

***TACHICARDIE QRS LARGO: MONO-POLIMORFISMI, FOCALITÀ
ABERRANZE, LINKING E DINTORNI***

**Inquadramento fisiopatologico-
classificativo delle tachicardie a QRS
largo**

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***Conflict of Interest (COI):
Nothing to declare***



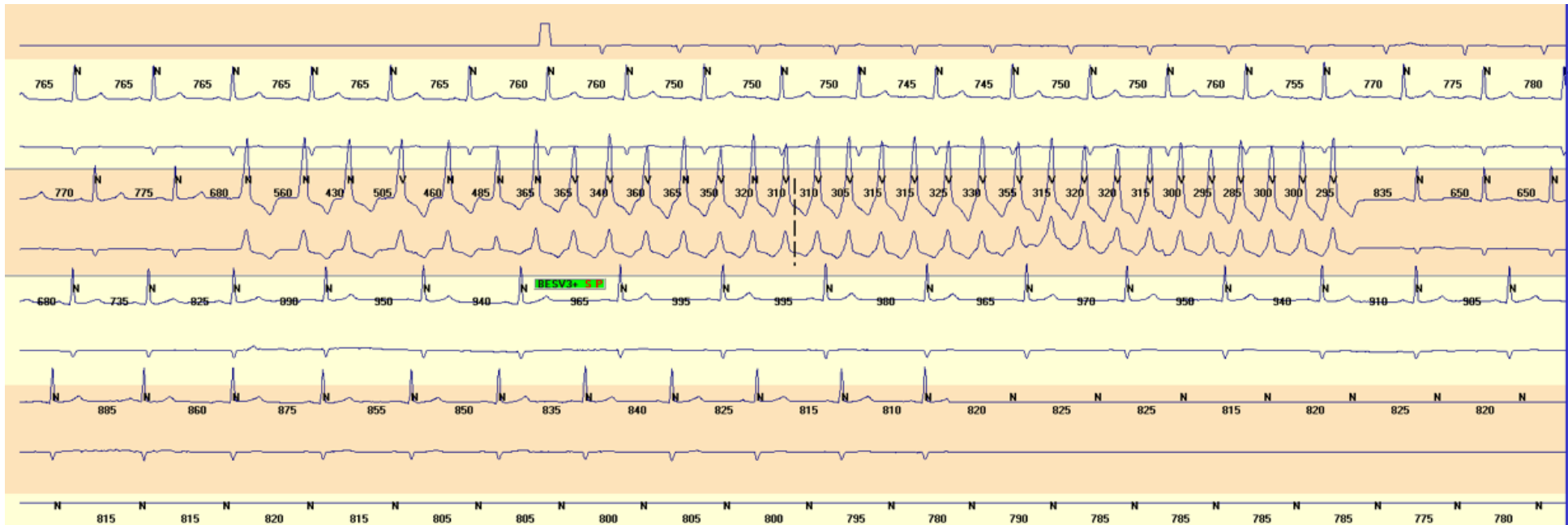
Cos'è una tachicardia a QRS largo?

Definizione:

- frequenza >100 bpm
- durata del QRS ≥ 120 ms

QRS largo indica:

- attivazione ventricolare anomala
- conduzione intraventricolare rallentata
- attivazione miocardica non fisiologica.



Cos'è una tachicardia a QRS largo?

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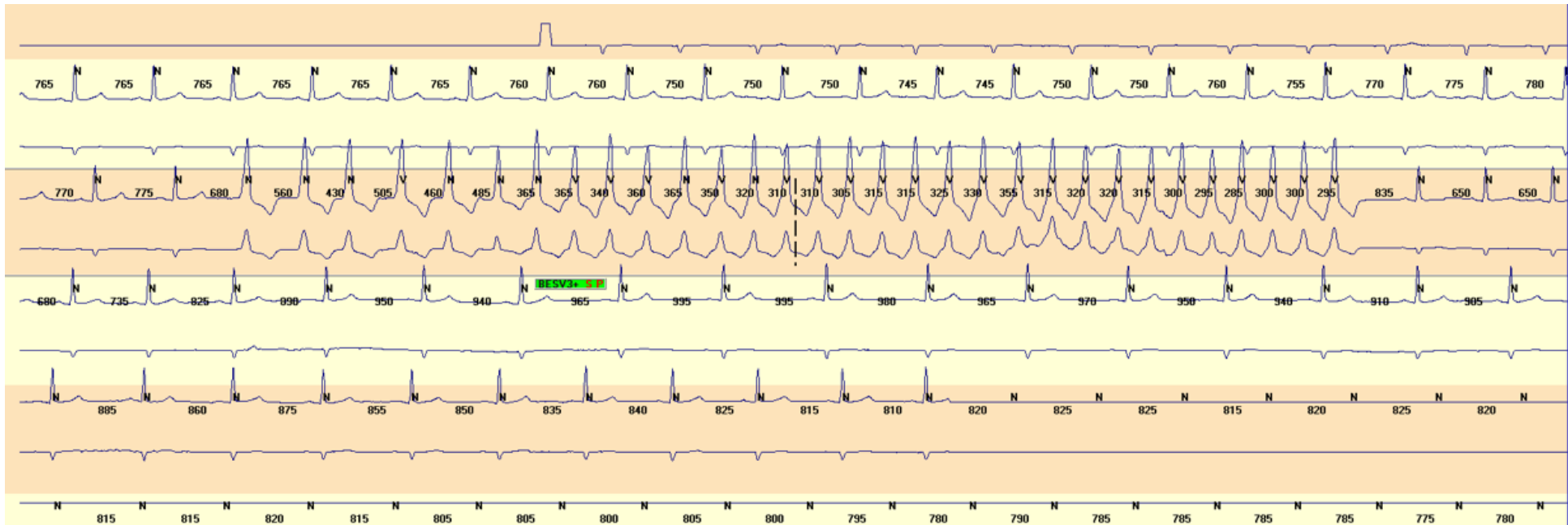
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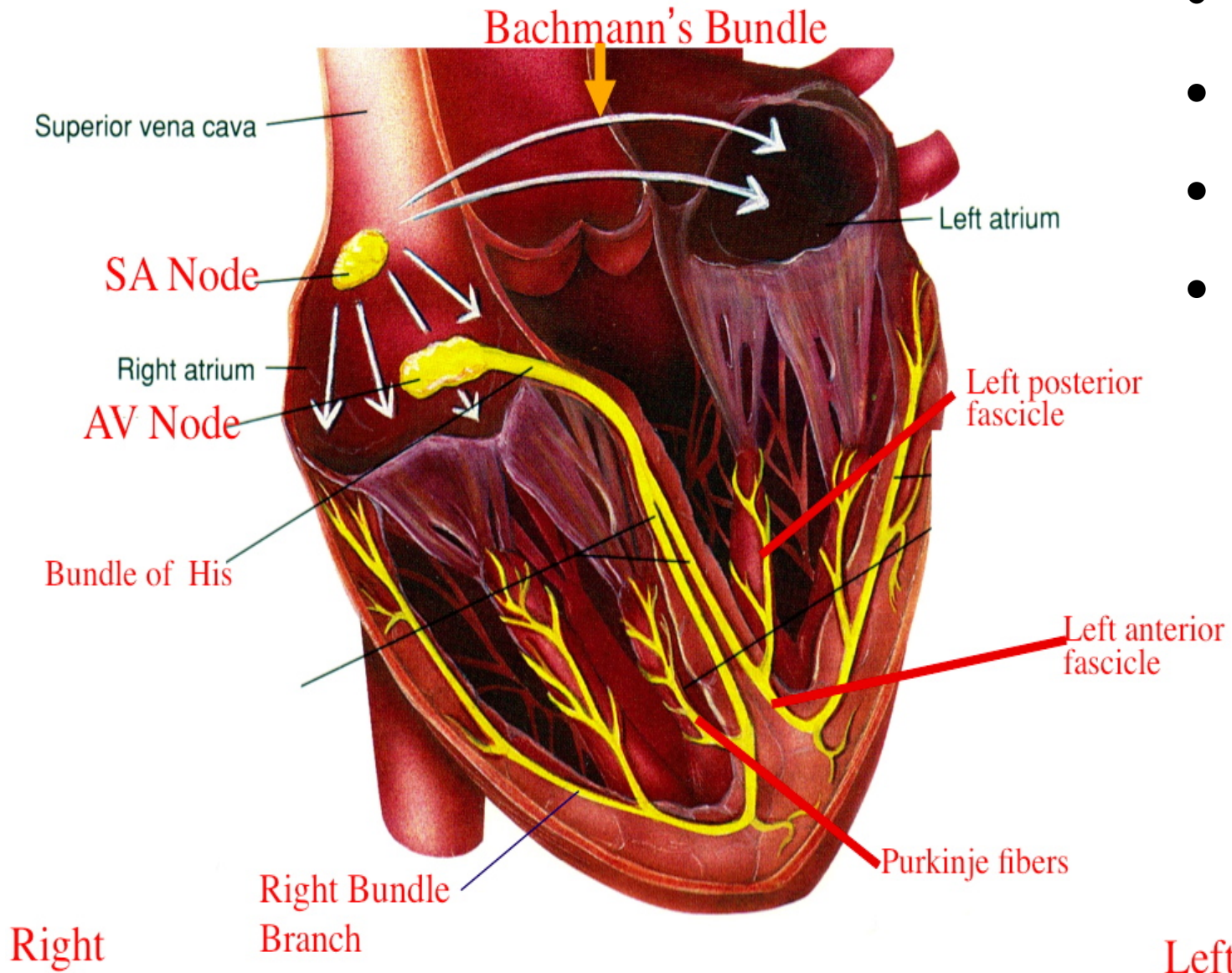
Perché il QRS è largo?

Tre possibili cause:

- origine ventricolare
- origine sopraventricolare con aberranza
- attivazione ventricolare attraverso una via accessoria



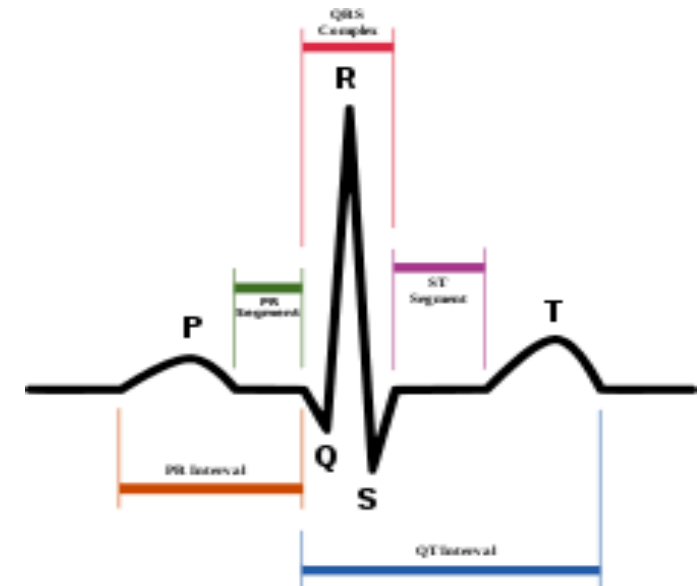
Sistema di Conduzione



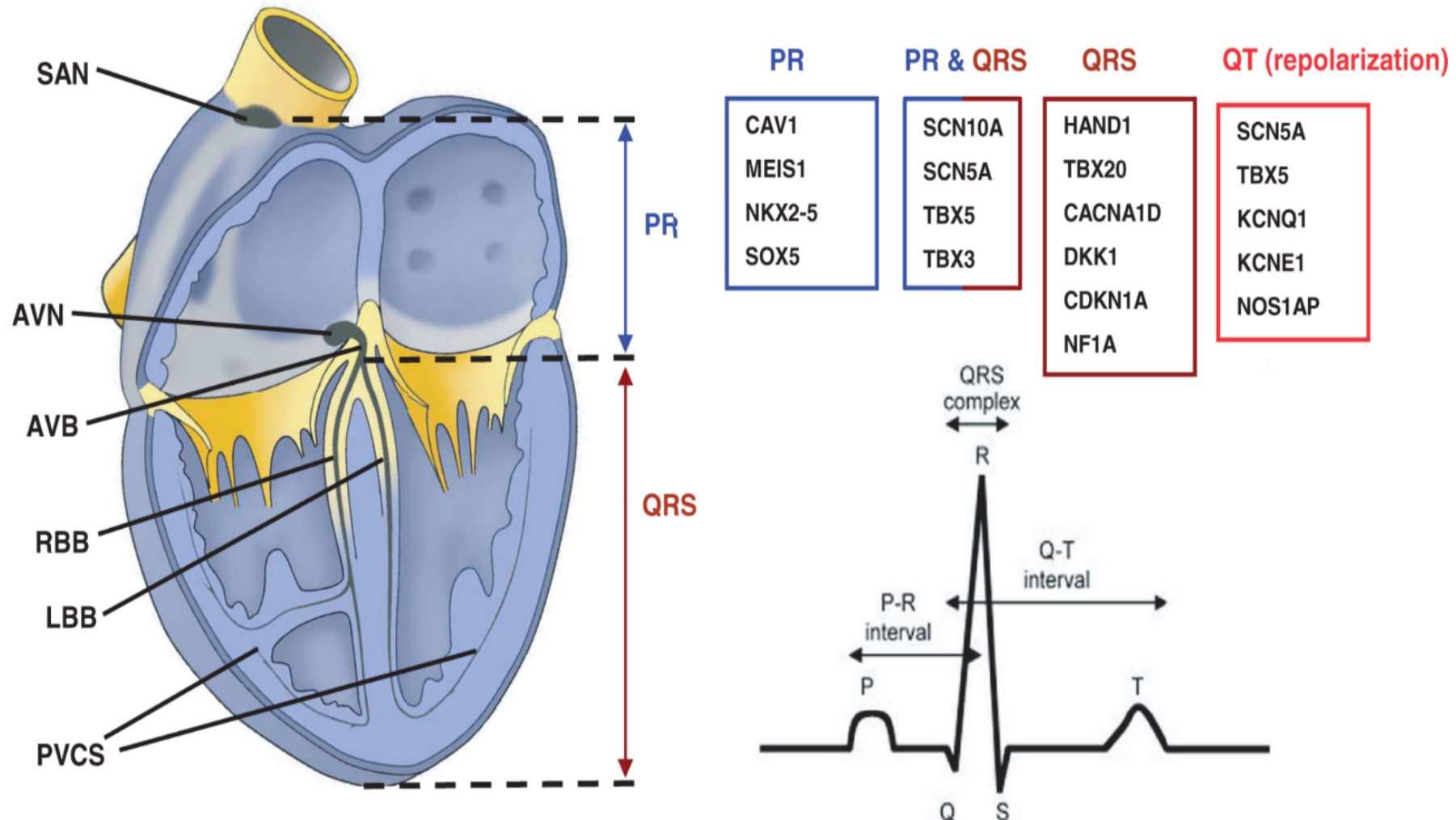
Attivazione ventricolare normale

- sistema His-Purkinje
- velocità di conduzione elevata
- sincronia ventricolare
- QRS stretto (<120 ms)

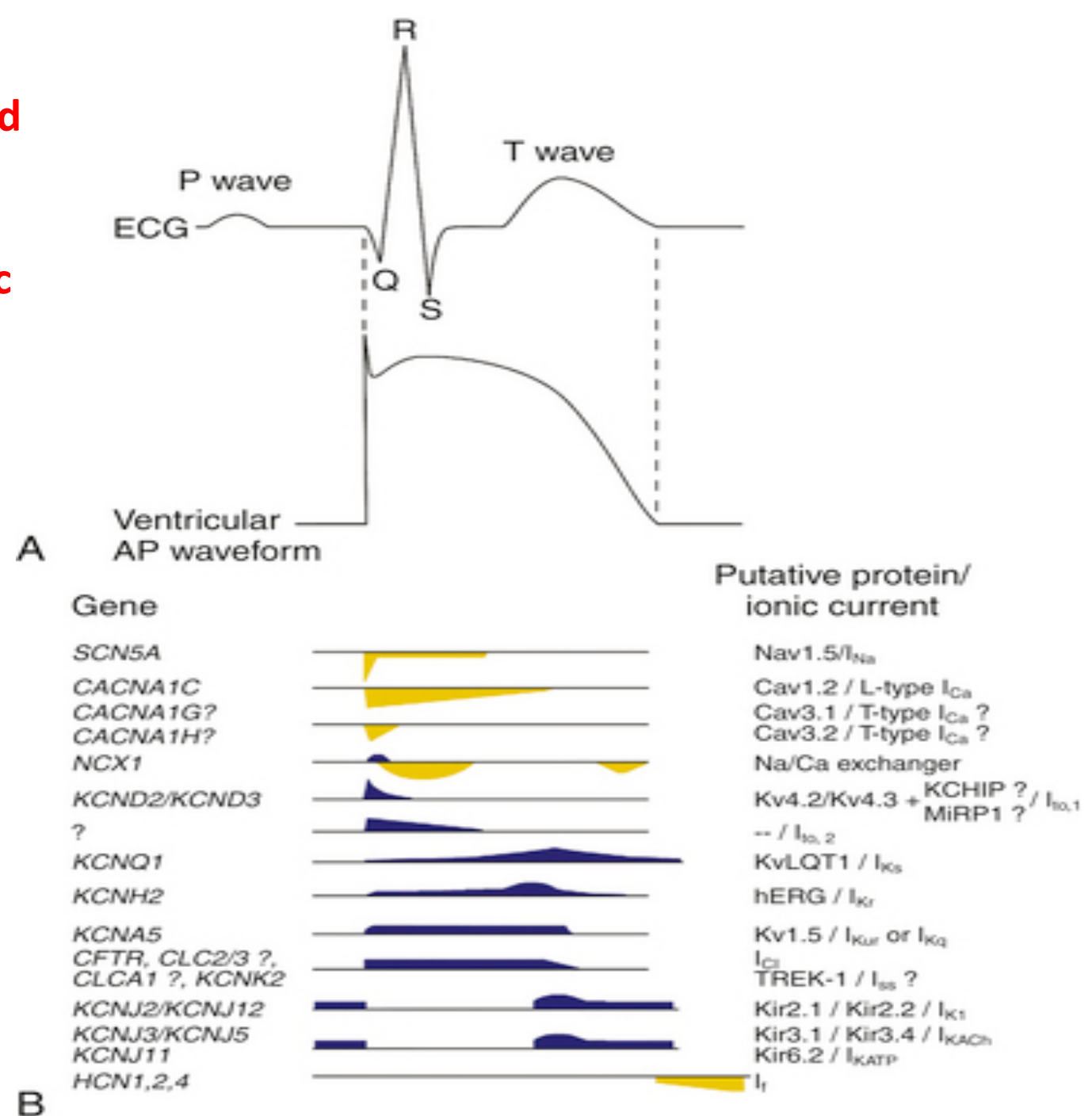
ECG Normale



GENE REGULATORY NETWORKS, CARDIAC CONDUCTION SYSTEM AND SURFACE ECG WAVEFORMS

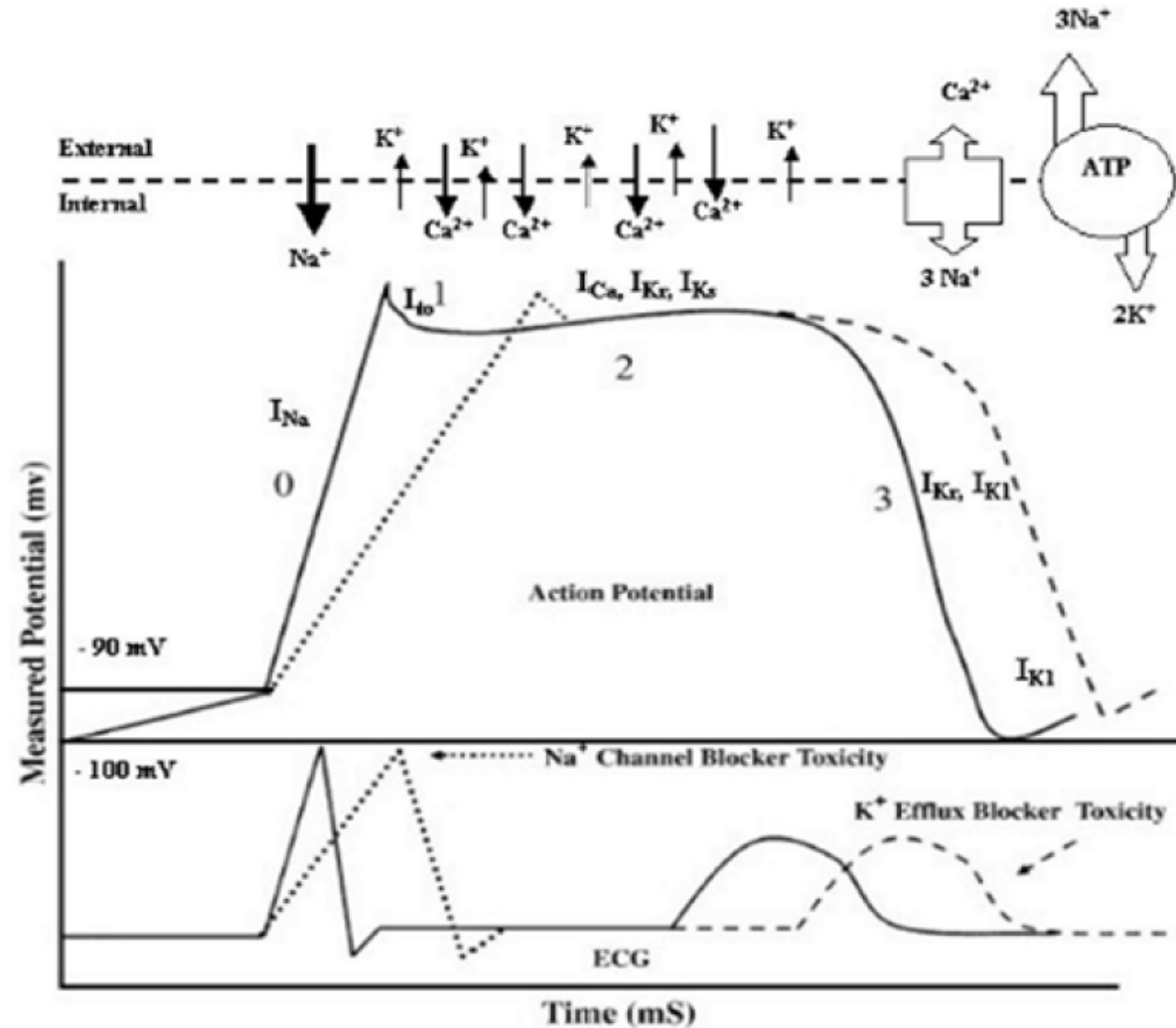


Cardiac Ionic Currents and Ion Channel Genes Responsible for the Generation of the Cardiac Action Potential



Il QRS si allarga quando:

- L'attività proviene dal tessuto ventricolare miocardico alterato
- il sistema His-Purkinje è bloccato
- Esiste una via di conduzione antidromica.



