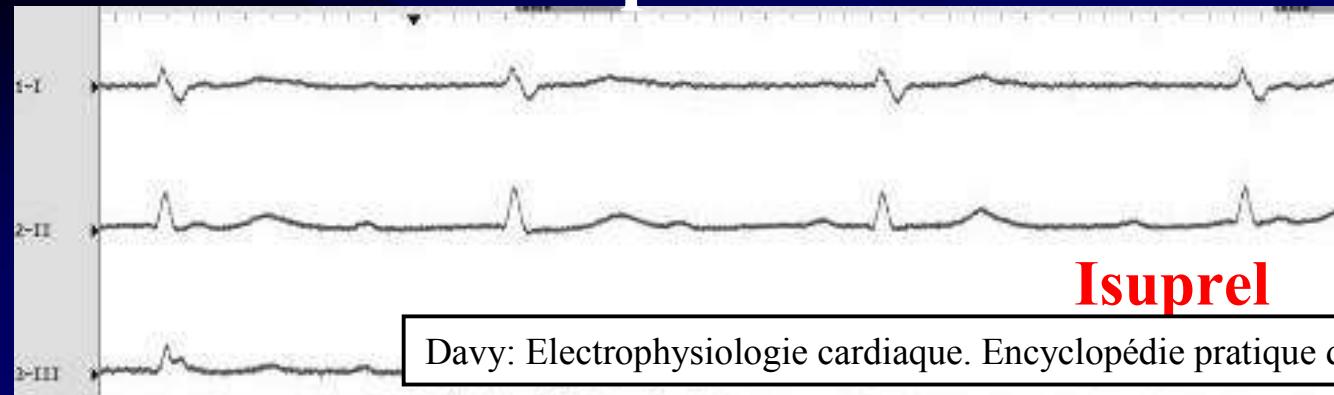
Region	Resting Potential	Depolarization	Repolarization	Pacemaker Current	Responsiveness Recovery	Parasympathetic Effect	Sympathetic Effect
Ventricular Cells	-90 mV - maintained by K permeability and intracellular K concentration; close to K equilibrium potential	- caused by rapid Na influx via channel activation - characterized by H and M gates	- large plateau phase - caused mainly by delayed rectifier channels (I_K)	Possible HVG CMD Ischémique	- voltage dependent - recovered as soon as polarity restored	- inhibit effects of sympathetics by preventing norepinephrine release	- alter course of repolarization by increasing flow of L-type depolarizing force, increasing plateau and increase contractile force of muscle
Atrial Cells	- similar to ventricle	- shorter, weaker calcium current than ventricle	- faster than ventricle - no plateau	- in some special atrial cells in crista terminalis	- voltage dependent - recovered as soon as polarity restored	- inc. outward K+ flow shortens refractory period, enables more frequent excitation	
SA Node	-no true resting potential - max at -60 mV - not completely K permeable, and permeable to other ions	- spontaneous depolarization caused by I_f - goes until reaches threshold - then Ca influx via L type Ca channel - weaker than Na current, slower than ventricle	- delayed rectifier K channel similar to ventricle	- normal pacemaker - carried by Na^+ and Ca^{++} , not by L type Ca current - I_f	- recovers after polarity restored - time-dependent - slower due to rate of Ca channel recovery since depolarization not caused by Na	- outward K+ current blocks inward I_f lowering cell depolarization rate - more K+ conductance makes for more neg. resting potential - overall, slower heart rate	- more positive resting potential and increased inward pacemaker current cause higher rate of spontaneous depolarization reaching threshold for faster heart rate
AV Node	- -60mV due to fewer gK1 channels and permeability to other ions (Na)	- mainly inward Ca flow through L type Ca channel - similar to SA node	- inactivation of L type Ca influx	- in some cells - weaker than SA node, small drift towards positive - no normal spontaneous impulses	- same as SA node - time dependent - slow recovery causes blocking and filtering of premature and too-rapid impulses	- block L-type Ca flow, slowing depolarization, lowering action potential amplitude, slowing conduction - delay L-type removal of inactivation, cause longer refraction, lower # of possible impulses	- increase L type Ca influx cause faster depolarization and lower refraction for overall faster conduction
Purkinje Fibers		- larger inward Na current for very fast depolarization				- inhibit effects of sympathetics by preventing norepinephrine release	-increase inward pacemaker, may result in impulse initiation in abn circumstances



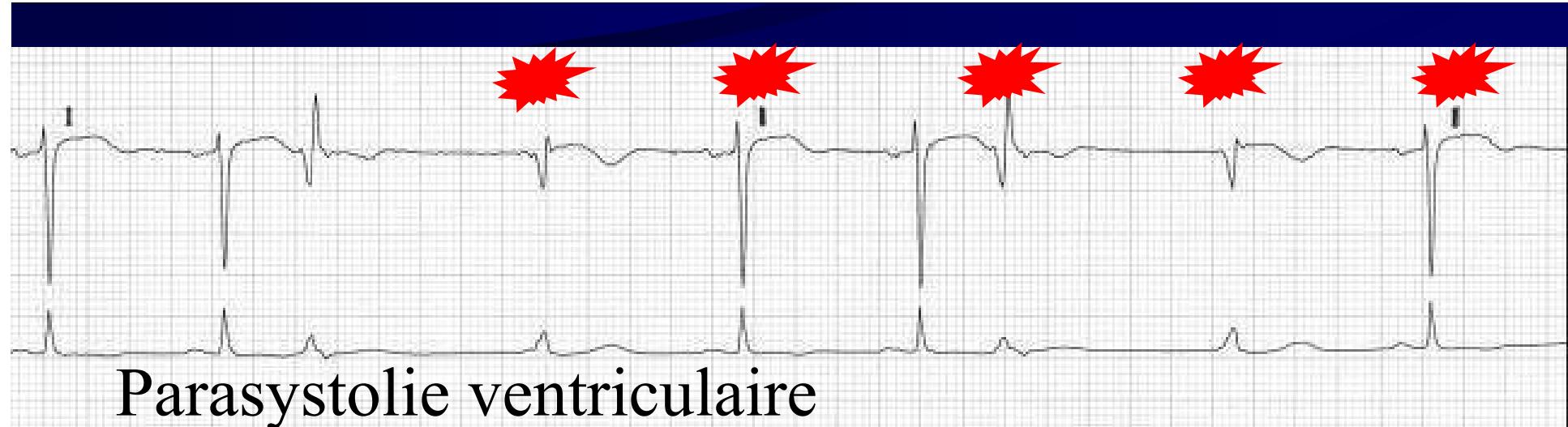
Hyperautomatisme Situations cliniques



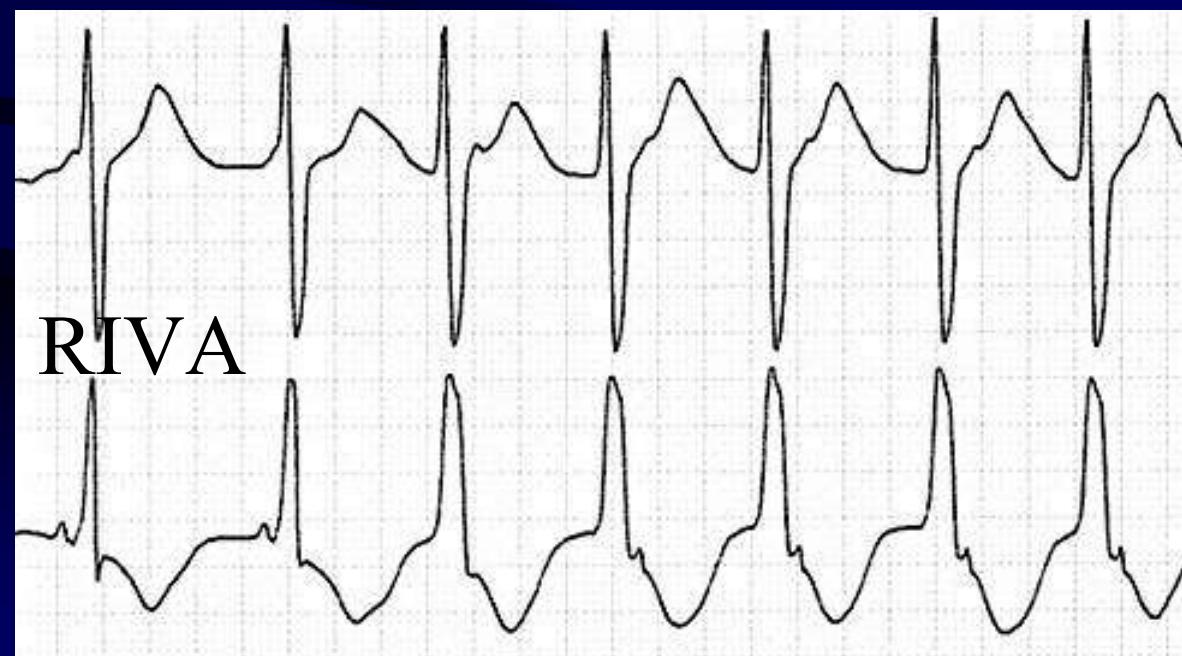
Overdrive suppression – Échauffement
Warm-up & Cool-down



Davy: Electrophysiologie cardiaque. Encyclopédie pratique du Cœur 2002



Parasystolie ventriculaire

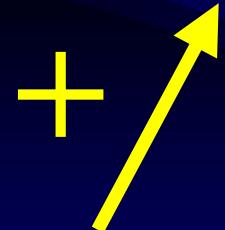


Automatismes Anormaux

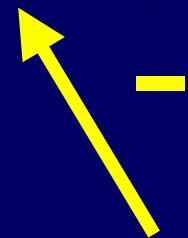
Toutes les Cellules myocardiques

Cœur pathologique source de
dépolarisation diastolique (phase 4)

Diminution du courant sortant
potassique et augmentation des
courant calcique entrant



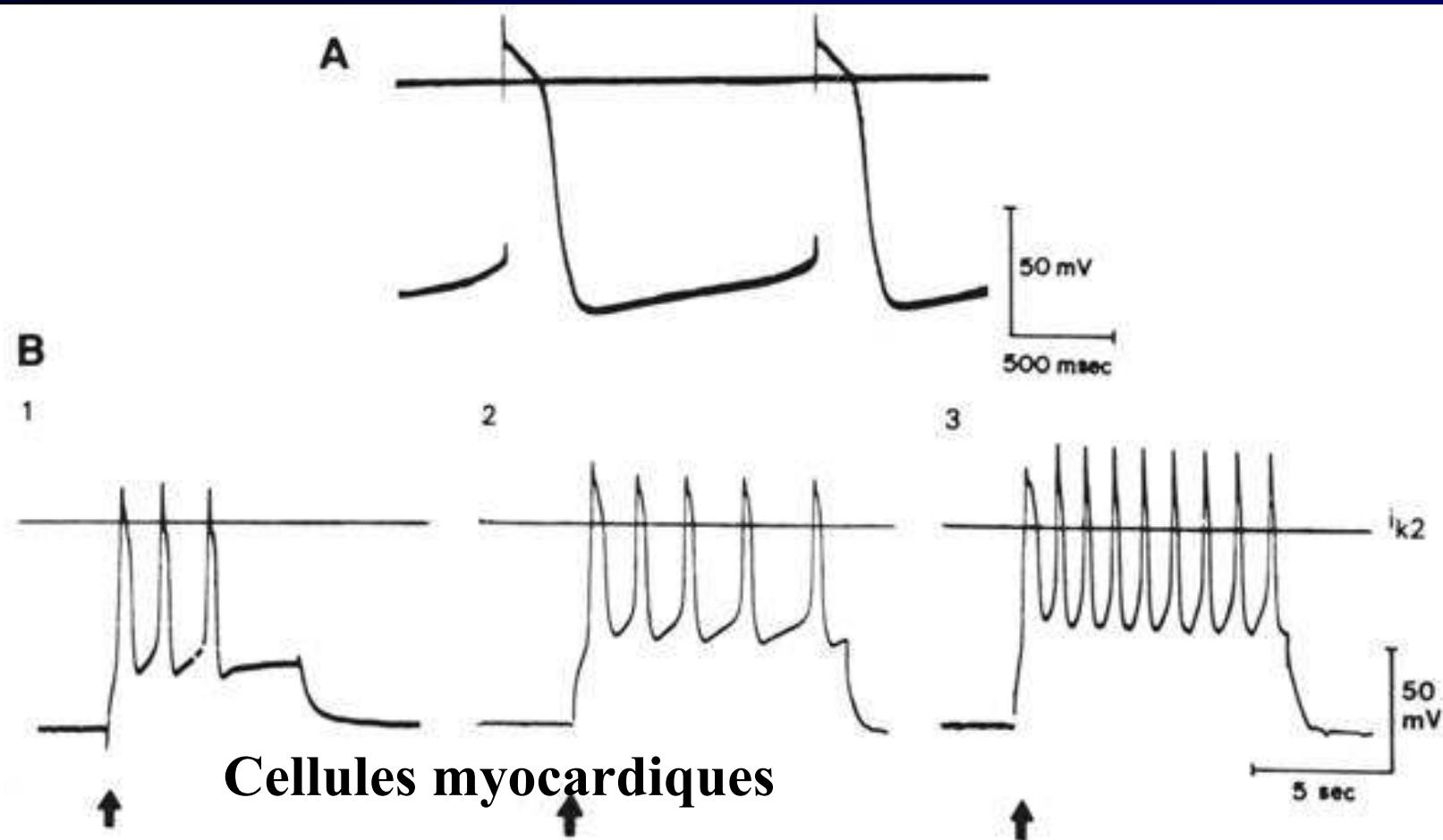
$\downarrow K+e - \downarrow PO_2 - \downarrow pH$



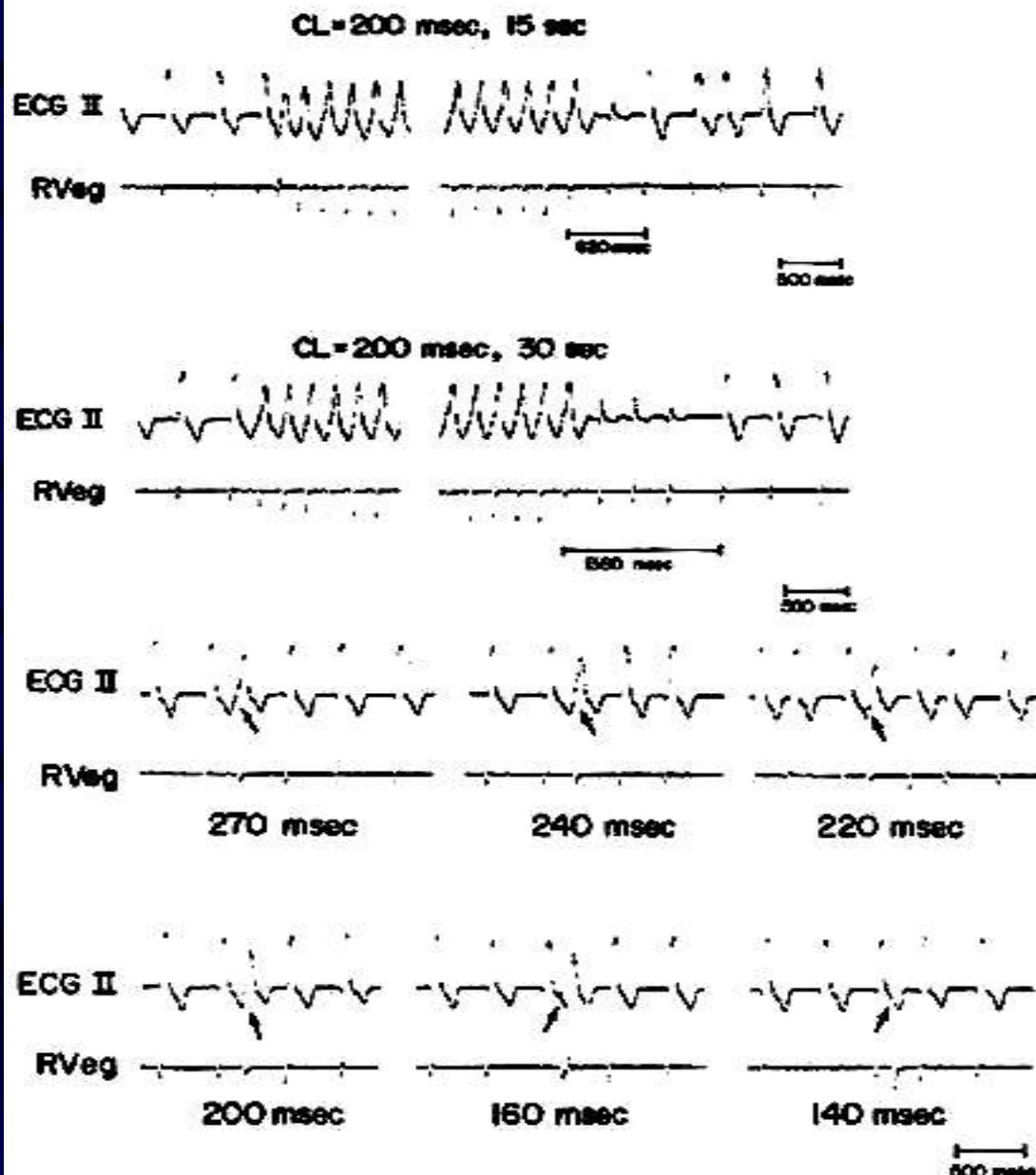
Verapamil - $\uparrow K+e$

Mg

Cellules purkinje



Arch int med 1975; 135: 459



Overdrive - SVP : aucun effet Mécanisme \neq Réentrée

Automatismes Anormaux

Situations cliniques

- Tachycardies ventriculaires de l'IDM
- RIVA

TABLE 18-1. The Nature, Time Course, Mechanisms, Site of Origin, and Response to Drugs of Ventricular Arrhythmias After Coronary Artery Occlusion

	Phase I	Phase II	Phase III
Nature	VT/VF	VT	VT/VF
Time course	15–30 min	6–72 h	3–12 d
Site of origin	Ischemic myocardial cells Purkinje fibers? Normal zone bordering the ischemic zone	Subendocardial Purkinje fibers in infarct zone Subepicardial muscle overlying the infarct	Subepicardial muscle cells overlying the infarct Surviving intramural muscle cells Purkinje fibers?
Mechanism(s)	Automaticity (early after depolarization)?	Adrenergic excitability Reentry?	Reentry Triggered automaticity?
Response to drugs	Usually resistant	Usually suppressed	Usually resistant

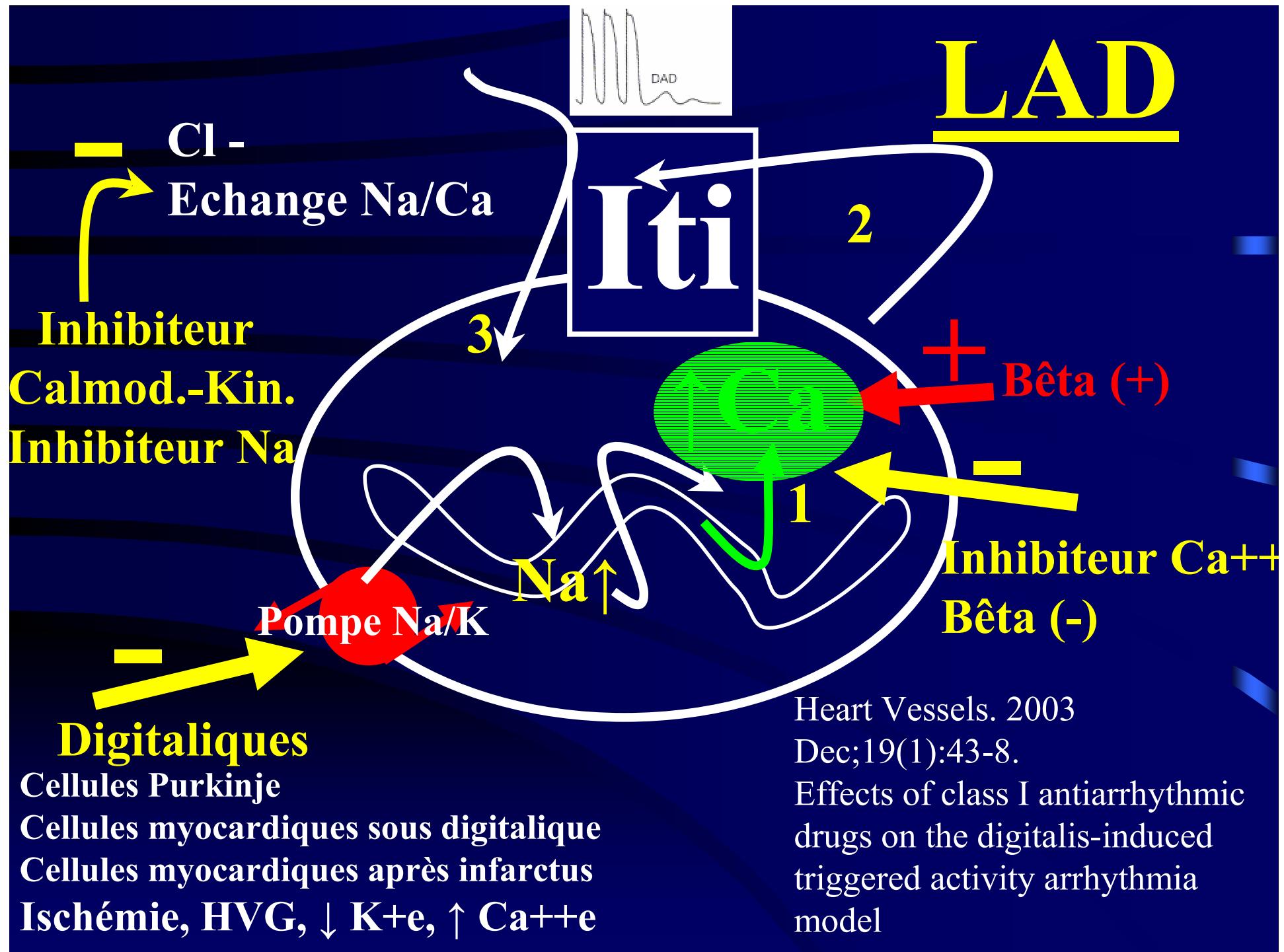
VT, ventricular tachycardia; VF, ventricular fibrillation, ?, evidence uncertain. Time course refers to postocclusion period.

Activités Déclenchées

Post-dépolarisations

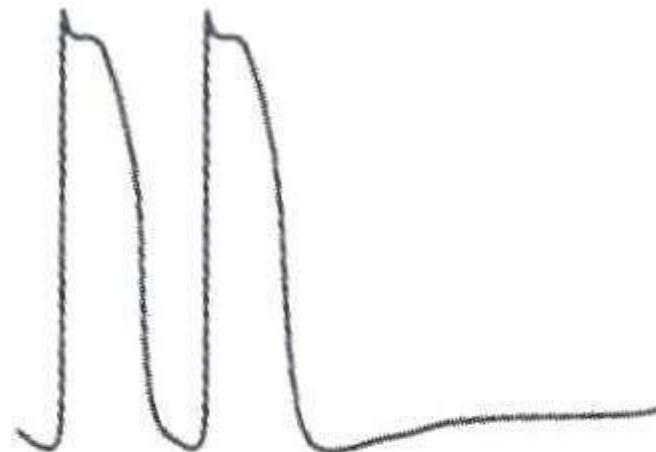
Précoces EAD

Tardives LAD

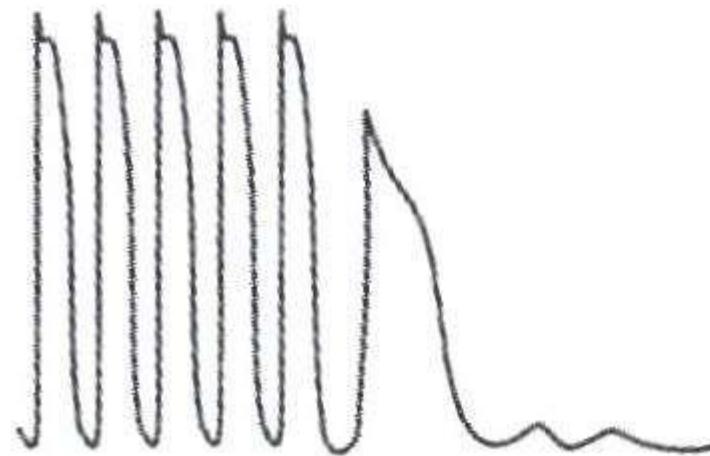


Tachycardie-Dépendance

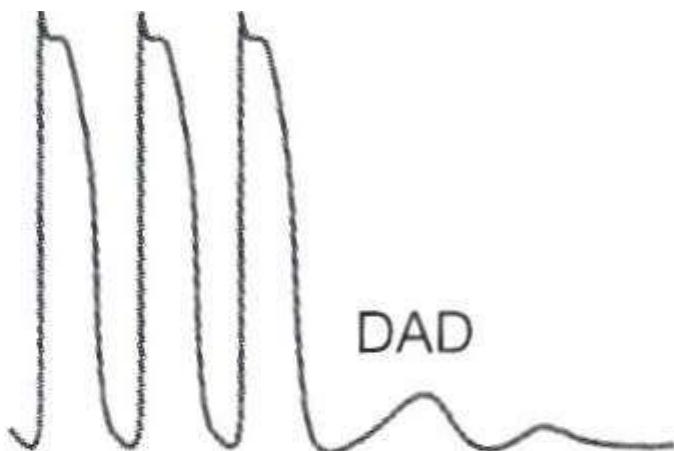
a



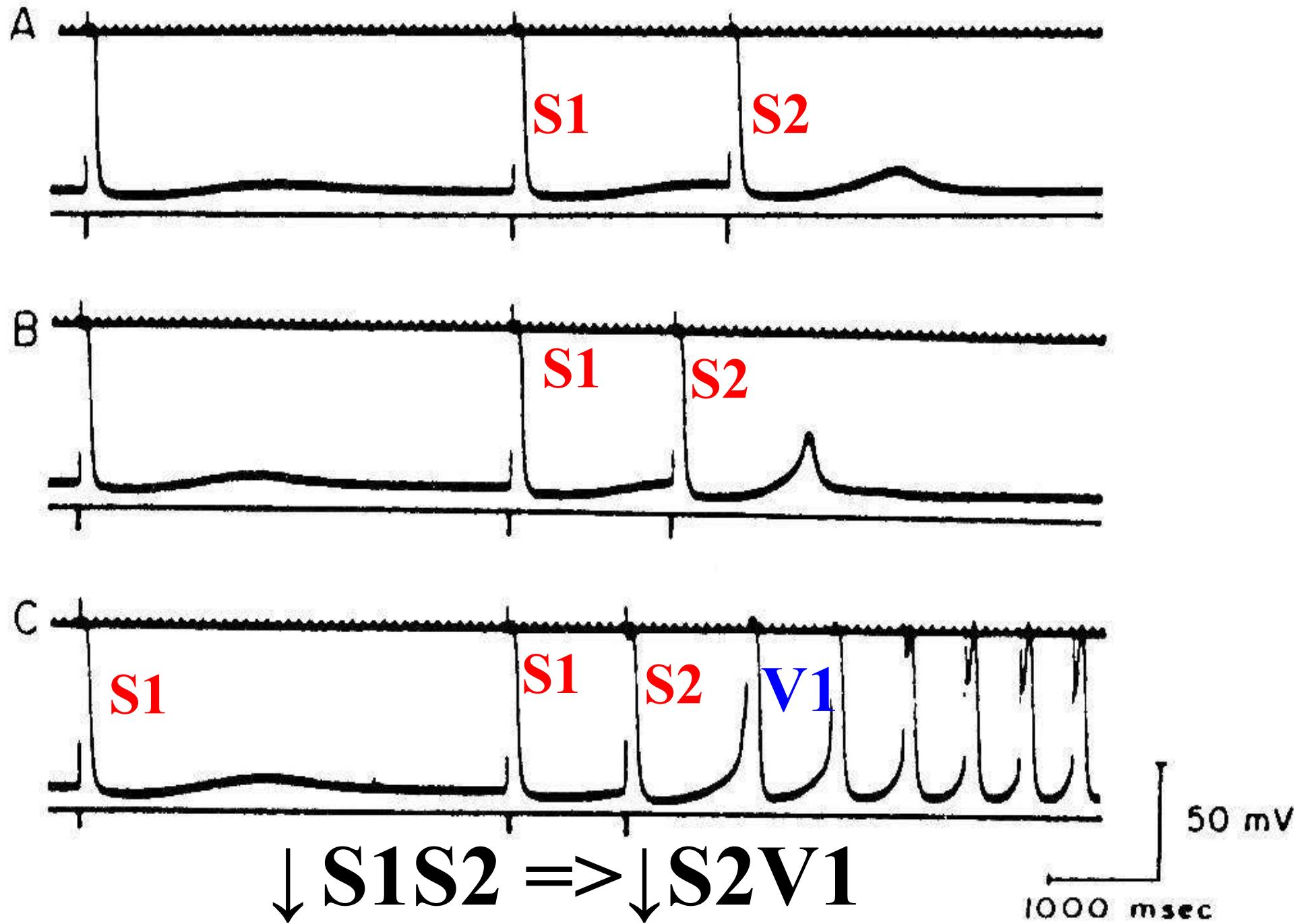
c



d

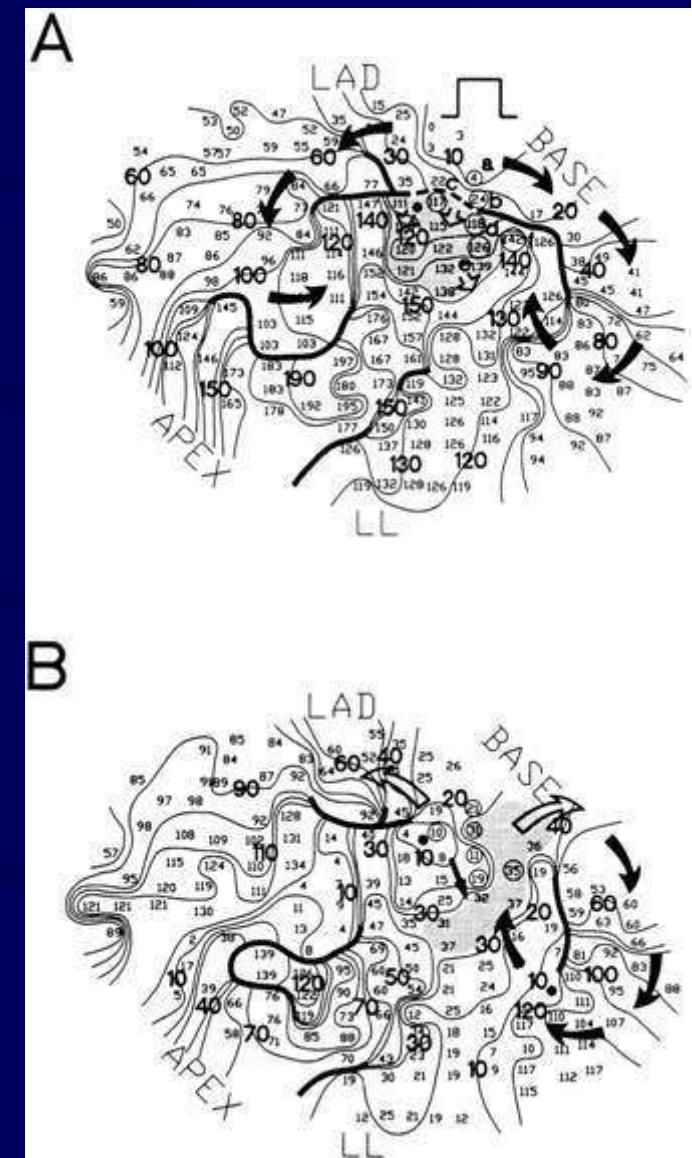
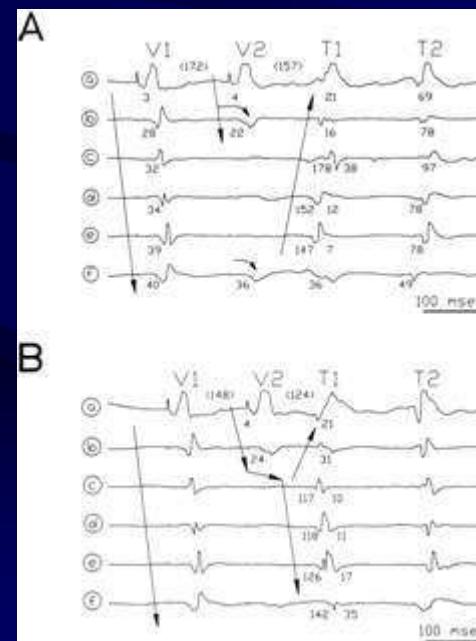
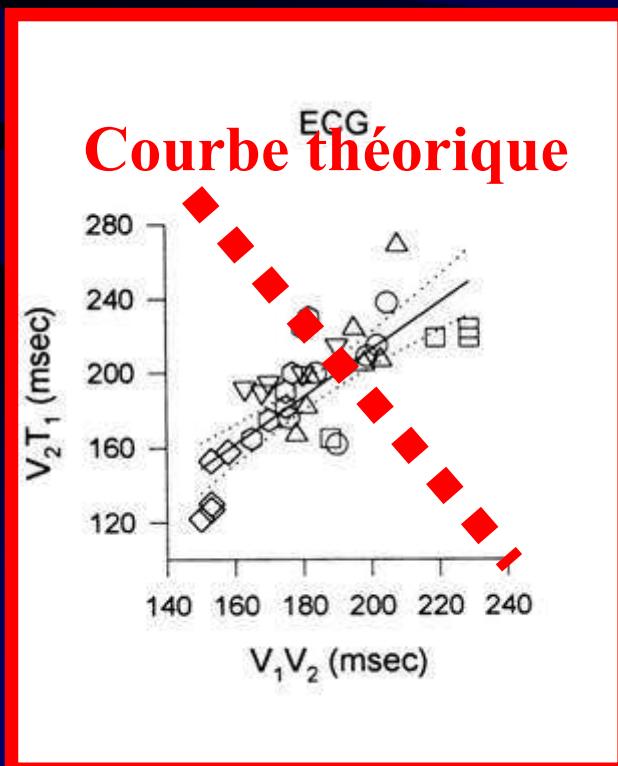


↑ stimulation ↑



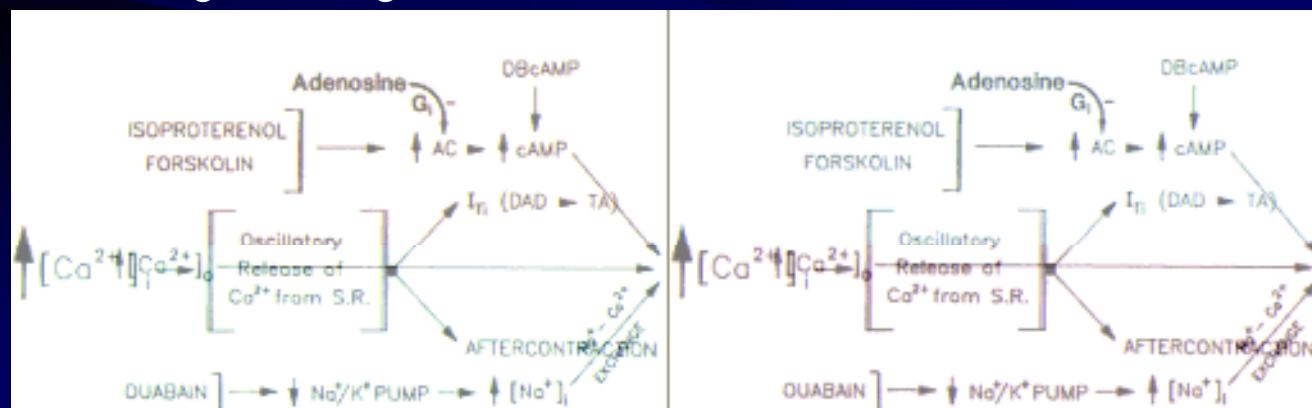
•Mechanisms for Absence of Inverse Relationship Between Coupling Intervals of Premature Impulses Initiating Reentrant Ventricular Tachycardia and Intervals Between Premature and First Tachycardia Impulses

C Cabo, Circulation. 1997;96:3136-47.