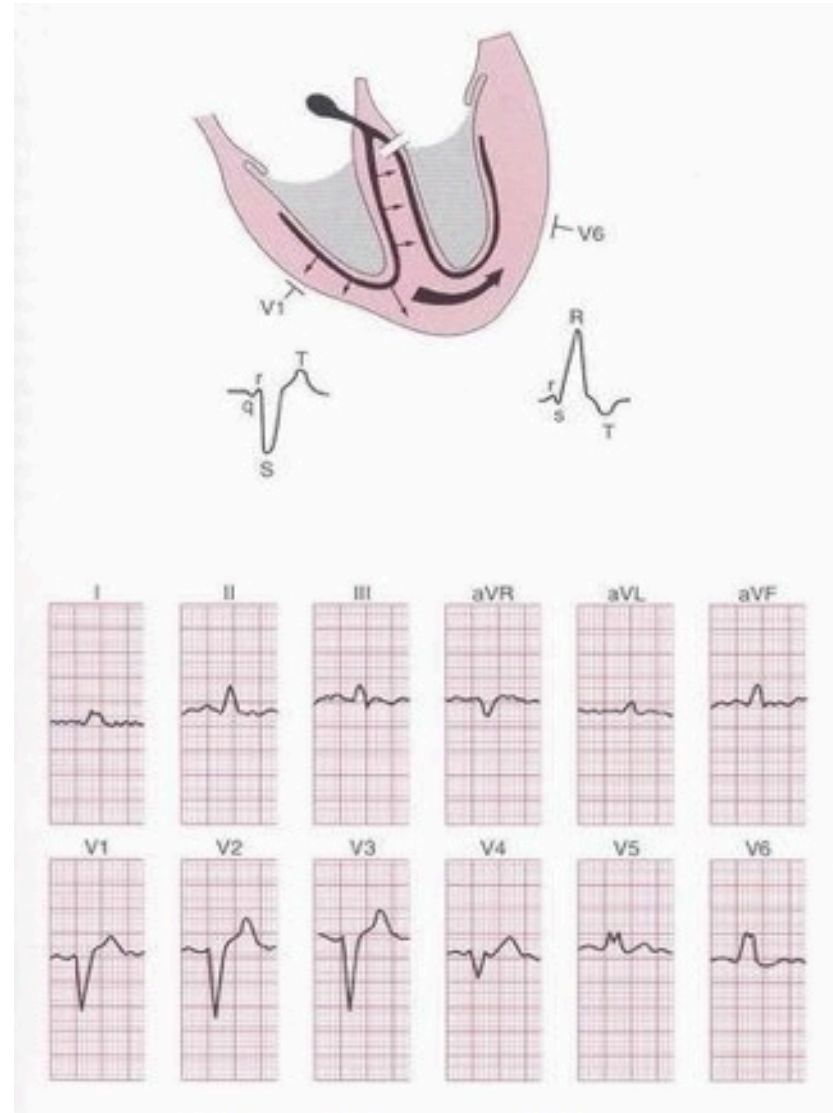
Dotted line indicates the changes associated with Na⁺ channel blocker toxicity. Dashed line indicates the changes associated with K⁺ efflux blocker toxicity.

I_{to} = transient outward K⁺ current; **I_{Ca}** = L-type Ca²⁺ current; **I_{Na}** = late sodium channel current; **I_{Kr}** = rapidly activating delayed-rectifier K⁺ current; **I_{Ks}** = slowly activating delayed rectifier K⁺ current; **I_{K1}** = inward rectifier K⁺ current (Adapted from Holstege et al., 2005)

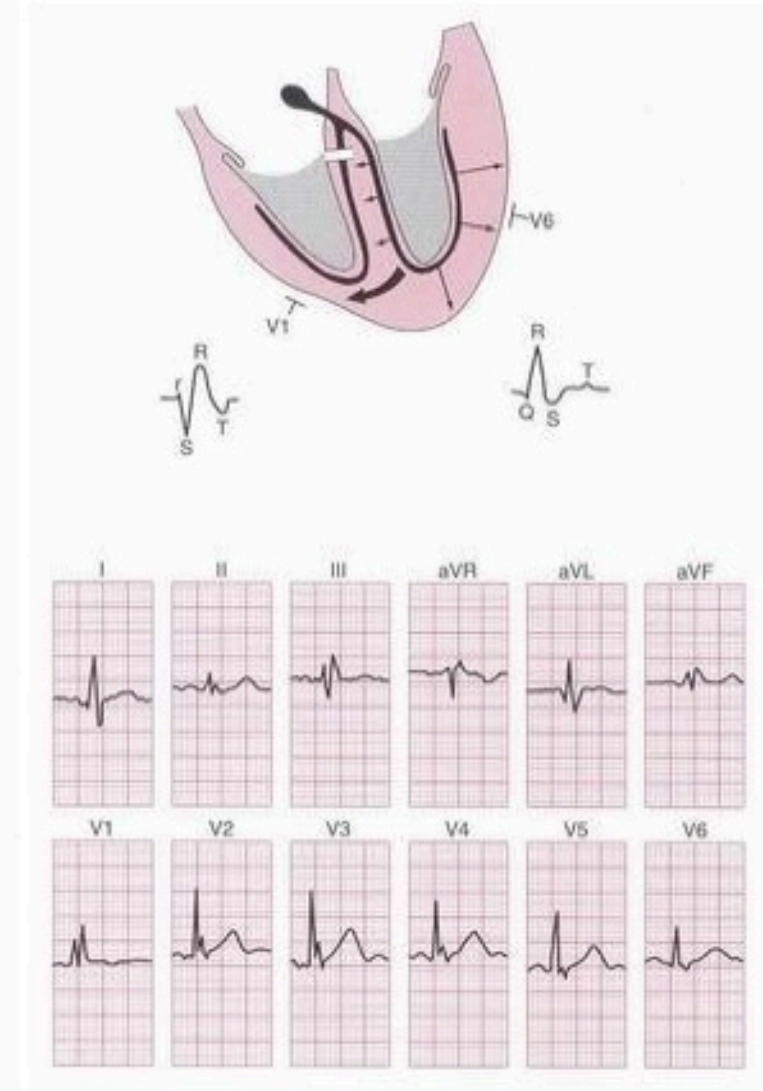
Blocchi di branca sinistro e destro

Il QRS si allarga quando:

- L'attività proviene dal tessuto ventricolare miocardico alterato
- **il sistema His-Purkinje è bloccato**
- Esiste una via di conduzione antidromica.



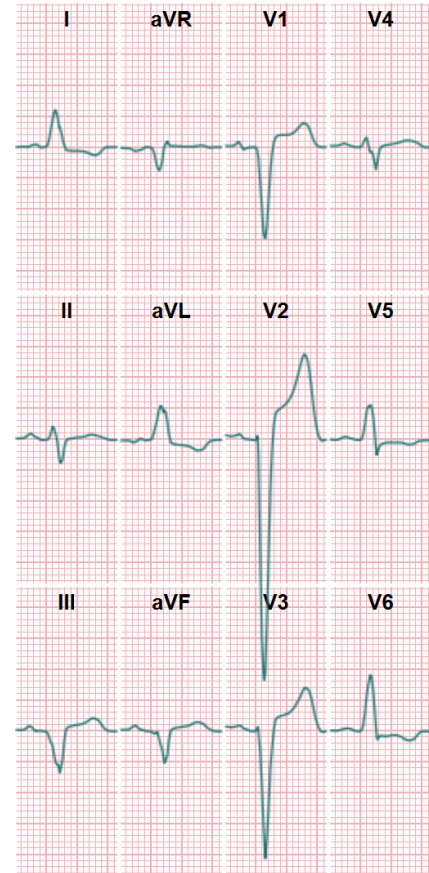
Blocco di Branca Sinistro



Blocco di Branca Destro

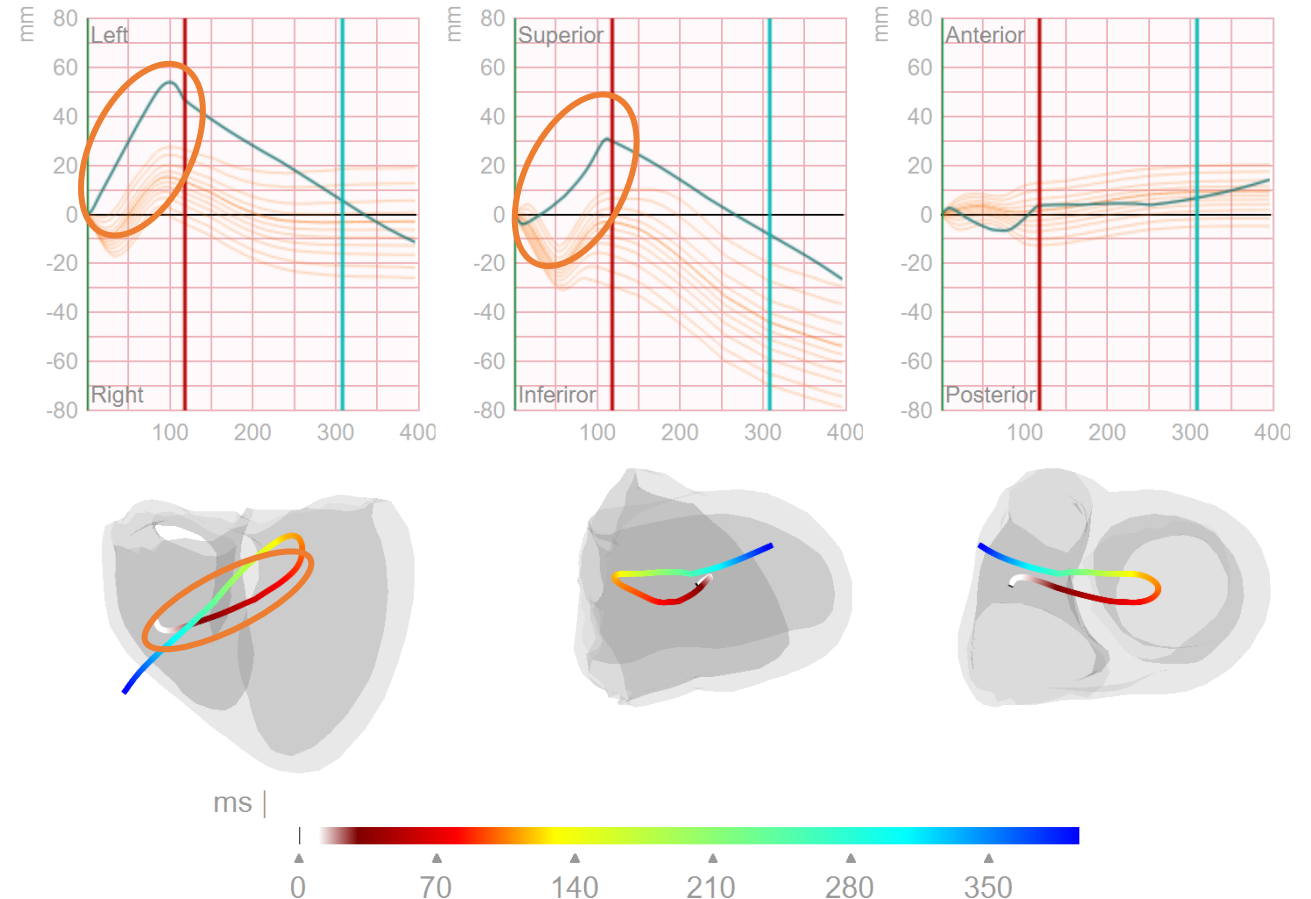
CineECG in Left Bundle Branch Block (LBBB)

- The terminal QRS **direction** moves from the right chamber to the left base
- T-wave moves in the opposite direction



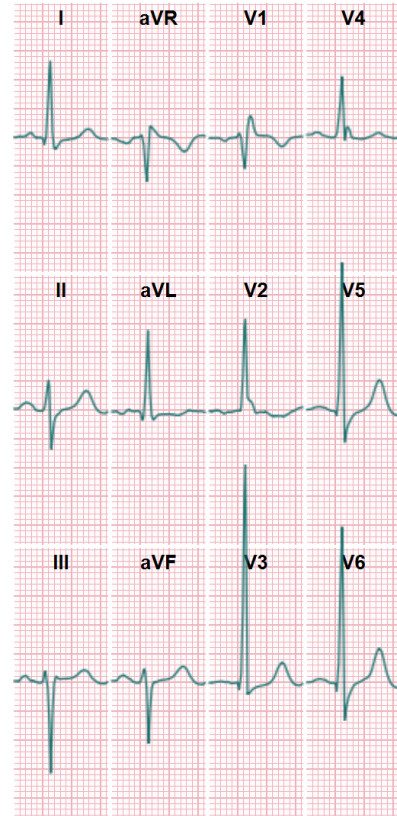
25 mm/s, 10 mm/mV

Electrical pathway



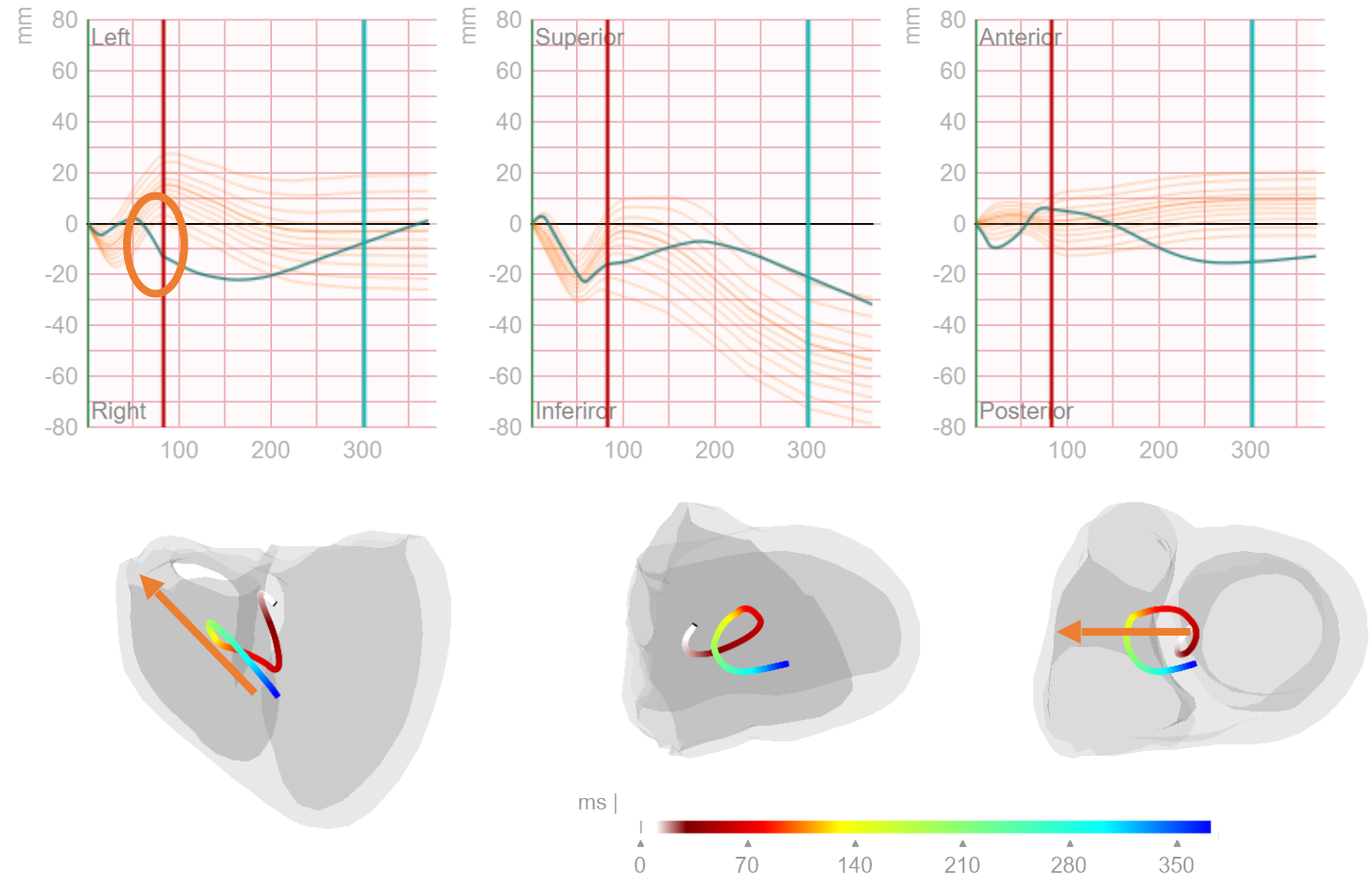
CineECG in Right Bundle Branch Block (RBBB)