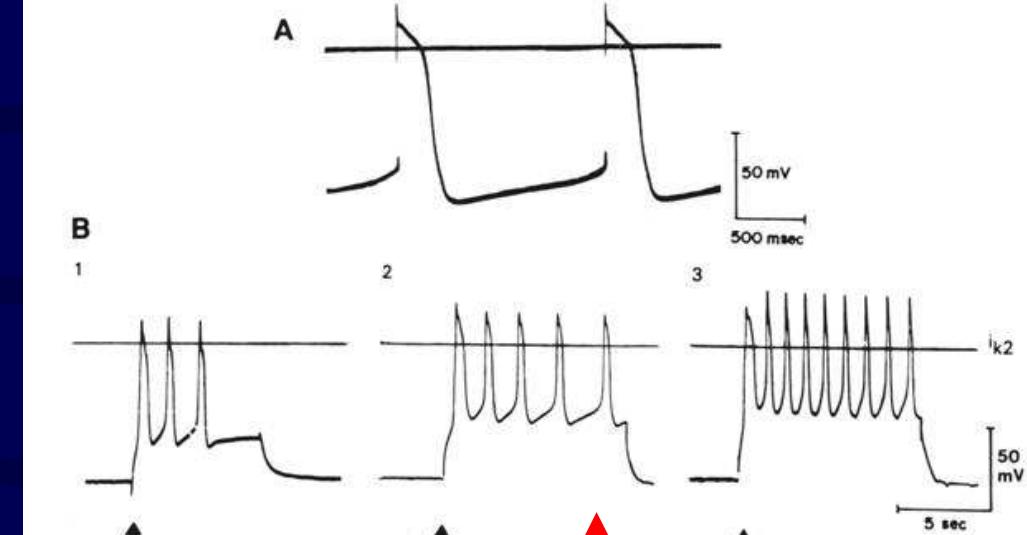
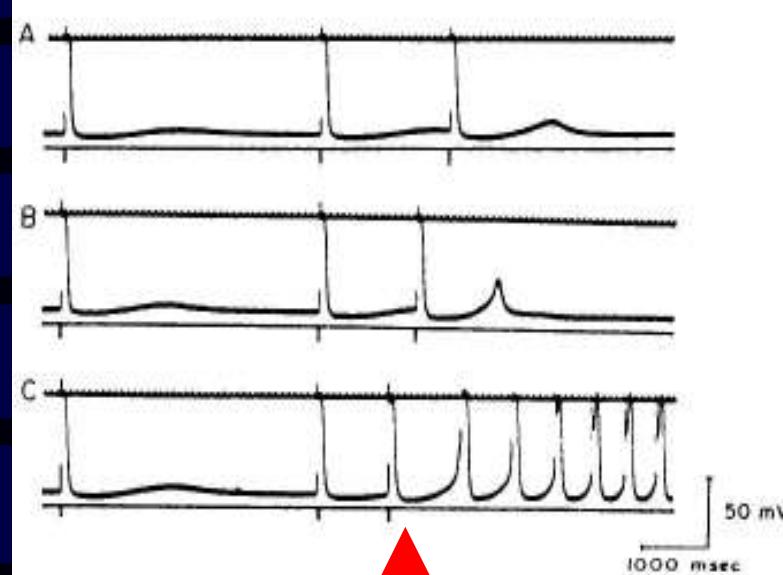


LAD

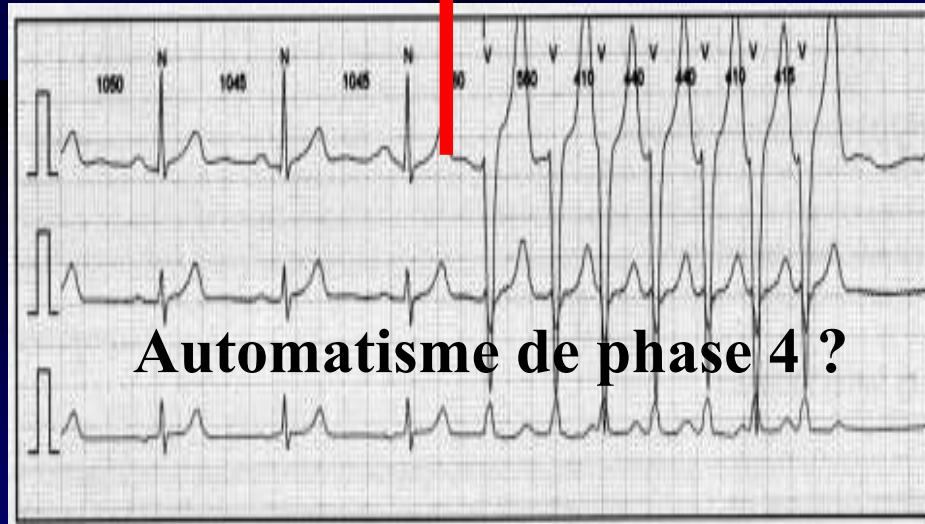
- SVP peut supprimer les LAD (Réentrée) !!
- Adénosine peut supprimer les LAD secondaires à une inhibition de l'adenyl-cyclase !!



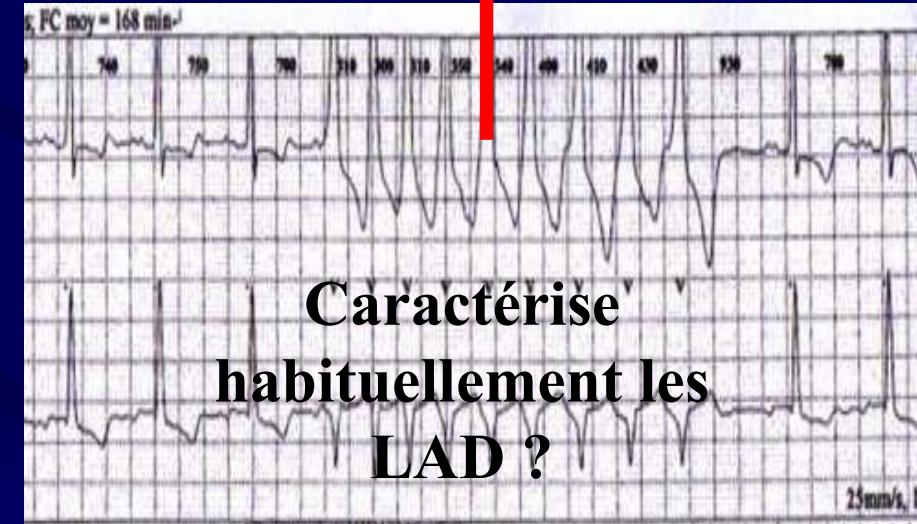
(Circulation Research 1992;70:743–753)



Mécanisme ?



Automatisme de phase 4 ?



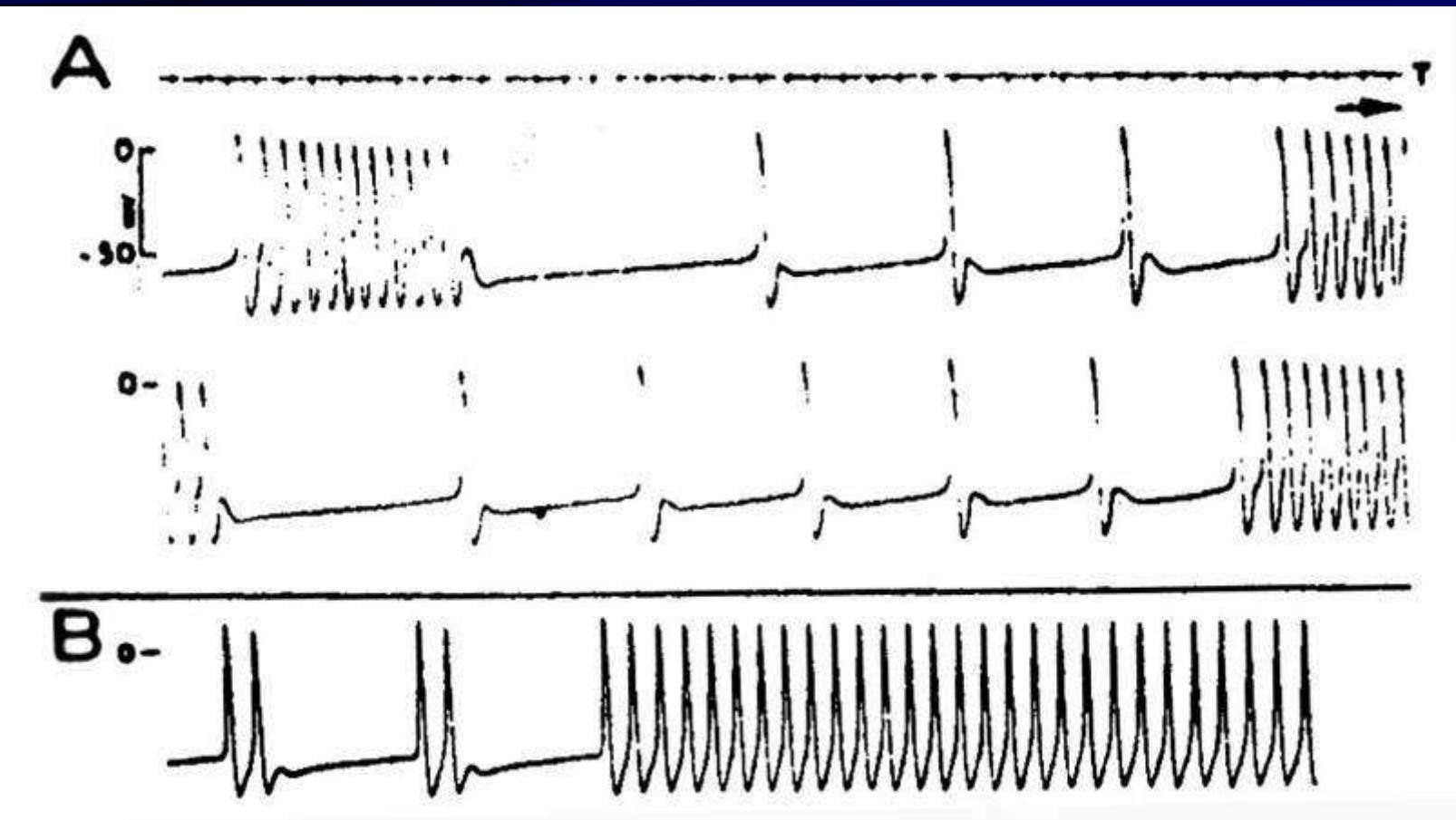
Caractérise
habituellement les
LAD ?

LAD

- Intox Digitaliques
- TV IDM et reperfusion
- TV infundibulaire ou en salves de Galavardin
- Hypertrophie
- TV Catécholergiques

Pace 1997; 20 : 936

Ischémie Myocardique



LAD cellule de purkinje de chien 1 jour après
IDM circ res 1983; 52: 566

TV Infundibulaire

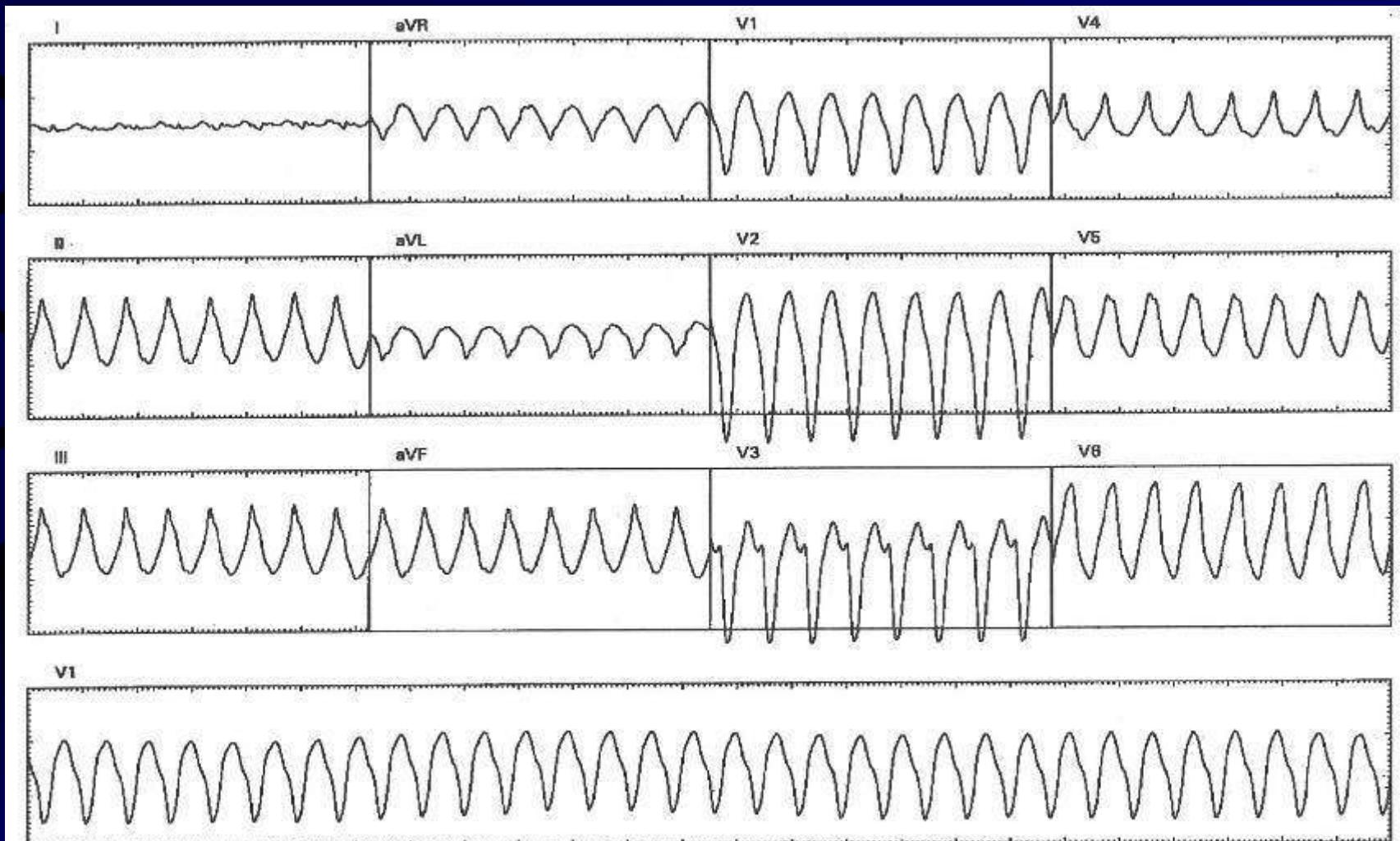


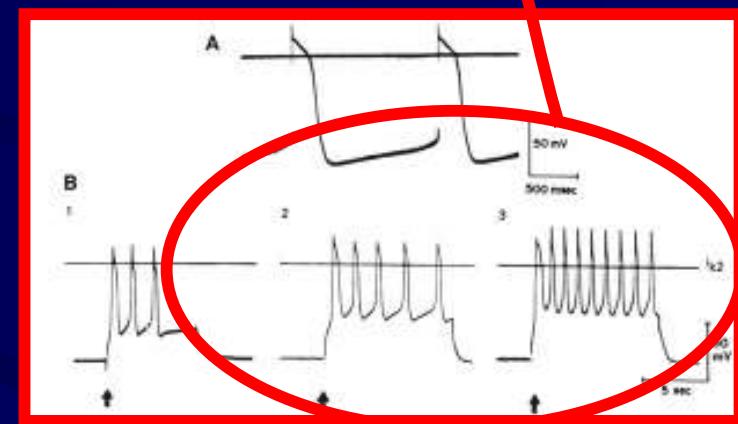
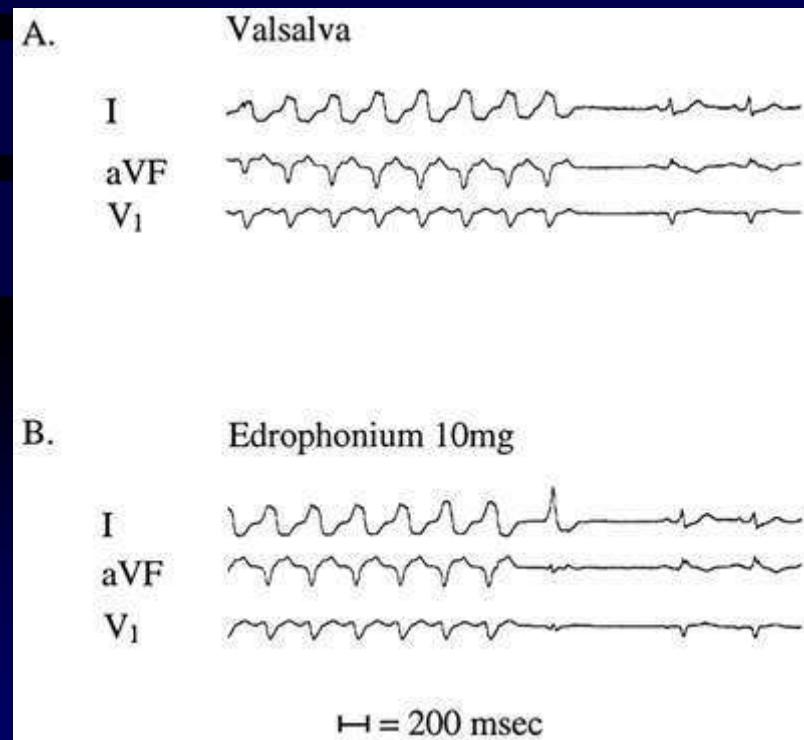
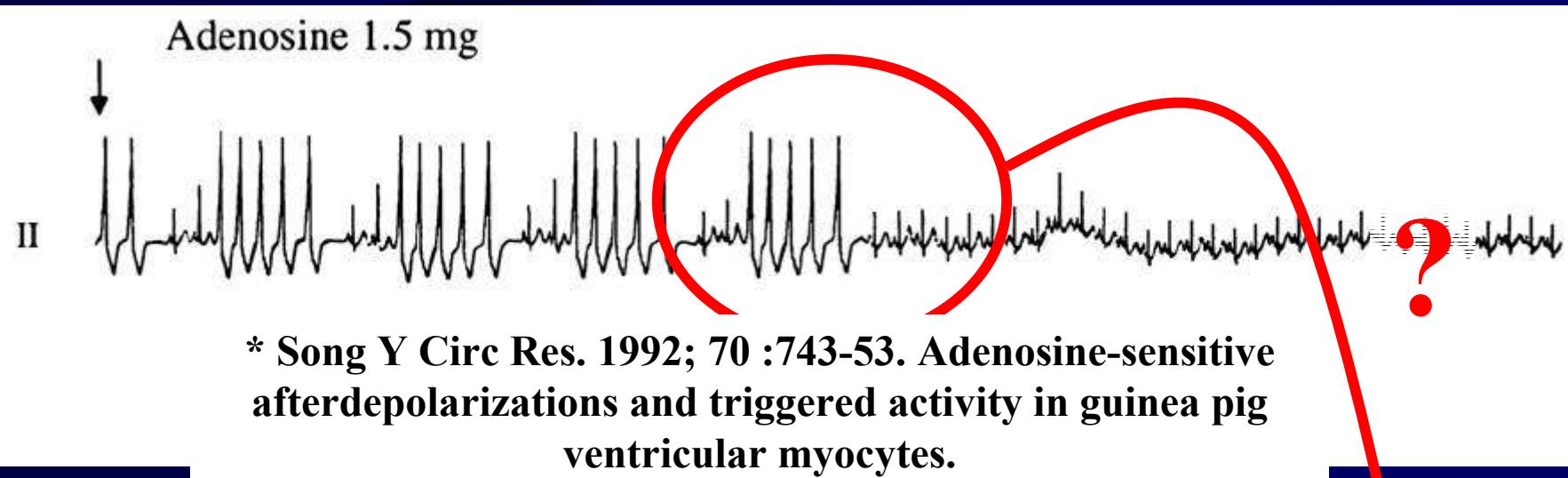
Figure 10. Electrocardiogram of tachycardia arising from the anterior free wall. Note nearly isoelectric QRS in lead I typical of this location, and precordial transition zone at V₄.

TV Infundibulaire

	Isuprel	SVP	ATP	Man. Vag.	BB
RVOT VT	++	+	+	50 %	-

~~origin Cinn房室瓣膜电生理学 Cinn房室瓣膜电生理学
心房颤动的治疗策略 心房颤动的治疗策略 Outflow tract fibrillation Outflow tract fibrillation (J Am Coll Cardiol 2007;49:2035-43)~~





* Mechanism of Repetitive Monomorphic Ventricular Tachycardia
 B B. Lerman, *Circulation*.
 1995;92:421-429

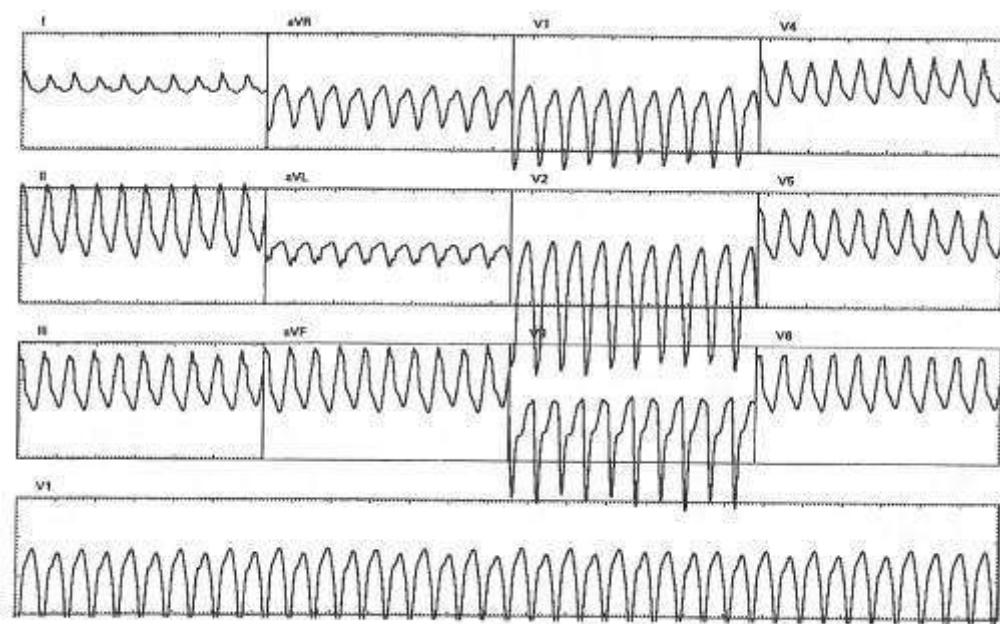
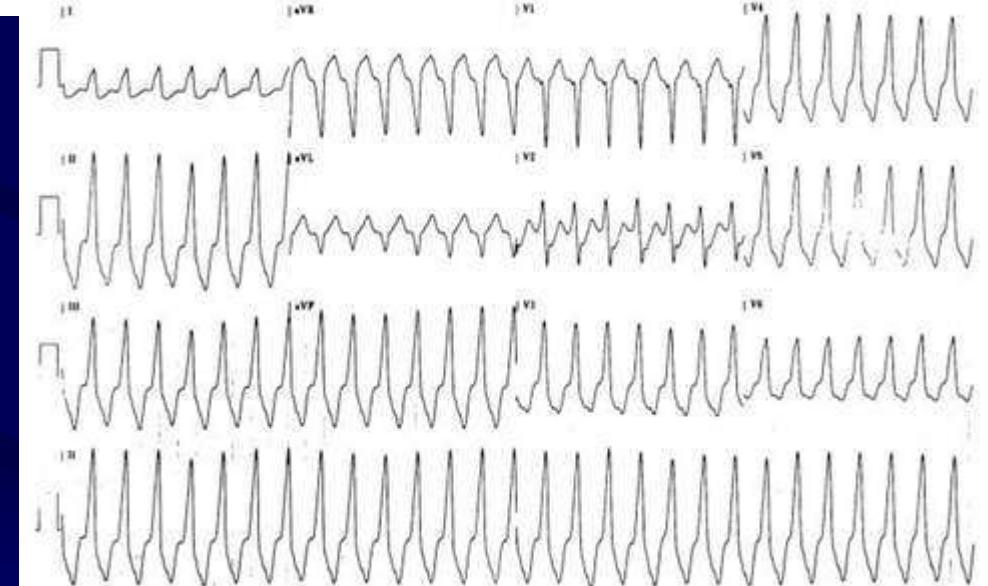


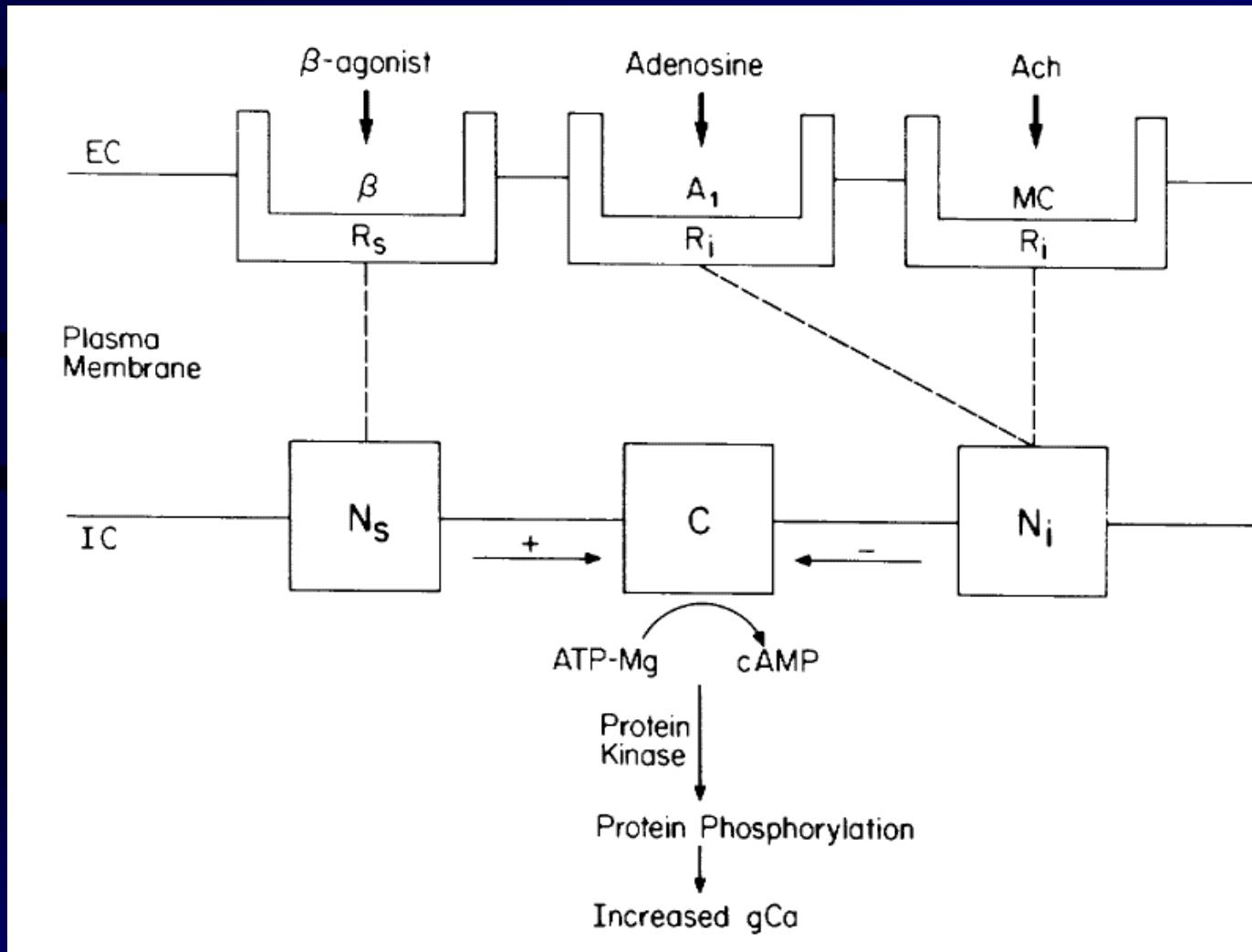
Figure 8. Electrocardiogram of tachycardia arising from the mid/posterior free wall. Note monophasic R waves in lead I, and precordial transition zone at V₄.

* Repetitive Monomorphic Tachycardia From the Left Ventricular Outflow Tract: Electrocardiographic Patterns Consistent With a Left Ventricular Site of Origine. DJ Callans J Am Coll Cardiol 1997;29:1023–7

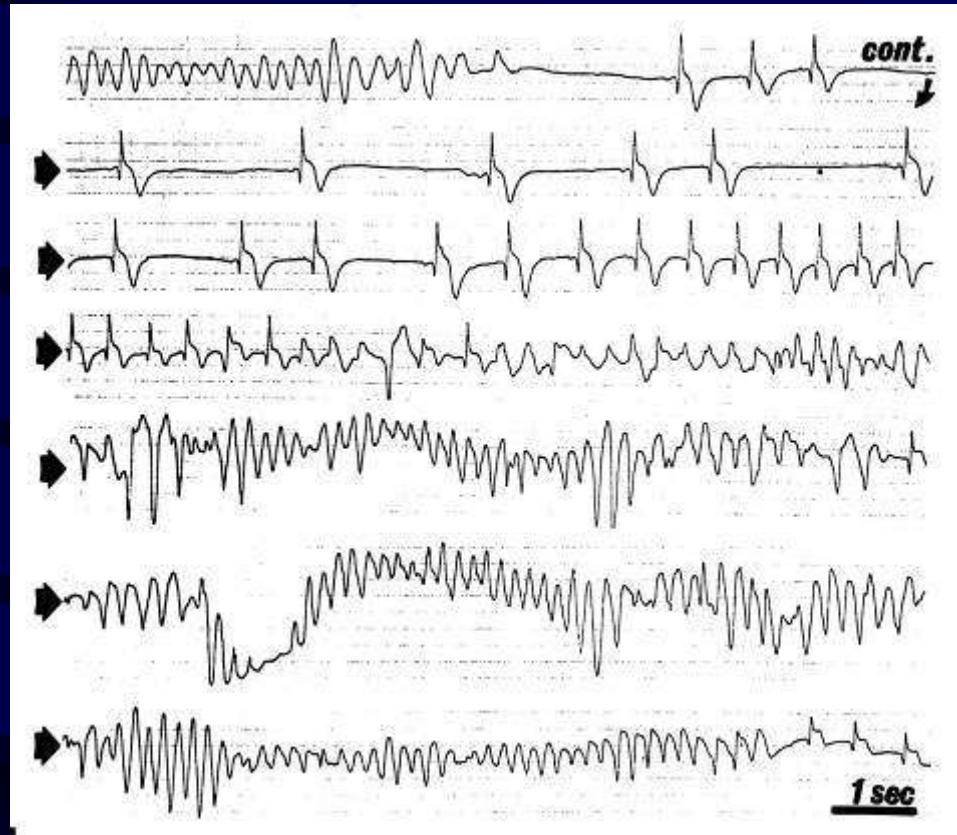
* Localization of Optimal Ablation Site of Idiopathic Ventricular Tachycardia from Right and Left Ventricular Outflow Tract by Body Surface ECG
S Kamakura, MD; *Circulation*. 1998;98:1525-1533.