Terminal
QRS
direction
moves to
the right
base

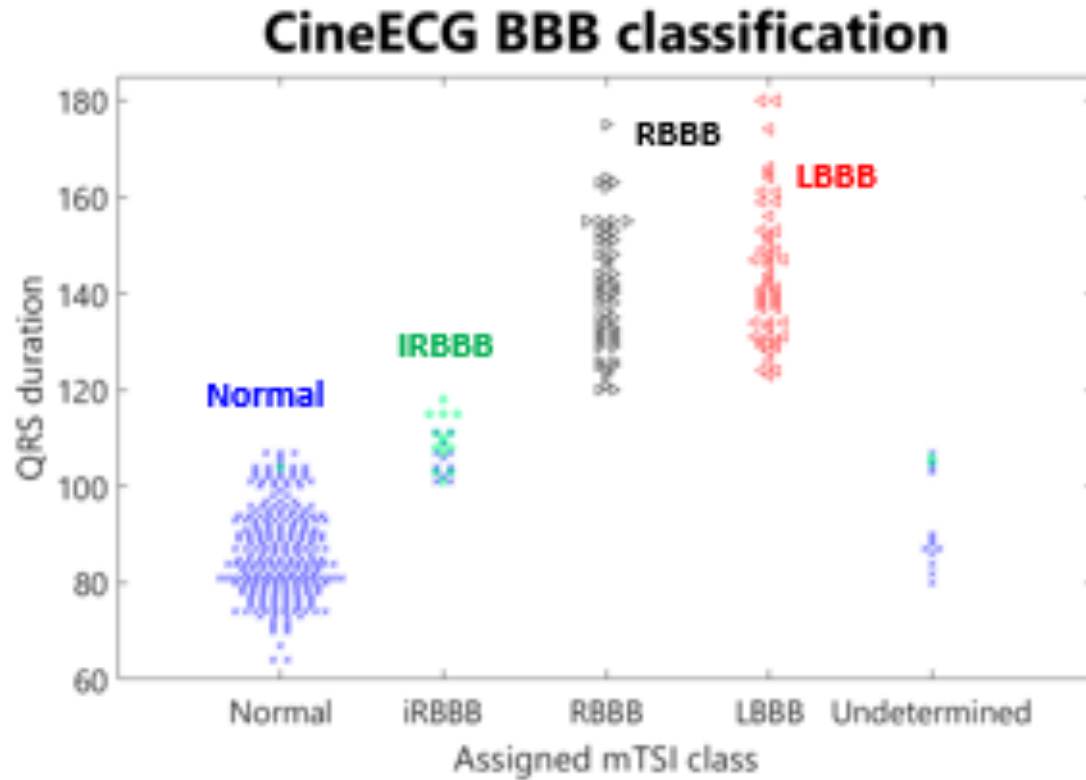


25 mm/s, 10 mm/mV

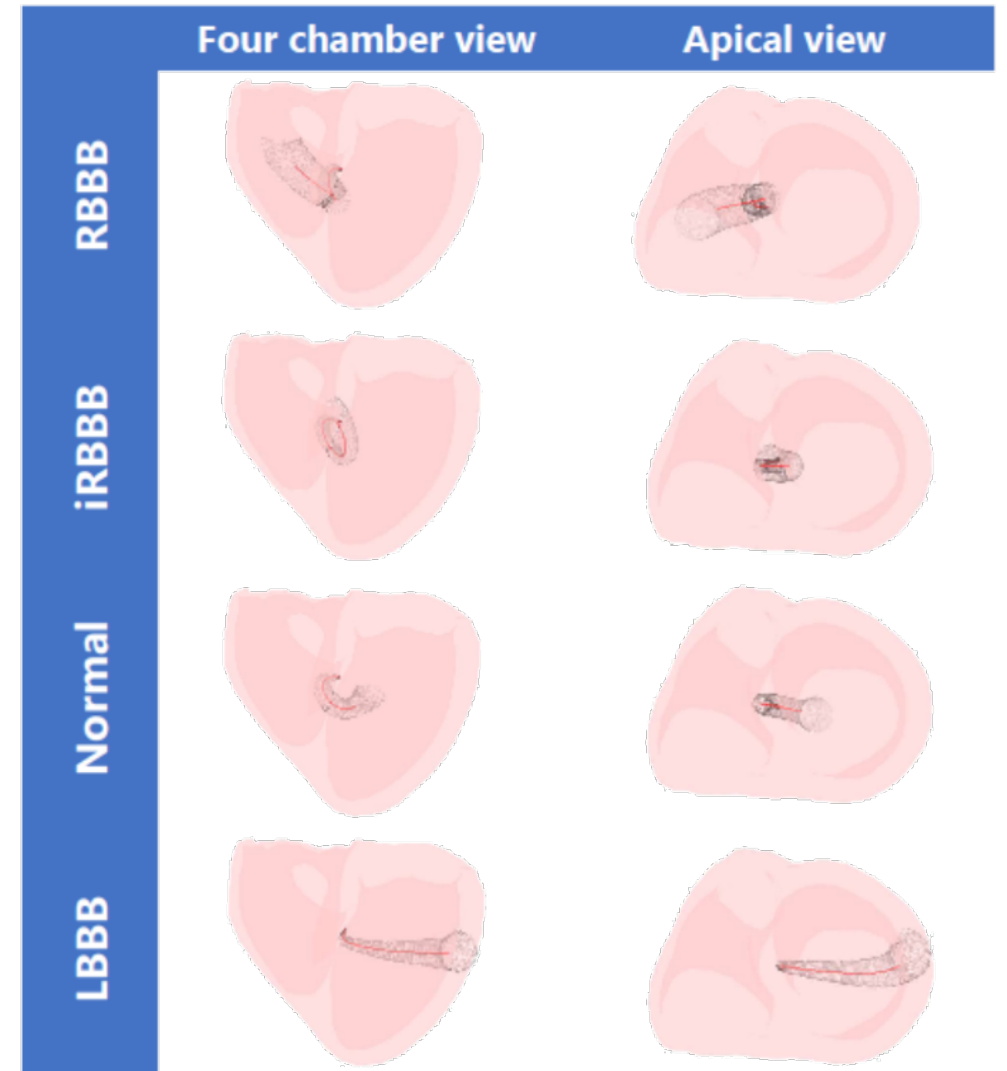
Electrical pathway



CineECG Detection of Conduction Disorders



Boonstra, M.J., et al., *Novel CineECG enables anatomical 3D localization and classification of bundle branch blocks*. EP Europace, 2021. 23(Supplement_1): p. i80-i87.

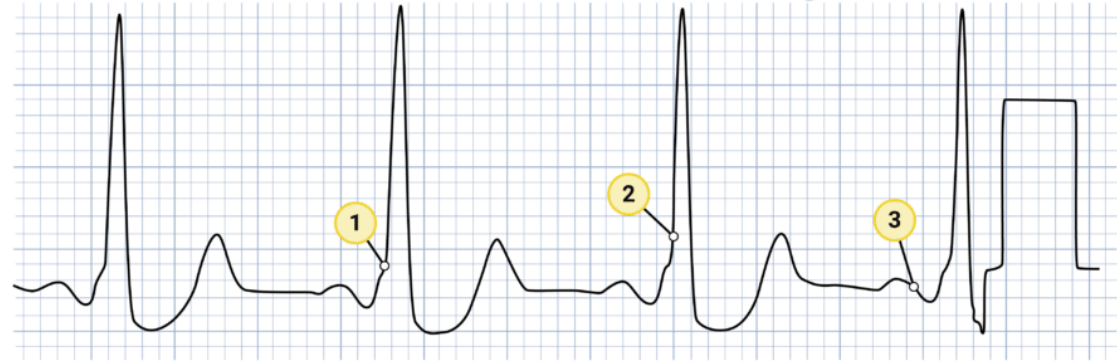


Alterazioni della conduzione

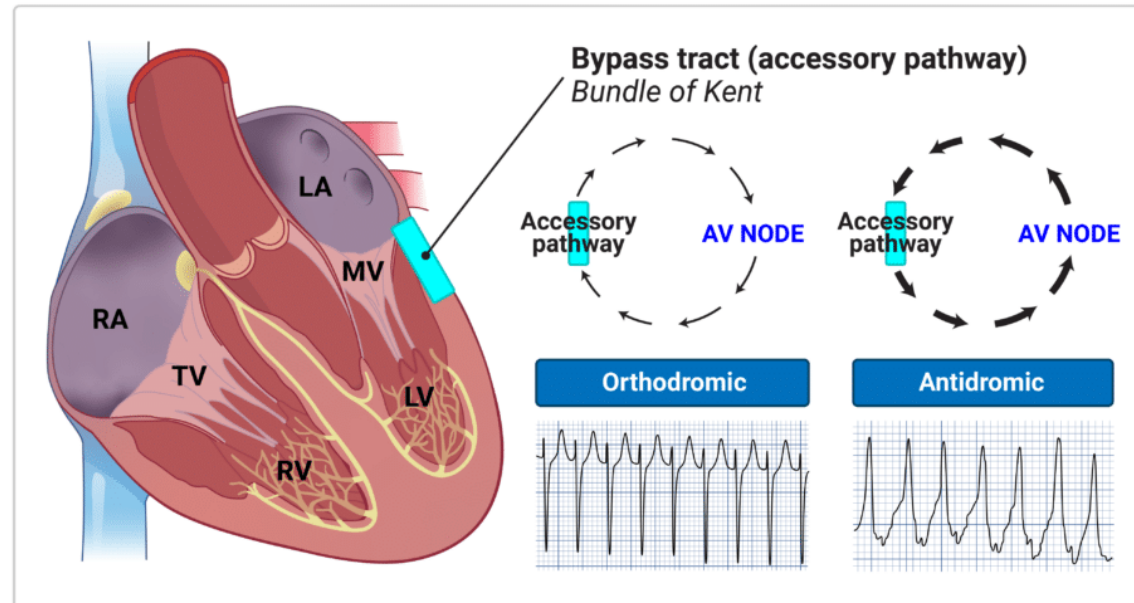
Il QRS si allarga quando:

- L'attività proviene dal tessuto ventricolare miocardico alterato
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- Esiste una via di conduzione antidromica.

Wolff-Parkinson-White Syndrome



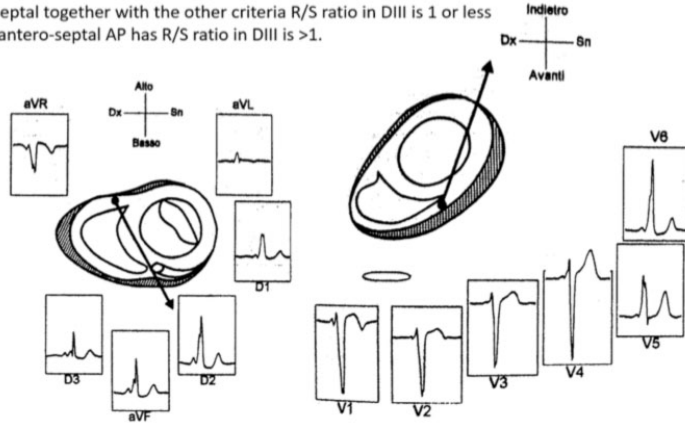
- 1 Slurred upstroke of QRS complex (delta wave)
- 2 Wide QRS complex (QRS > 120 msec)
- 3 Short PR interval (PR < 120 msec)



Possible (Multiple) Localizations of Ventricular Preexcitation

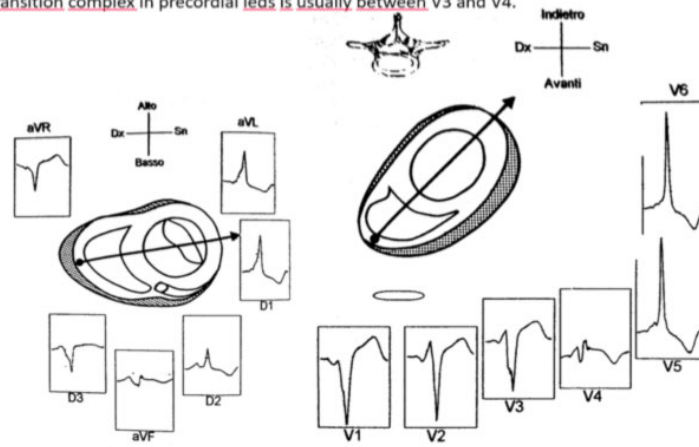
ANTEROSEPTAL AP

Positive Delta waves in inferior leads (DII, aVF and DIII).
If truly anterospetal very late (V5-V6) transition is observed.
If parahissian QS complexes or negative Delta wave in V1 and V2.
If midseptal together with the other criteria R/S ratio in DIII is 1 or less
While antero-septal AP has R/S ratio in DIII is >1 .



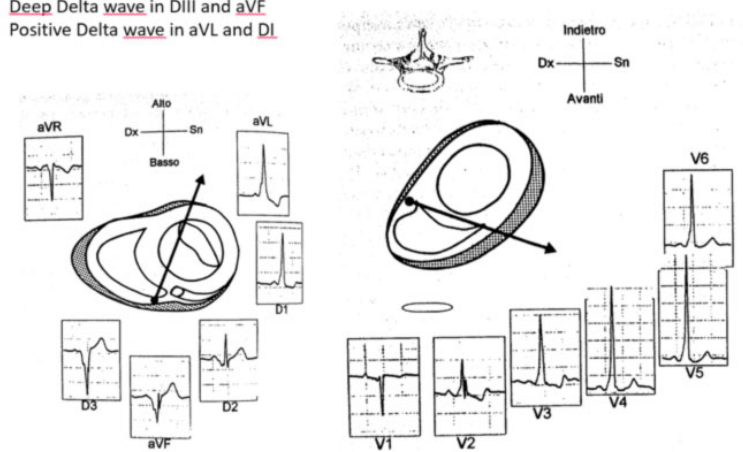
RIGHT LATERAL AP

Positive delta wave in left leads (DI aVL).
Deep negative delta waves in aVR and DIII.
Transition complex in precordial leads is usually between V3 and V4.



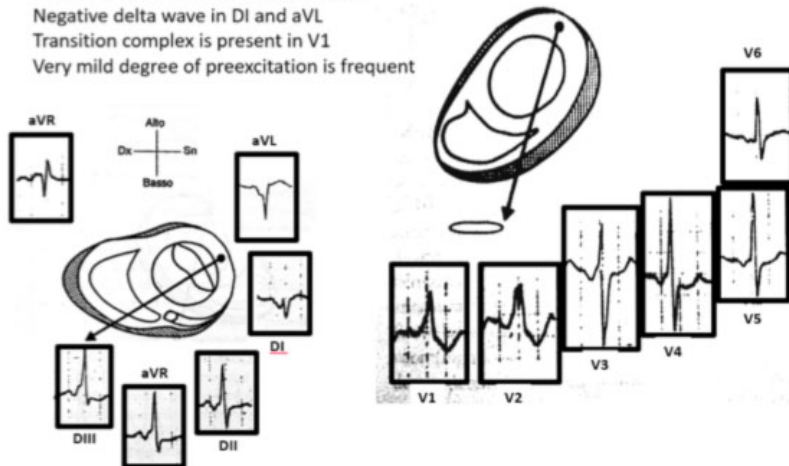
RIGHT POSTEROSEPTAL AP

Transition complex is present in V2
Deep Delta wave in DIII and aVF
Positive Delta wave in aVL and DI



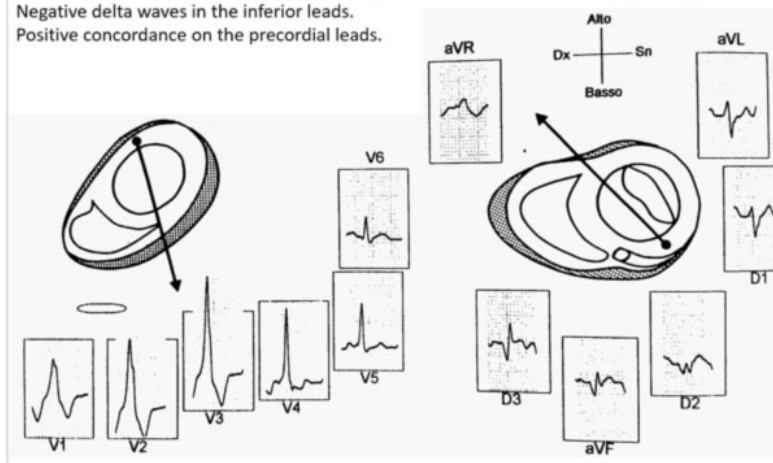
LEFT LATERAL AP

Positive delta wave in the inferior leads
Negative delta wave in DI and aVL
Transition complex is present in V1
Very mild degree of preexcitation is frequent



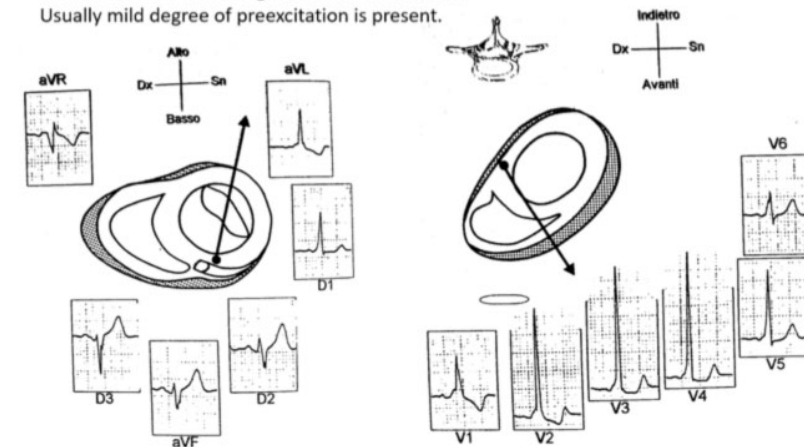
LEFT POSTERIOR AP

Posterior and anterior left AP can also show positive delta wave in left leads (DI aVL).
Negative delta waves in the inferior leads.
Positive concordance on the precordial leads.



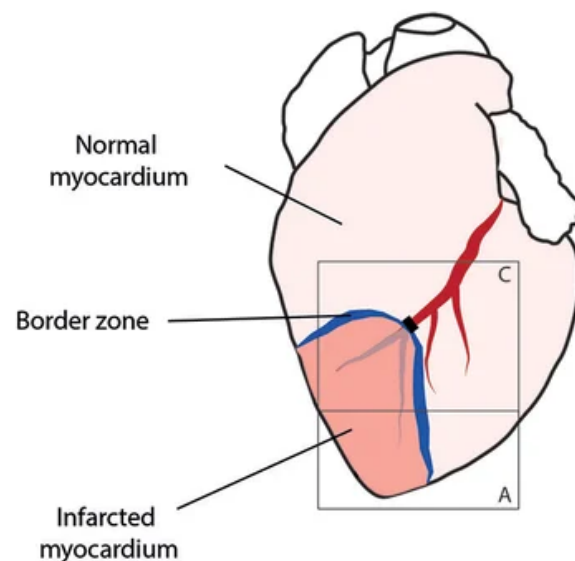
LEFT POSTEROSEPTAL AP

Left postero-septal AP usually presents positive Delta waves in precordial leads.
Positive Delta waves are also visible in aVR and aVL.
DII delta wave is more negative than DIII and aVF.
Usually mild degree of preexcitation is present.