Thycaid acid-induced arrhythmias suggesting myocardial tachycardia
A triggered activity AMP-mediated triggered activity AMP-mediated
GA West, RM Bell, D Metherell, G A West, RM Bell, D Metherell, J
Circulation 1984;74:270-280 Circulation 1984;74:270-280



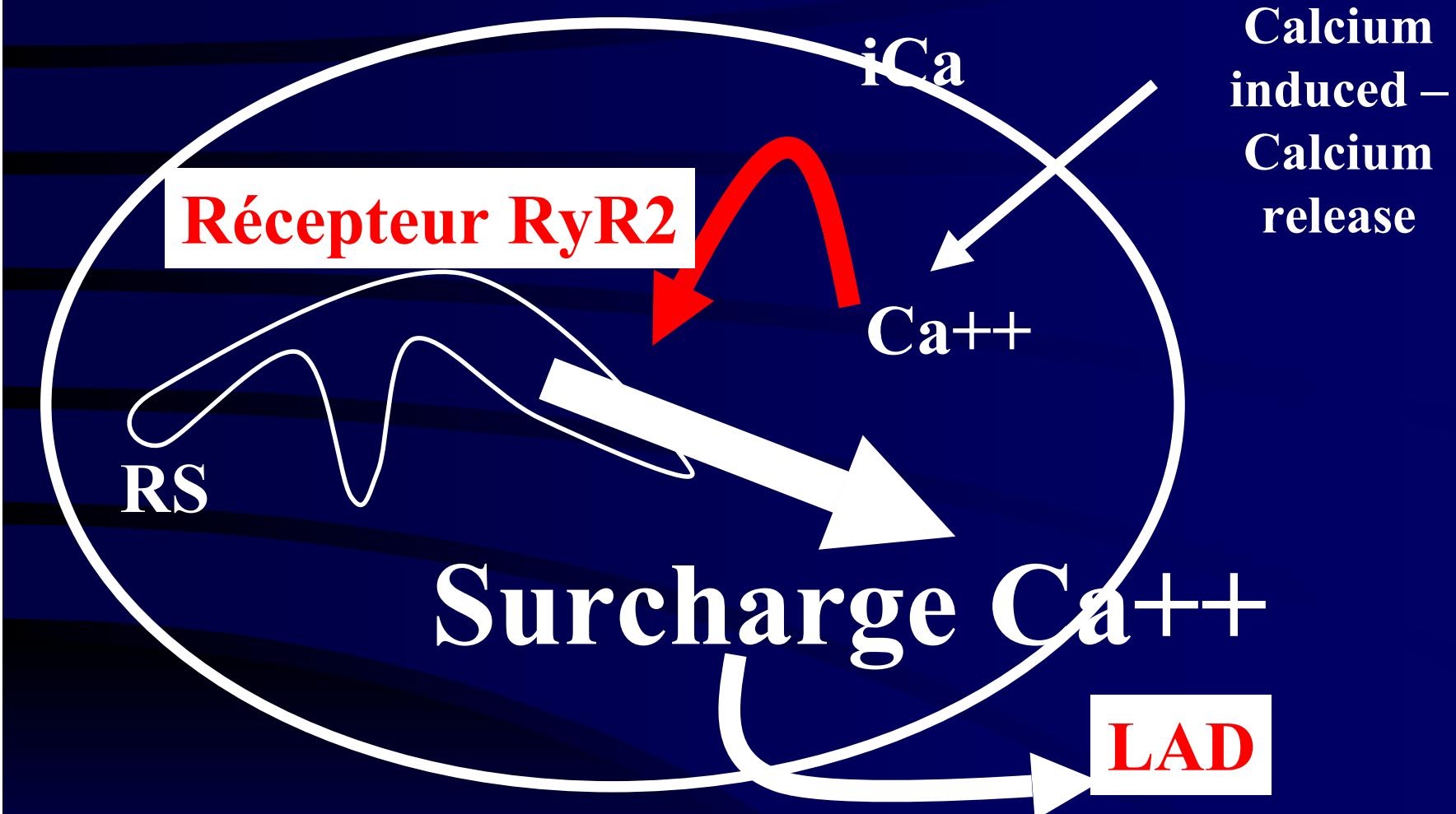
TV catécholergiques



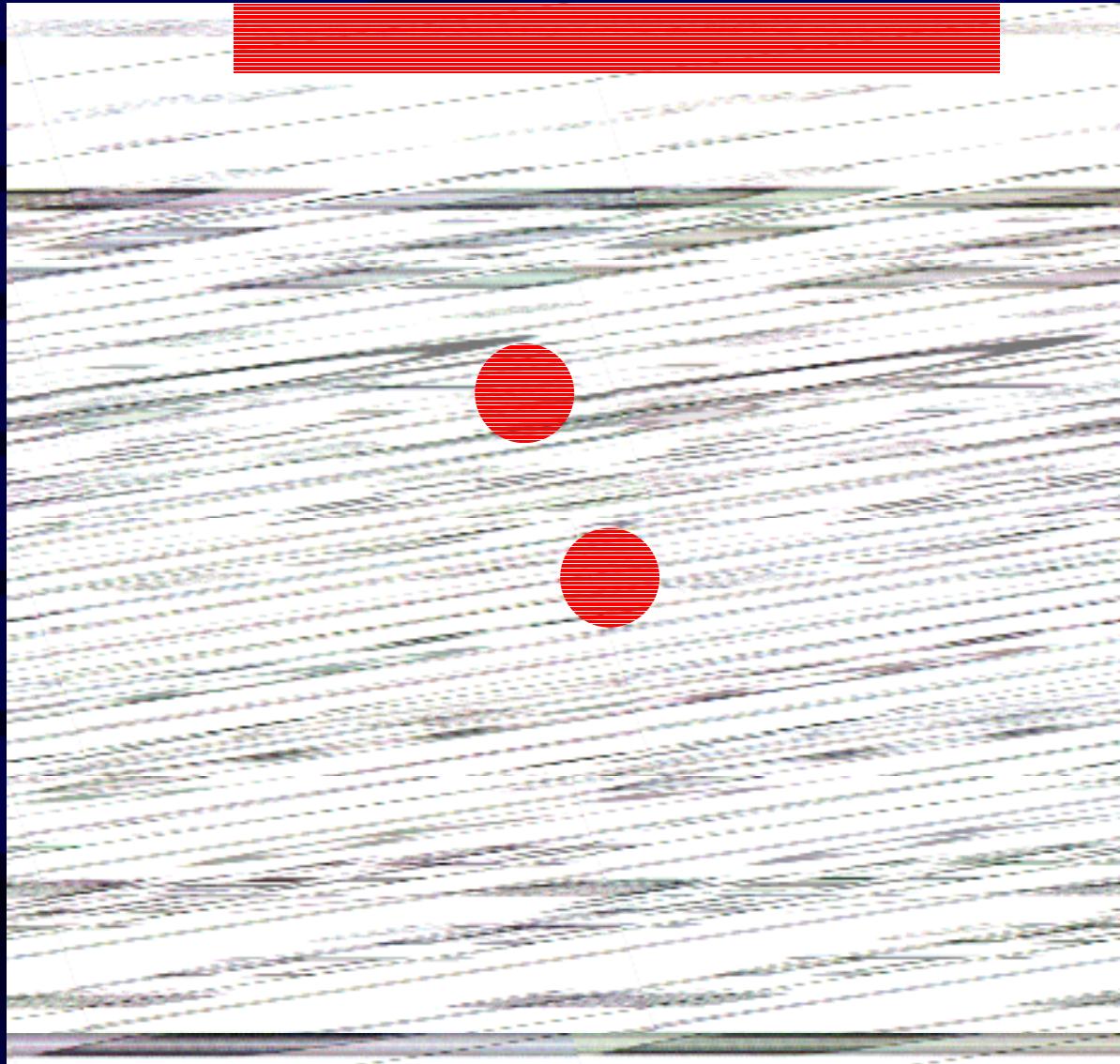
* Catecholaminergic Polymorphic Ventricular Tachycardia in Children
A 7-Year Follow-up of 21 Patients A Leenhardt, *Circulation*. 1995;91:1512-1519.

* **Ryanodine Receptor** Mutations Associated With Stress-Induced Ventricular Tachycardia.
George et al. *Circ Res*.2003; 93: 531-540.

CPVT # Intox Digitalique



* Enhanced Basal Activity of a Cardiac Ca²⁺ Release Channel (Ryanodine Receptor) Mutant Associated With Ventricular Tachycardia and Sudden Death. Dawei Jiang,. (*Circ Res.* 2002;91:218-225.)

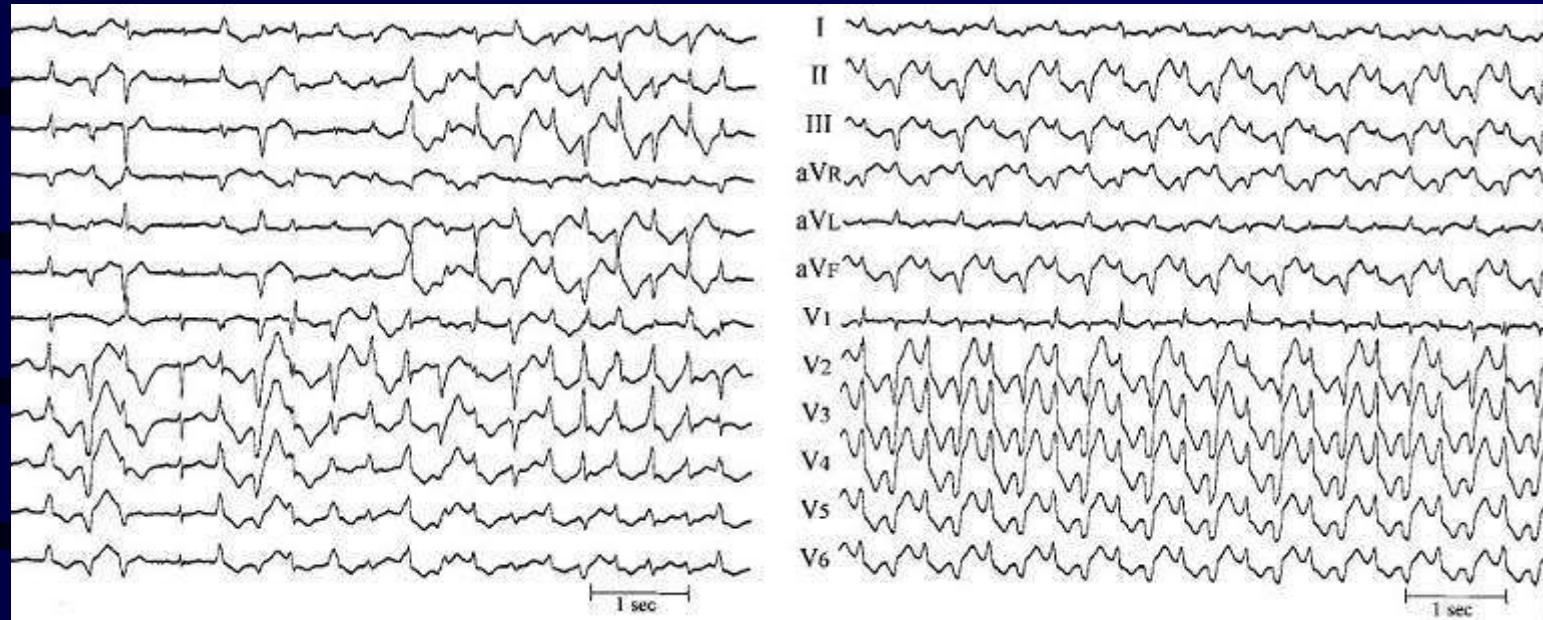


* Clinical and Molecular Characterization of Patients With Catecholaminergic Polymorphic Ventricular Tachycardia Silvia G. Priori,
Circulation. 2002;106:69-74.

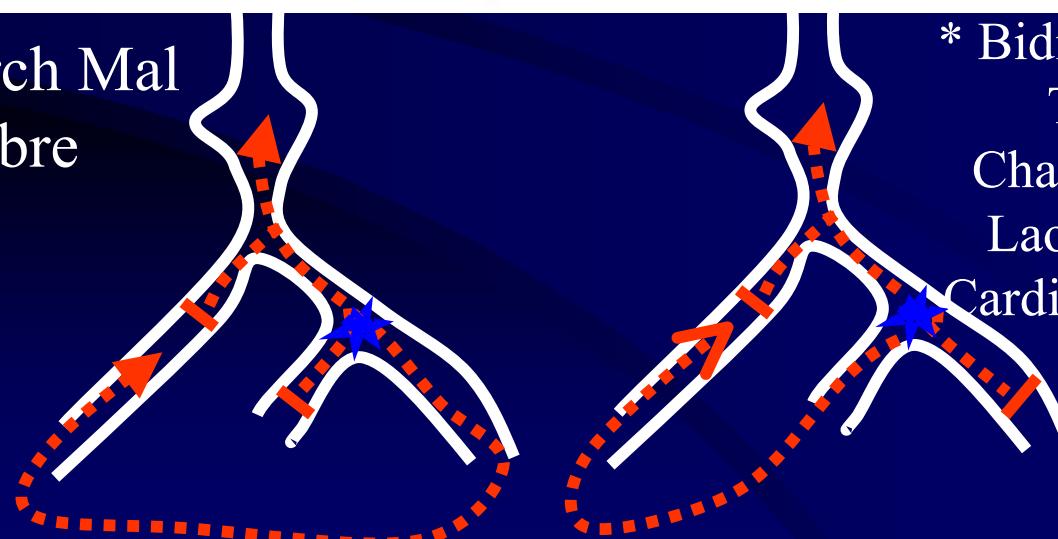
Absence of Calsequestrin 2 Causes Severe Forms of Catecholaminergic Polymorphic Ventricular Tachycardia
Alex V. Postma, *Circ Res*. 2002; 91:e21-e26.

TV Bidirectionnelle

* Ito JCE 2002



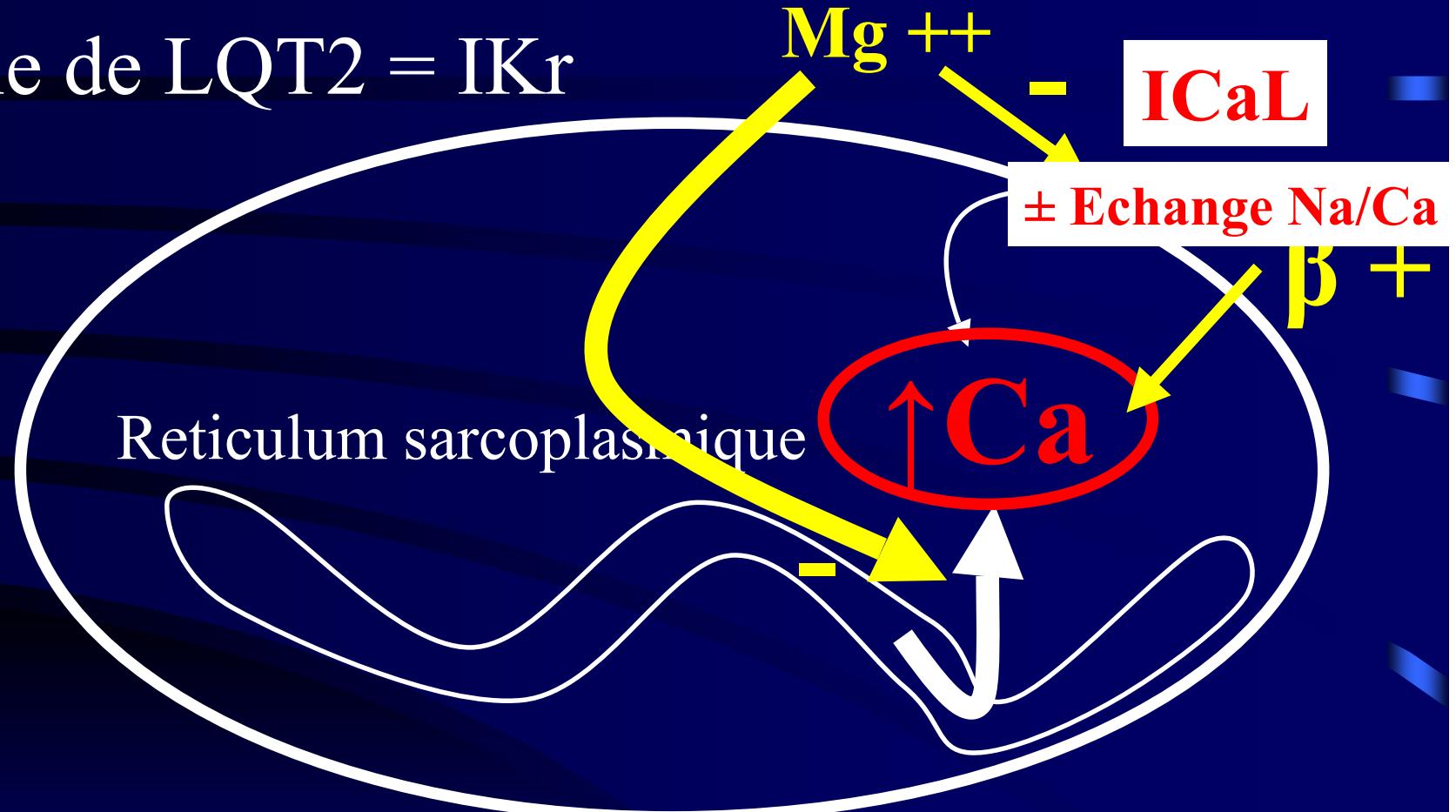
Leenhardt Arch Mal
Cœur Décembre
2003



* Bidirectional Ventricular
Tachycardia and
Channelopathy Preecha
Laohakunakorn, Am J
Cardiol 2003;92:991–995.

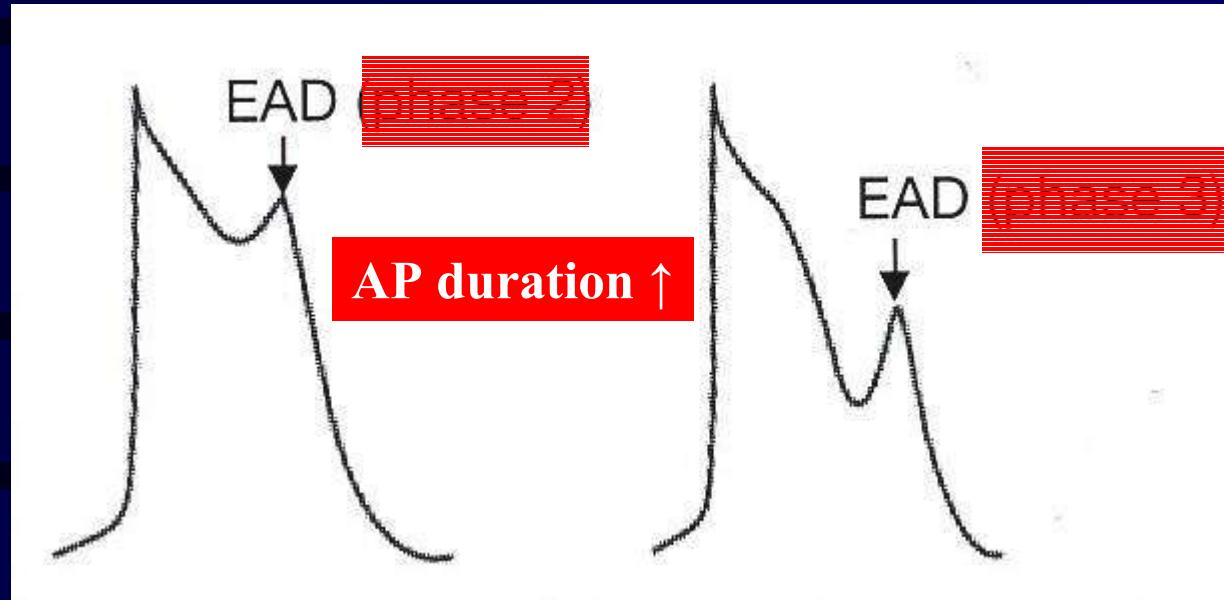
Early Afterdepolarizations

Modèle de LQT2 = IKr



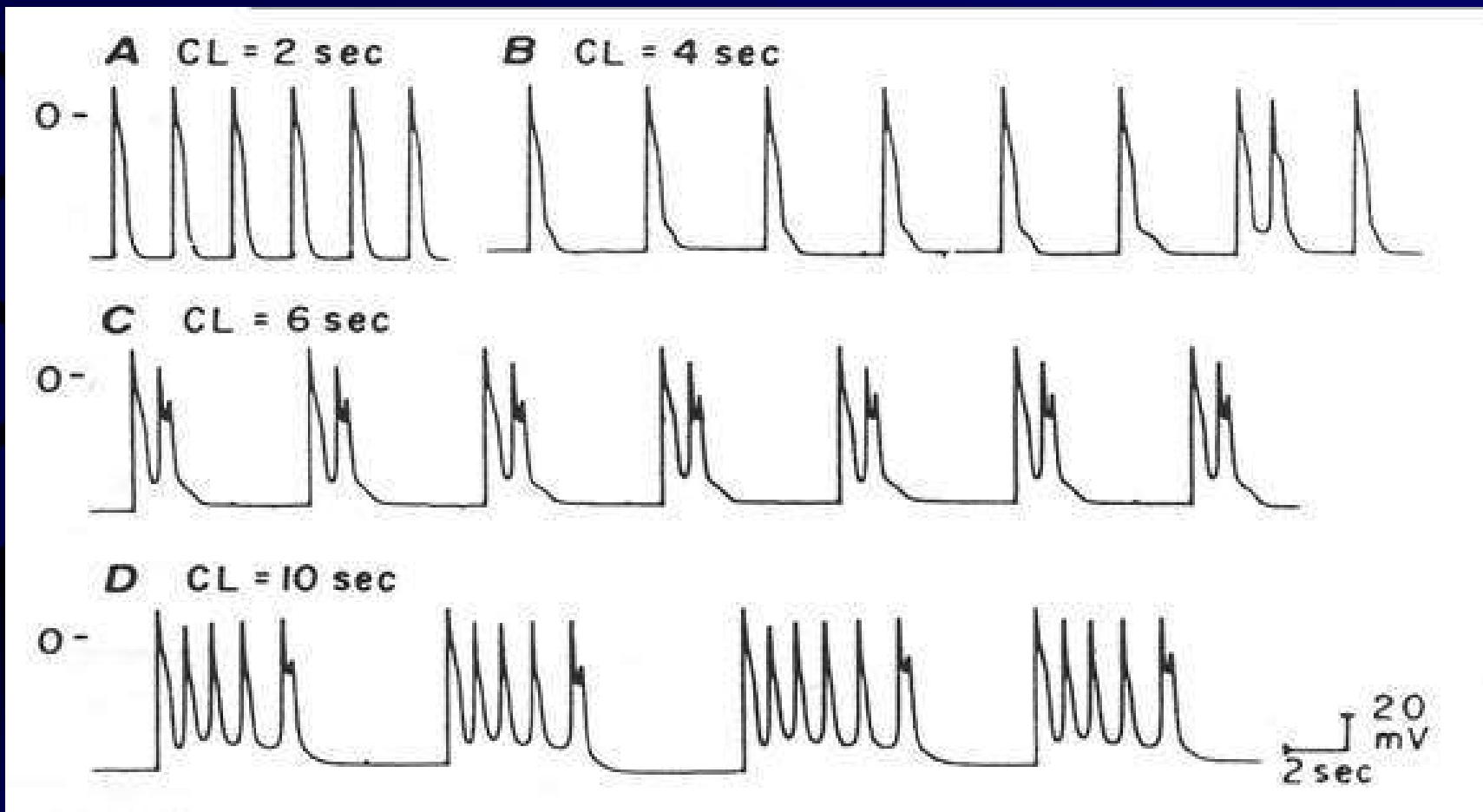
* Cytosolic Ca^{2+} triggers early afterdepolarizations and Torsade de Pointes in rabbit hearts with type 2 long QT Syndrome BR Choi Journal of Physiology 2002, 543, 615–631

Early Afterdepolarizations

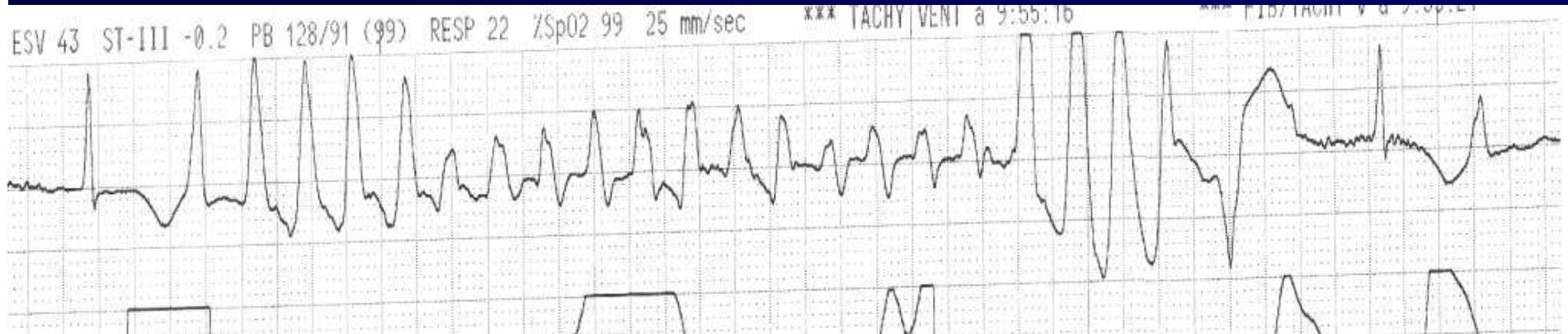


- Diminution des courants sortants K⁺ : Ikr, Iks, Ito
- Augmentation des courant entrants sodique ou calcique
- Hypo K⁺, Ca⁺⁺, hypoxie, AAR III, Césium, Quinidine neuroleptiques, antihistaminiques, érythromycine
BRADYCARDIE

Rôle de la fréquence



Torsades de Pointes



EAD ?

Réentrée ?

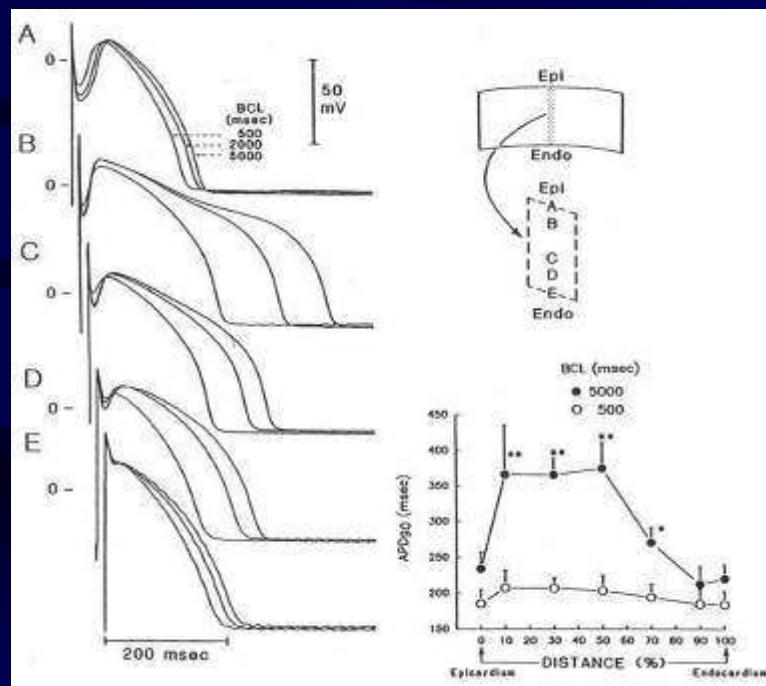
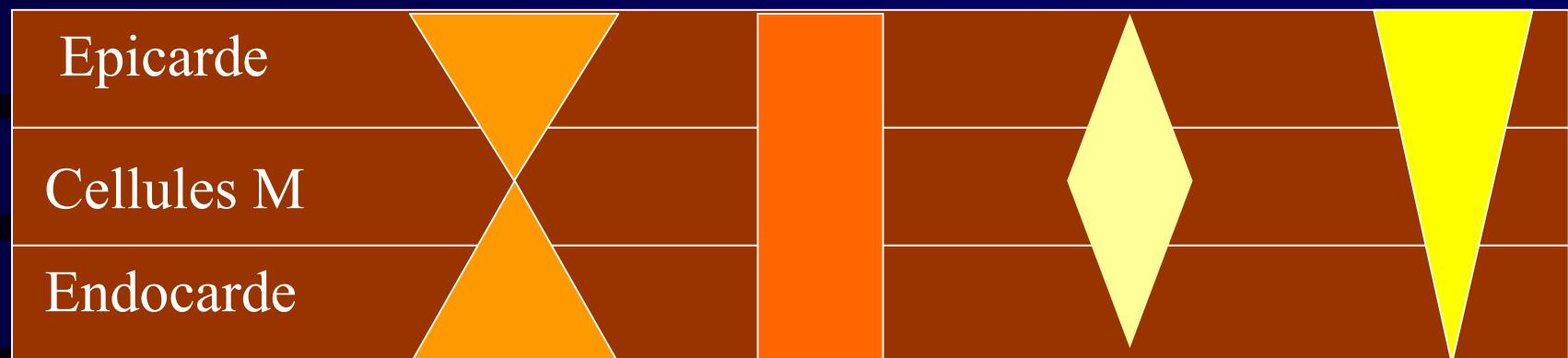
- QT long 1 et 5 Iks
- QT long 2 et 6 Ikr
- QT long 3 Na
- QT long acquis