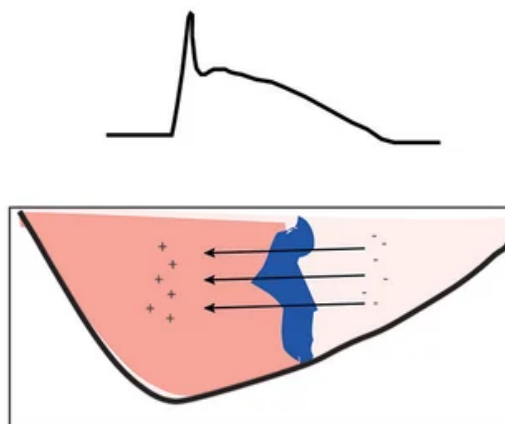


Meccanismi elettrofisiologici delle tachicardie a QRS largo

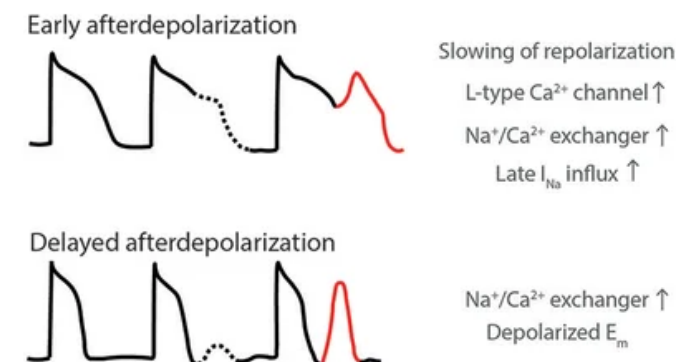
1. Aumentato automatismo
2. Attività triggerata (triggered activity): EAD and DAD
3. Rientro: Anatomico e funzionale



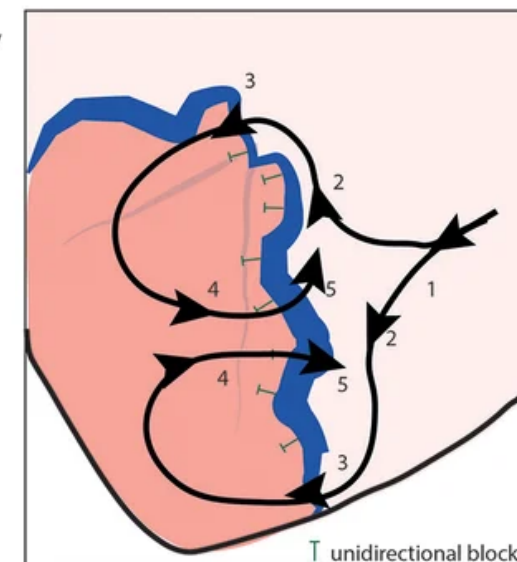
A Automaticity (Injury current)



B Triggered activity



C Reentry



1. Aumentato Automatismo

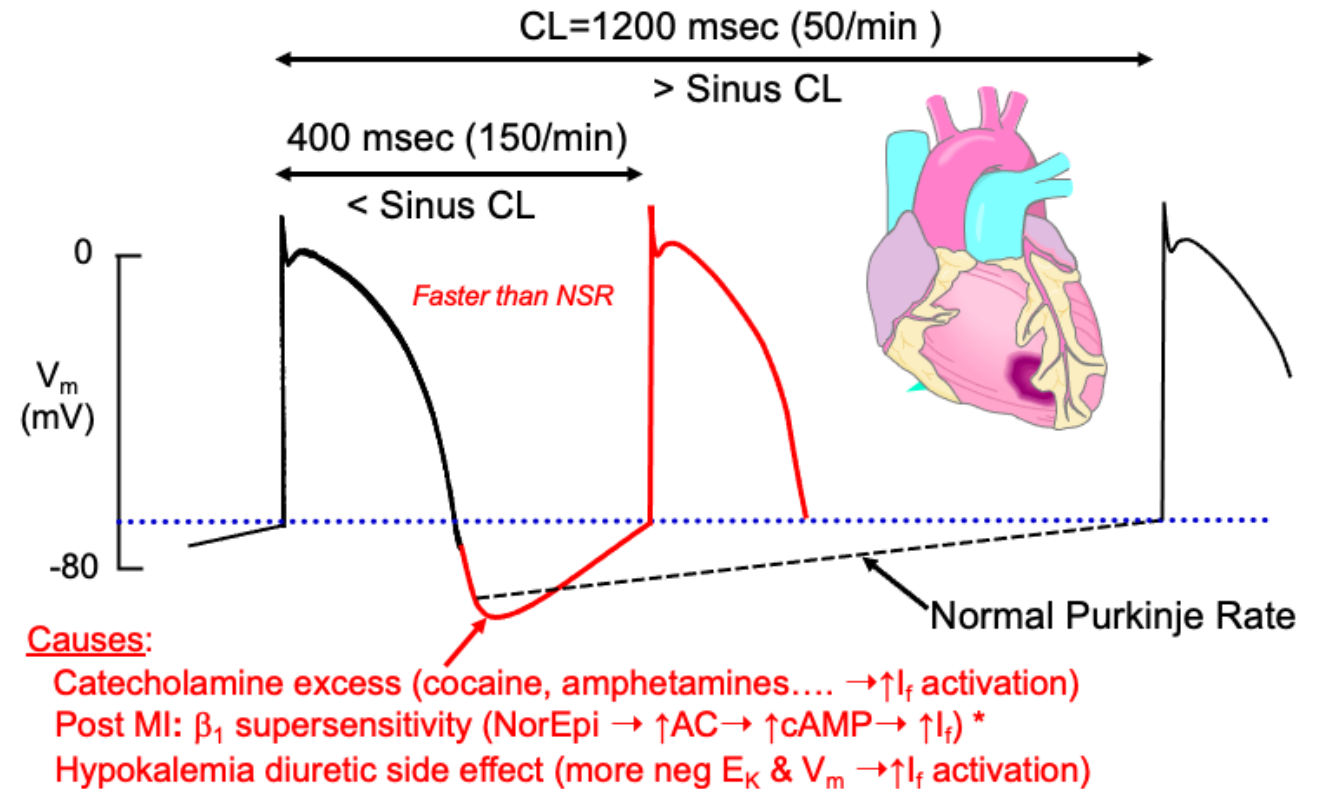
Caratteristiche:

- depolarizzazione spontanea durante la fase diastolica
- warm-up
- cool-down
- frequenza variabile

Esempi:

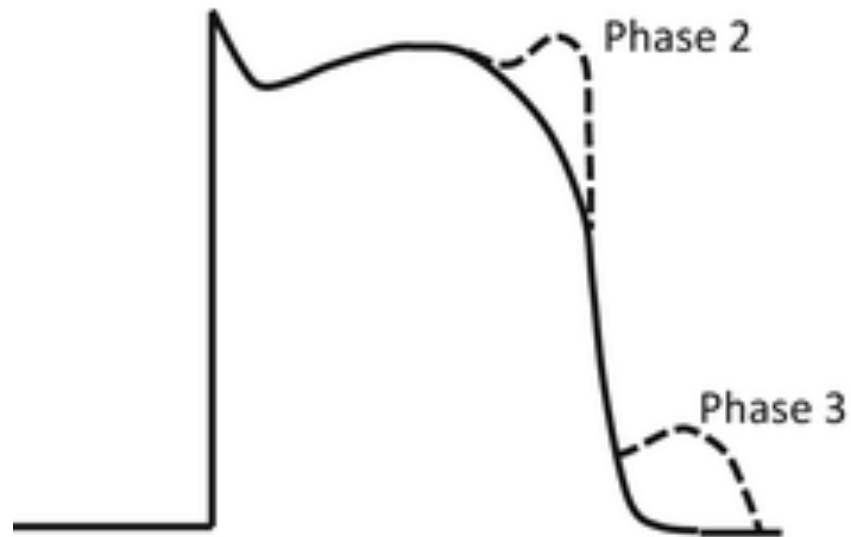
- TV automatica
- Tachicardie fascicolari

Ectopic Pacemaker (Enhanced Normal Automaticity)



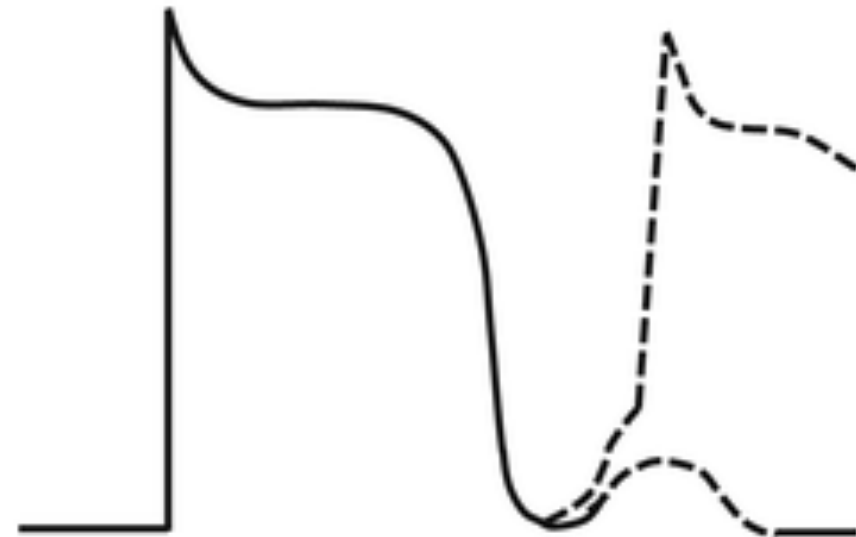
2. Early and Delayed Triggered Activity

Early afterdepolarization
(EAD)



- Reduced outward current (I_K)
- Increased inward current ($I_{Ca,L}$; I_{NCX})
- Exacerbated by bradycardia

Delayed Afterdepolarization
(DAD)



- Increased intracellular calcium
- Digoxin toxicity
- Exacerbated by catecholamines
- Catecholaminergic polymorphic ventricular tachycardia

3. Rientro

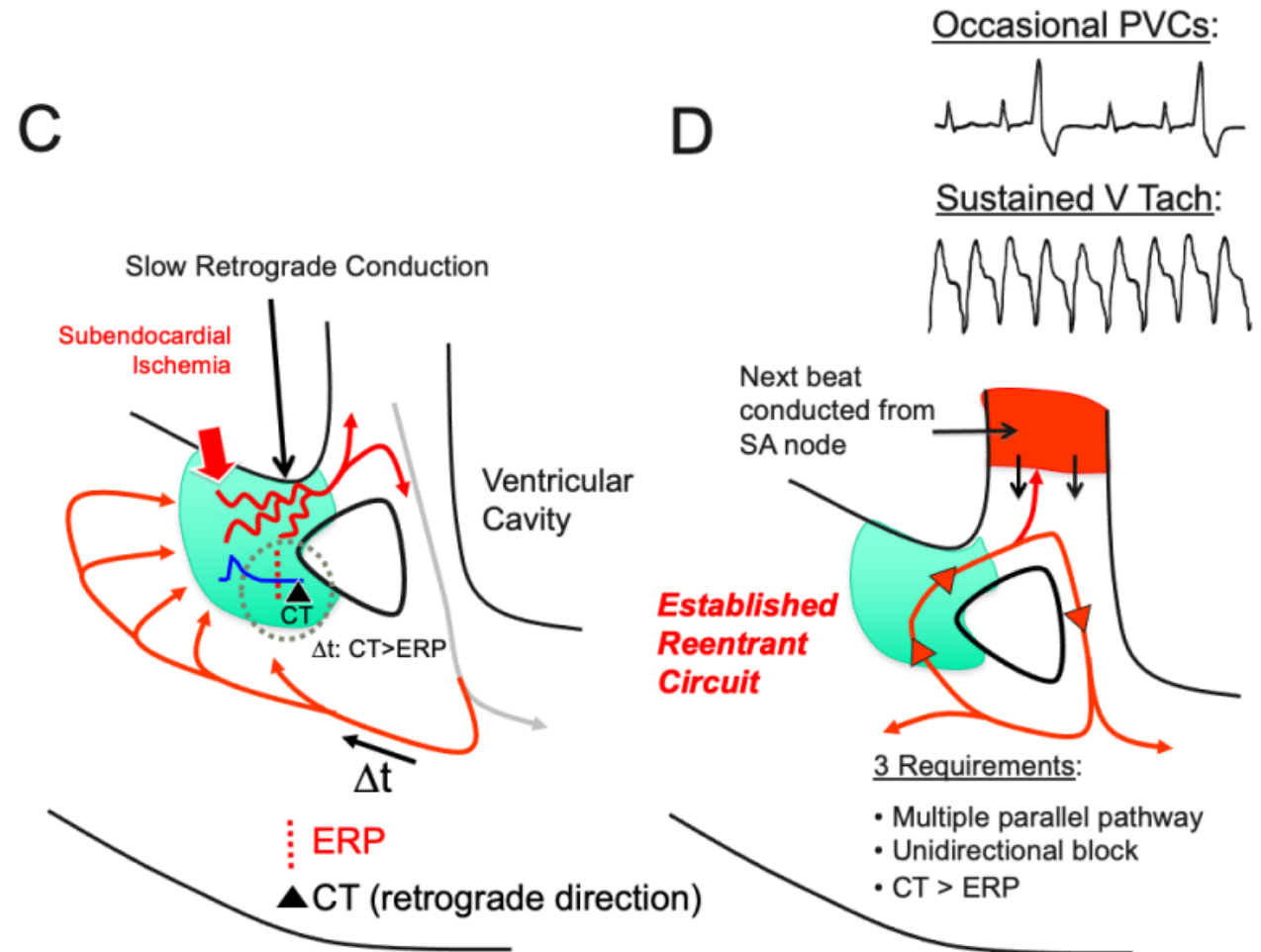
Condizioni necessarie:

- Doppia via
- Blocco unidirezionale
- Rallentamento della conduzione

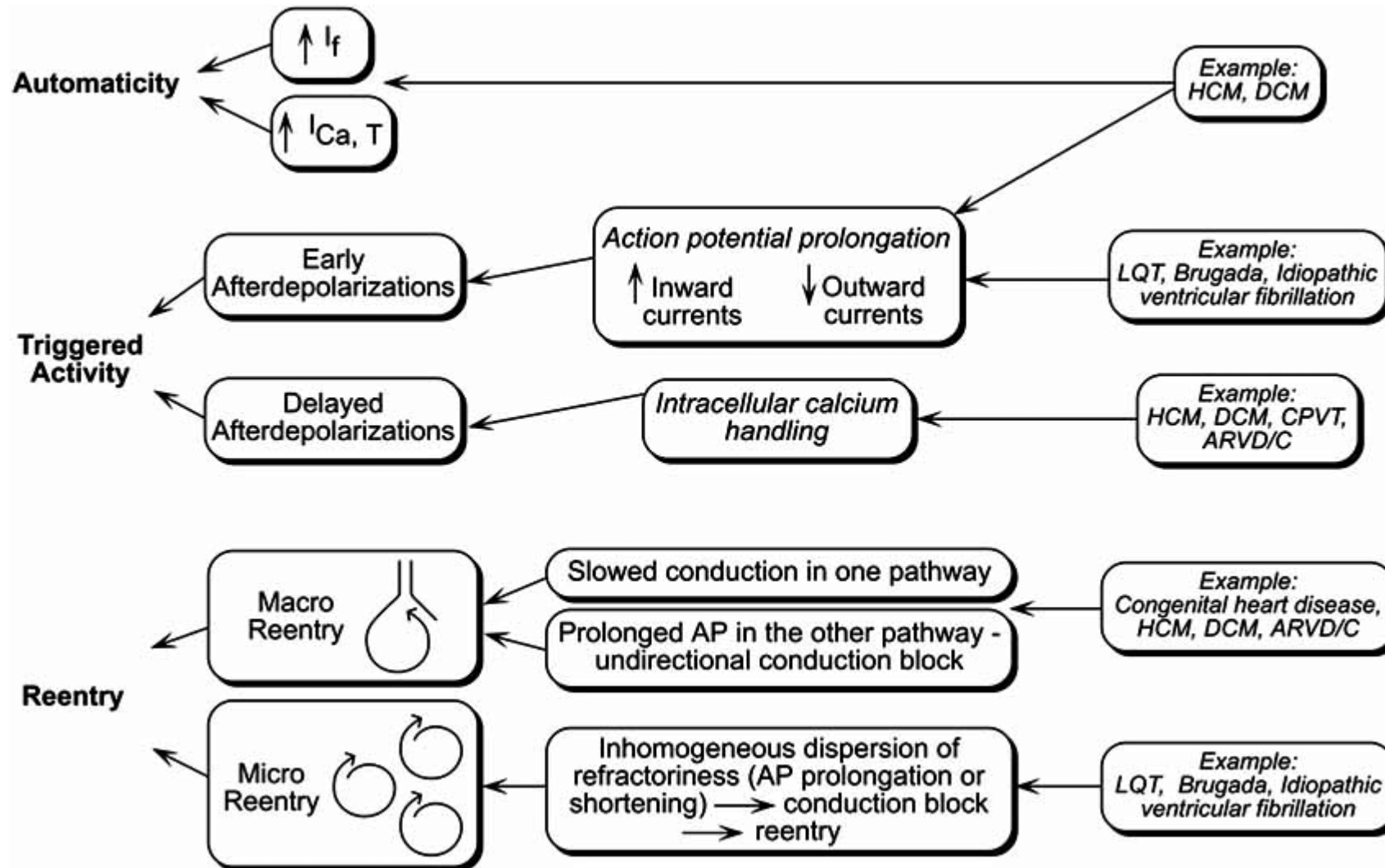
Possibili variabili:

- Micro e macro rientro
- Circuiti anatomici e funzionali

Il rientro rappresenta il meccanismo più frequente delle tachicardie a QRS largo



Electrophysiologic Mechanisms of Tachyarrhythmias



Classificazione Tachicardie a QRS largo

Ventricolari

- TV monomorfa
- TV polimorfa
- TV bidirezionale
- flutter ventricolare
- fibrillazione ventricolare

Sopraventricolari con aberranza

*Conduzione con blocco di
branca, fenomeno di
Ashman*

- Fibrillazione atriale
- Flutter atriale
- AVNRT
- AVRT
- Tachicardia atriale

Sopraventricolari preeccitate

*Conduzione attraverso via
accessoria*

- AVRT antidromica
 - FA preeccitata
- QRS molto largo, elevato rischio di FV

Tachicardie ventricolari monomorfe

Meccanismo prevalente:

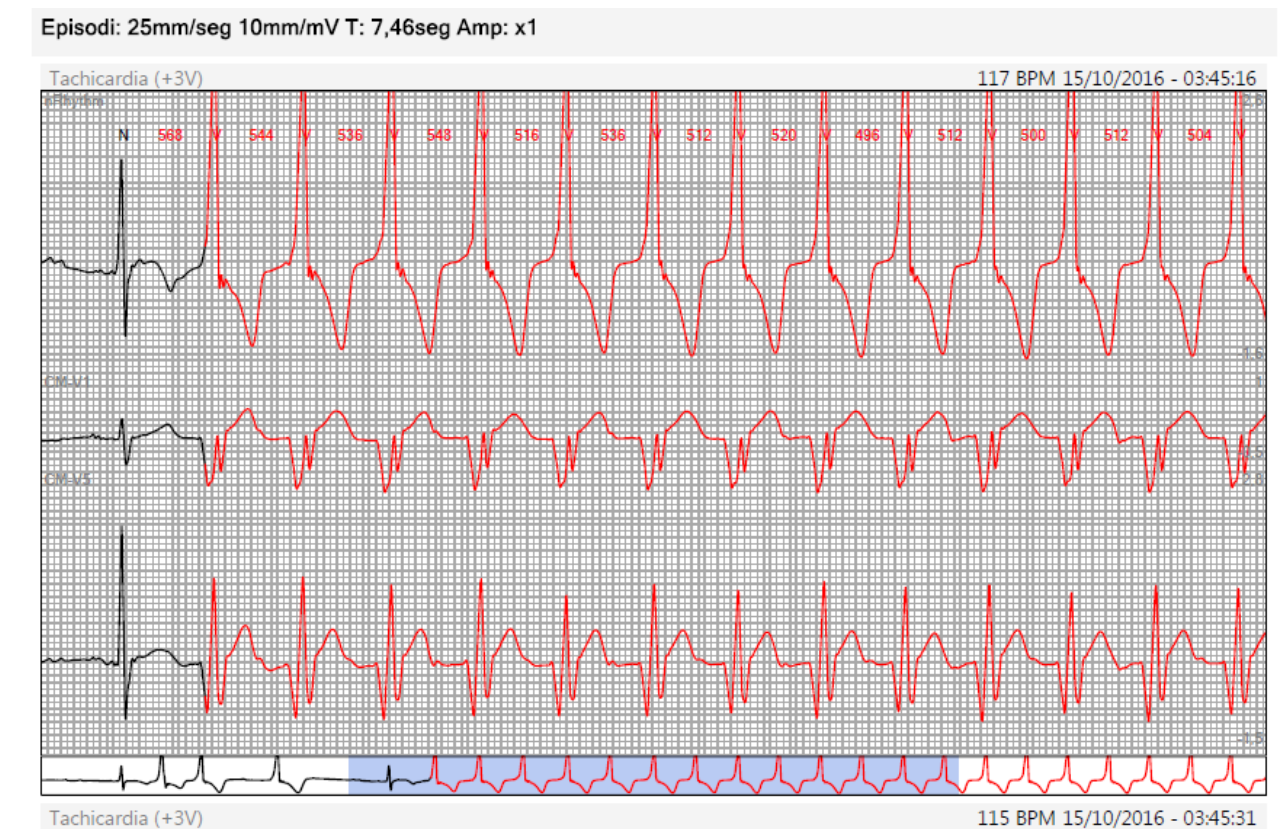
→ Rientro (+ automaticità)

Cause:

- Cardiopatia ischemica
- Cicatrici/fibrosi/sostituzione adiposa/miocardipatie da accumulo
- Cardiomiopatie (HCM, RVAD)

Aspetto ECG:

→ QRS a morfologia costante.



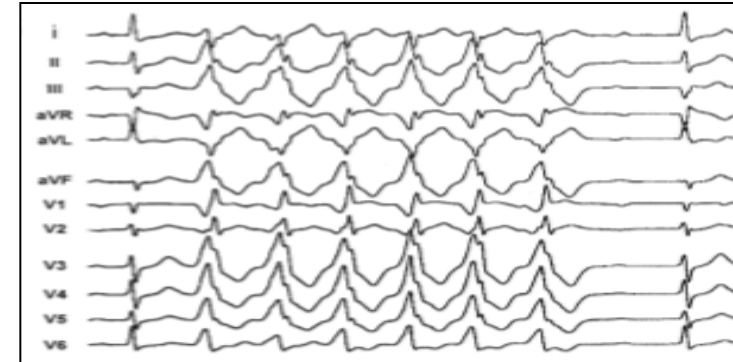
TF, Male, 20 yrs
HCM, Hx of Palpitation at effort

ARITMIE VENTRICOLARI DA TRATTO DI EFFLUSSO

FENOTIPI DI PRESENTAZIONE

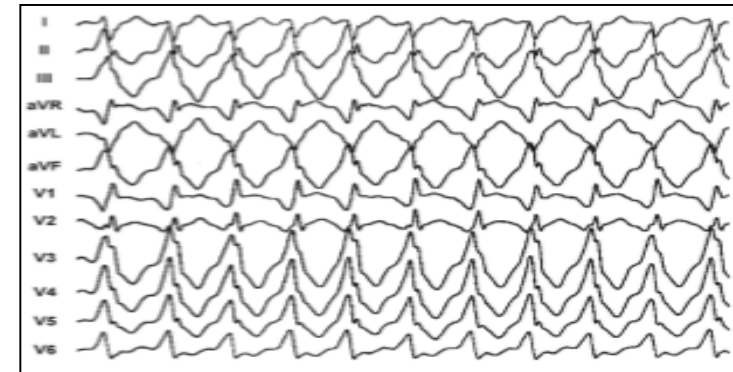
TVNS MONOMORFE, RIPETITIVE A RIPOSO

60-92% dei casi, talora carattere incessante



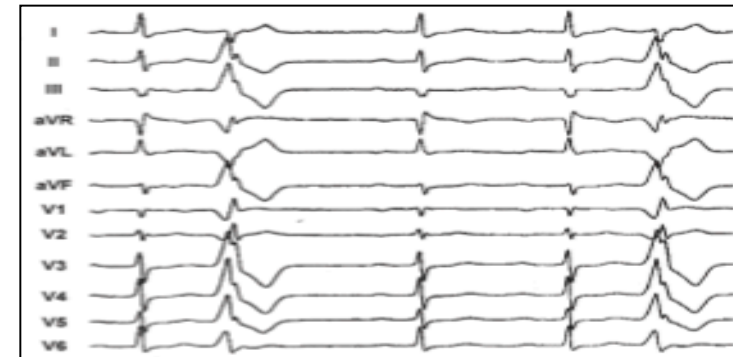
TVS MONOMORFA INDOTTA DA ESECIZIO O STRESS

solo occasionali

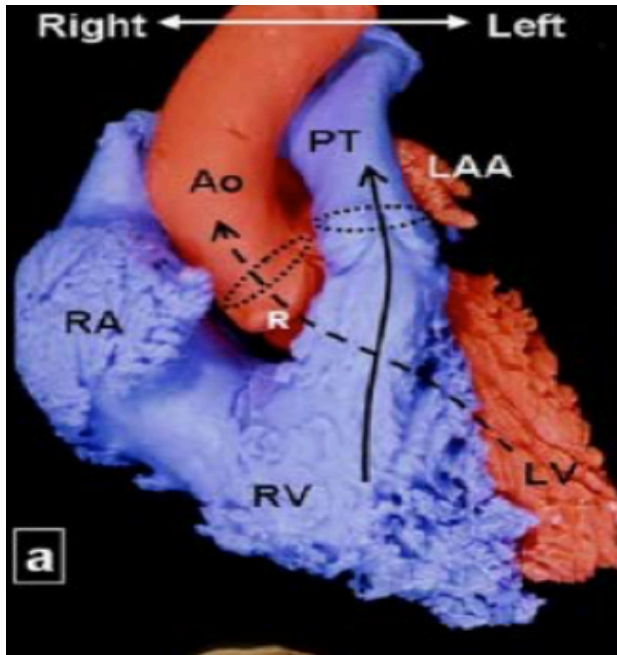


BATTITI EXTRASISTOLICI

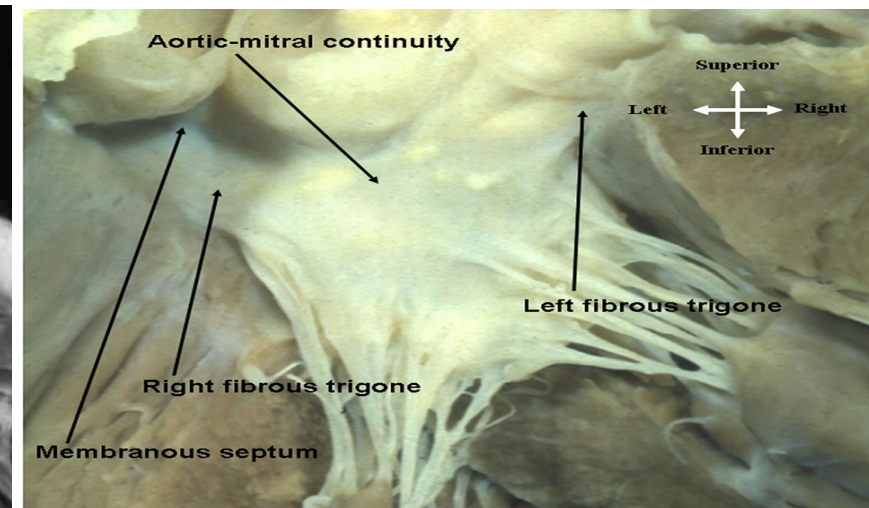
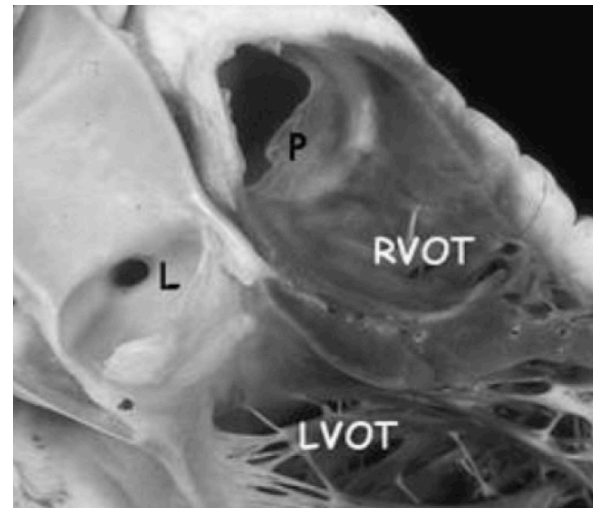
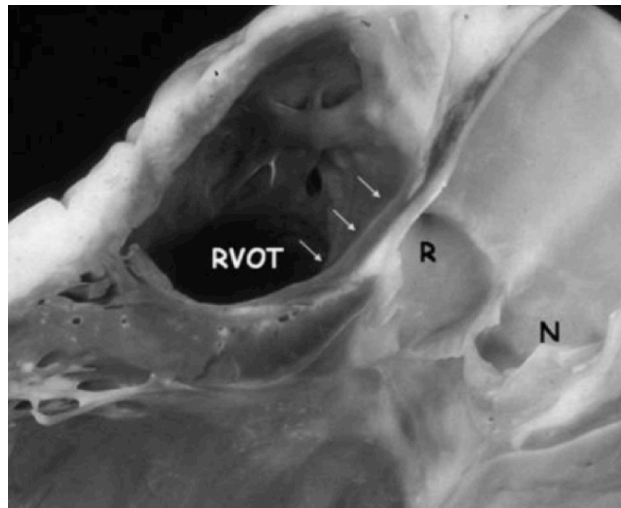
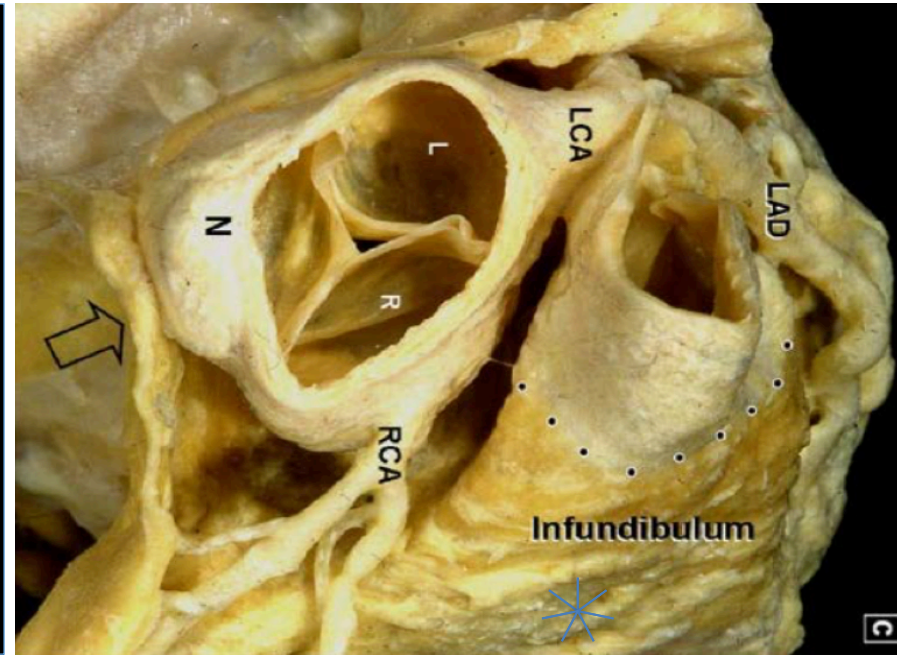
extrasistolia ventricolare isolata o in coppia;
in genere a riposo, nel recupero dallo
sforzo, si riducono durante l'esercizio



TV MONOMORFE IN ORIGINE DAL TRATTO DI EFFLUSSO



FIBRE
MIOCARDICHE
("MUSCULATURE
SLEEVES") SI
ESTENDONO AL DI
SOPRA DELLE
VALVOLE
SEMILUNARI NELLE
GRANDI ARTERIE



PATTERN ECG: RVOT VS LVOT TV

LA LOCALIZZAZIONE DEL “FOCUS” DELLA TV PUÒ ESSERE PREDETTA USANDO LA MORFOLOGIA DEL QRS DI SUPERFICIE

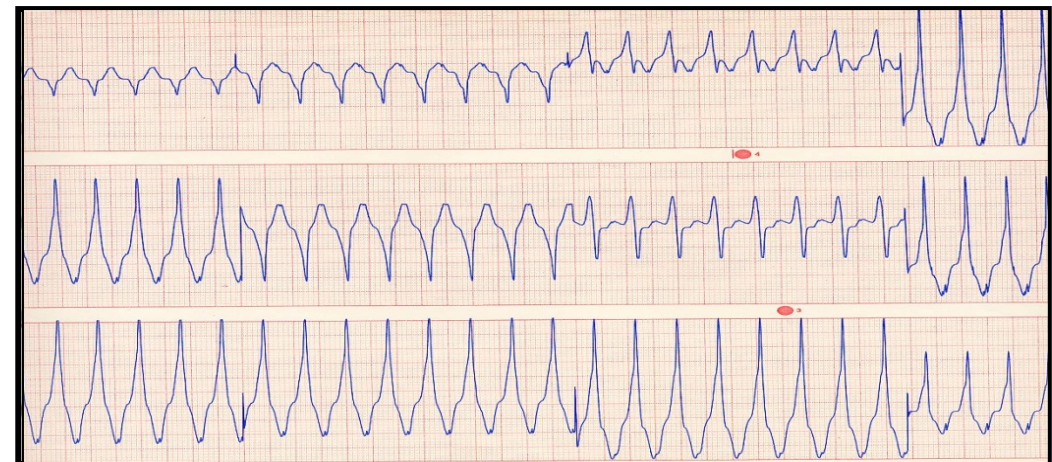
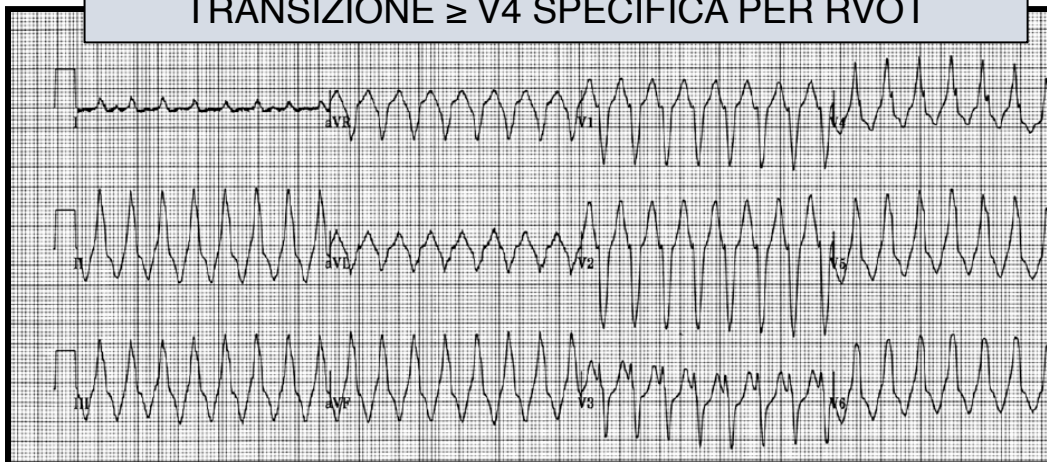
RVOT TV

- QRS allargato
- BBS
- Asse frontale inferiore
- **Transizione precordiale tardiva (V3-V5)**
- QS in aVR e aVL
- QS o rS in V1

LVOT TV

- QRS allargato
- BBS (70%) / BBDx (30%)
- Asse frontale inferiore
- **Transizione precordiale precoce (V1-V3) o BBDx**
- Asse più deviato a dx
- rS, RS o R in V1
- R alte nelle derivazioni inferiori

TRANSIZIONE $\geq$ V4 SPECIFICA PER RVOT



The site of origin of torsade de pointes

Edo Y Birati,¹ Bernard Belhassen,¹ Abdennasser Bardai,² Arthur A M Wilde,² Sami Viskin¹

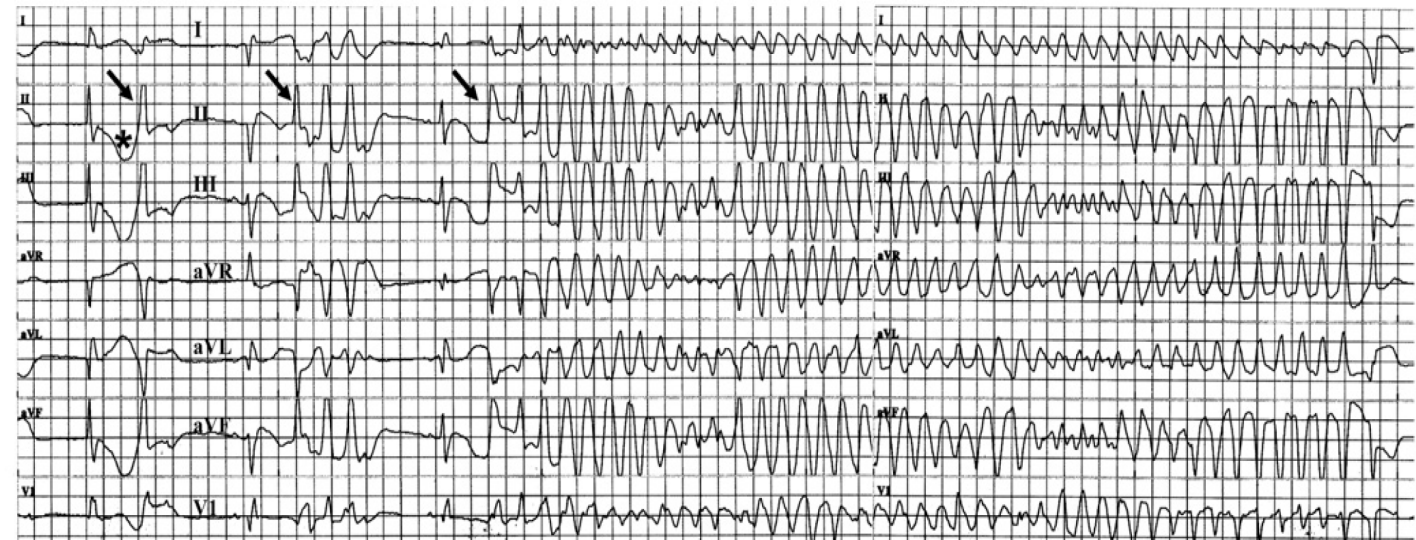
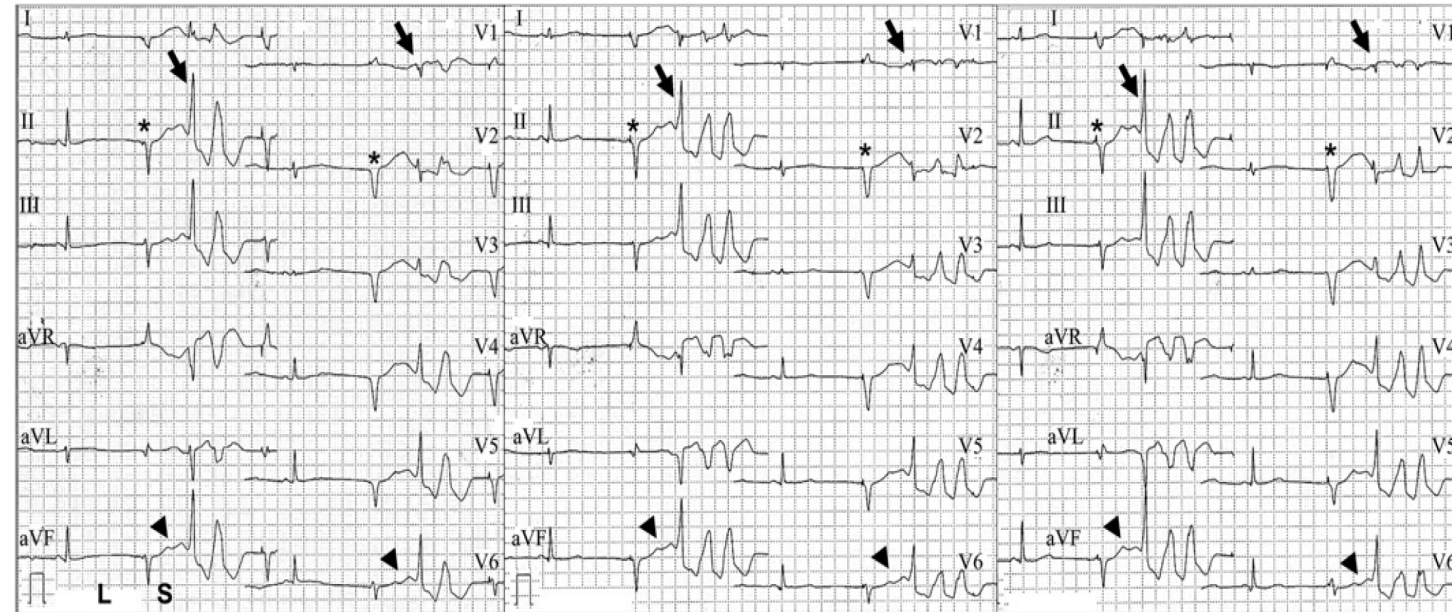
151 episodes of TdP and 874 QT-related extrasystoles (impending TdP) were available for **50 patients**.

The majority of episodes of TdP (56%) and most QT-related extrasystoles (70%) originated from the **outflow tract**.

There was no correlation between site of origin and the aetiology of LQTS or the QT duration.

Conclusions

The most frequent site of origin of TdP is the outflow tract. Further studies are needed to understand why this relatively small area of the ventricle is a **predominant site of origin of**



“MALIGNANT VARIANT” OF RVOT VT

The “Short-Coupled” Variant of Right Ventricular Outflow Ventricular Tachycardia: A Not-So-Benign Form of Benign Ventricular Tachycardia?