iKs QT1

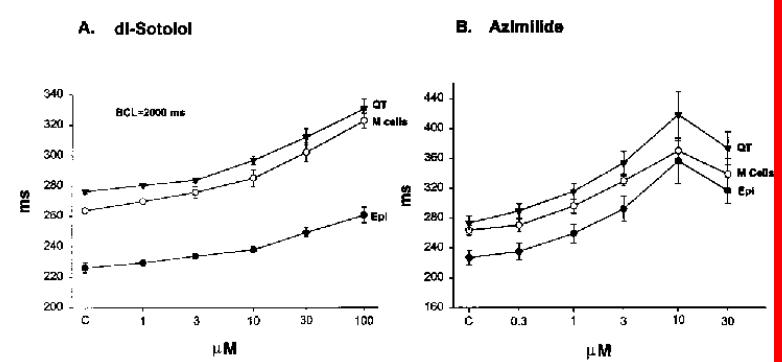
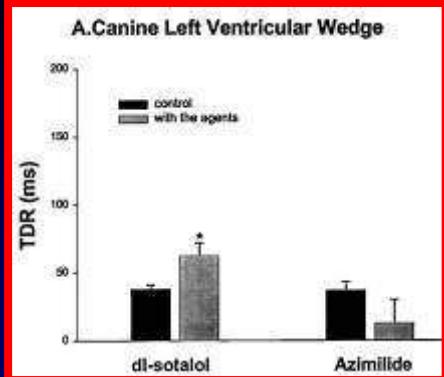
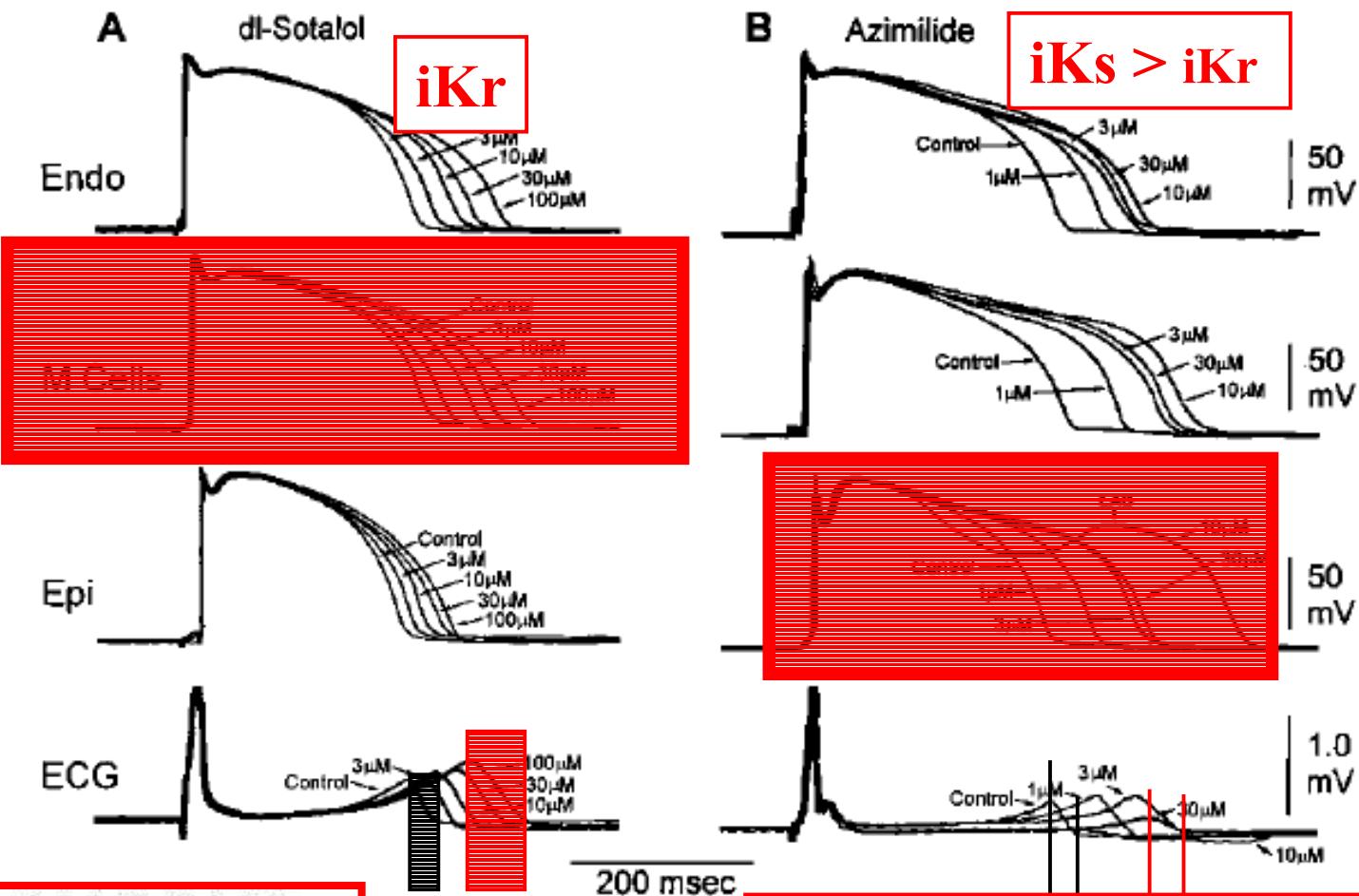
iKr QT2

iNa lent QT3

Ito

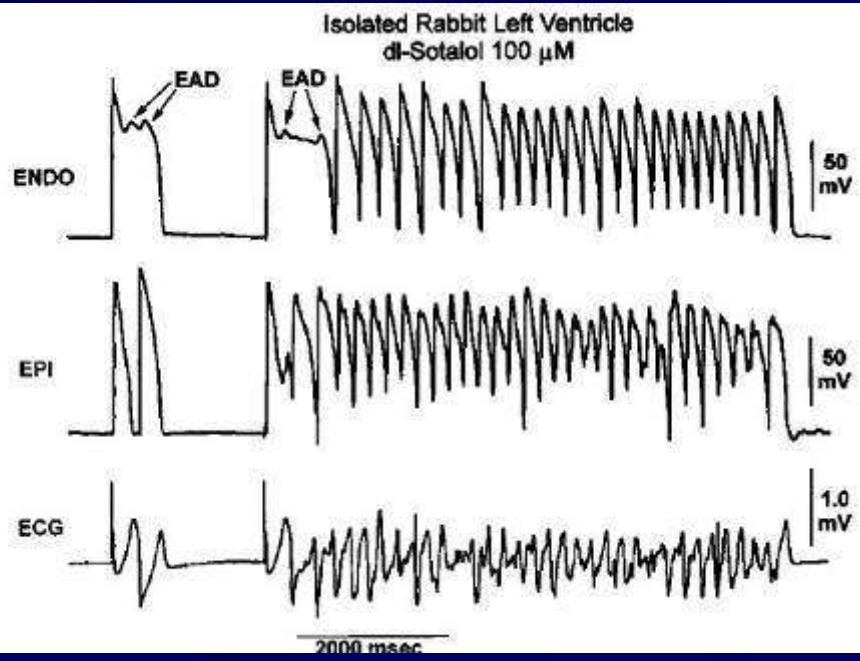
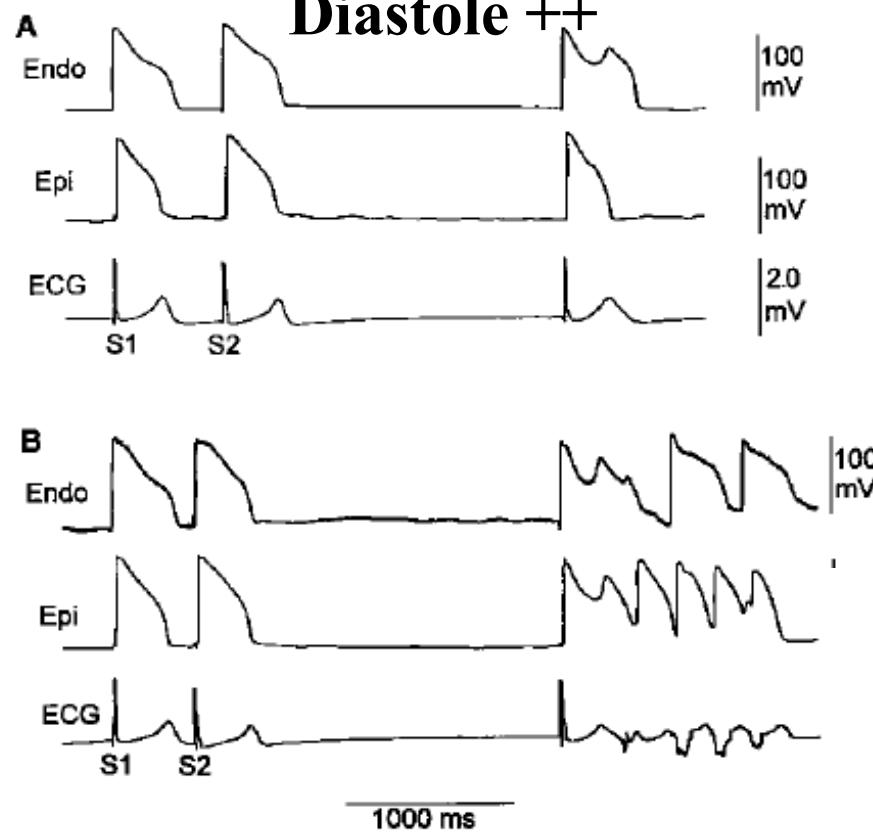


* Cellular Basis for the ECG Features of the LQT1 Form of the Long-QT.
W Shimizu, *Circulation*. 1998;98:2314-2322.



Isolated Rabbit Left Ventricle
dl-sotalol 100 μ M

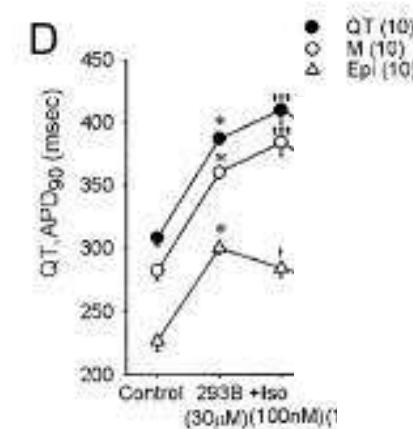
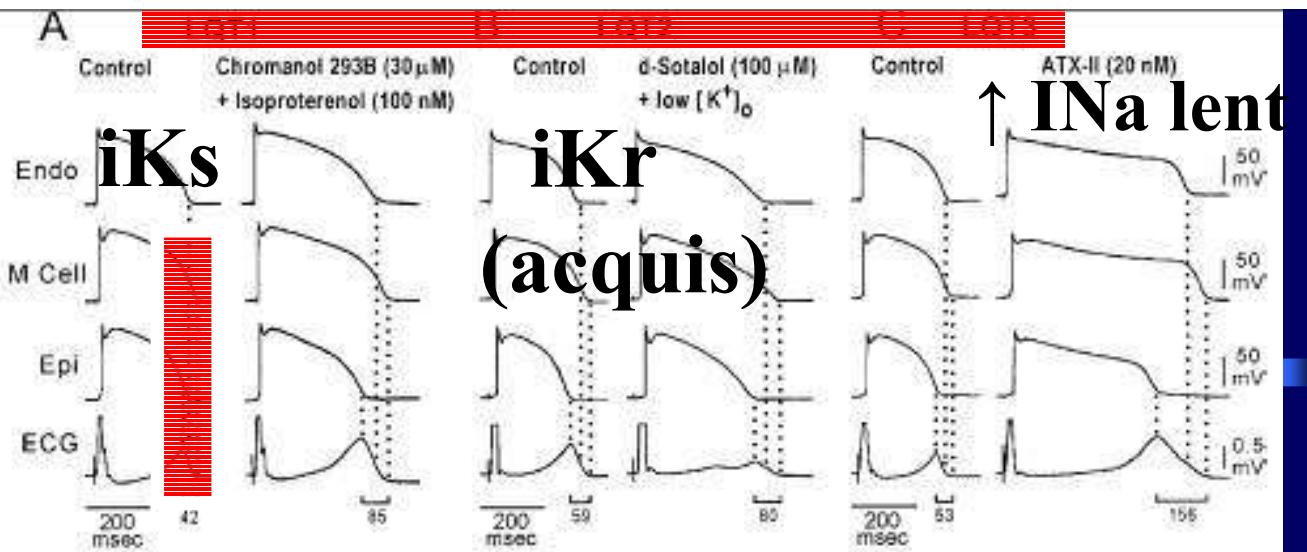
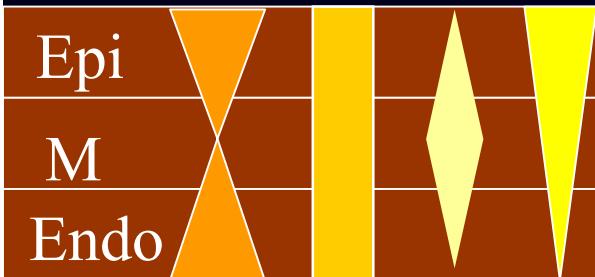
Diastole ++



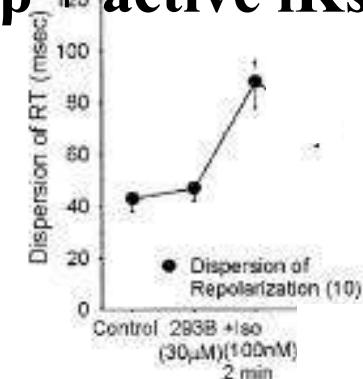
* Antzelevitch JCE
2003 13 1259-72

* Cellular Basis for the ECG Features of the LQT1 Form of the Long-QT. W Shimizu,
Circulation.
1998;98:2314-2322.

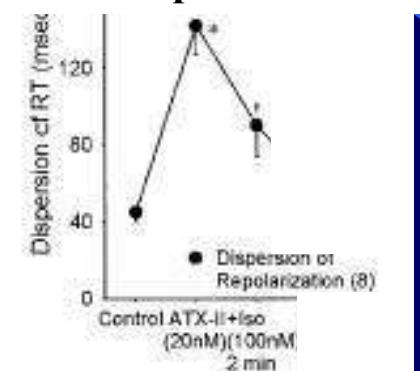
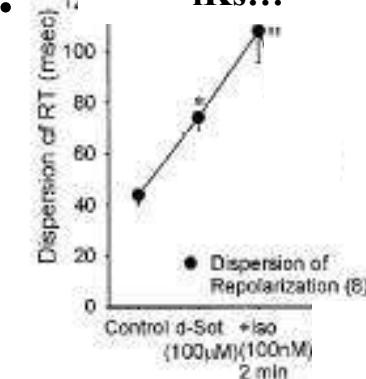
Ina
iKs iKr lent Ito

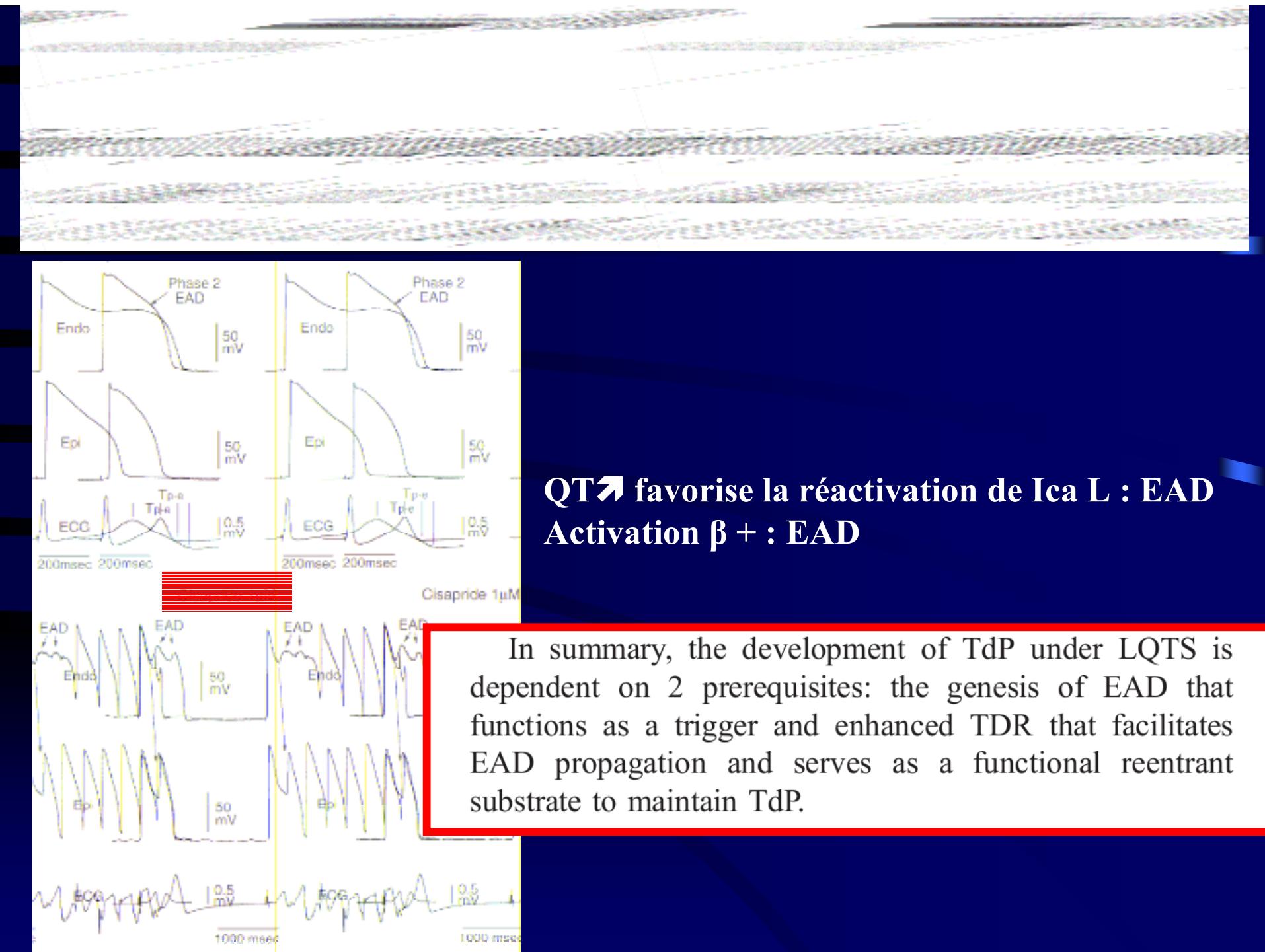


β + active Ina/ca plus vite que iKs...

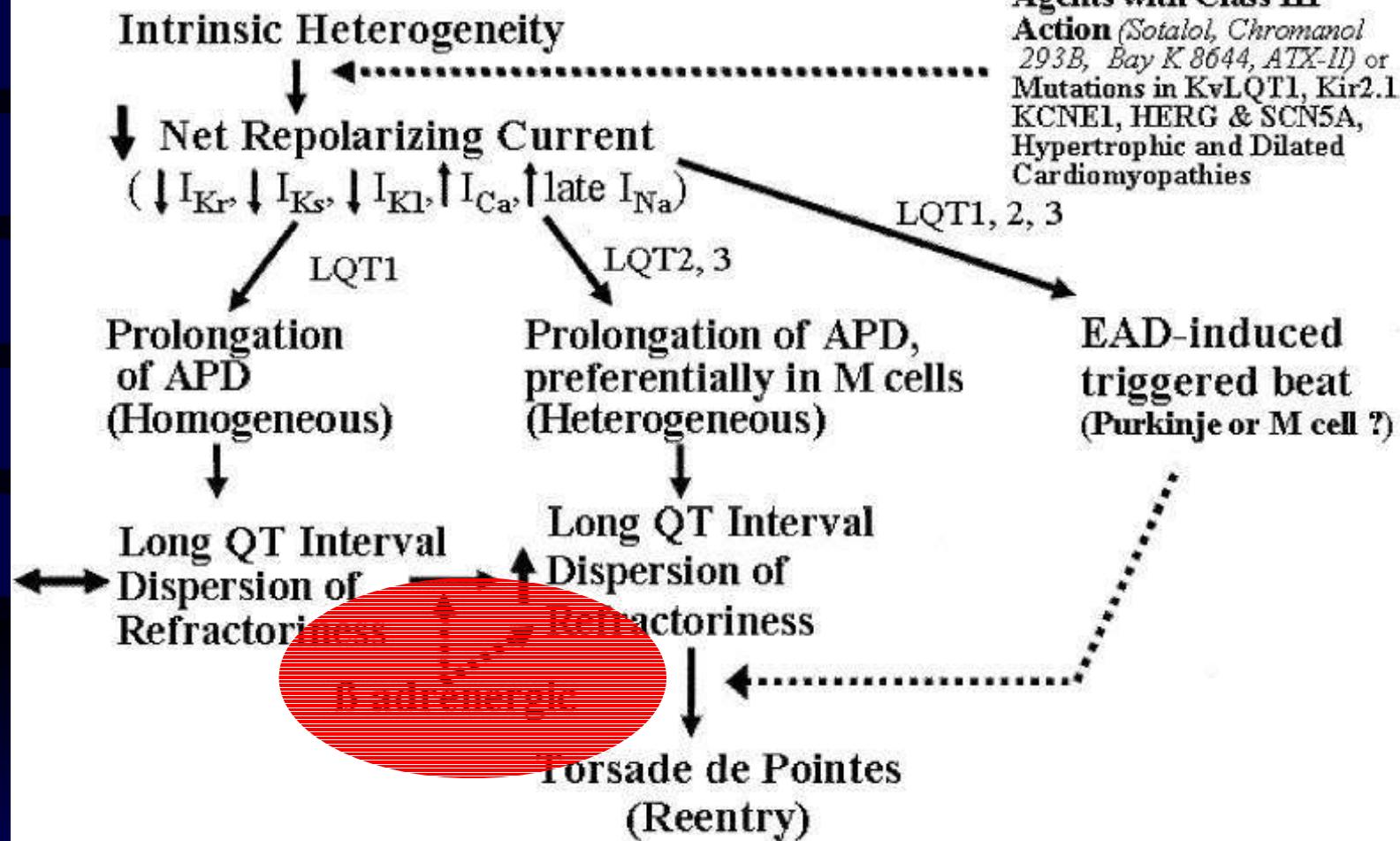


Effet protecteur β + TDR repos nocturne

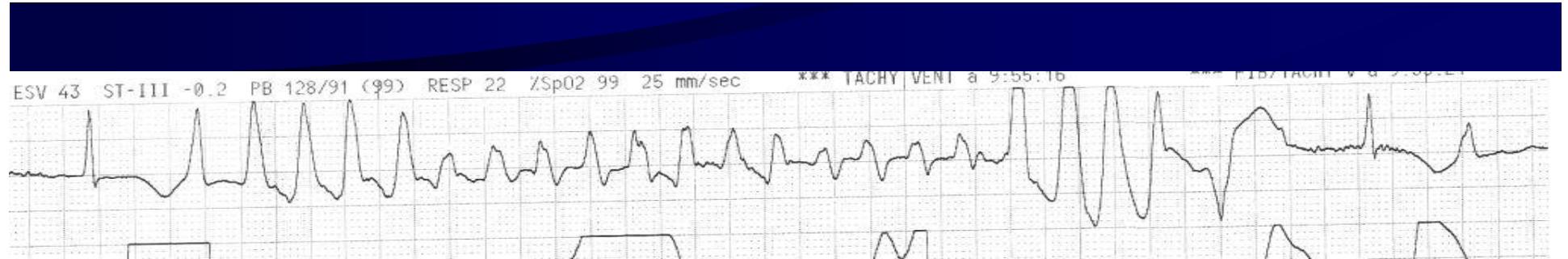




Long QT Syndrome



* Antzelevitch JCE 2003 13 1259-72



EAD = initialisent Torsades de pointes

La torsade ?

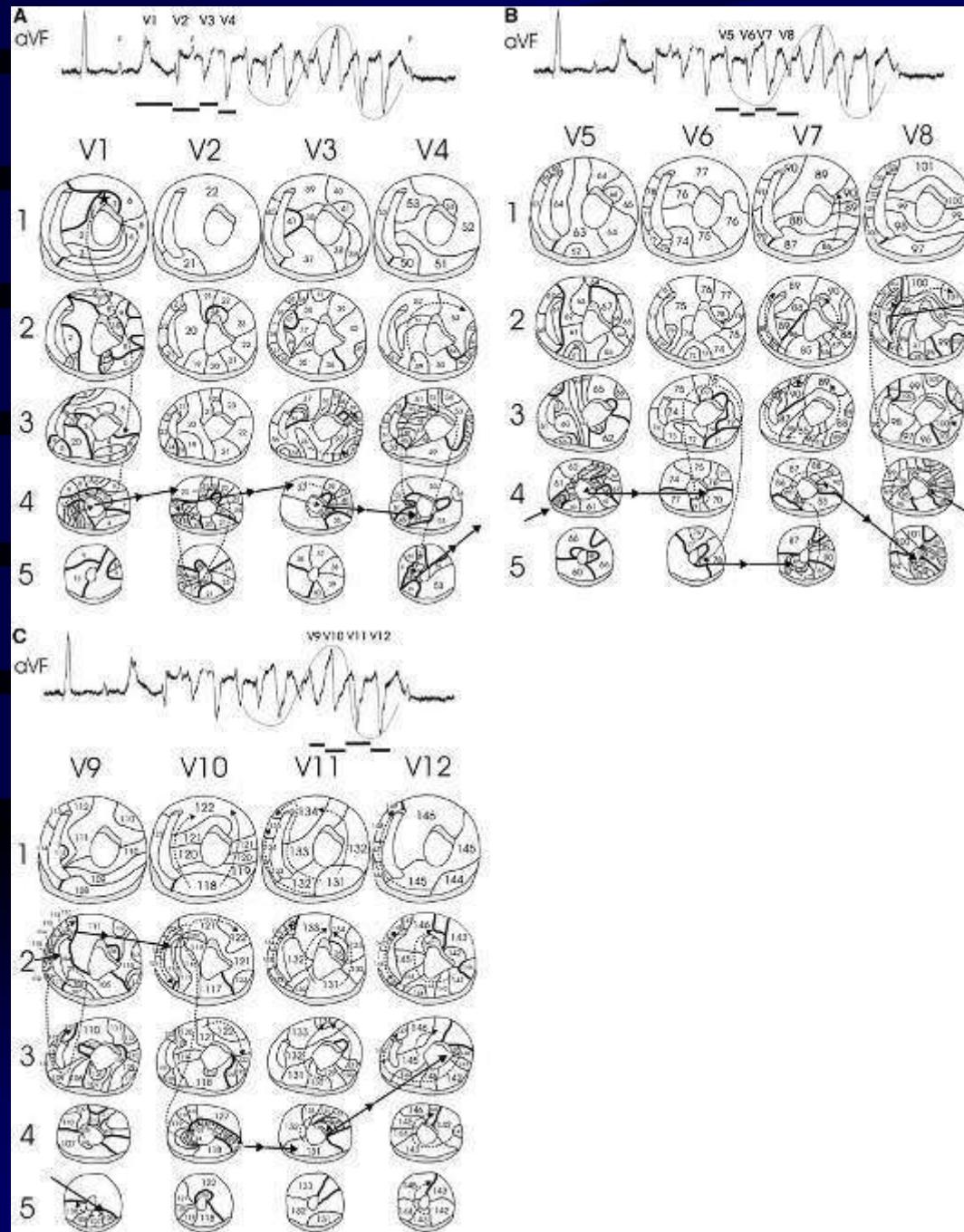
EAD de différents foyers ?

Deux foyers à une fréquence proche
peuvent reproduire l'aspect ECG

Réentrée avec différents points de sortie

Arythmie non inducible par SVP

Réentrée en phase 2



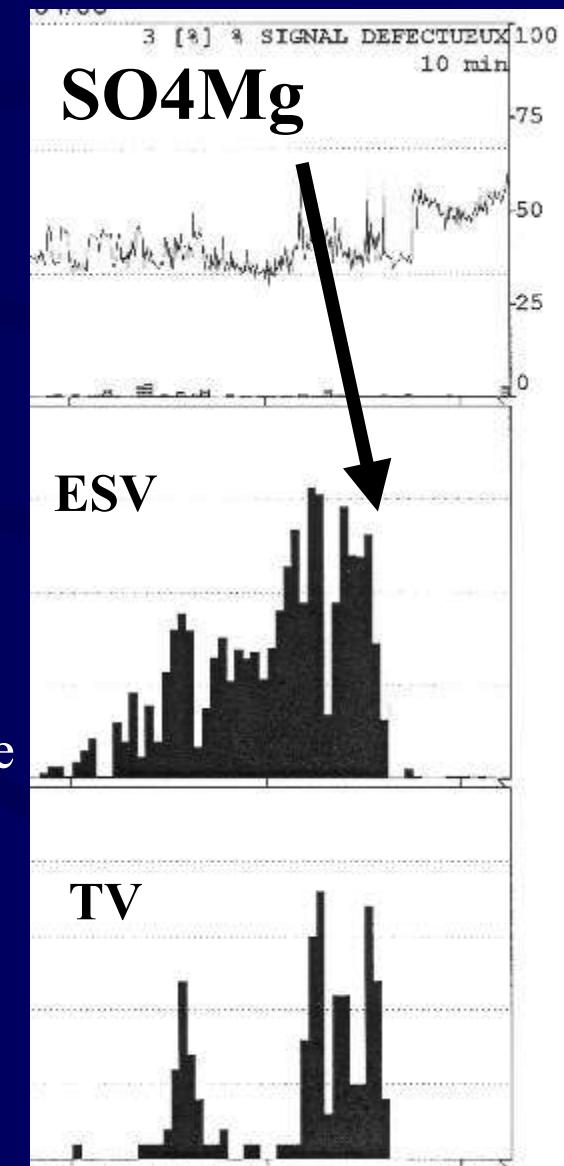
* Nabil El-Sherif,
Electrophysiological Mechanism
of the Characteristic
Electrocardiographic
Morphology of Torsade de
Pointes Tachyarrhythmias in the
Long-QT Syndrome Detailed
Analysis of Ventricular
Tridimensional Activation
Patterns. *Circulation*.
1997;96:4392-4399.

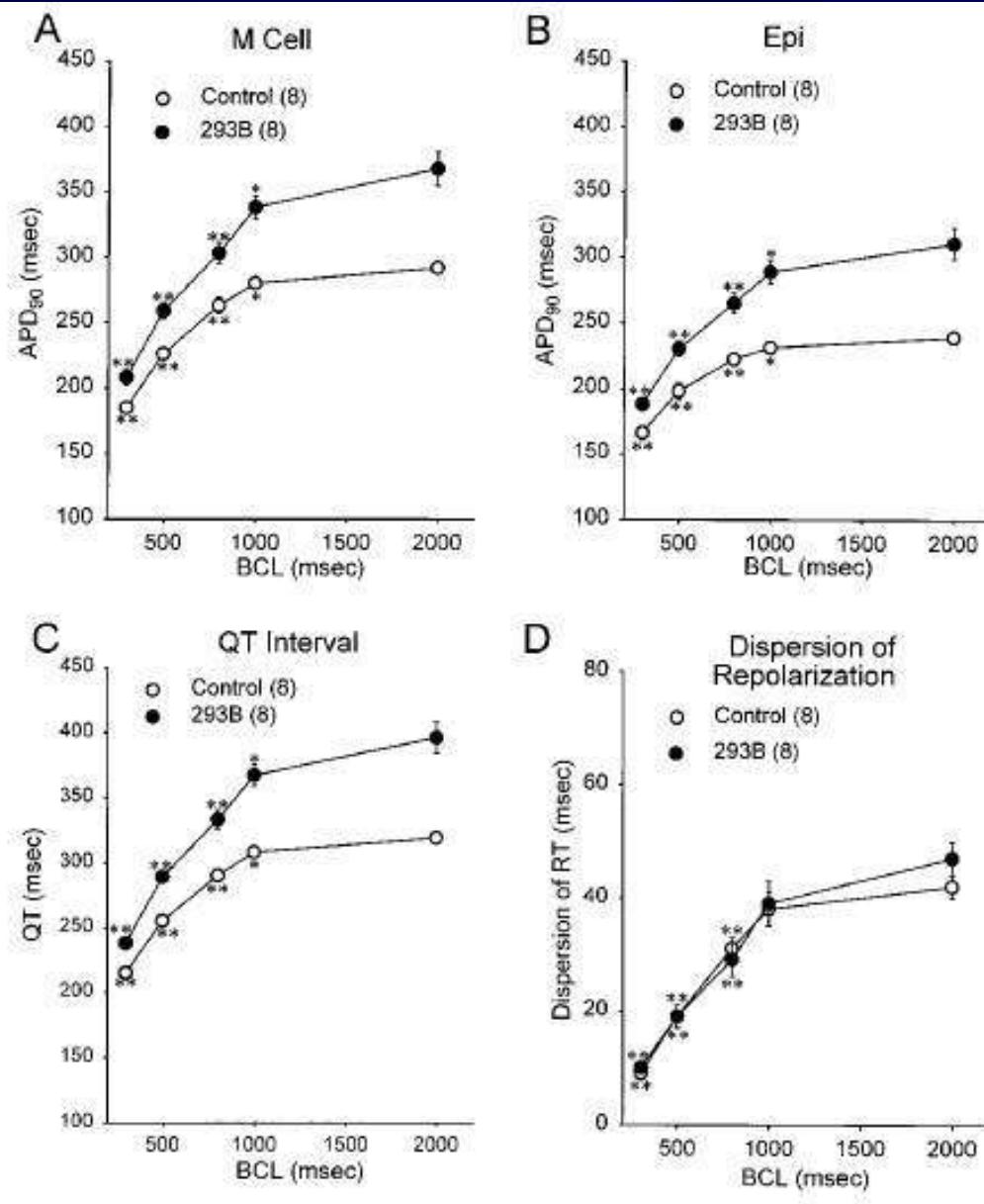
Effet du Mg++

APPLICATION CLINIQUE

* Treatment of torsade de pointes with magnesium sulfate
D Tzivoni, Circulation, 1988; 77: 392-397

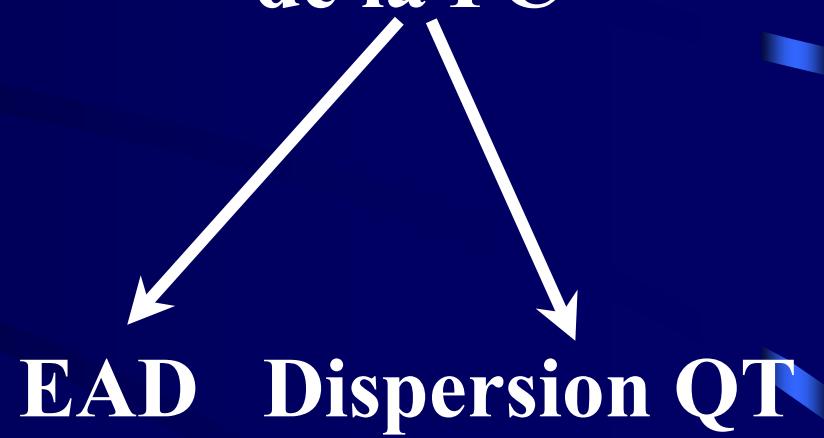
Diminution de la dispersion
de la repolarisation





**Chromanol 293B
Inhibiteur de Iks
QT > QT contrôle**

**Effet protecteur
de la FC**



* Cellular Basis for the ECG Features of the LQT1 Form of the Long-QT.
W Shimizu, *Circulation*. 1998;98:2314-2322.

Pause-Dependent Torsade de Pointes Following Acute Myocardial Infarction

A Variant of the Acquired Long QT Syndrome

Amir Halkin, MD,
Sami Viskin, MD
Tel Aviv, Israel

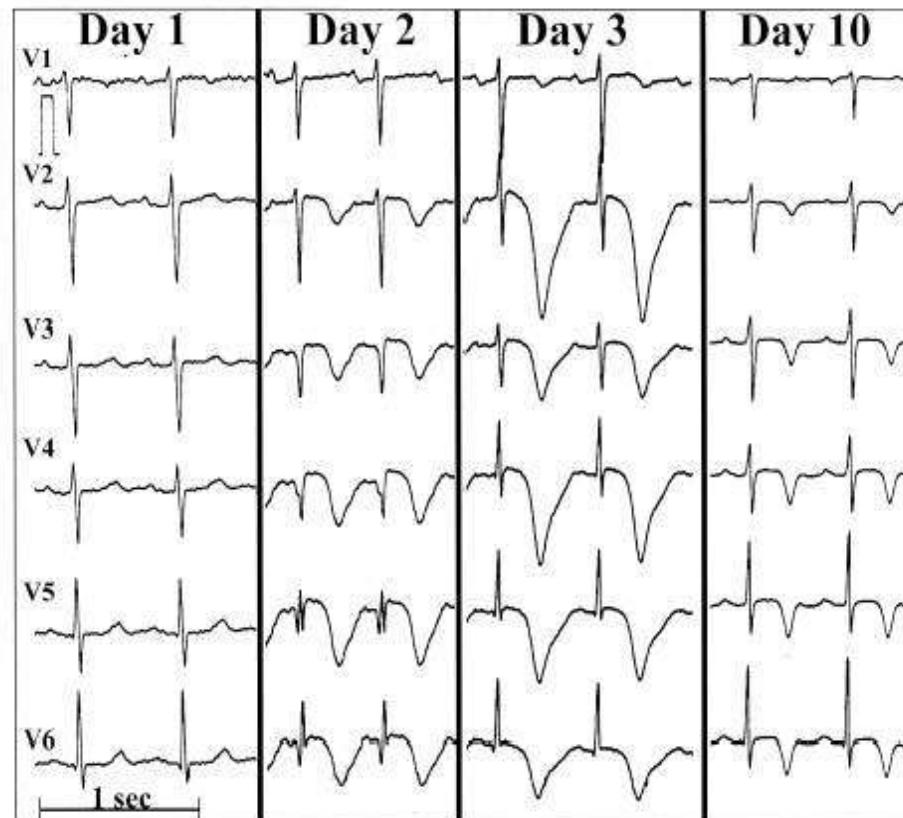
Ad Belhassen, MD,

OBJECTIVES

BACKGROUND

METHODS

RESULTS



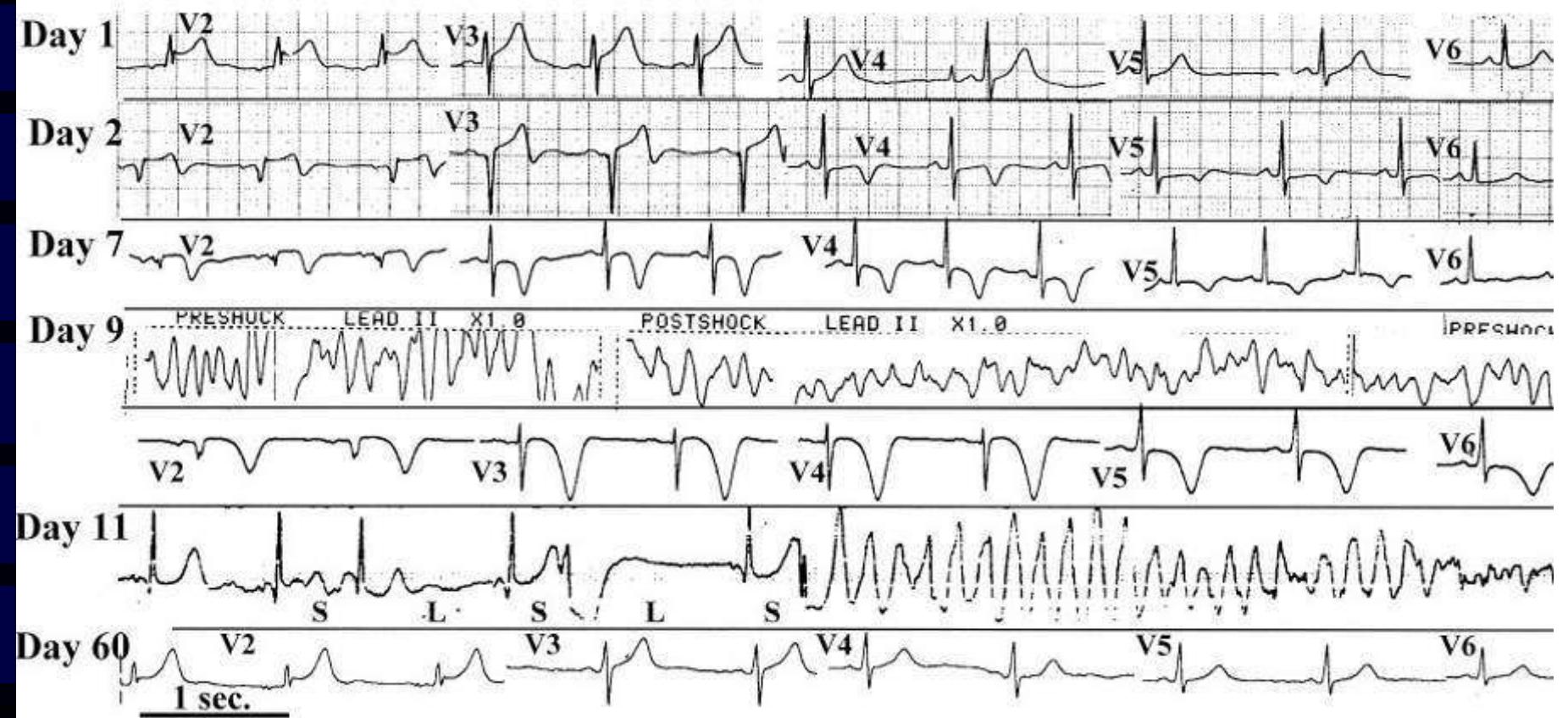
syndrome (QT interval
a [VT]) entirely related

the hyperacute phase, is
ough QT prolongation
T (torsade de pointes)

: QT prolongation that
hemia or other known
nsecutive patients with

idence interval 0.8% to
admission. The QTc
rsade de pointes (from
4 ms, respectively [$p <$
d 3 to 11 days after
d beta-blockers. Three
eventful.

CONCLUSIONS Infarct-related torsade de pointes is uncommon but potentially lethal. An acquired long QT syndrome should be considered in patients recovering from MI who experience polymorphic VT as specific therapeutic measures are mandatory. (J Am Coll Cardiol 2001;38:1168-74)
© 2001 by the American College of Cardiology



Physiopathologie : Cellules de Purkinje ischémées: PA \downarrow puis $\uparrow \Rightarrow$ EAD
 Cycle long-court : Ca⁺⁺ Dispersion QT =EAD
 Remodeling des zones saines : Ikr Iks \downarrow
 Dénervation

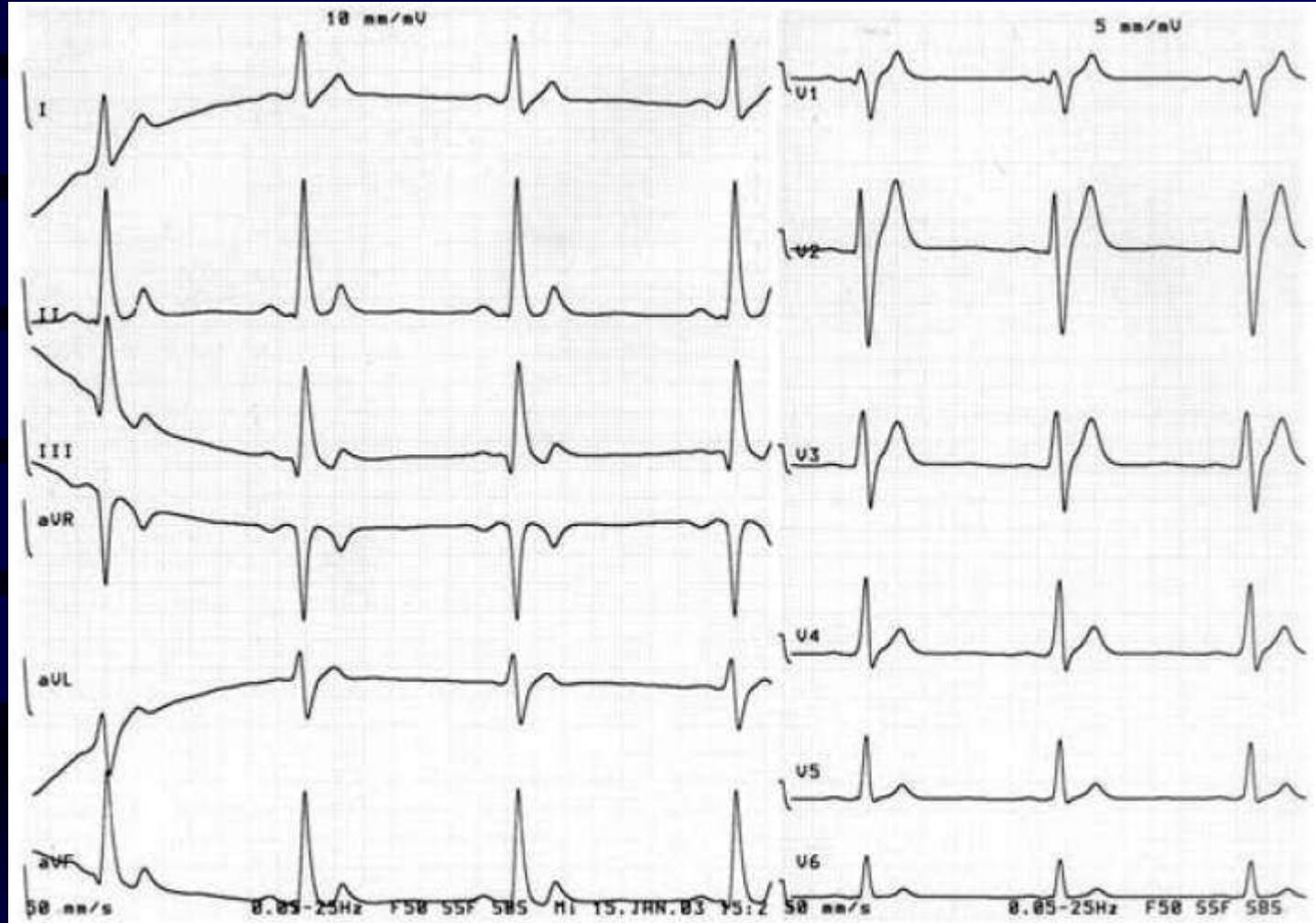
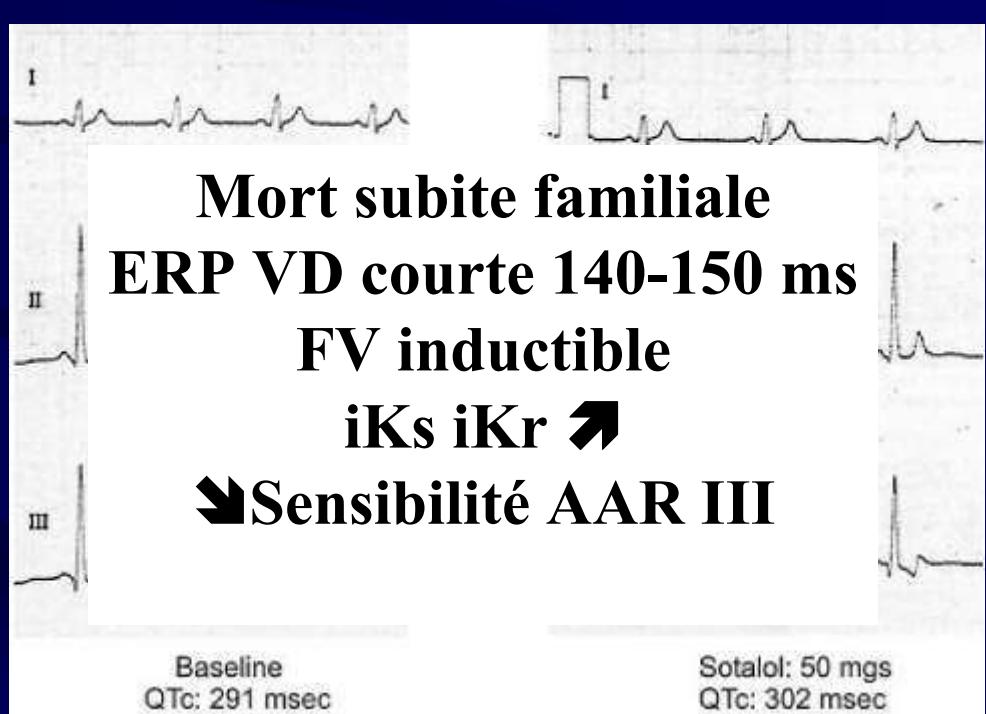
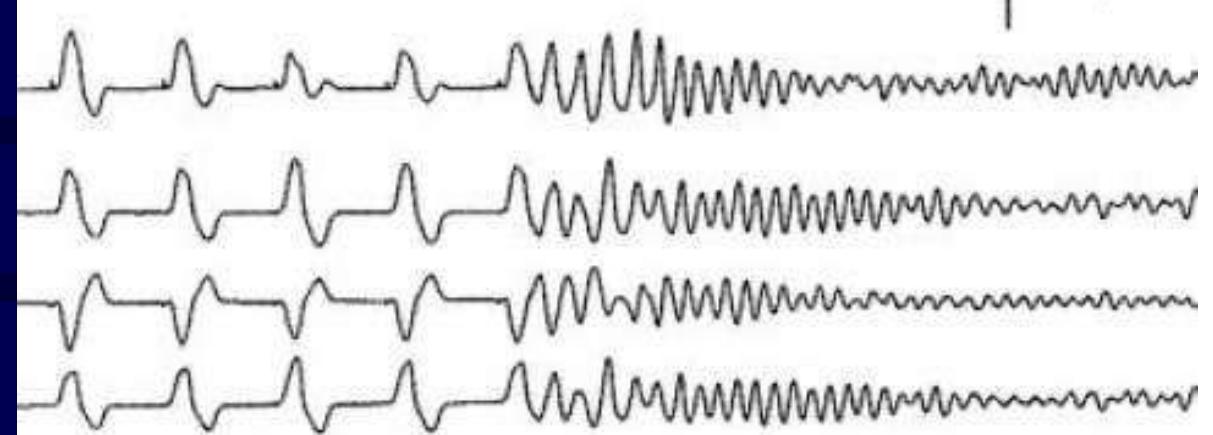
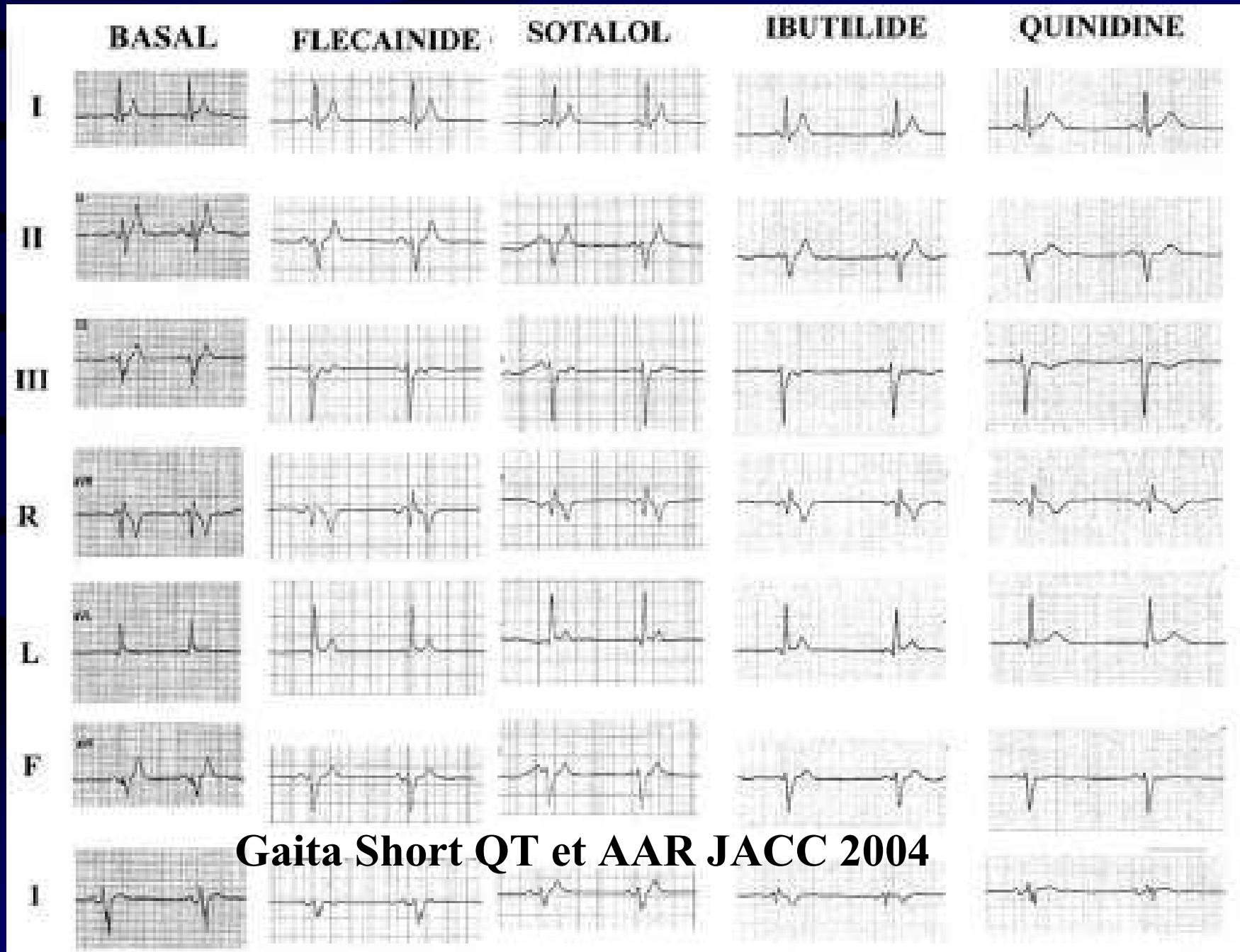


Figure 1. Twelve-lead surface ECG from patient I with congenital short QT syndrome. QT interval = 240 ms (QTc 275 ms).

QTc < 300 ms



* Gaita Circulation 2003; * Schimpf JCE 2003; * Brugada Circulation 2004

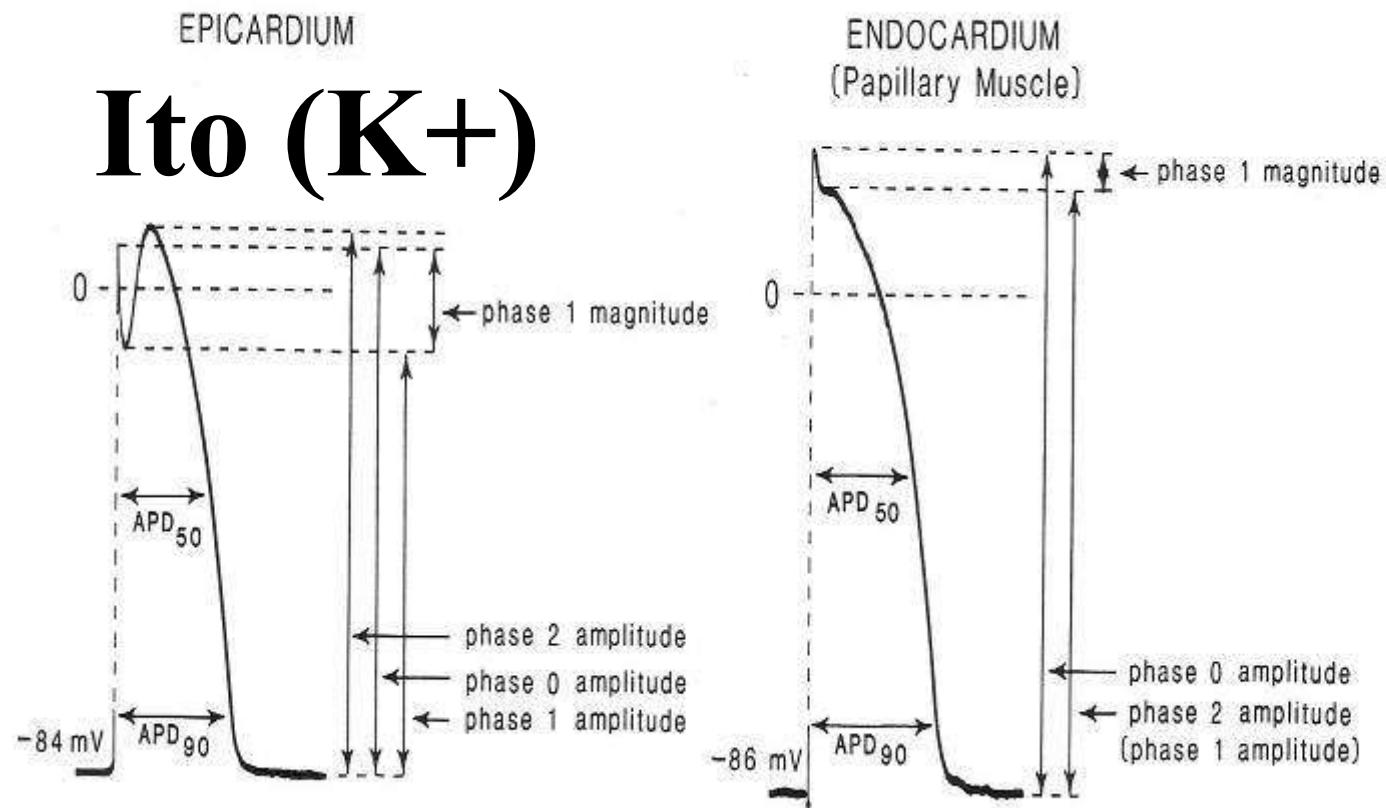


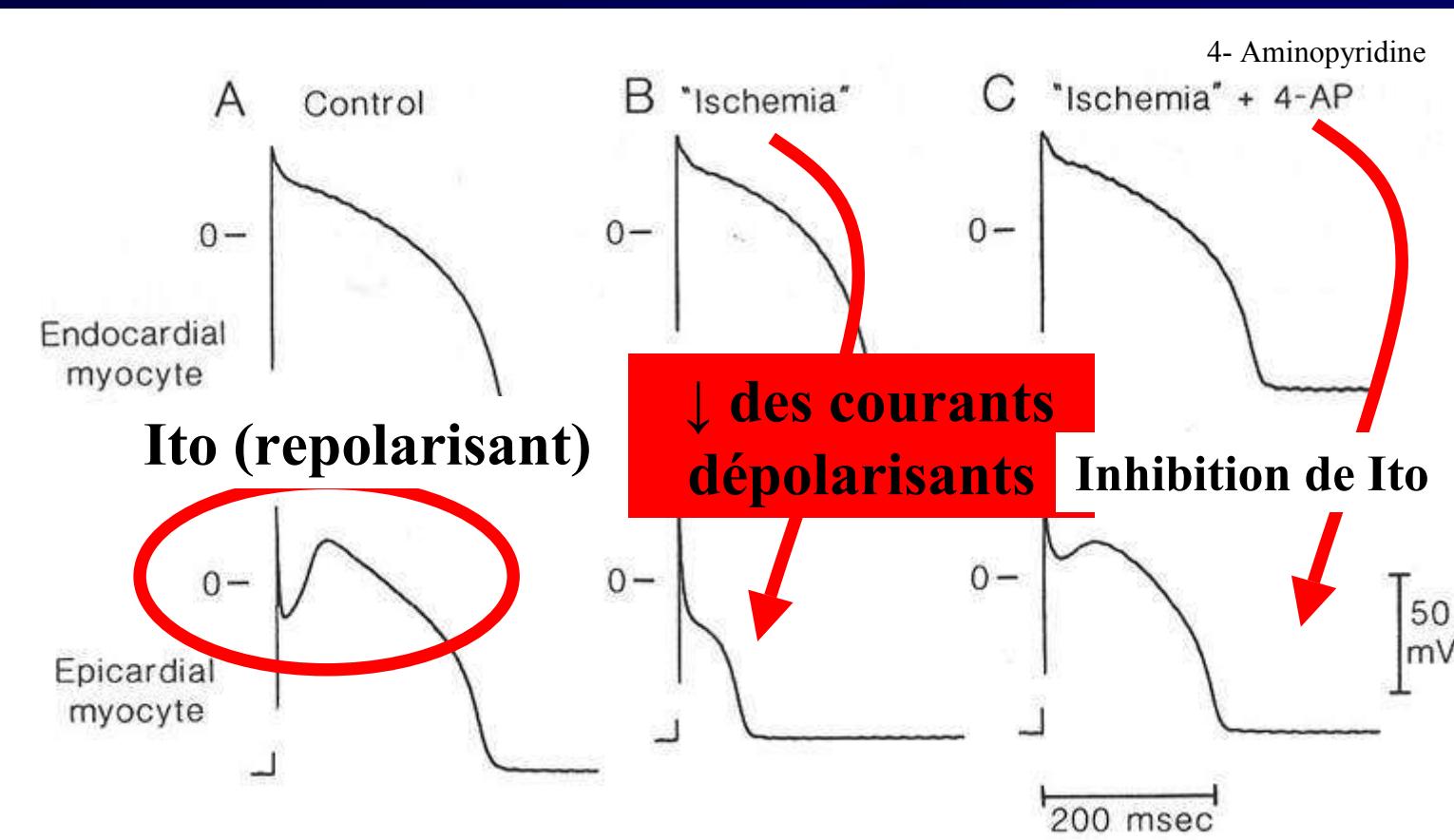
Gaita Short QT et AAR JACC 2004

ST Segment Elevation and Sudden Cardiac Death: From the Brugada Syndrome to Acute Myocardial Ischemia

GAN-XIN YAN, M.D., PH.D., and PETER R. KOWEY, M.D.

From the Lankenau Hospital and the Main Line Health Heart Center, Wynnewood,
and Jefferson Medical College, Philadelphia, Pennsylvania



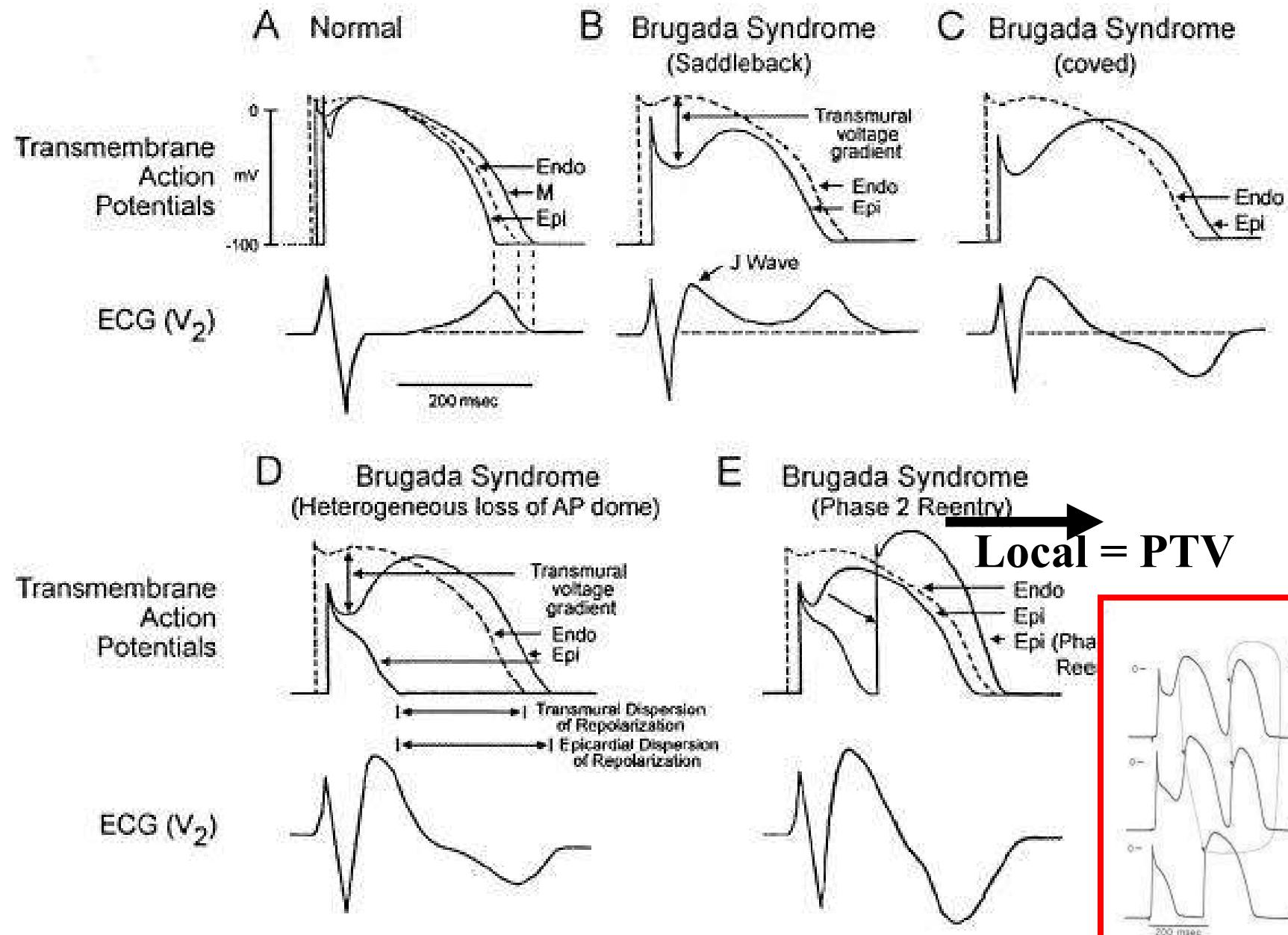


* Lukas Differences in the electrophysiological response of canine ventricular epicardium and endocardium to ischemia. Role of the transient outward current
Circulation 1993; 88: 2903

Syndrome de BRUGADA

Accélération de l'inactivation de iNa
Diminution du nombre des canaux Na⁺

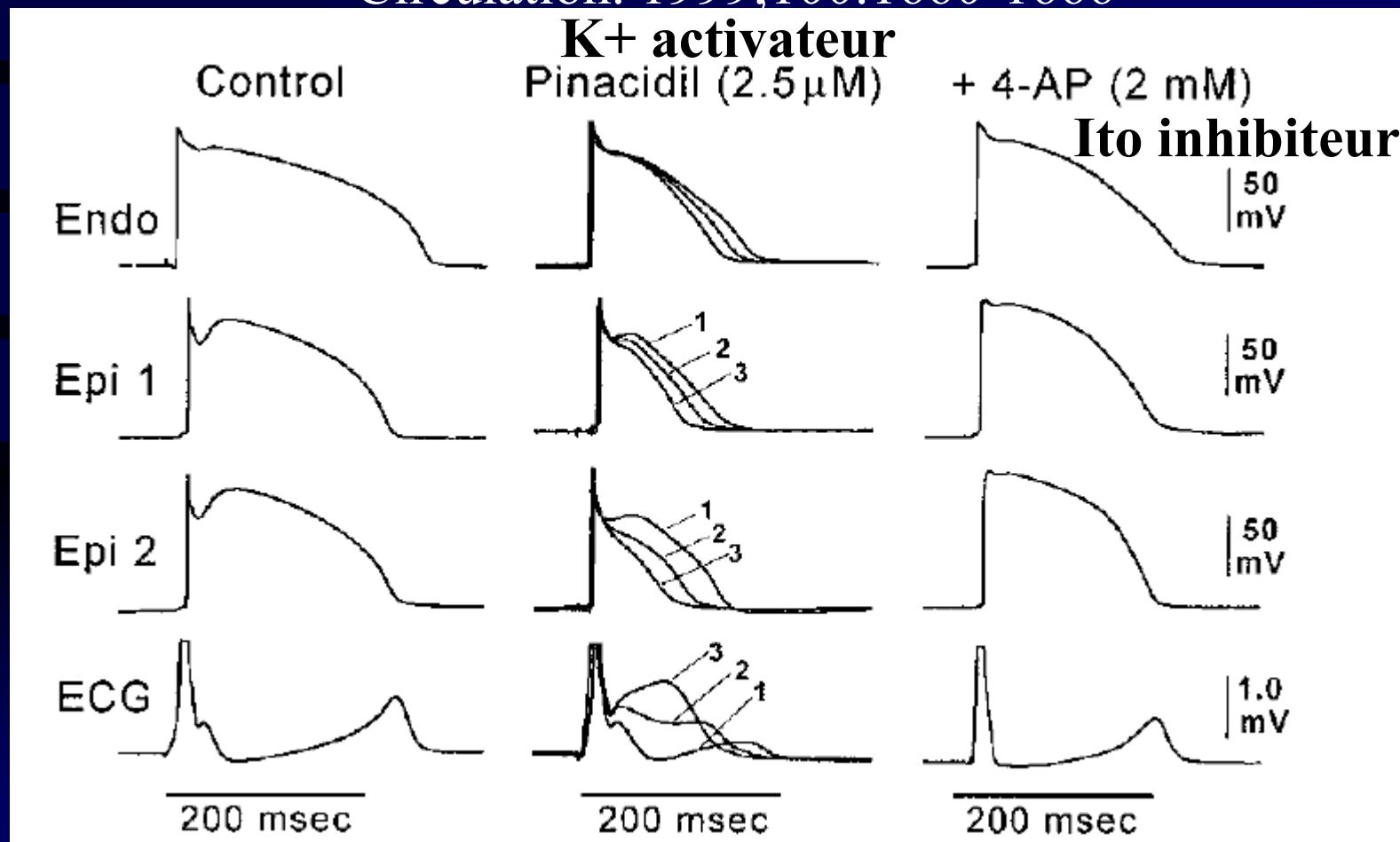
.....