Viskin S, et al. J Cardiovasc Electrophysiol 2005;Vol.16,912-6

3 pz

relatively short coupling interval
(mean of 340 ms) of the PVCs

Malignant Entity of Idiopathic Ventricular Fibrillation and Polymorphic Ventricular Tachycardia Initiated by Premature Extrasystoles Originating From the Right Ventricular Outflow

Noda T, et al. JACC 2005;Vol.46,N.7

16 pz

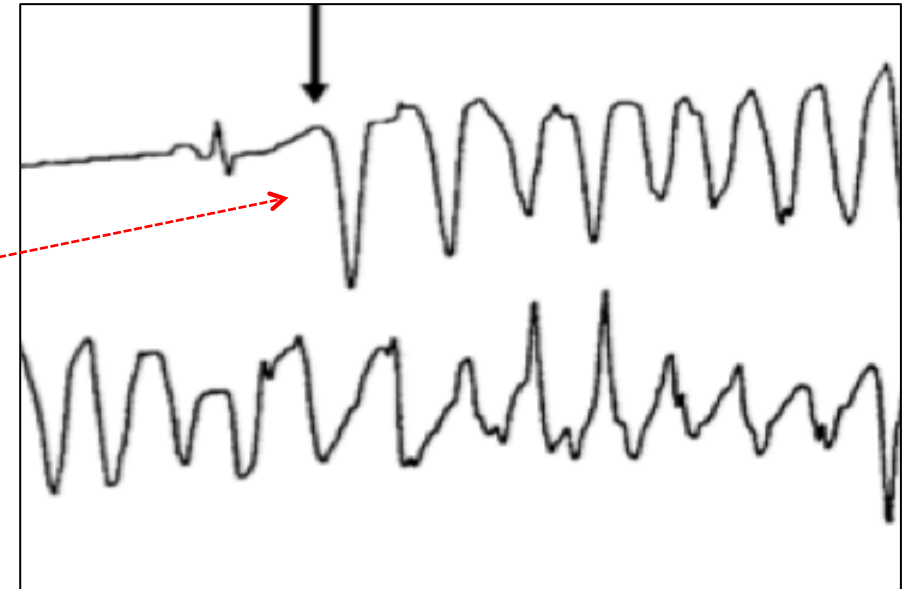
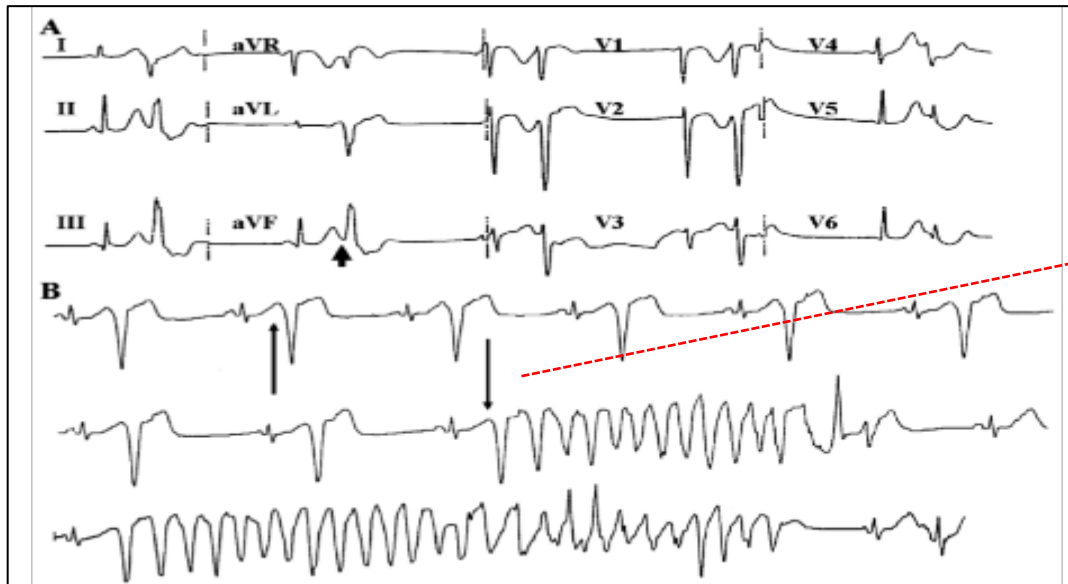
not “short” coupling interval
(mean 409 ms) of the PVCs

Mapping and Ablation of Idiopathic Ventricular Fibrillation

Haissaguerre M, et al. Circulation 2002;106:962-7

23 pz

very short-coupled
(mean 297 ms) of the PVCs



Tachicardia Ventricolare Polimorfa

Meccanismo prevalente:

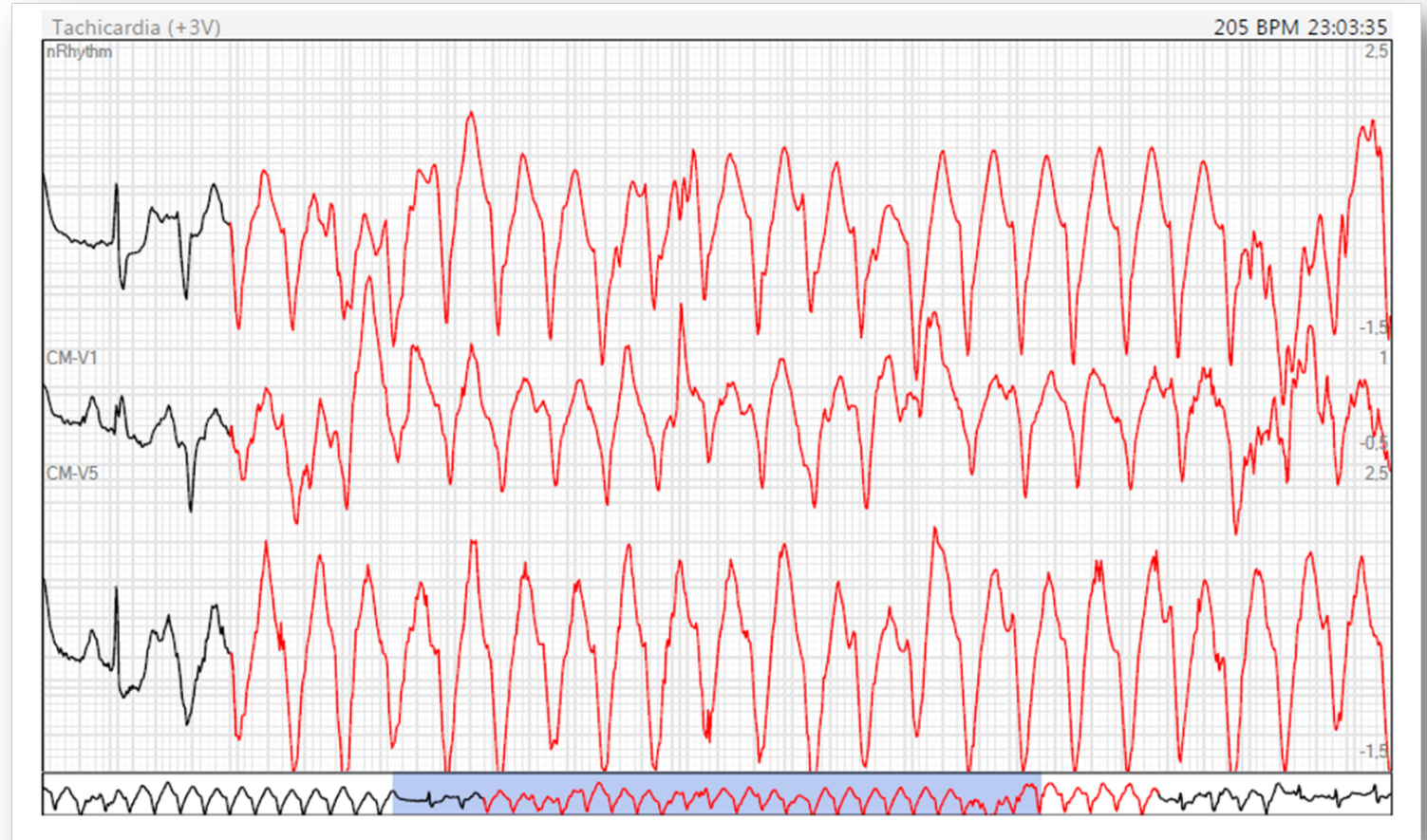
→ Triggered activity/Rientro

Cause:

- Canalopatie (LQTS, Brugada)
- Catecolamine PCVT
- Effetto proaritmico (QT lungo iatrogeno)
- Ischemia

Aspetto ECG

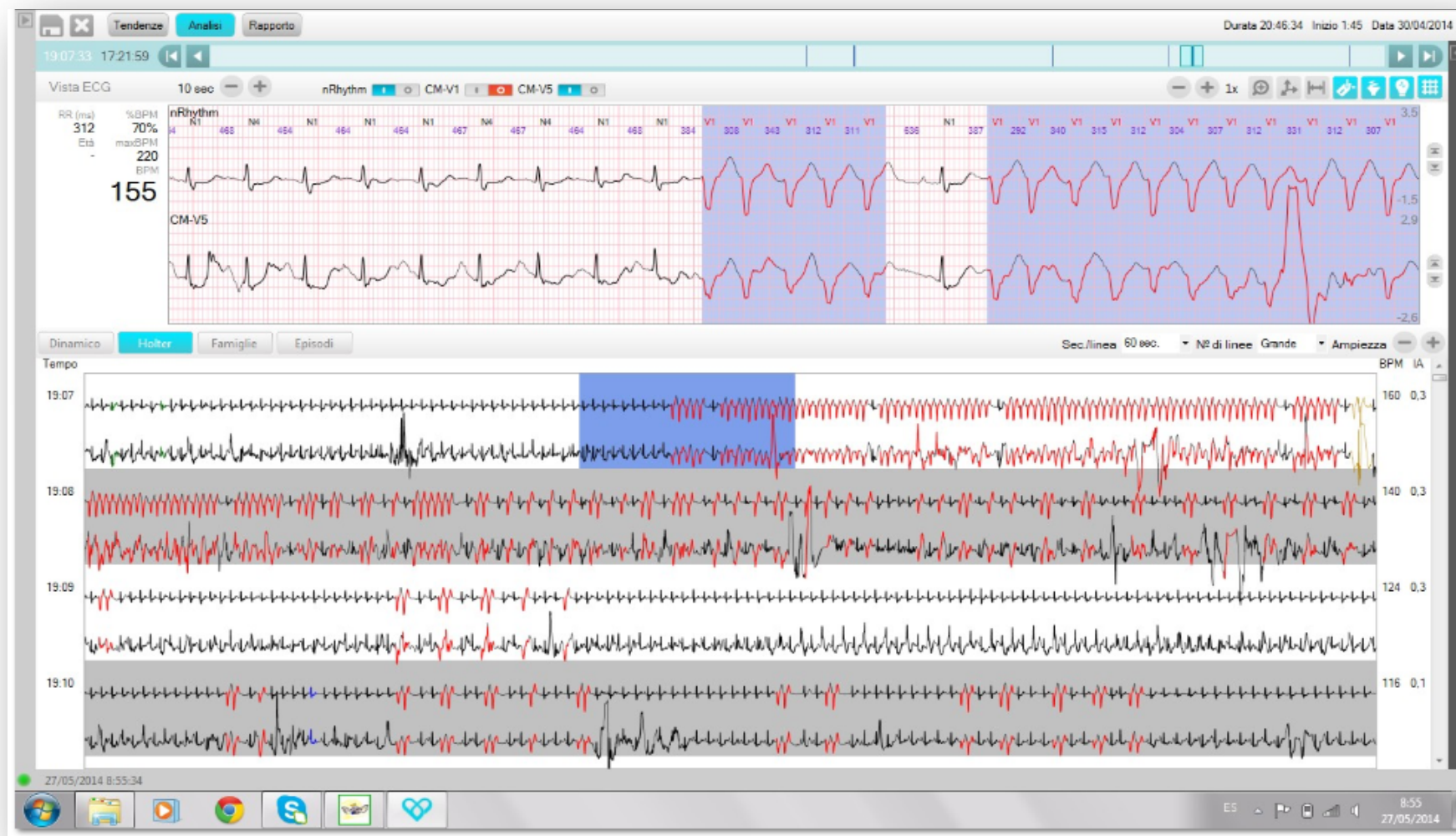
→ Morfologia QRS continuamente variabile



Tachicardia Ventricolare Polimorfa

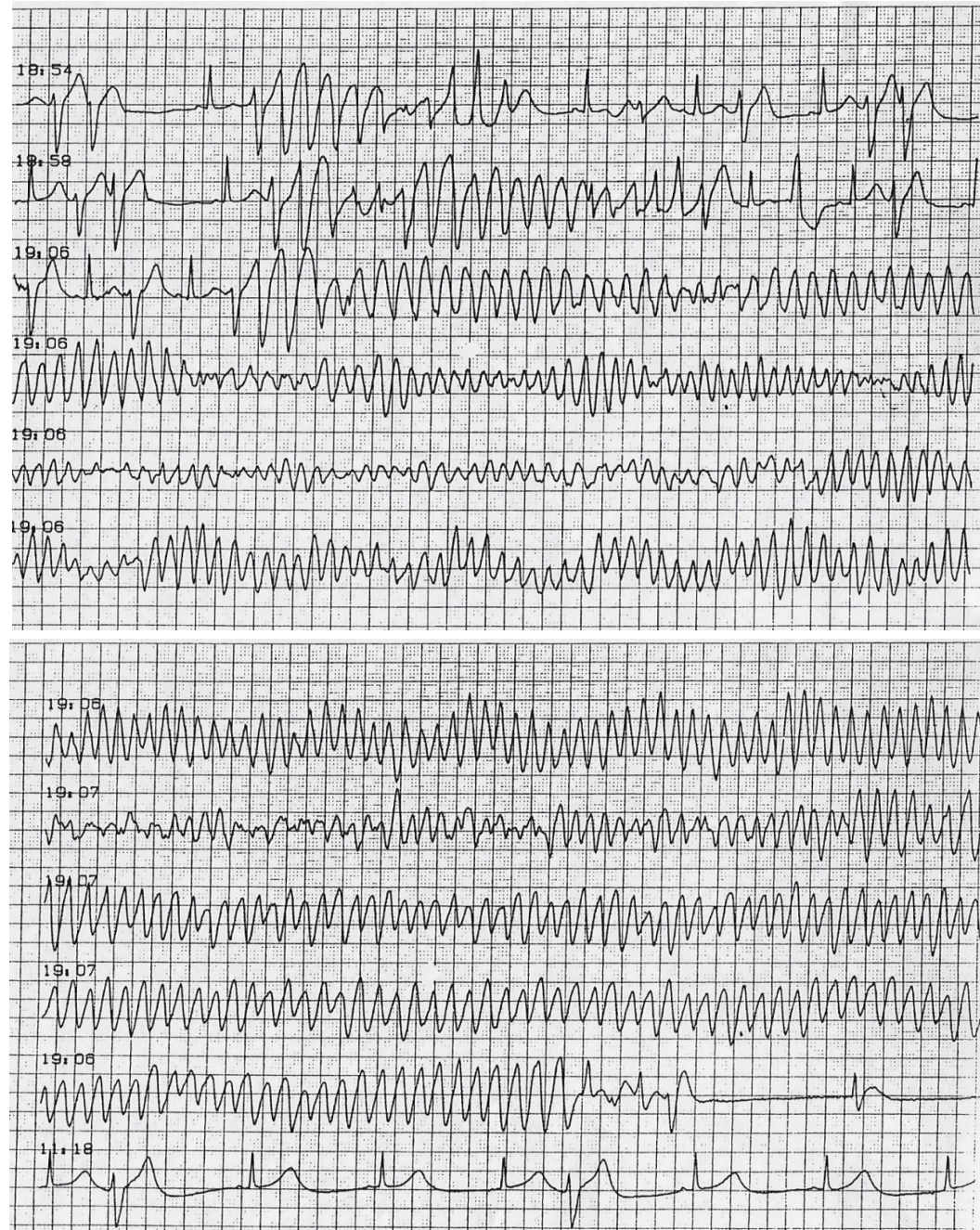
AMG,
Femmina
24 anni
Palpitazioni
Da sforzo,
Sorella con
morte
improvvisa

ECG: QT lungo
RMNC: Strie
fibrotiche
multiple
Genotipo:
mutazione di
RYR2

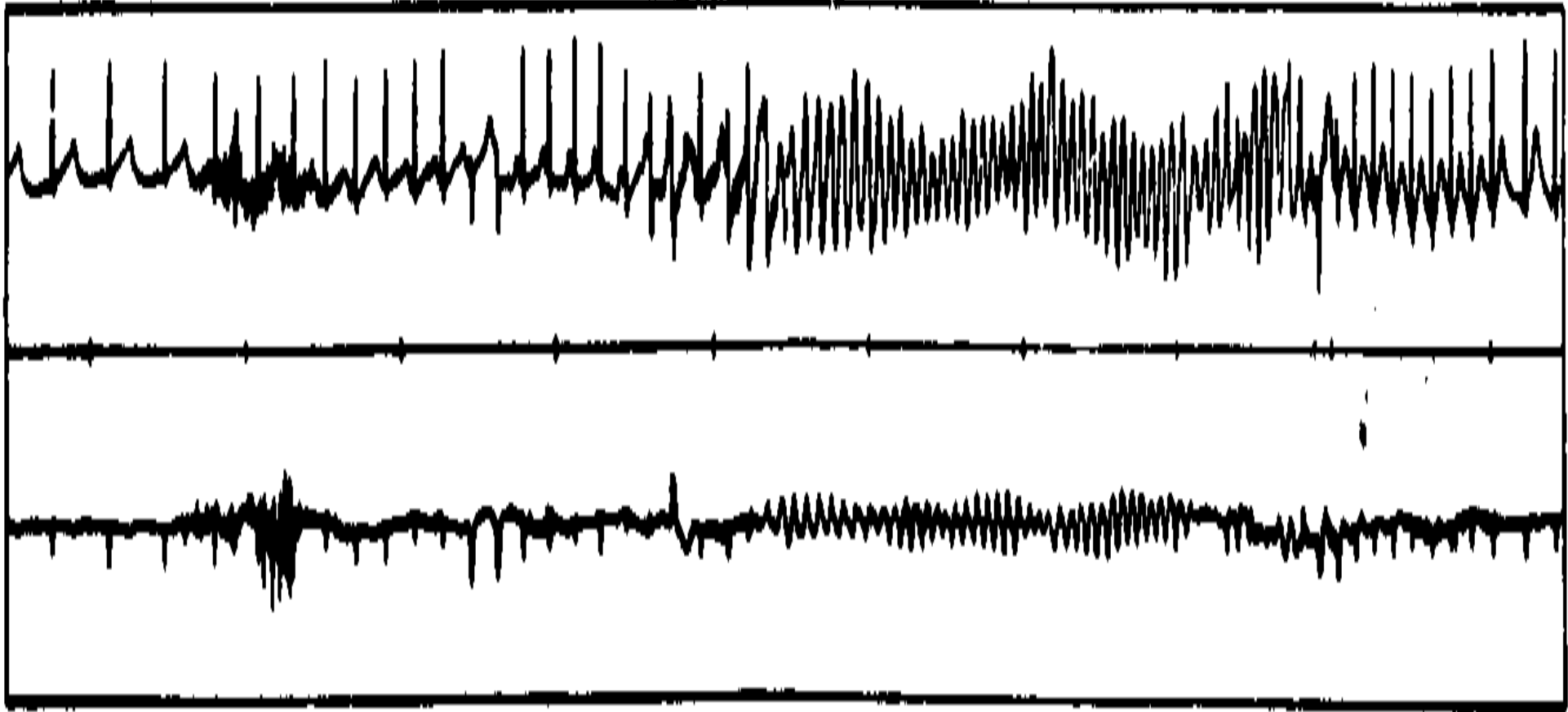


Self-terminating Torsades-de-pointes may explain frequent syncope in congenital LQTS

19 yr old female, history of
repetitive syncope, QTc interval
540 msec, TdP lasting about 2
minutes

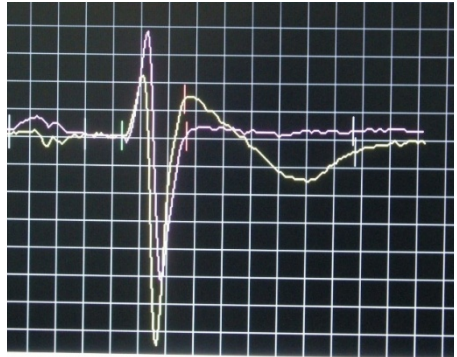


TdP in Congenital LQT2 During Sleep



Female, 19 yrs, Syncope during nocturnal arousal and nightmares

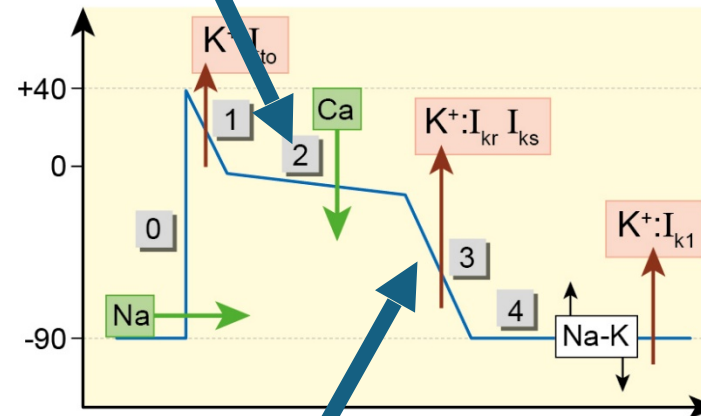
Due modelli di Tachicardie Ventricolari da rientro secondarie a «dispersione della ripolarizzazione»



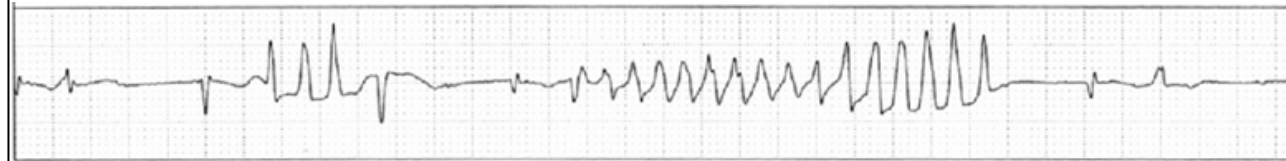
Dispersione da QT accordato:
modello IperK, SQTS, S.Brugada, isch:
Rientro in fase 2



Tachicardia Polimorfa

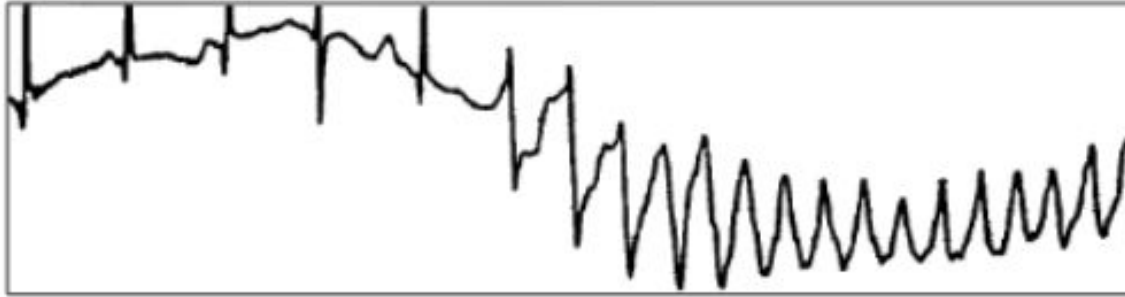


Dispersione da QT prolungato
modello IpoK, LQTS, AAD classe III :
Rientro in fase 3



Torsione di Punta:

Initiation of TdP by Genotypes in LQTS

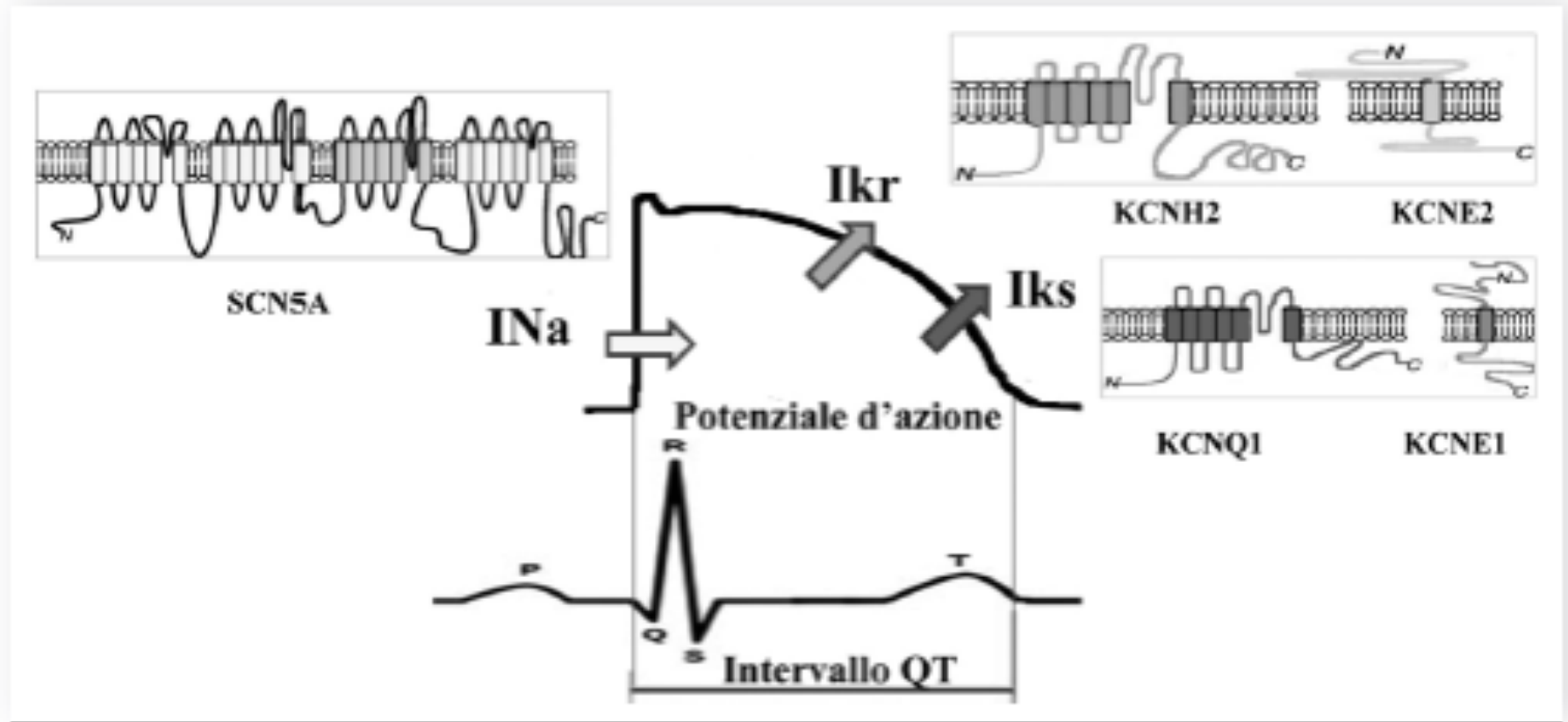


- Non-pause dependent torsade de pointes
- Mainly in LQT1



- Pause dependent torsade de pointes
- Mainly in LQT2

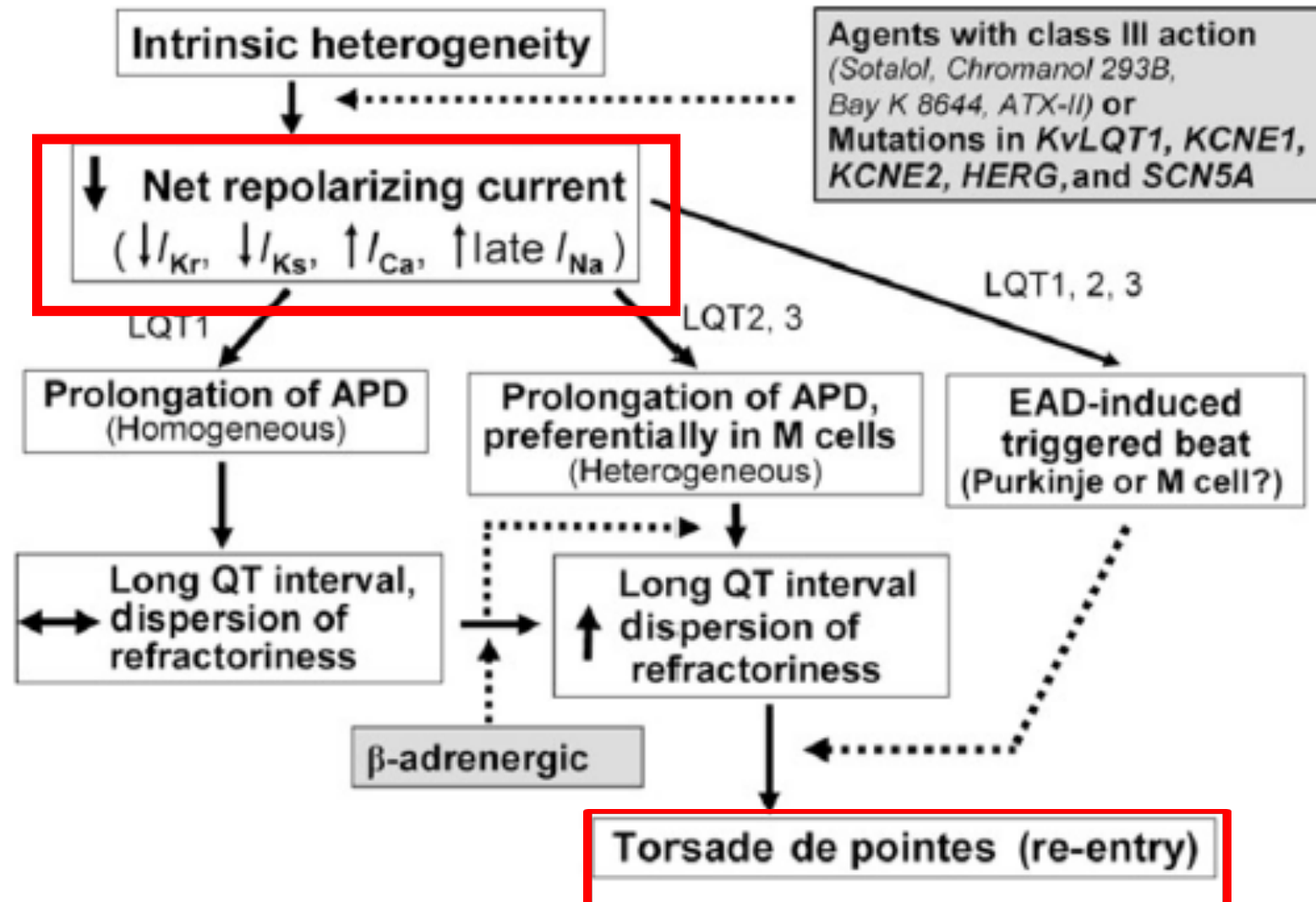
Main Cardiac Ion Channels Involved in the Genesis of the 3 Main Variants of Congenital LQTS



- **KCNQ1 (LQT1)** and **KCNE1 (LQT5)** encode respectively for the subunits α and β of the channel carrying the current **I_{Ks}** .
- **KCNH2 (LQT2)** and **KCNE2 (LQT6)** genes encode respectively the α and β subunits of the channel conducting the **I_{Kr} current**.
- **SCN5A gene (LQT3)** encoding for the α subunit of the sodium channel that conducts the **current I_{Na}** .

I_{Na} , sodium depolarizing current; **I_{Kr}** , rapid component of potassium repolarizing current; **I_{Ks}** , slow component of

Cellular and Ionic Mechanisms for the LQTS



THE MUTATIONS RESPONSIBLE FOR LONG QT SYNDROME

Type	Locus	Gene	Protein	Current	Effect	Frequency, %
Romano-Ward (autosomal dominant)						
LQTS1	11p15.5	<i>KCNQ1/KVLQT1</i>	Principal, I_{Ks} α -subunit	K	↓	30-35
LQTS2	7q35-36	<i>KCNH2/HERG</i>	Principal, I_{Kr} α -subunit	K	↓	25-30
LQTS3	3p21-p24	<i>SCN5A</i>	Principal, I_{Na} α -subunit	INa	↑	5-10
LQTS4	4q25-q27	<i>ANKB</i>	Accessory, ankyrin- β	Na/Ca	↑	<1
LQTS5	21q22.1	<i>KCNE1/minK</i>	Accessory, I_{Ks} β -subunit	K	↓	<1
LQTS6	21q22.1	<i>KCNE2/MiRP1</i>	Accessory, I_{Kr} β -subunit	K	↓	<1
LQTS7 ^a	17q23	<i>KCNJ8</i>	Principal, K_{ir} 2.1 α -subunit	K	↓	<1
LQTS8 ^b	12p13.3	<i>CACNA1</i>	Principal, C_{av} 1.2 α -subunit	Ca type L	↑	<1
LQTS9	3p25	<i>CAV3</i>	Accessory, caveolin 3	Na	↑	<1
LQTS10	11q23	<i>SCN4B</i>	Accessory, I_{Na} β 4-subunit	Na	↑	<1
Jervell-Lange-Nielsen (autosomal recessive)						
JLN1	11p15.5	<i>KCNQ1/KVLQT1</i>	Principal, I_{Ks} α -subunit	K	↓	>90.5
JLN	21q22.1	<i>KCNE1/minK</i>	Accessory, I_{Ks} β -subunit	K	↓	<0.5

^aAndersen-Tawil Syndrome

^bTimothy syndrome

Genetic screening identifies a disease-causing mutation in 70% of LQTS cases and three main genes (*KCNQ1*, *KCNH2* and *SCN5A*) account for 90% of positively genotyped cases.



An International, Multicentered, Evidence-Based Reappraisal of Genes Reported to Cause Congenital Long QT Syndrome

Clinical Perspective

What Is New?

- Of 17 genes previously reported as causing long QT syndrome (LQTS), 9 were found to have limited or disputed evidence for disease causation.
- Only 3 genes were found to have definitive evidence for causation of typical LQTS.
- Four genes are definitively associated with LQTS with atypical features that can include QT prolongation associated with neonatal heart block or autosomal-recessive inheritance.

What Are the Clinical Implications?

- Genes with limited or disputed evidence for disease causation should not be routinely tested in the evaluation of patients and families with LQTS, as interpretation of any identified variants cannot appropriately be classified in relation to disease.
- Genetic testing for genes (*CALM1*, *CALM2*, *CALM3*, *TRDN*) in LQTS with atypical features should be considered in patients with clinical observations concordant with the specific phenotypic expression demonstrated in reported cases.

Table 2. Classification of Genetic Evidence for Genes Previously Reported as Causing LQTS

Gene	LQTS	aLQTS	Syndromic*
<i>AKAP9</i>	Disputed		
<i>ANK2</i>	Disputed		
<i>CACNA1C</i>	Moderate		Definitive (Timothy syndrome)
<i>CALM1</i>	Definitive†		
<i>CALM2</i>	Definitive†		
<i>CALM3</i>	Definitive†		
<i>CAV3</i>	Limited		
<i>KCNE1</i>	Limited	Strong	
<i>KCNE2</i>	Disputed	Strong	
<i>KCNH2</i>	Definitive		
<i>KCNJ2</i>	Limited		Definitive (Andersen-Tawil syndrome)
<i>KCNJ5</i>	Disputed		
<i>KCNQ1</i>	Definitive		
<i>SCN4B</i>	Disputed		
<i>SCN5A</i>	Definitive		
<i>SNTA1</i>	Disputed		
<i>TRDN</i>	Strong‡		

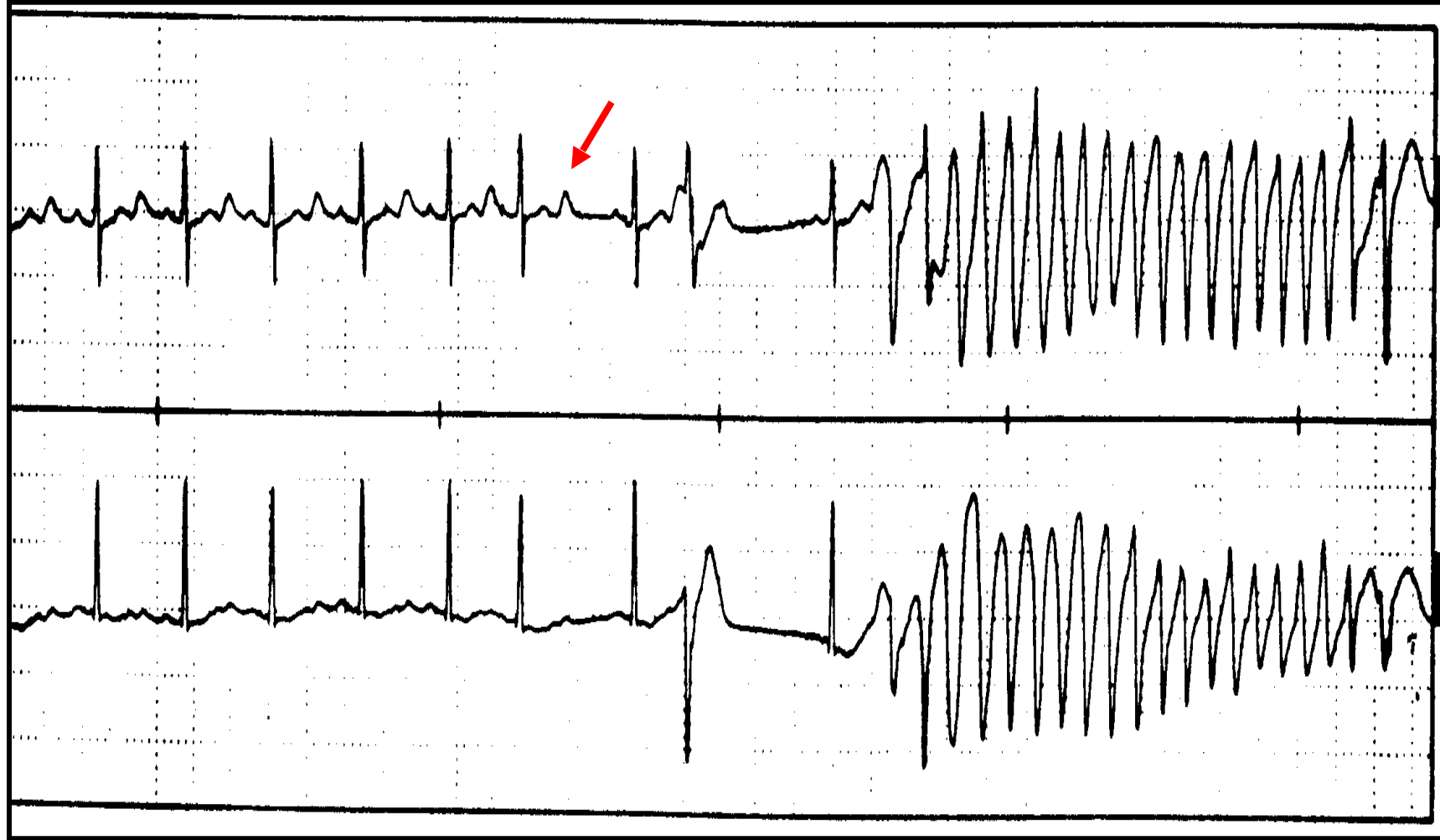
aLQTS indicates acquired long QT syndrome; and LQTS, congenital long QT syndrome.

*Multiorgan syndrome including QT prolongation and cardiac arrhythmias.

†LQTS presenting in infancy or early childhood with heart block and severe QT prolongation.

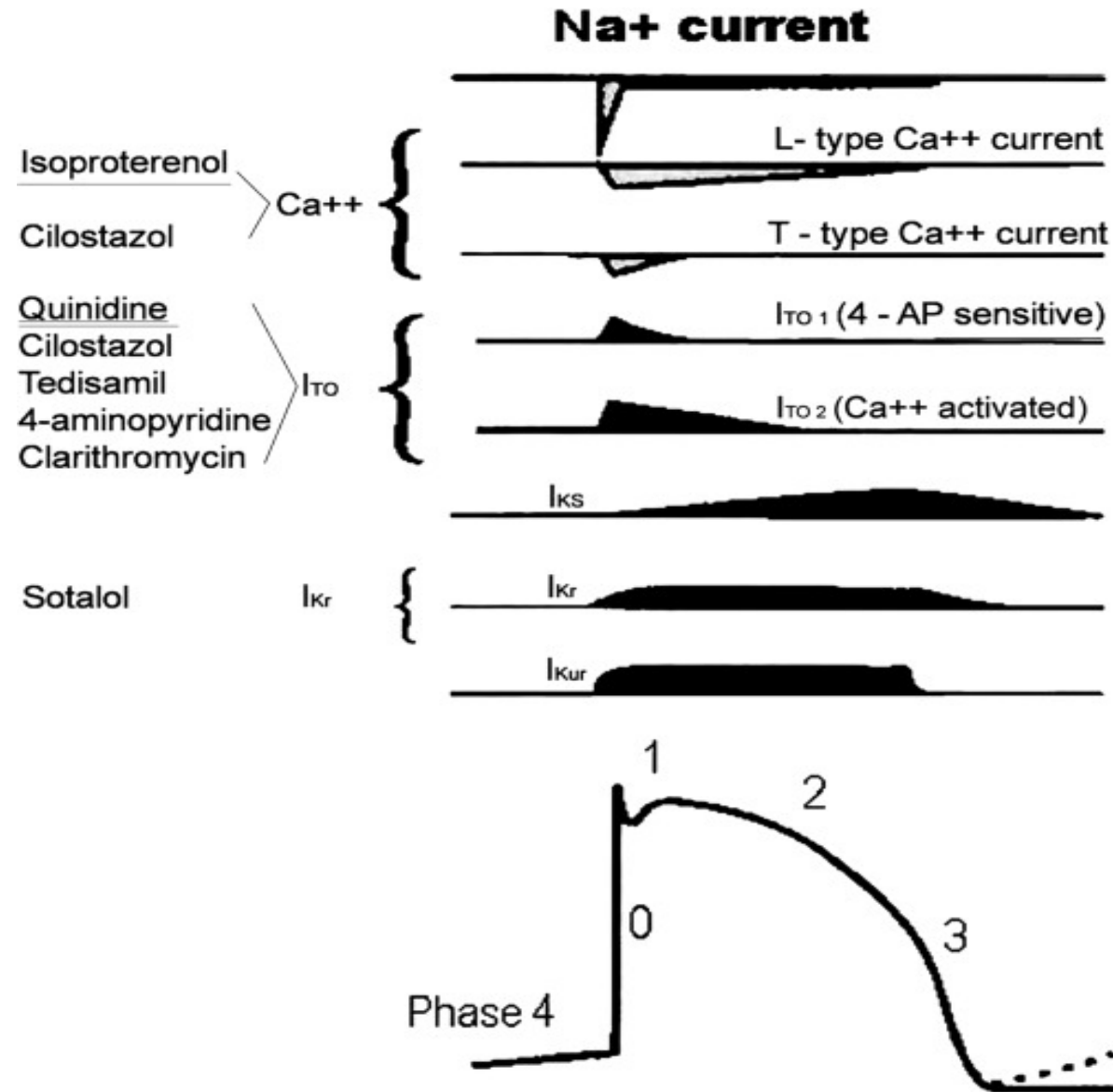
‡QT prolongation, negative T waves in precordial leads, and exercise-induced arrhythmias in early childhood related to homozygous or compound heterozygous frameshift mutations.