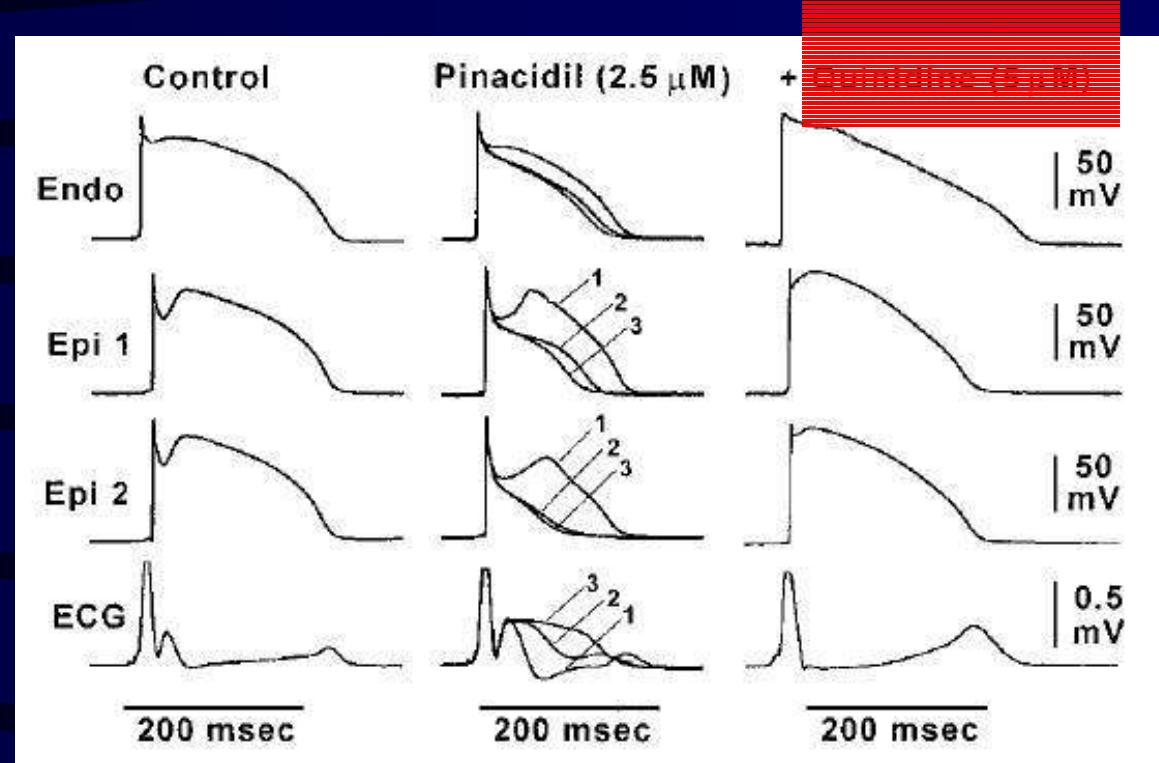


* Cellular Basis for the Brugada Syndrome and Other Mechanisms of Arrhythmogenesis Associated With ST-Segment Elevation

Gan-Xin Yan, MD, PhD; Charles Antzelevitch, PhD

Circulation. 1999;100:1660-1666



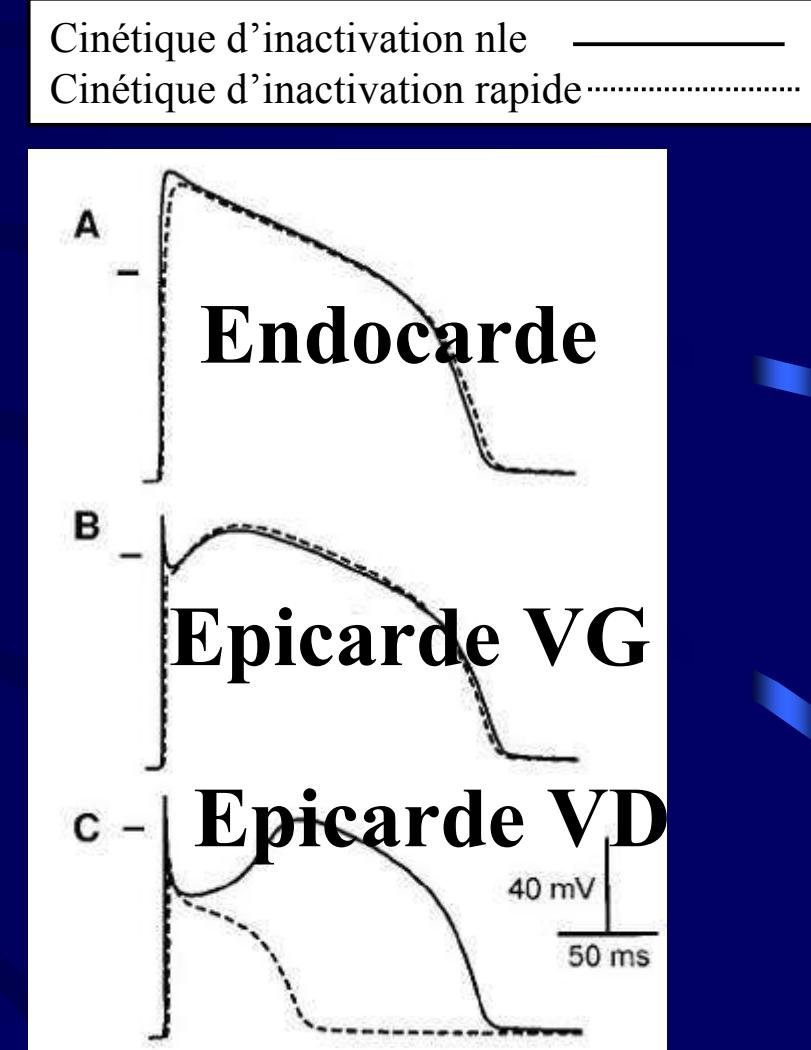
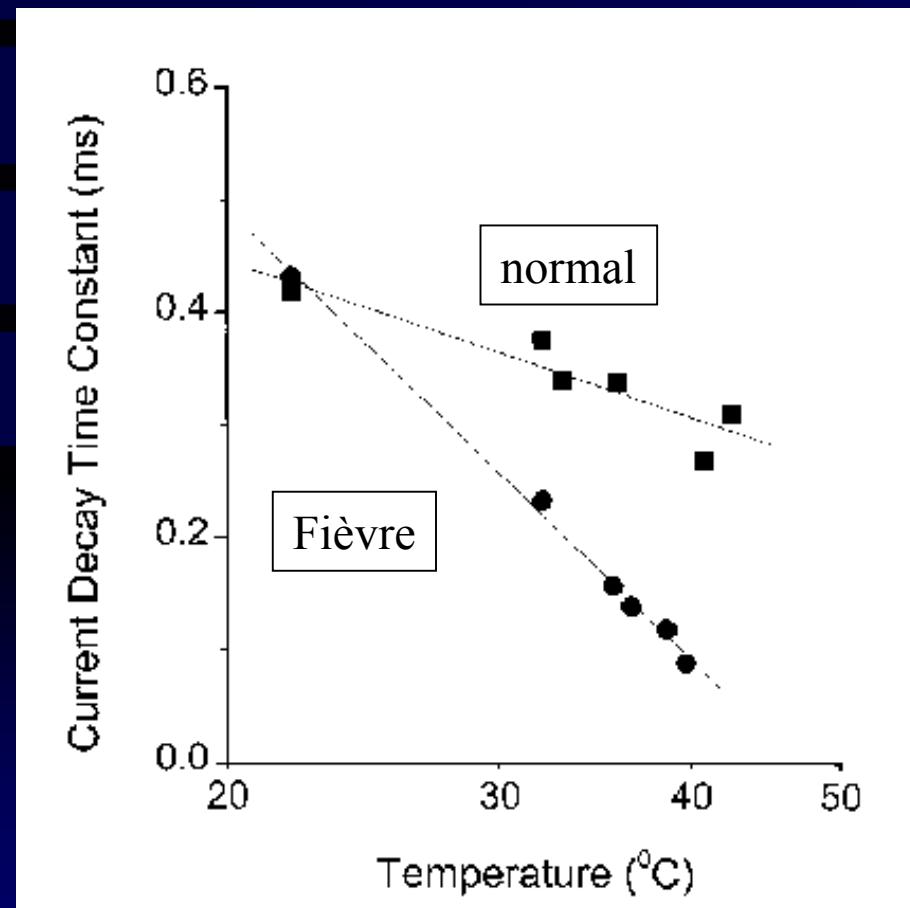


Belhassen B. Effects of electrophysiologic-guided therapy with **Class IA antiarrhythmic drugs** on the long-term outcome of patients with idiopathic ventricular fibrillation with or without the Brugada syndrome. J Cardiovasc Electrophysiol. 1999 Oct; 10(10): 1301-12.

Alings M, **Quinidine induced electrocardiographic normalization** in two patients with Brugada syndrome. Pacing Clin Electrophysiol. 2001 Sep; 24: 1420-2.

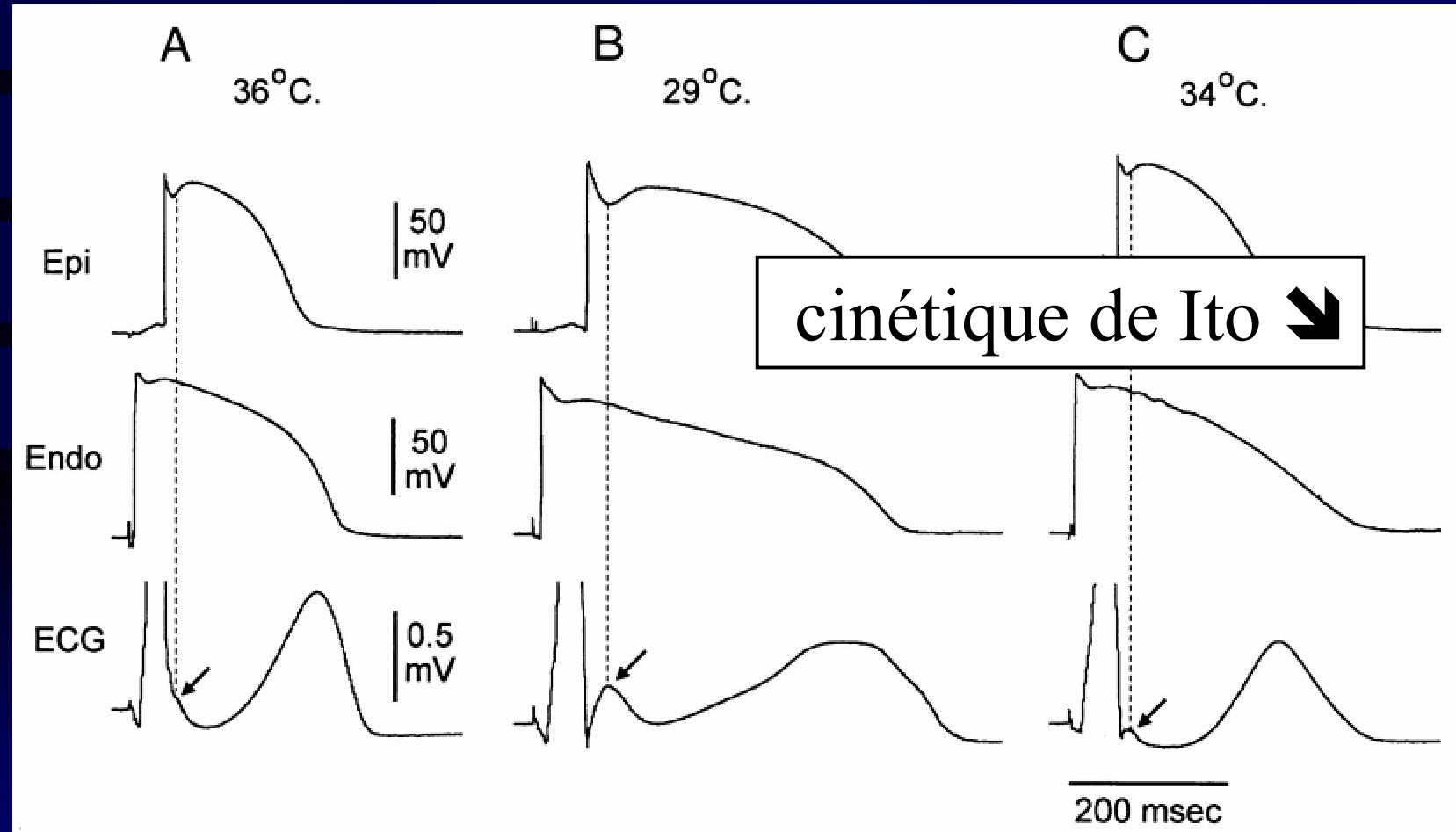
* Ionic Mechanisms Responsible for the Electrocardiographic Phenotype of the Brugada Syndrome Are Temperature Dependent

Robert Dumaine, Circ Res. 1999;85:803-809



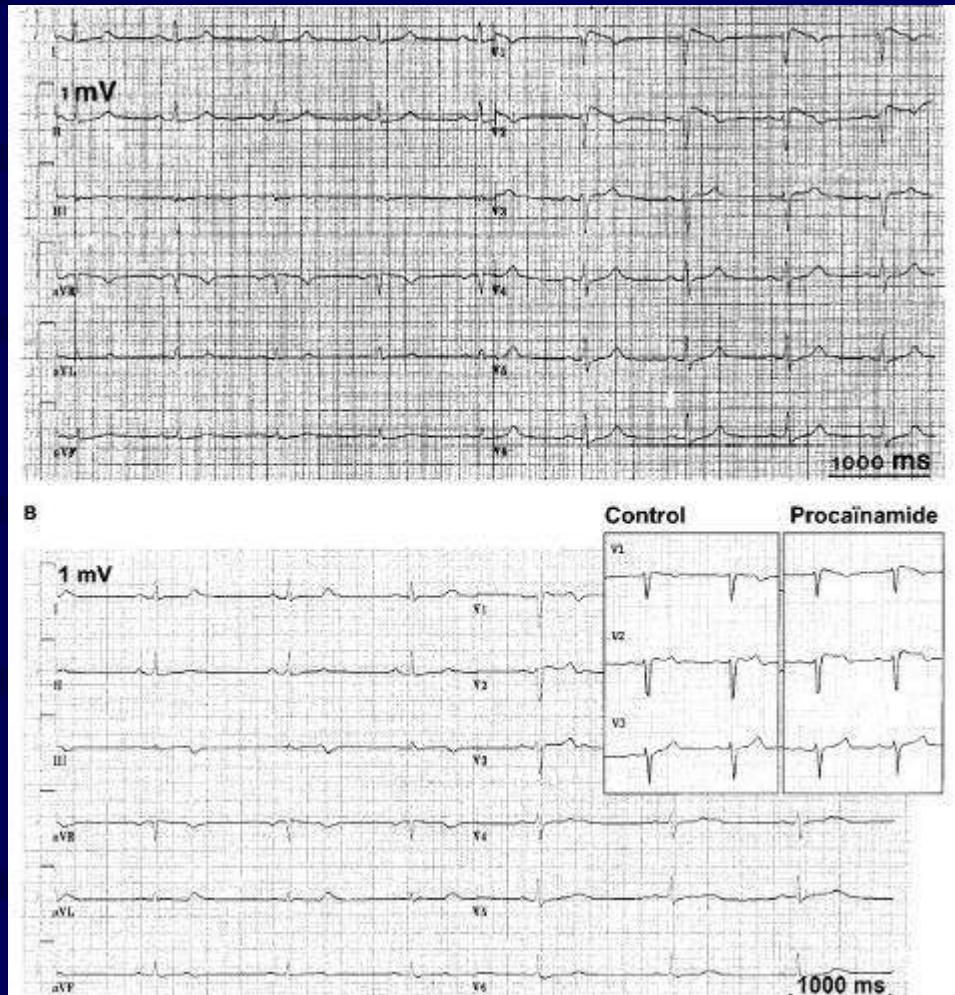
Cellular Basis for the Electrocardiographic J Wave

Gan-Xin Yan, MD, PhD; Charles Antzelevitch, PhD
Circulation. 1996;93:372-379



* A Single Na Channel Mutation Causing Both Long-QT and Brugada Syndromes. C Bezzina. *Circ Res.* 1999;85:1206-1213.

The Elusive Link Between LQT3 and Brugada Syndrome
The Role of Flecainide Challenge Silvia G. Priori, Circulation. 2000;102:945-947.

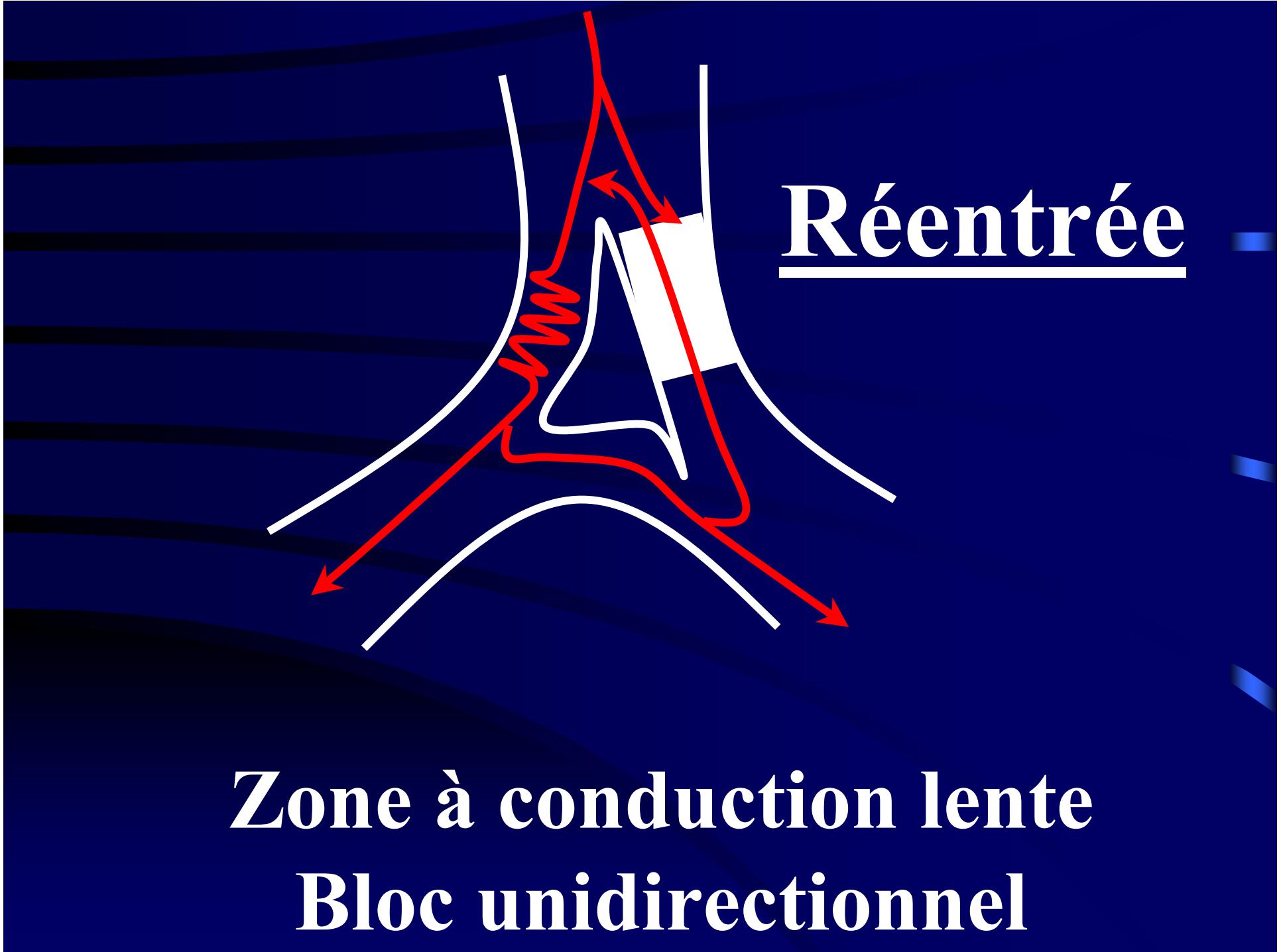


Conclusions—The data demonstrate that flecainide may induce ST segment elevation in LQT3 patients, raising concerns about the safety of flecainide therapy and demonstrating the existence of an intriguing overlap between LQT3 and BS.

Réentrées

□ Anatomique

□ Fonctionnelle (Polymorphique)
anisotropie
hétérogénéité des P Réfractaires



Leading Circle Model (Allessie, 1977)

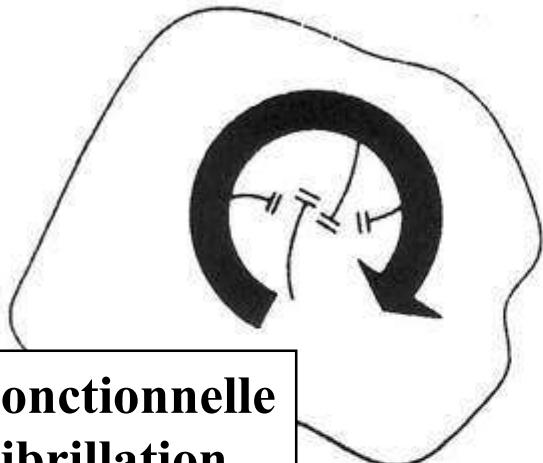
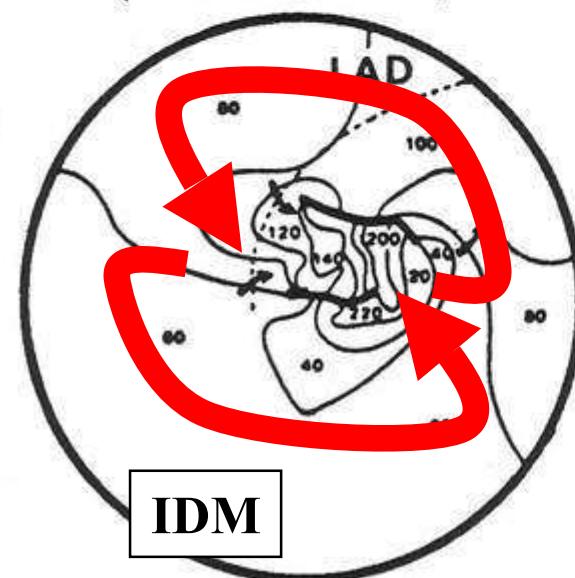
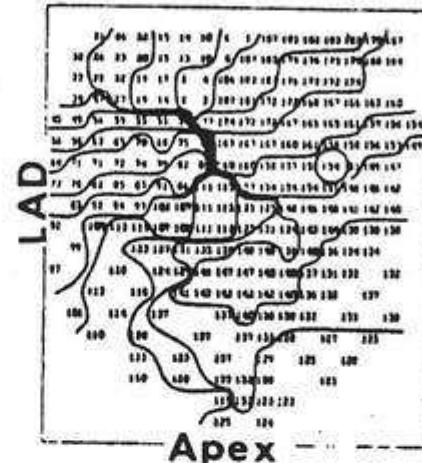


Figure 8 Model (El-Sherif, 1985)



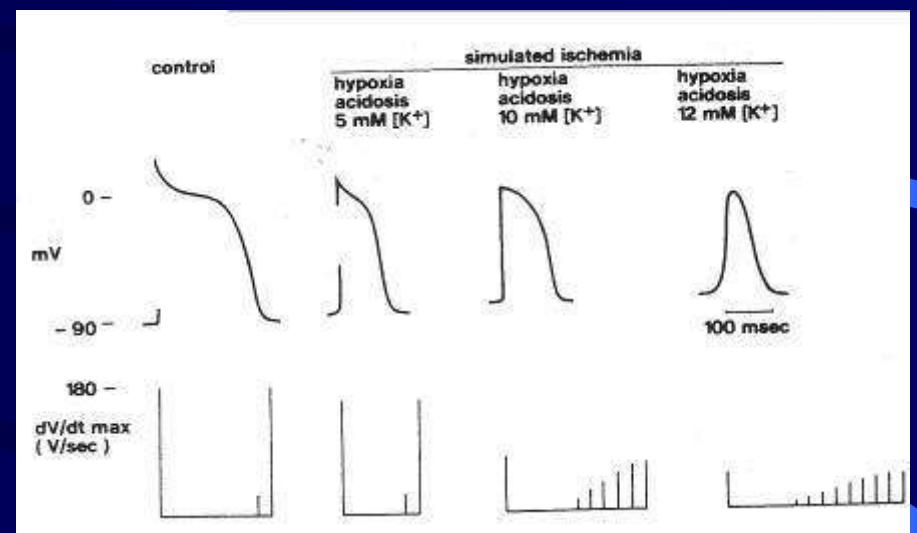
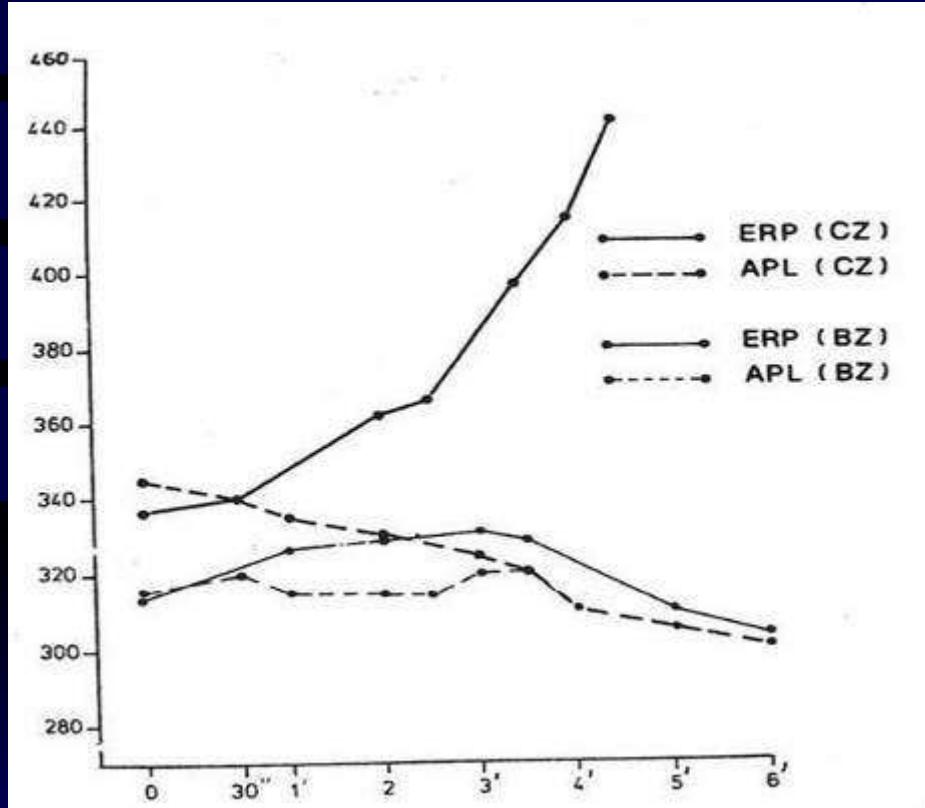
Anisotropic Model (Allessie, 1989)



Spiral Wave Model (Davidenko, 1992)

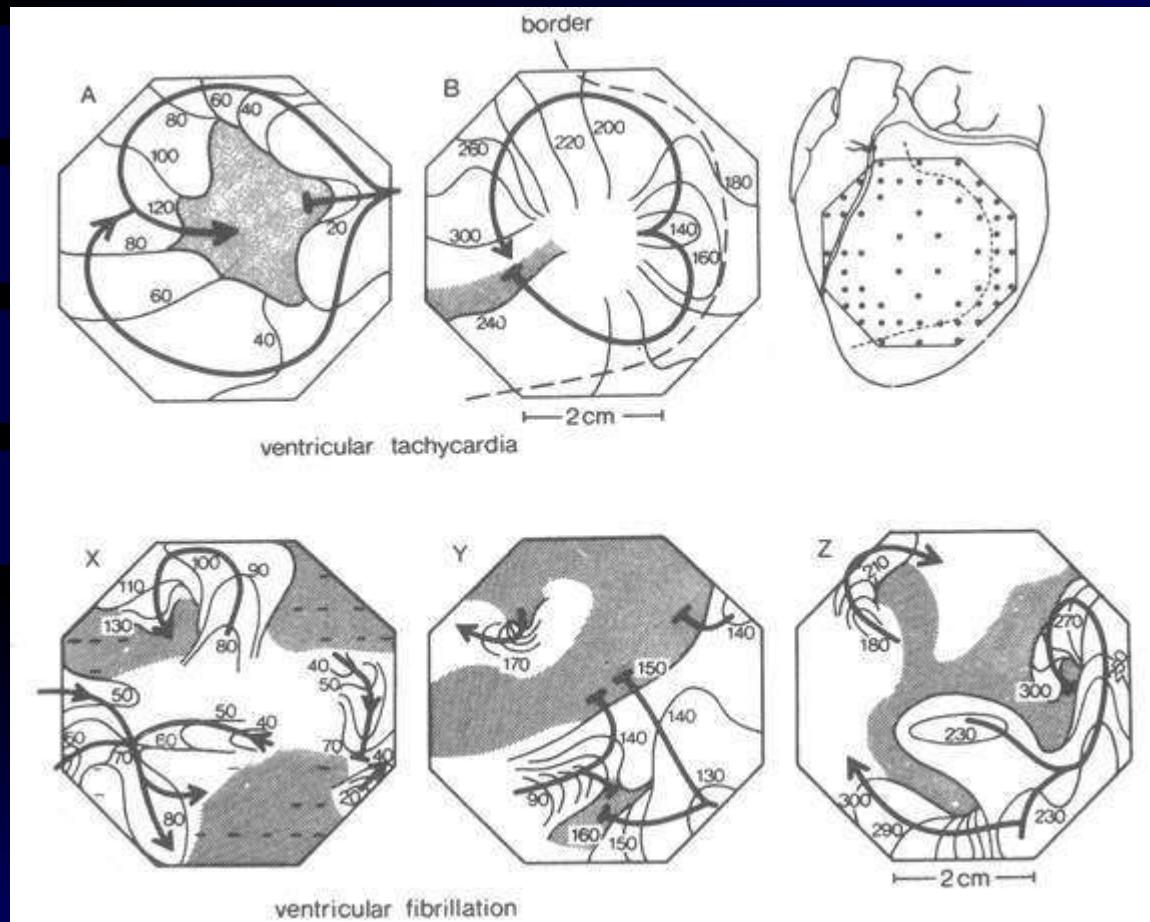


Réentrée pendant ischémie ERP s'allonge et APD diminue



Circulation 1980; 61:768.

Ischémie Myocardique



Mais AUSSI
Mécanismes focaux

* Arnar DO. Role of the Purkinje system in spontaneous ventricular tachycardia during acute ischemia in a canine model. Circulation. 1997; 96: 2421-9

Circ Res 1981; 49: 1069

Application Clinique

Successful Catheter Ablation of Electrical Storm After Myocardial Infarction

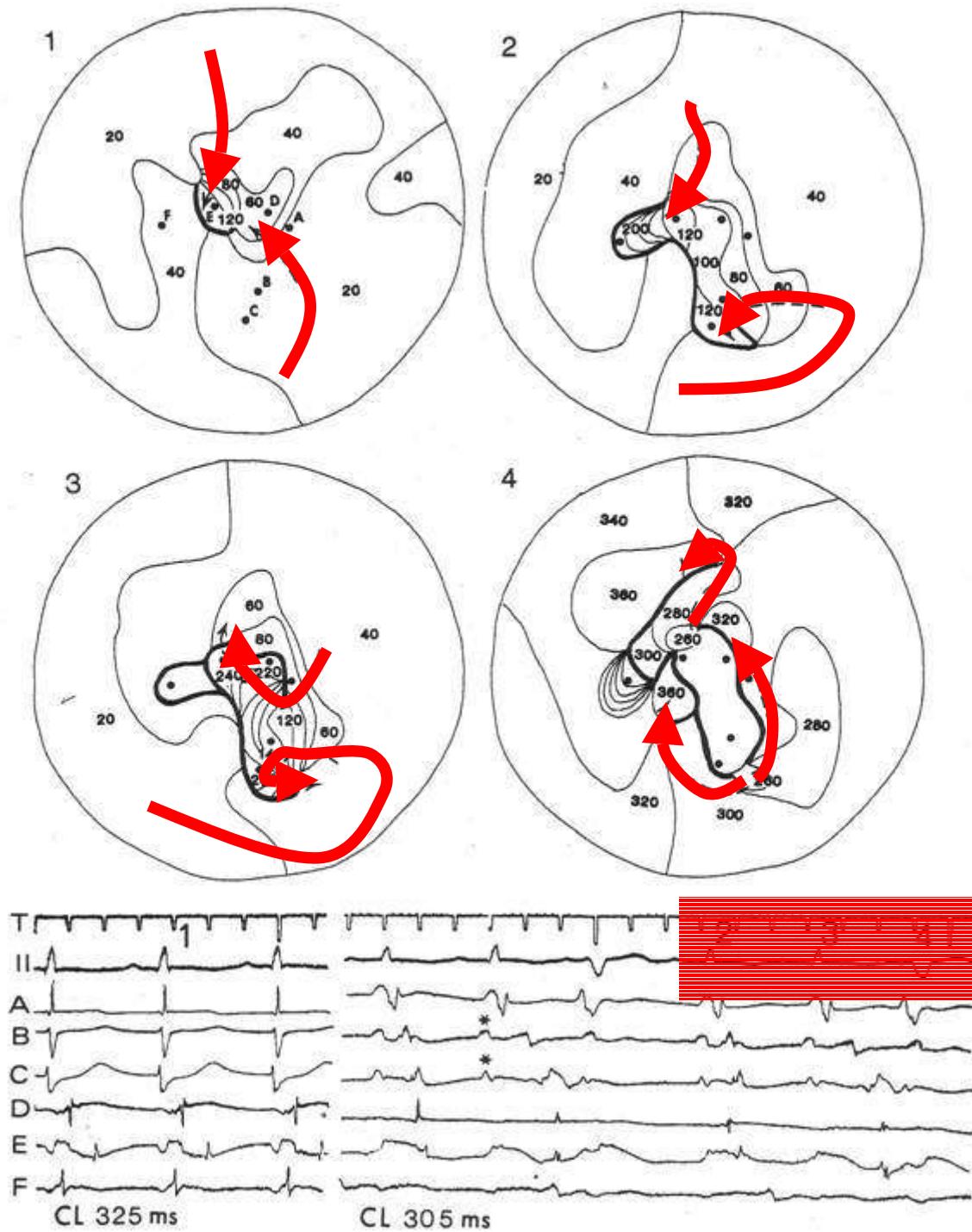
Dietmar Bänsch, MD*; Feifan Oyang, MD*; Matthias Antz, MD; Thomas Arentz, MD; Reinhold Weber, MD; Jesus E. Val-Mejias, MD; Sabine Ernst, MD; Karl-Heinz Kuck, MD

Background—We report on 4 patients (aged 57 to 77 years; 3 men) who developed drug-refractory, repetitive ventricular tachyarrhythmias after acute myocardial infarction (MI). All episodes of ventricular arrhythmias were triggered by monomorphic ventricular premature beats (VPBs) with a right bundle-branch block morphology (RBBB).

Methods and Results—Left ventricular (LV) mapping was performed to attempt radiofrequency (RF) ablation of the triggering VPBs. Activation mapping of the clinical VPBs demonstrated the earliest activation in the anteromedial LV in 1 patient and in the inferomedial LV in 2 patients. Short, high-frequency, low-amplitude potentials were recorded that preceded the onset of each extrasystole by a maximum of 126 to 160 ms. At the same site, a Purkinje potential was documented that preceded the onset of the QRS complex by 23 to 26 ms during sinus rhythm. In 1 patient, only pace mapping was attempted to identify areas of interest in the LV. Six to 30 RF applications abolished all local Purkinje potentials at the site of earliest activation and/or perfect pace mapping and suppressed VPBs in all patients. No episode of ventricular tachycardia or fibrillation has recurred for 33, 14, 6, and 5 months in patients 1, 2, 3, and 4, respectively.

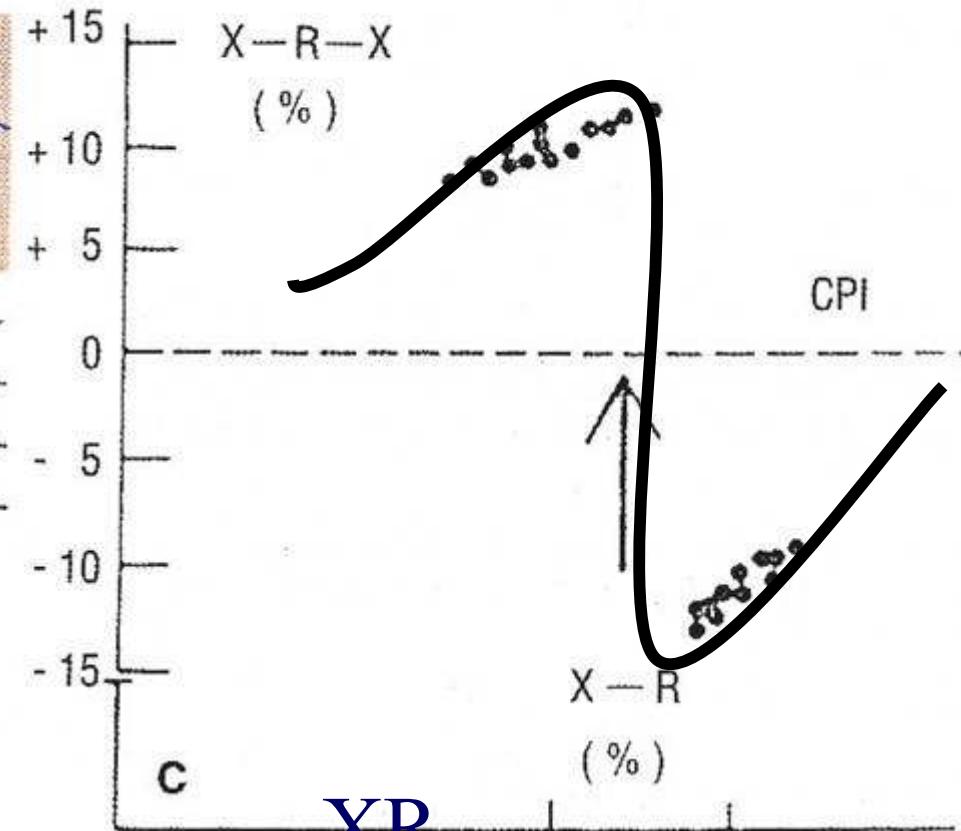
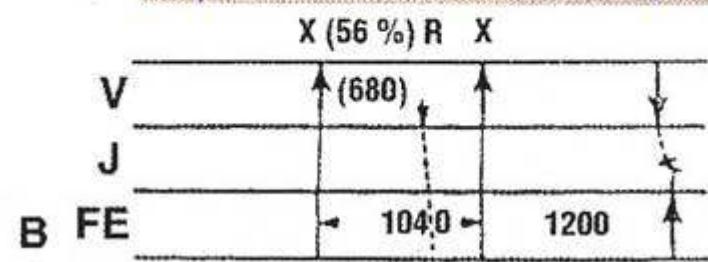
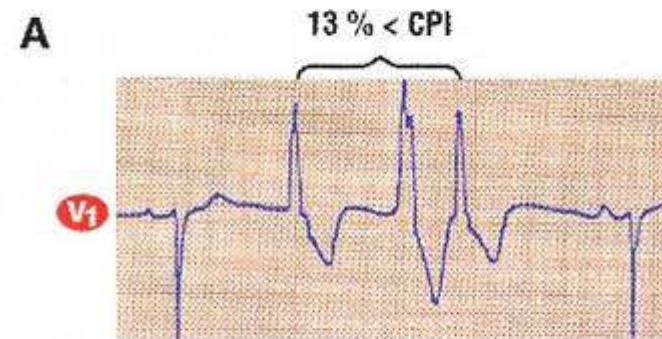
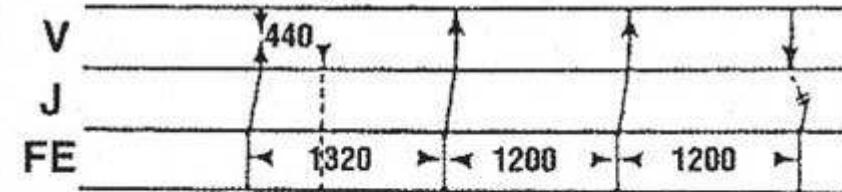
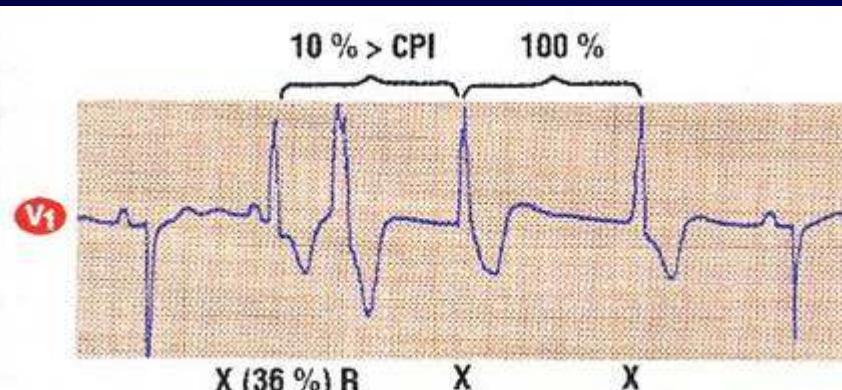
Conclusions—Recurrent ventricular tachyarrhythmias after MI may be triggered by VPBs. RF ablation of the triggering VPBs may be successful and safe. These findings support the hypothesis that catheter ablation of the Purkinje system and VPBs may be used as a bailout therapy in these patients. (Circulation. 2002;105:3011-3016.)

Key Words: fibrillation ■ myocardial infarction ■ ablation ■ tachycardia



Trigéminisme
Bigéminisme
=
Reentrée
?

Modulation Electrotonique

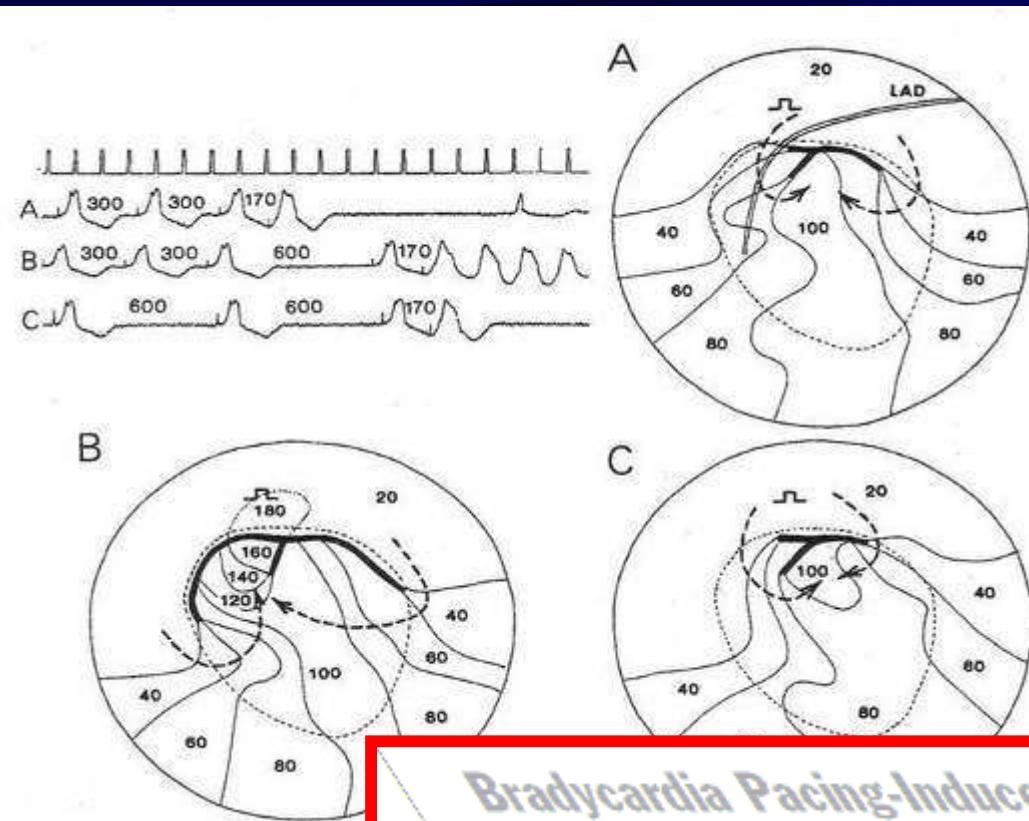




This is a summary of the abstract of the article. This is a summary of the abstract of the article.

Background: The rule of bigeminy is commonly explained by a reentrant mechanism.

Cycle long – Cycle court

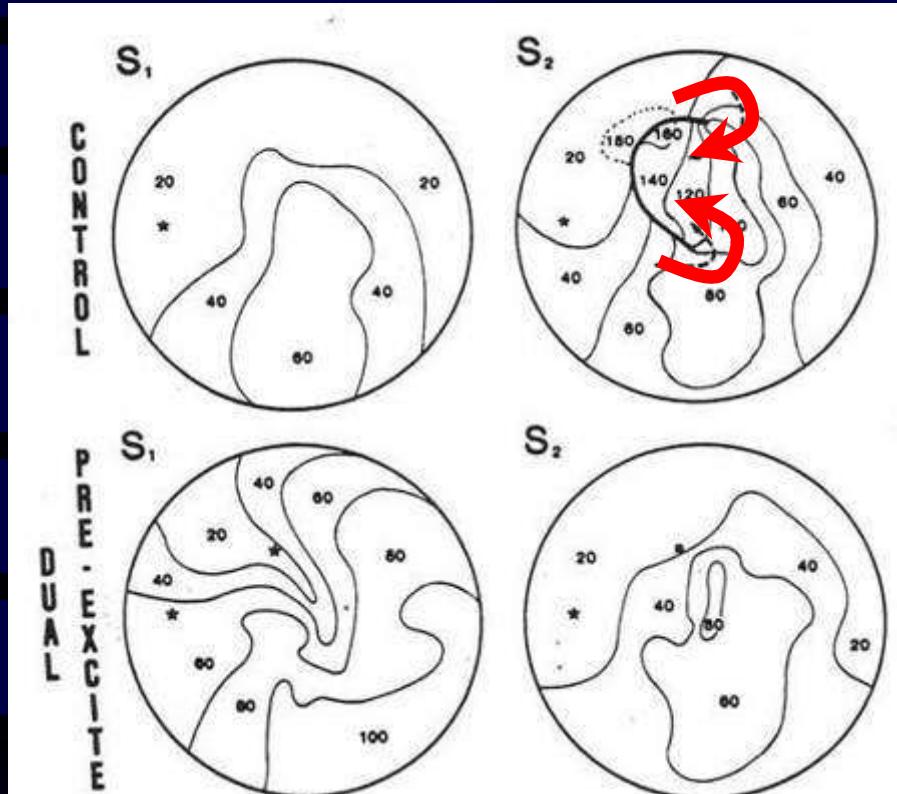


**Bradycardia Pacing-Induced
Long-Short Sequences at the Onset of Ventricular Tachyarrhythmia? A Possible Mechanism of Proarrhythmia?**

Michael Delibes, Michael J. Ohman, Paul D. Tracy, Michael Delibes, Michael J. Ohman, Paul D. Tracy, James W. Johnson, Michael J. Ohman, Paul D. Tracy

(J Am Coll Cardiol 2007;50:614-22)

Stimulation Biventriculaire



?

* Biventricular Pacing Decreases the Inducibility of Ventricular Tachycardia in Patients With Ischemic Cardiomyopathy J D . Zagrodzky Am J Cardiol 2001; 87: 1208 1210

* Effect of Epicardial or Biventricular Pacing to Prolong QT Interval and Increase Transmural Dispersion of Repolarization. Does Resynchronization Therapy Pose a Risk for Patients Predisposed to Long QT or Torsade de Pointes? VA. Medina-Ravell, Circulation 2003; 10:740-46.

* Increase in VT frequency after biventricular ICD upgrade JCE 2003; 14: 1245-47.

14.12.04 11:20:42

InSync III 8042 14.12.04 11:27:40

Test terminé

VITESSE 12.5 mm/s

VITESSE 12.5 mm/s

Test temporaire - VD

VVI, 90 min-□

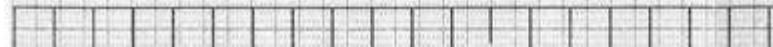
ECG DER. III 0.2 mV/mm

ECG DER. VD 0.2 mV/mm



MARQUEUR CANAL

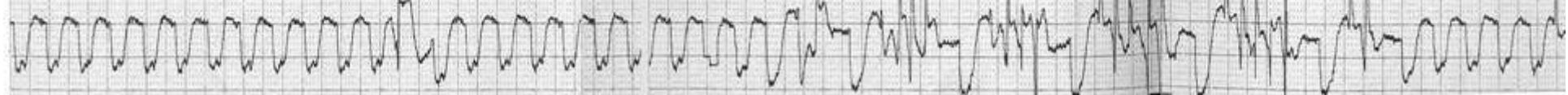
MARQUEUR CANAL



EGM VD 0.5 mV/mm



EGM VD 0.5 mV/mm



Diagnostic de Réentrée ?

RR = 370

RR = 320

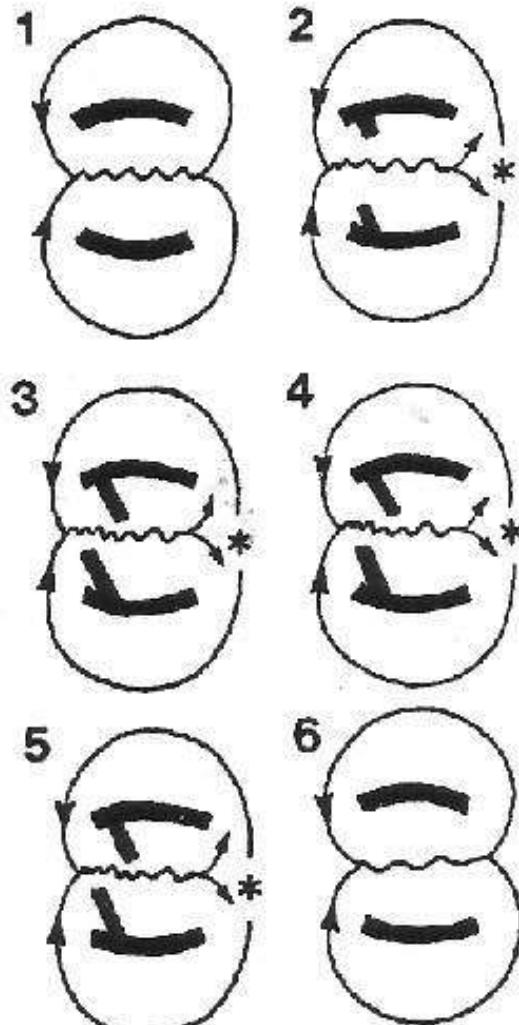


Entraînement
=
Réentrée

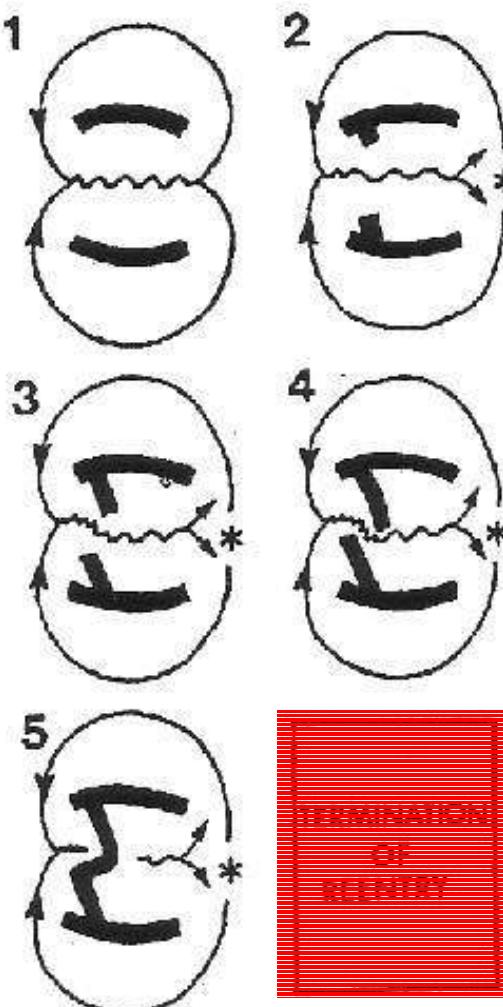
Foyer non protégé

Foyer
protégé

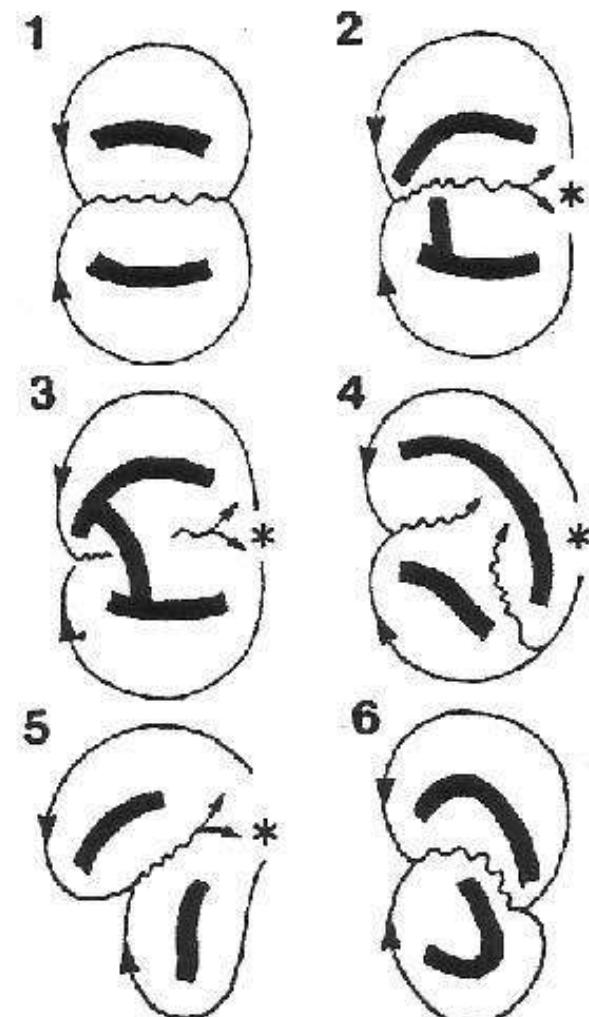
ENTRAINMENT
= Réentrée



TERMINATION

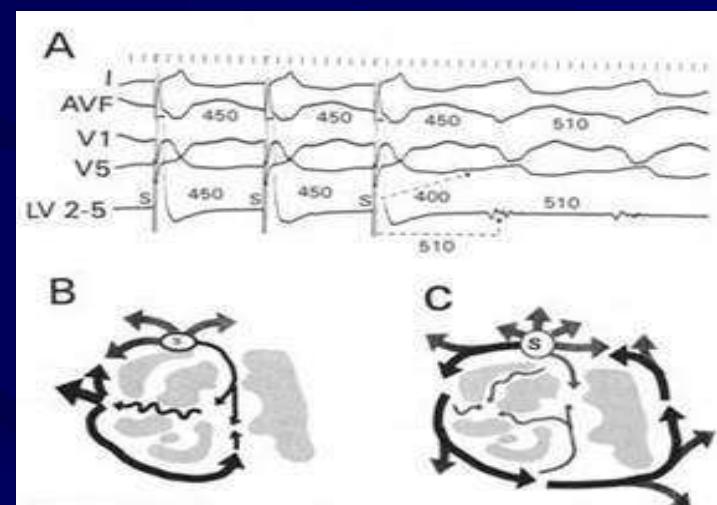
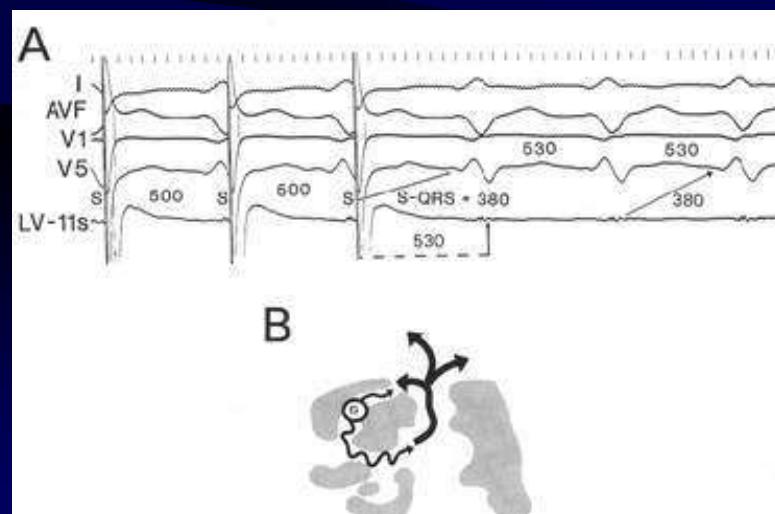
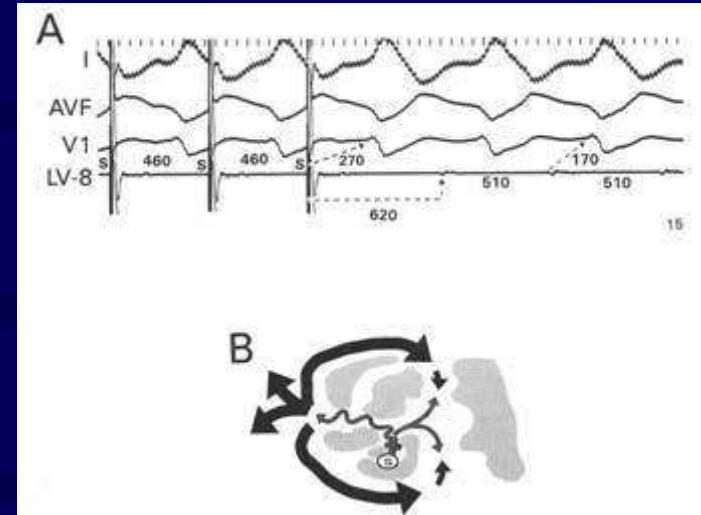
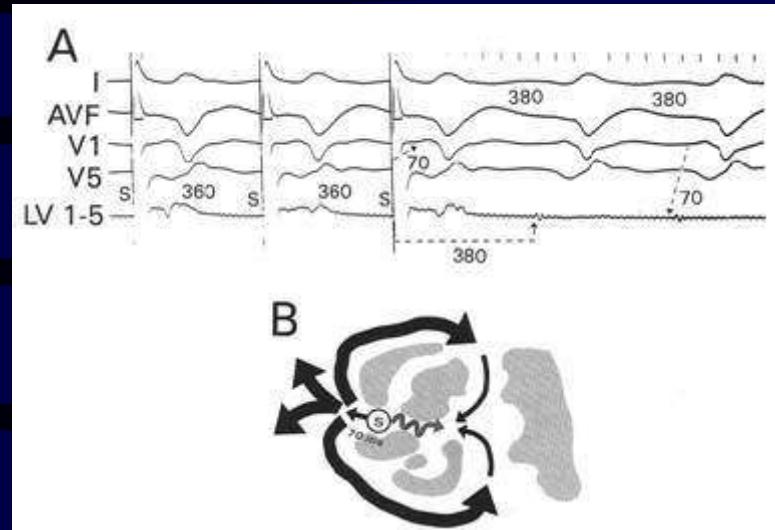


ACCELERATION

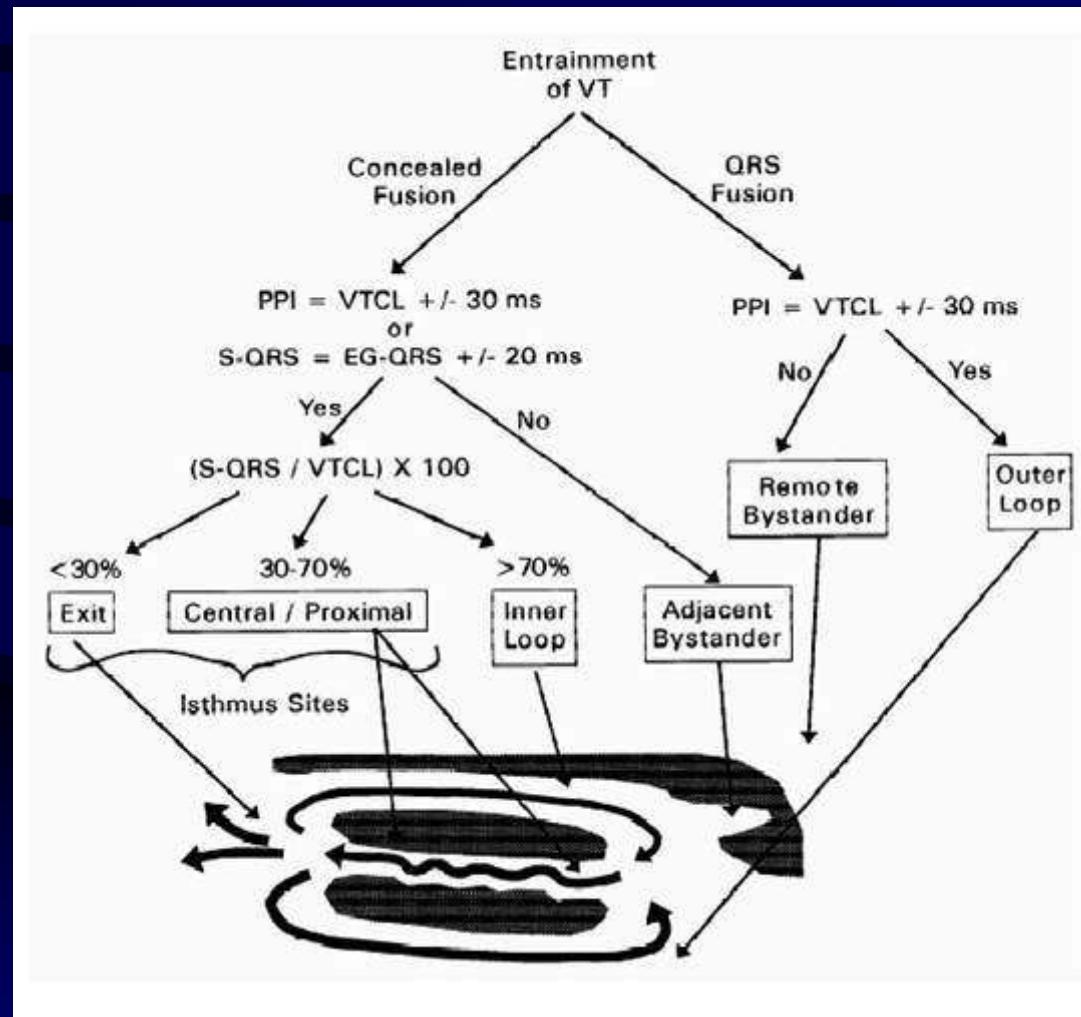


TERMINATION
OR
REENTRY

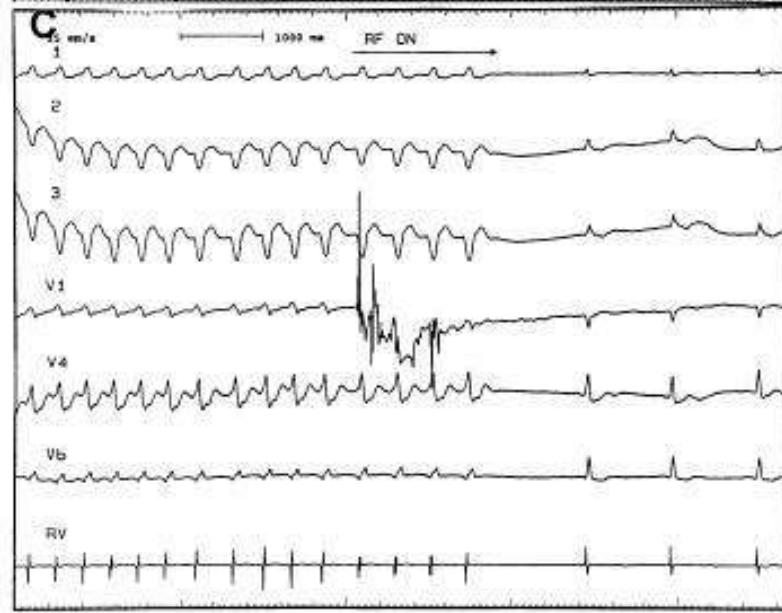
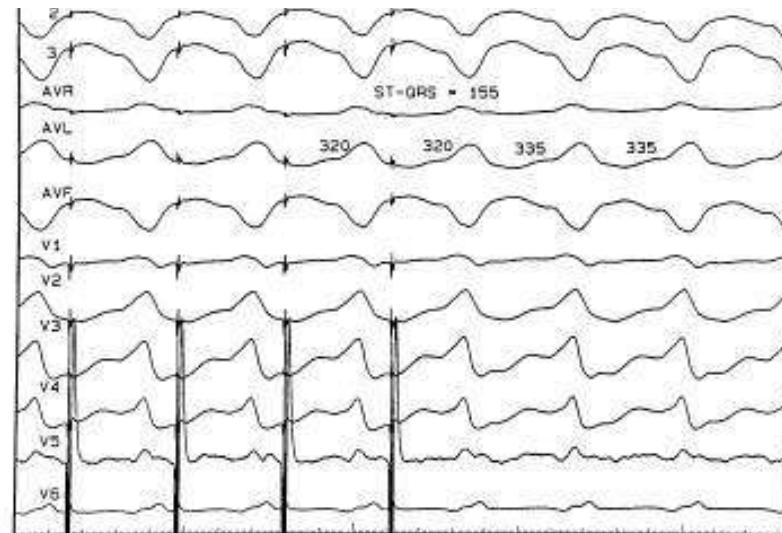
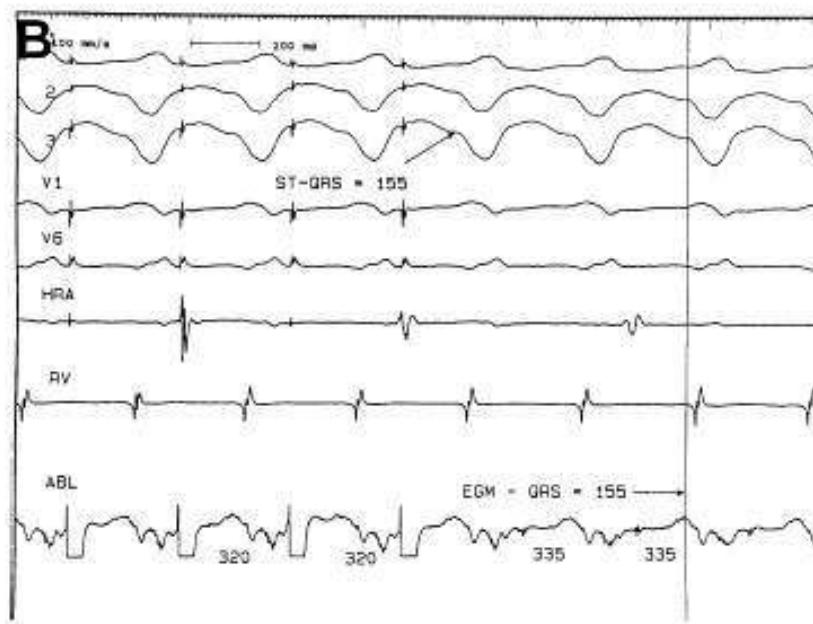
* Exploring Postinfarction Reentrant Ventricular Tachycardia With
Entrainment Mapping W. G. STEVENSON, MD, J Am Coll Cardiol 1997;29:1180–9



* Entrainment Mapping and Radiofrequency Catheter Ablation of
Ventricular Tachycardia in Right Ventricular Dysplasia
K E ELLISON, JACC; 1998; 32: 724.



PERFECT ENTRAINMENT MAP OF VT



* Electrophysiology of VT . Josephson JCE 2003;14 : 1133

Topostimulation
Post pacing interval
Potentiel diastolique Pot-QRS
Delai spike-QRS

TABLE 18-1. The Nature, Time Course, Mechanisms, Site of Origin, and Response to Drugs of Ventricular Arrhythmias After Coronary Artery Occlusion

	Phase I	Phase II	Phase III
Nature	VT/VF	VT	VT/VF
Time course	15–30 min	6–72 h	3–12 d
Site of origin	Ischemic myocardial cells Purkinje fibers? Normal zone bordering the ischemic zone	Subendocardial Purkinje fibers in infarct zone Subepicardial muscle overlying the infarct	Subepicardial muscle cells overlying the infarct Surviving intramural muscle cells Purkinje fibers?
Mechanism(s)	Reentry Automaticity (early after depolarization)?	Abnormal automaticity Triggered automaticity Reentry?	Reentry Triggered automaticity?
Response to drugs	Usually resistant	Usually suppressed	Usually resistant

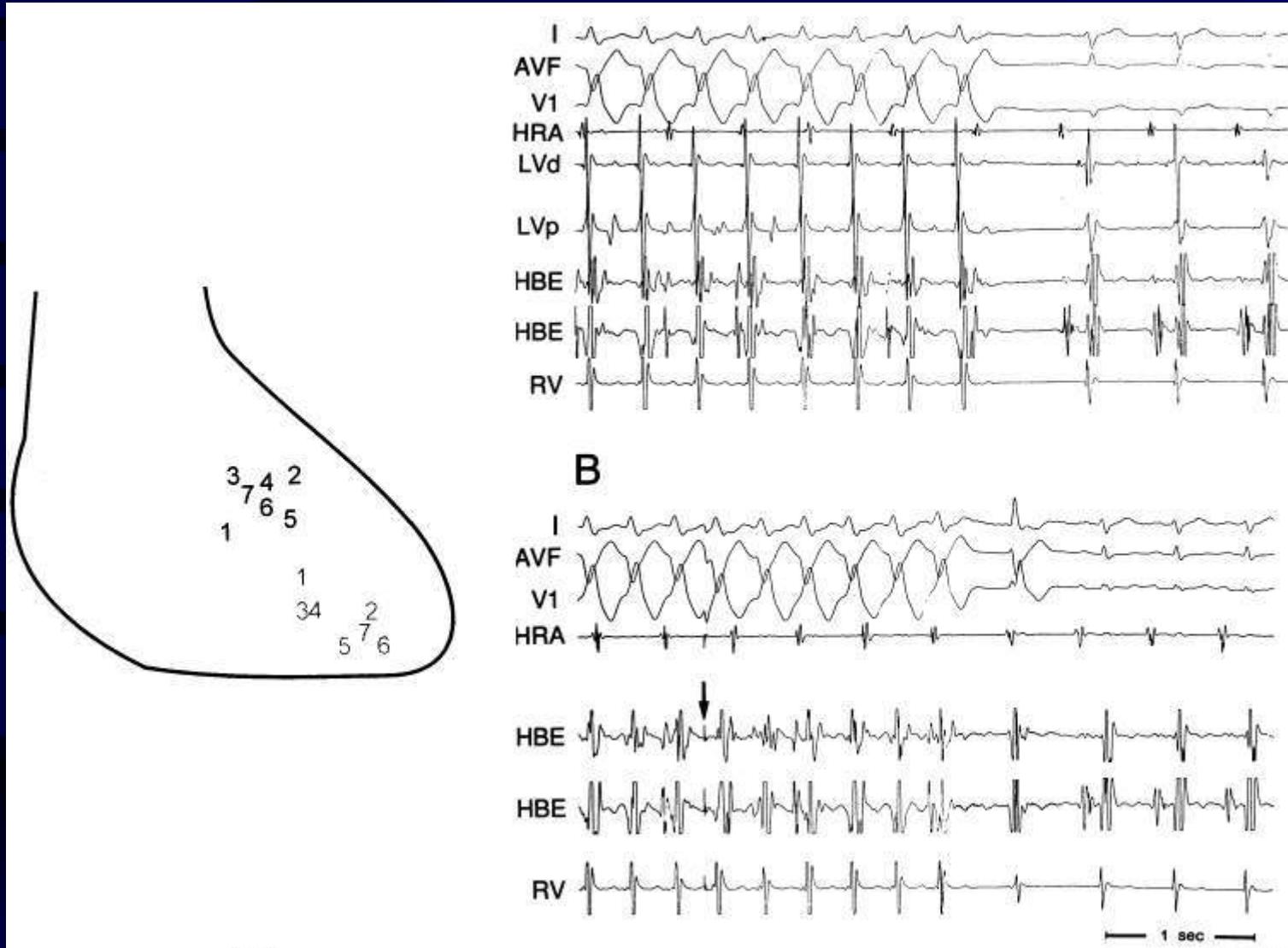
VT, ventricular tachycardia; VF, ventricular fibrillation, ?, evidence uncertain. Time course refers to postocclusion period.

Réentrée

- TV sur cicatrice d'IDM
- DVDA
- CMD (branche à branche)
- TV Fasciculaire



Réentrée - Automatique ????



* Successful Radiofrequency Ablation of Idiopathic Left Ventricular Tachycardia at a Site Away From the Tachycardia Exit
 MS WEN, J Am Coll Cardiol 1997;30:1024 –31

CMD non ischémique

Réentrée - Foyers

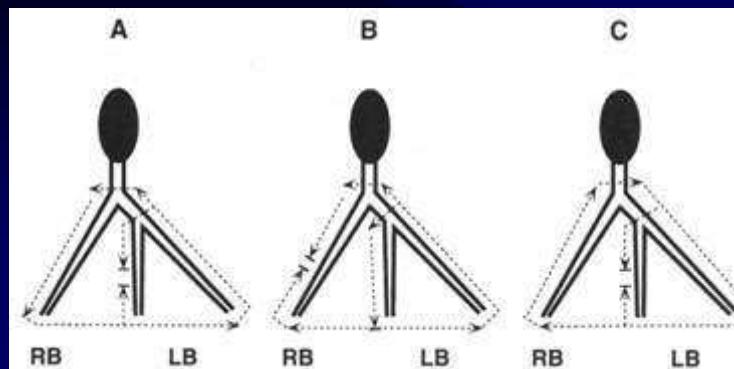
- Characterization of Endocardial Electrophysiological Substrate in Patients With Nonischemic Cardiomyopathy and Monomorphic Ventricular Tachycardia. Henry H. Hsia, *Circulation*. 2003;108:704-710. **Réentrée**
- Mechanisms Underlying Spontaneous and Induced Ventricular Arrhythmias in Patients With Idiopathic Dilated Cardiomyopathy Steven M. Pogwizd, *Circulation*. 1998;98:2404-2414. **Foyers**
- Dispersion des durée de PA - Dispersion des PR Antzelevitch JCE 2003 13 1259-72

Réentrée de branche à branche

Table 1

Diagnostic Criteria for Bundle Branch Reentry Ventricular Tachycardia

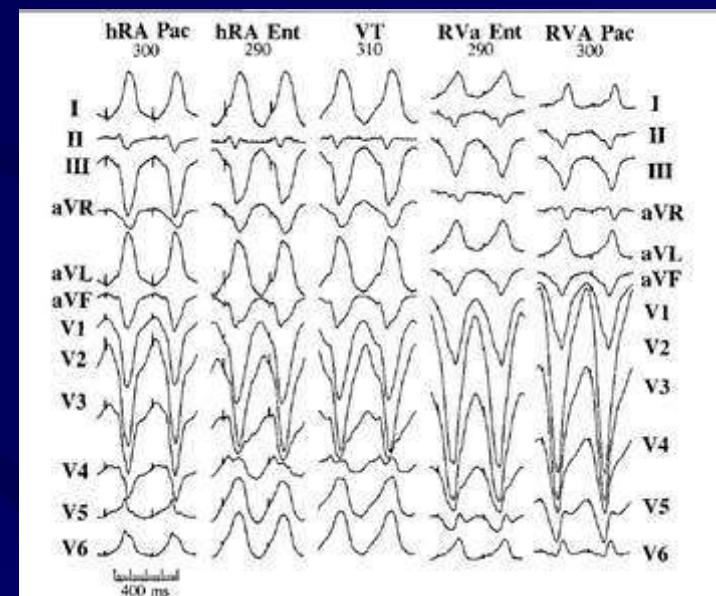
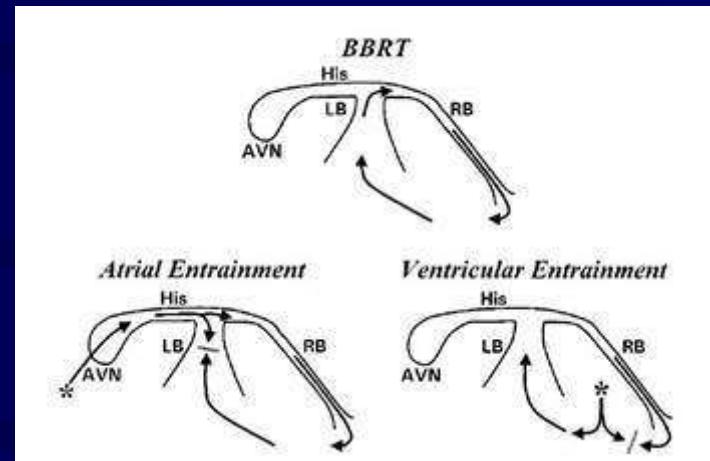
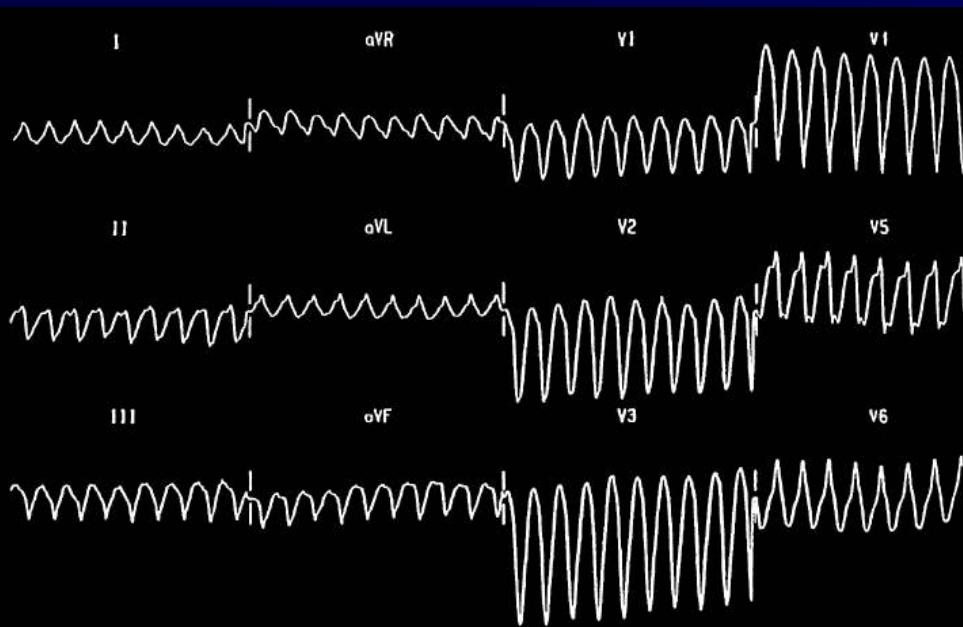
1. Prolonged HV interval during sinus rhythm (> 60 milliseconds) and usually somewhat longer during tachycardia. This finding (HV interval prolongation during tachycardia) has been described as the recording site being "more distal" than the site where the impulse turns around to descend the contralateral bundle branch.
2. Spontaneous variation in ventricle-to-ventricle (VV) intervals usually seen at initiation of tachycardia is preceded, rather than followed, by similar changes in H-H or RB-RB intervals.
3. Similar His-to-ventricle (HV) relations during tachycardia even if different programmed stimulation method was used for induction.
4. Single or multiple premature ventricular beats especially using a short-to-long sequence in the basic drive cycle length can usually induce the tachycardia. The first beat of tachycardia and all subsequent beats are preceded by H or RB potentials.
5. Resetting of tachycardia with a single premature ventricular beat by advancing the His or RB electrogram, indicative of association of HPS with the reentrant circuit of tachycardia.
6. Surface QRS morphology, as well as activation sequence of intracardiac electrograms, must be consistent with depolarization of the ventricle through 1 of the bundle branches.
7. Termination of tachycardia with retrograde conduction block within the HPS (V with no His or RB electrogram).
8. Absence of consistent H or RB deflection between QRS complexes during ventricular pacing at the cycle length of tachycardia.



* Li JCE 2002. BB
reentrant VT role of HP
system: Le HV long
n'est pas un pré
requis.....

Réentrée de branche à branche

Entraînement caché

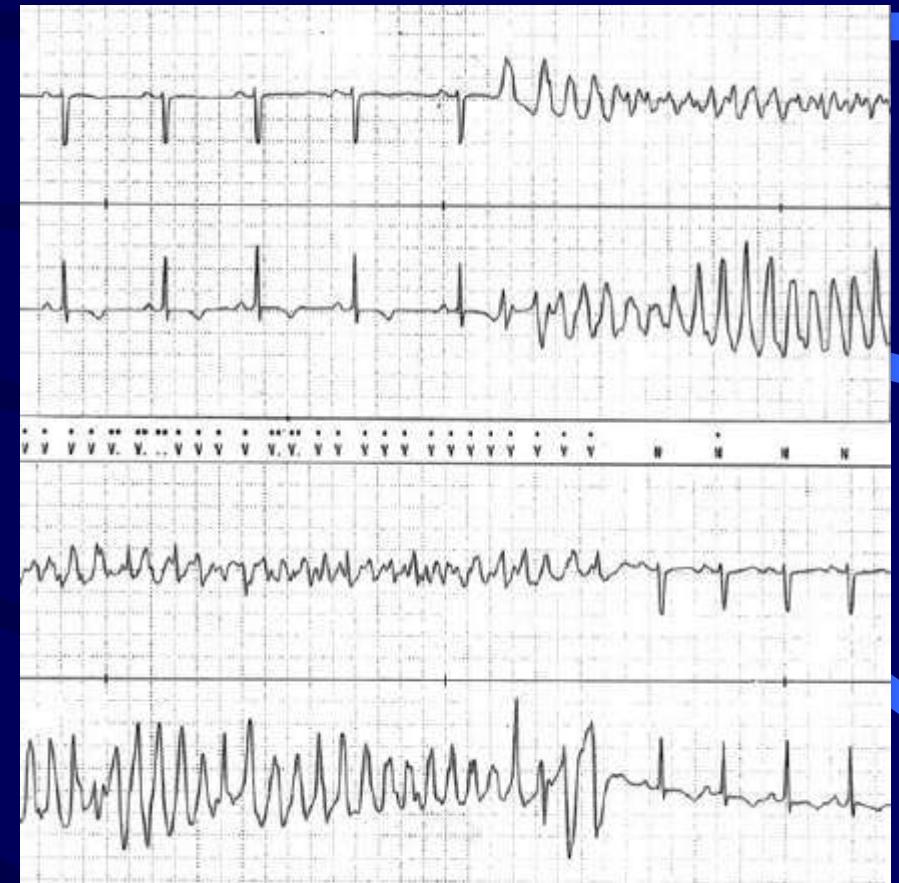
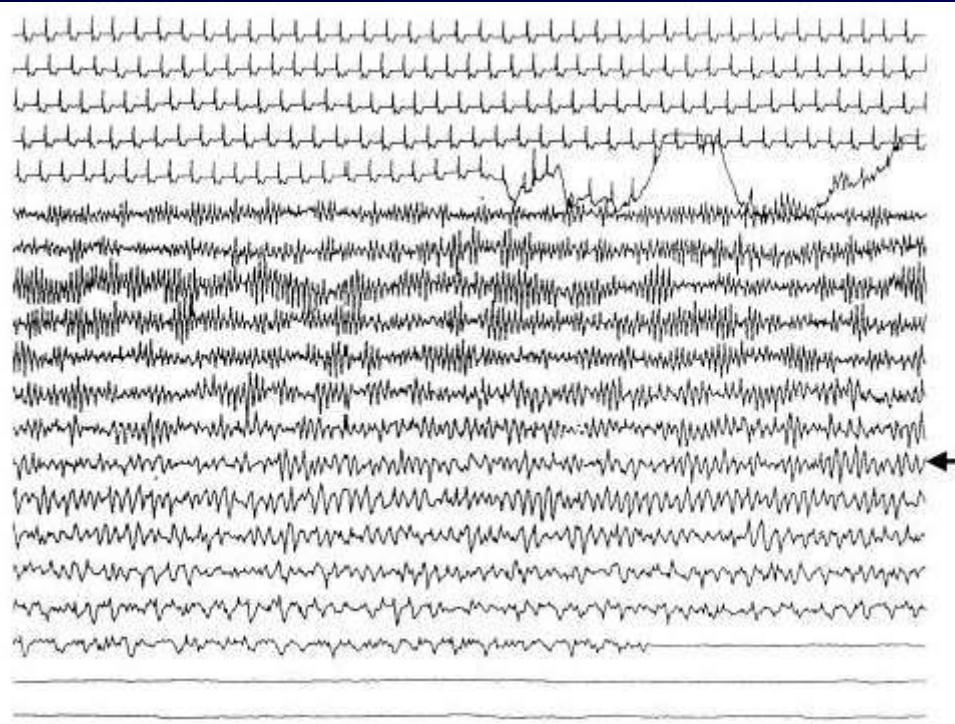


* Transient Entrainment of Bundle-Branch Reentry by Atrial and Ventricular Stimulation *Circulation*. 1999;100:1784-1790.

Réentrée de branche à branche



Fibrillation Ventriculaire

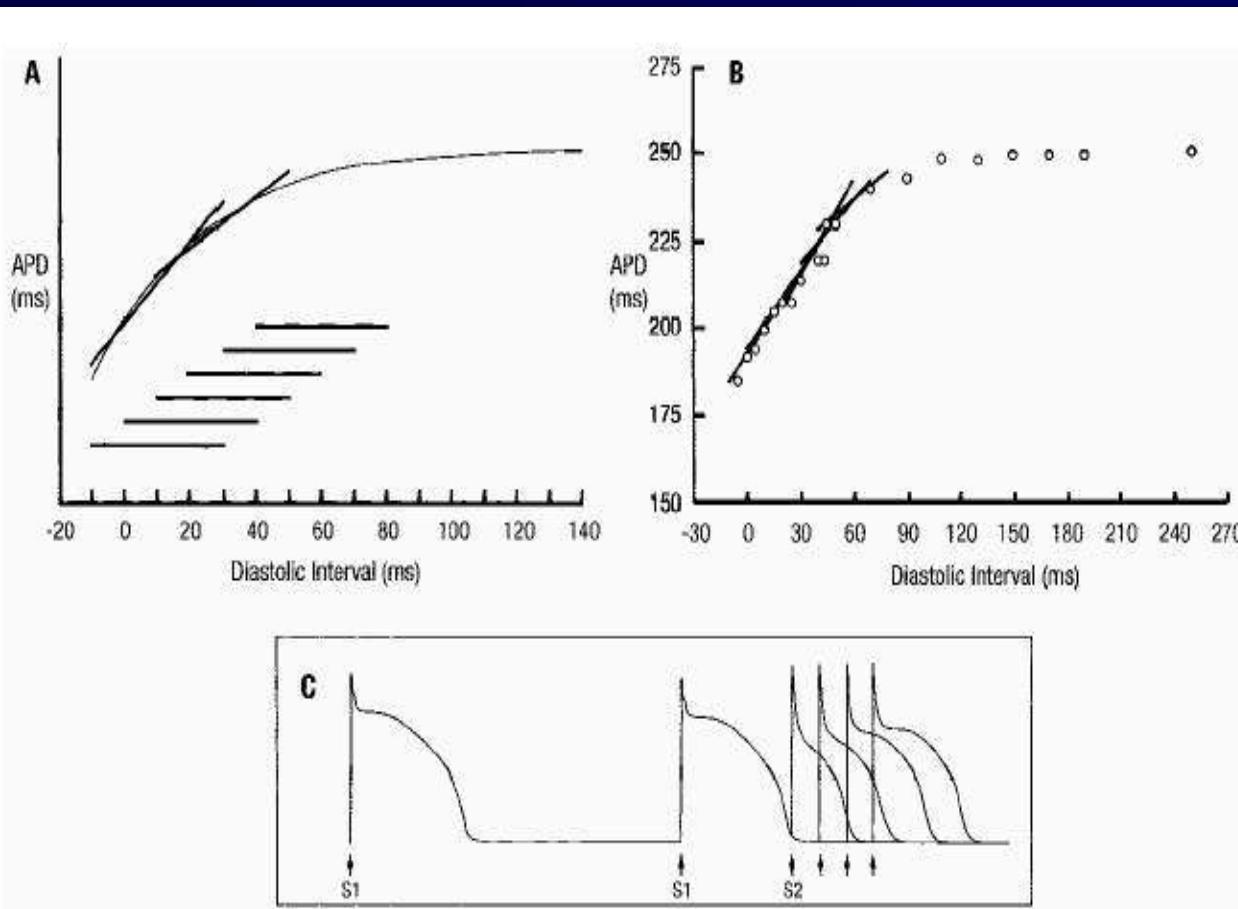


* A Tale of Two Fibrillations

PS Chen

Circulation 2003;108:2298-2303.

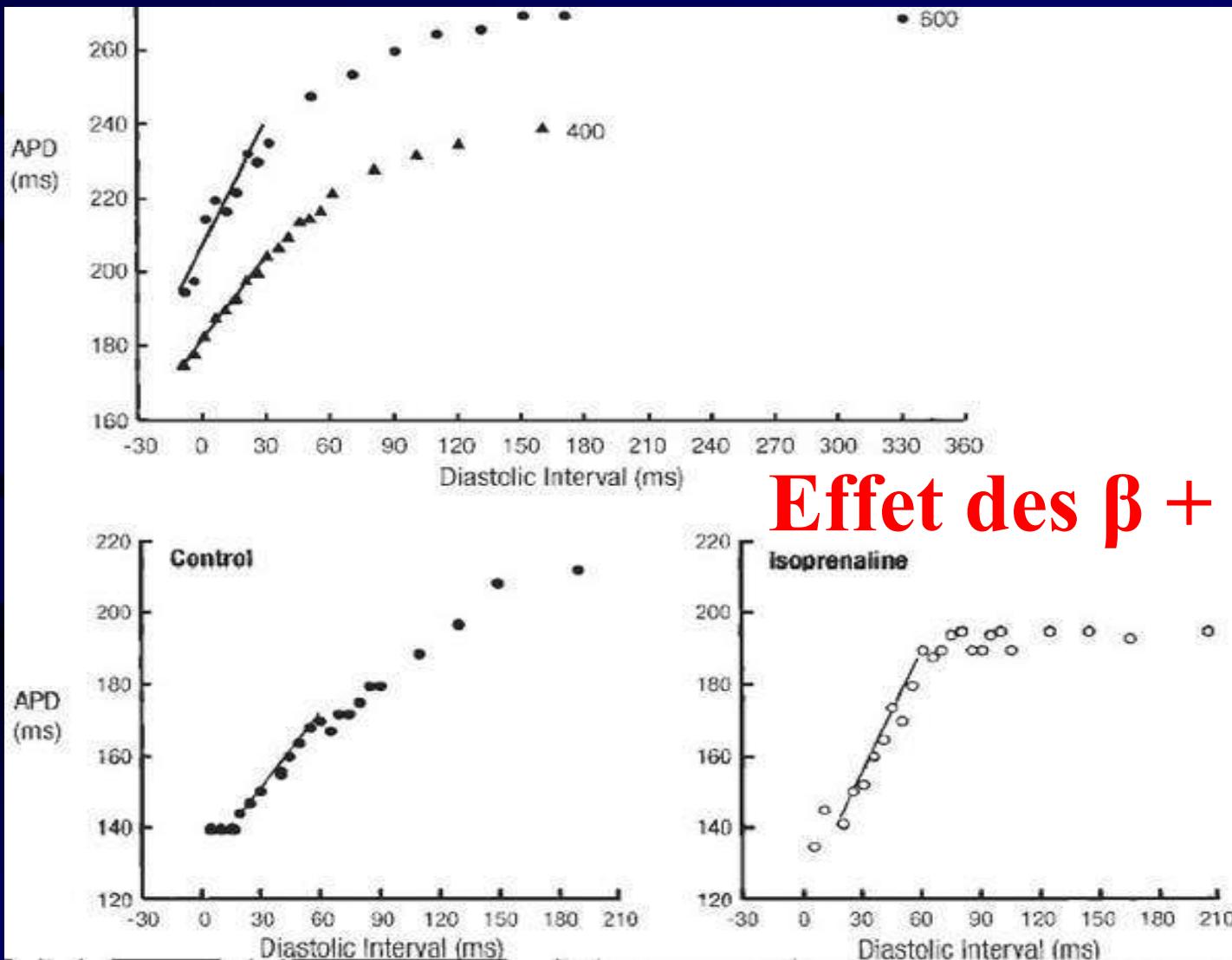
Fibrillation Ventriculaire



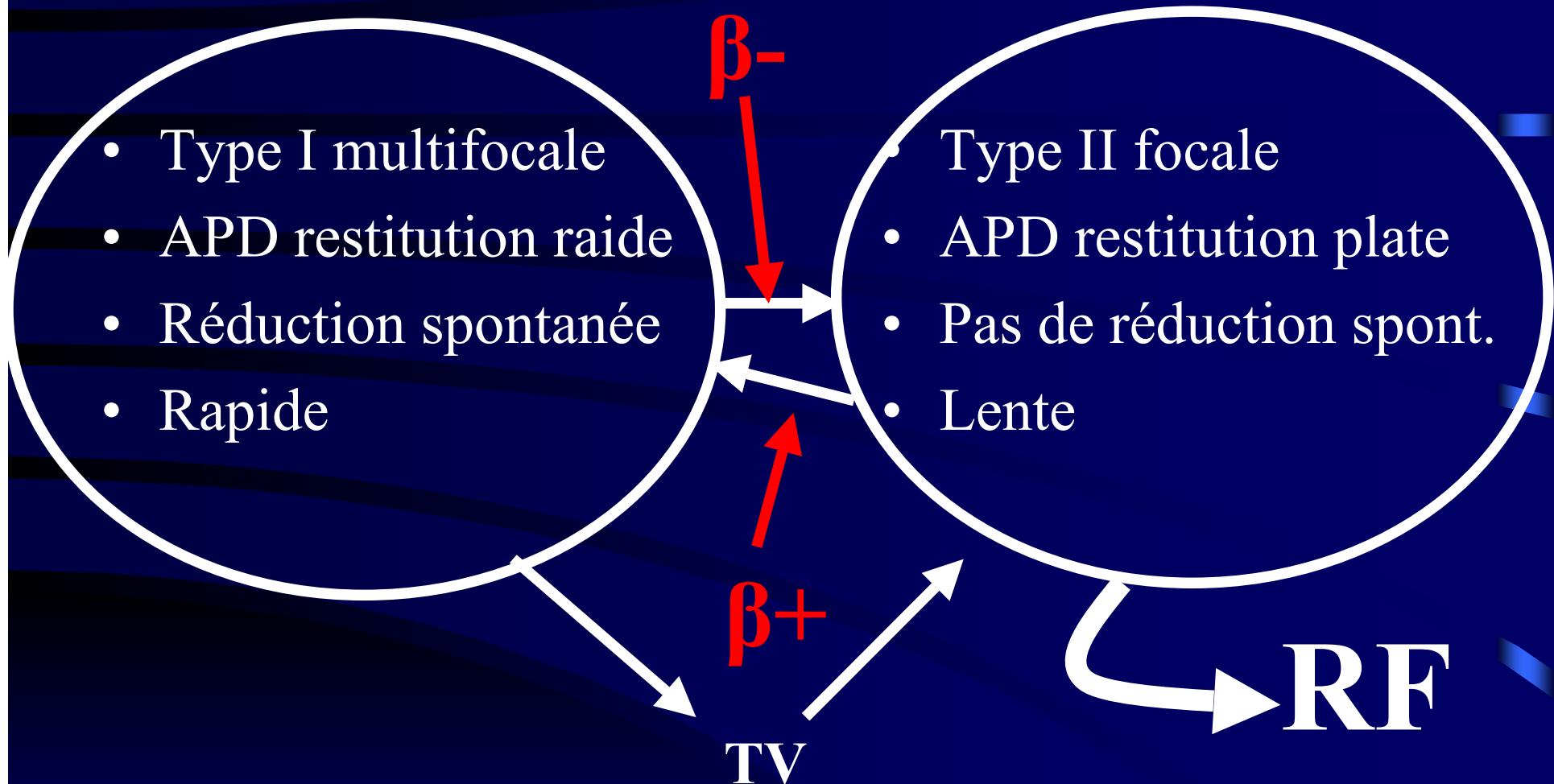
* Effect of Adrenergic Stimulation on Action Potential Duration Restitution in Humans
P Taggart, Circulation. 2003;107:285-289.

AP duration restitution

Fibrillation Ventriculaire



Fibrillation Ventriculaire



* A Tale of Two Fibrillations PS Chen Circulation. 2003;108:2298-2303.

* Two Types of Ventricular Fibrillation in Isolated Rabbit Hearts. Tsu-Juey Wu.
Circulation. 2002;106:1859-1866.

Conclusion

Mécanisme

Non univoque ++

Difficile à préciser++



Traitement