Quinidine-Induced Torsades-de-Pointes: *Long QT, Pause-Dependence and Tachycardia*

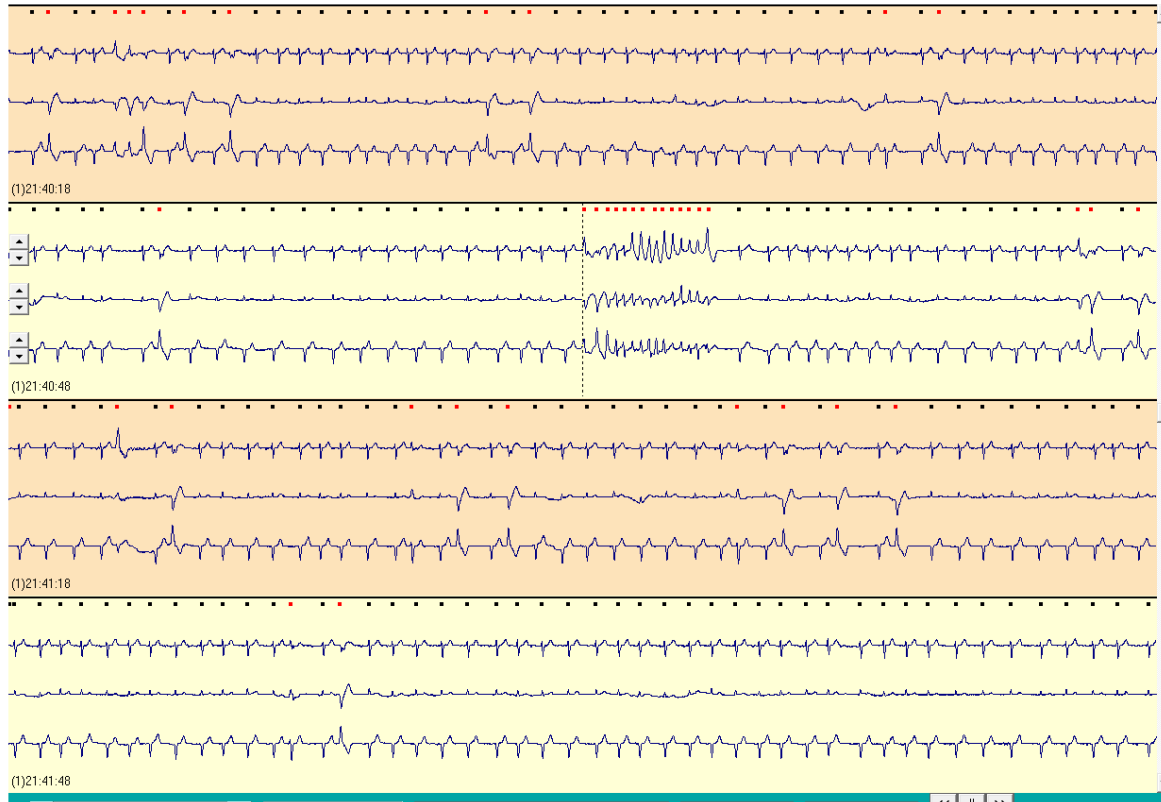


Locati EH et al, JACC 1992

Cardiac Ionic Currents and Antiahythmic Drugs



Un caso di morte improvvisa annunciata?



AR, maschio, 69 yrs, CAD, FAC, TVNS polimorfa a Holter, FE 50%, SEF negativo, morte improvvisa dopo 6 mesi dal SEF (non impiantato ICD)

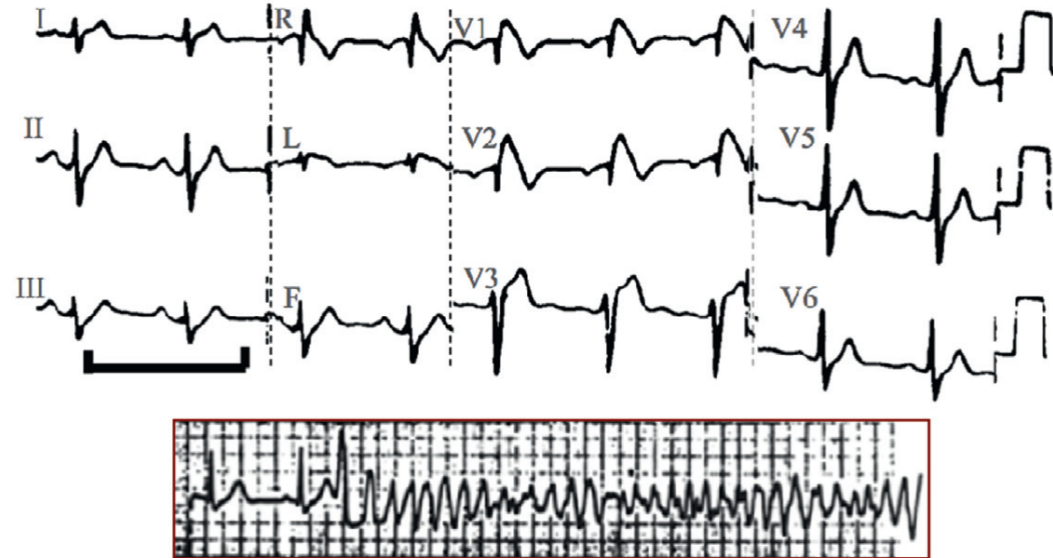
Right Bundle Branch Block, Persistent ST Segment Elevation and Sudden Cardiac Death: A Distinct Clinical and Electrocardiographic Syndrome

A Multicenter Report

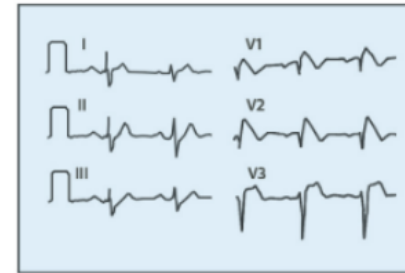
PEDRO BRUGADA, MD, JOSEP BRUGADA, MD*†

Aalst, Belgium and Barcelona, Spain

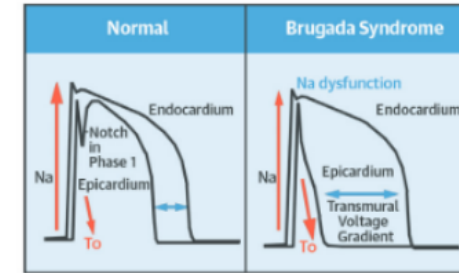
Objectives. The objectives of this study were to present data on eight patients with recurrent episodes of aborted sudden death whom ventricular biopsies were performed. The arrhythmia leading to (aborted) sudden death was a rapid polymorphic



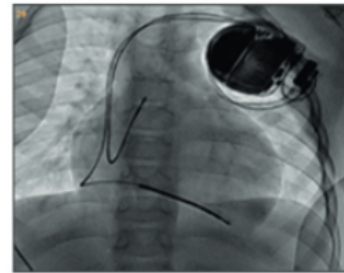
Brugada Syndrome



Coved type ST-segment in V1-V2

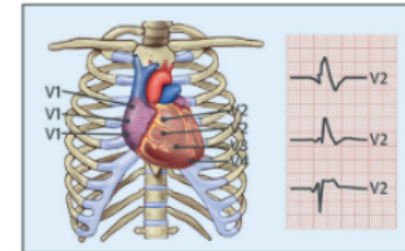


Loss of function of sodium channels

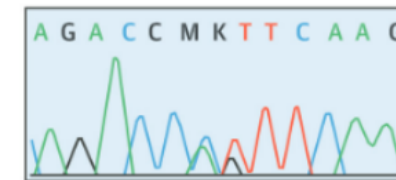


ICD is standard therapy, epicardial radiofrequency ablation a promising one

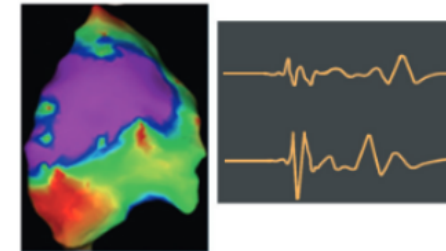
Diagnosis



Pathophysiology



Management



Brugada, J. et al. J Am Coll Cardiol. 2018;72(9):1046-59.

From Substrate to ECG and Symptoms in BRS

ECG



Symptoms



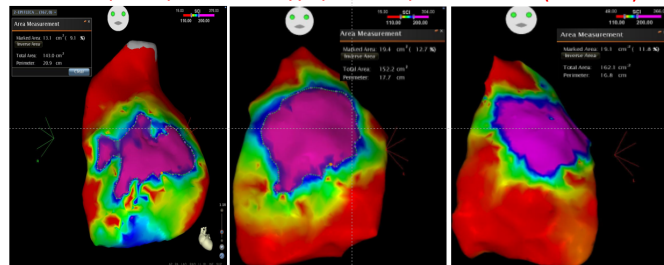
Family history

Monozygotic Twins

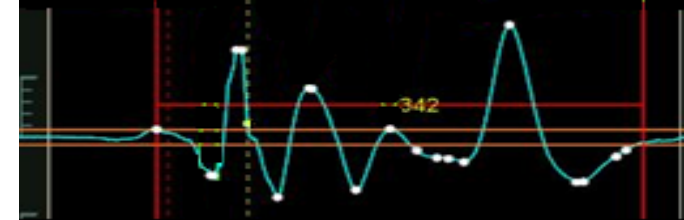
Paola (Mother)

Filippo (Son 1° Twin)

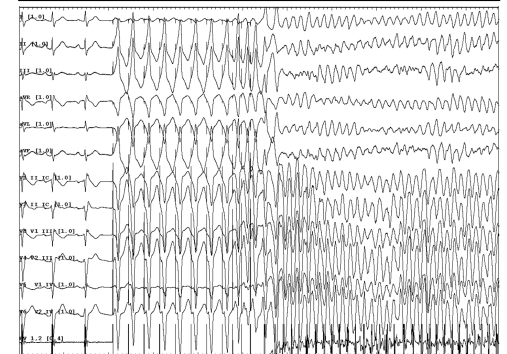
Lorenzo (Son 2° Twin)



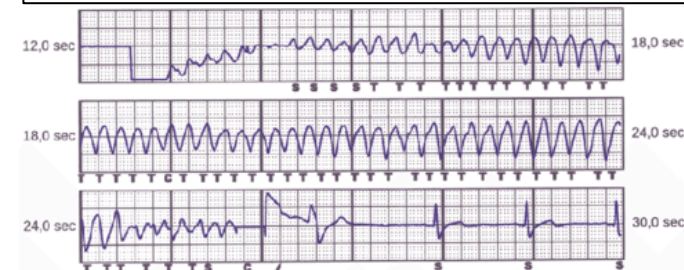
EGM Duration



Inducibility



VA





ESC

European Society
of Cardiology

European Heart Journal (2020) 00, 1–9
doi:10.1093/eurheartj/ehaa942

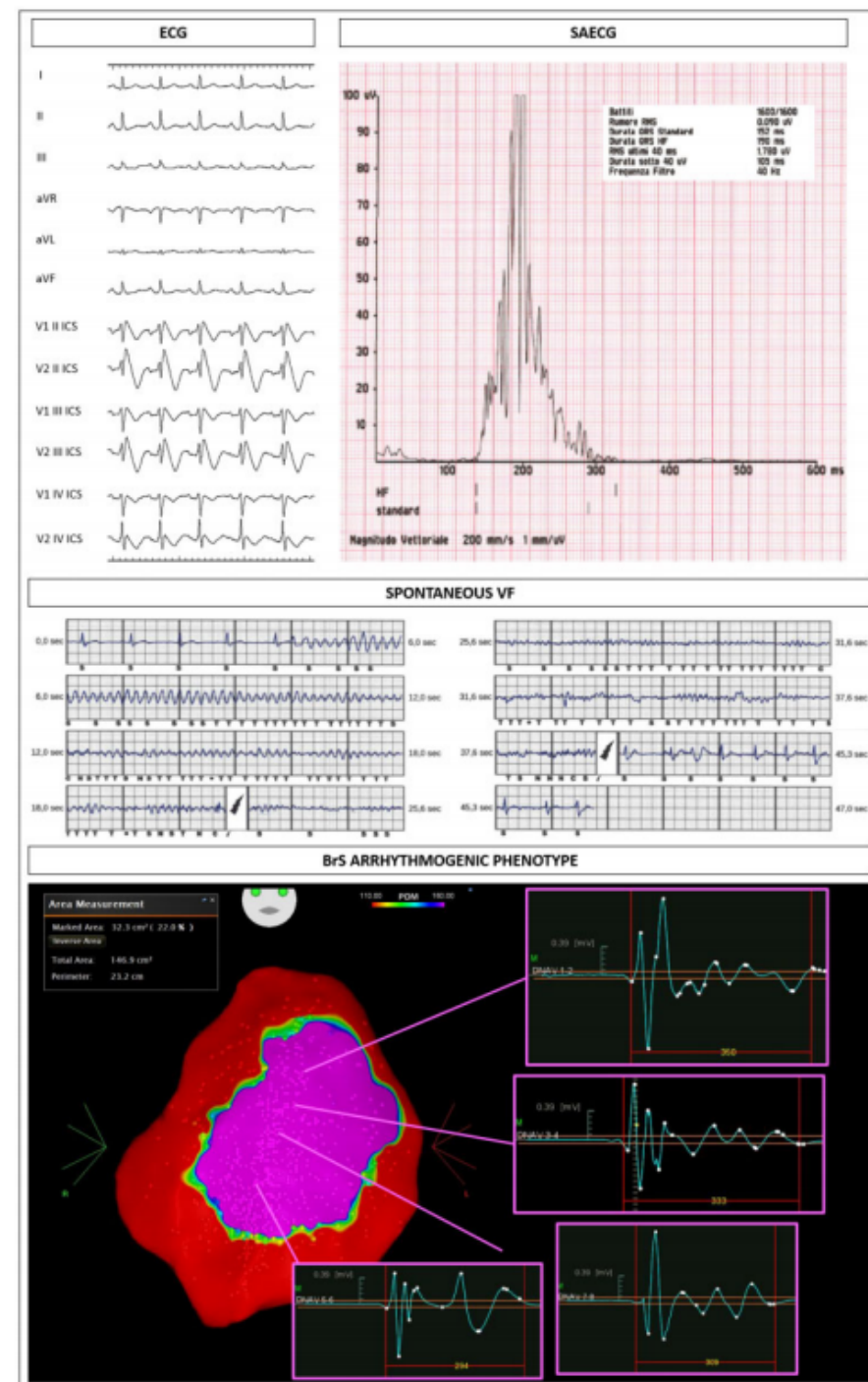
CLINICAL RESEARCH

Arrhythmias

Brugada syndrome genetics is associated with phenotype severity

Giuseppe Ciconte ¹, Michelle M. Monasky ¹, Vincenzo Santinelli¹, Emanuele Micaglio ¹, Gabriele Vicedomini¹, Luigi Anastasia ^{2,3}, Gabriele Negro¹, Valeria Borrelli ¹, Luigi Giannelli¹, Francesca Santini¹, Carlo de Innocentiis¹, Roberto Rondine ¹, Emanuela T. Locati ¹, Andrea Bernardini¹, Beniamino C. Mazza ¹, Valerio Mecarocci¹, Žarko Calović¹, Andrea Ghiroldi ², Sara D'Imperio ², Sara Benedetti ⁴, Chiara Di Resta^{3,4}, Ilaria Rivolta ⁵, Giorgio Casari ^{3,4}, Enrico Petretto ⁶, and Carlo Pappone ^{1,3*}

- 200 BrS Probands with genetic and phenotype assessment
- Pts with SCN5A mutations had **more cardiac arrest/VT-VF** and exhibited **larger epicardial arrhythmogenic substrate area**
- SCN5A mutations explained the 26% of the variation of the substrate area being the strongest predictor of a large substarte



Brugada
phenoc

Tal

.....

Mal

Age

Spc

Fan

Abc

Syn

Spc

Indi

Pre

A

A

Pre

PQ

QR

f-Q

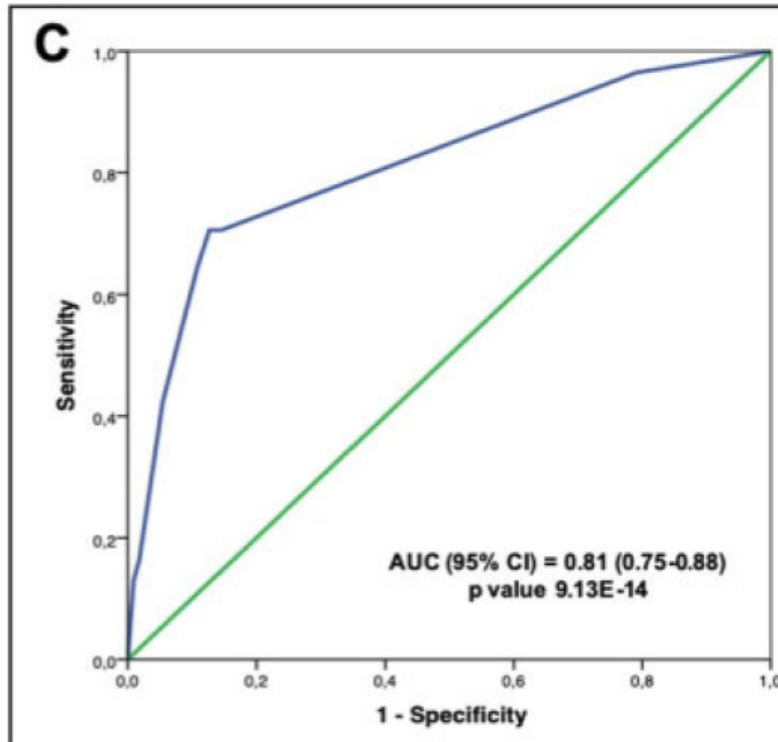
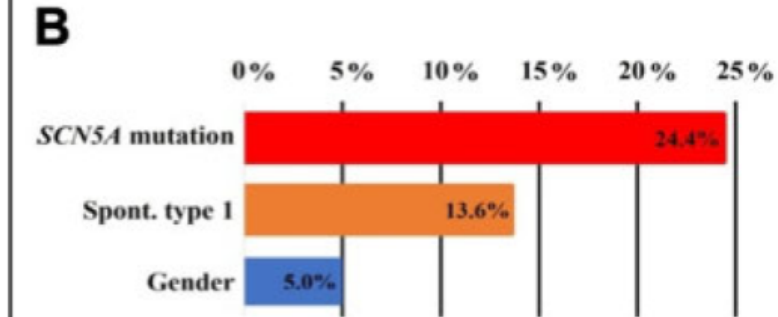
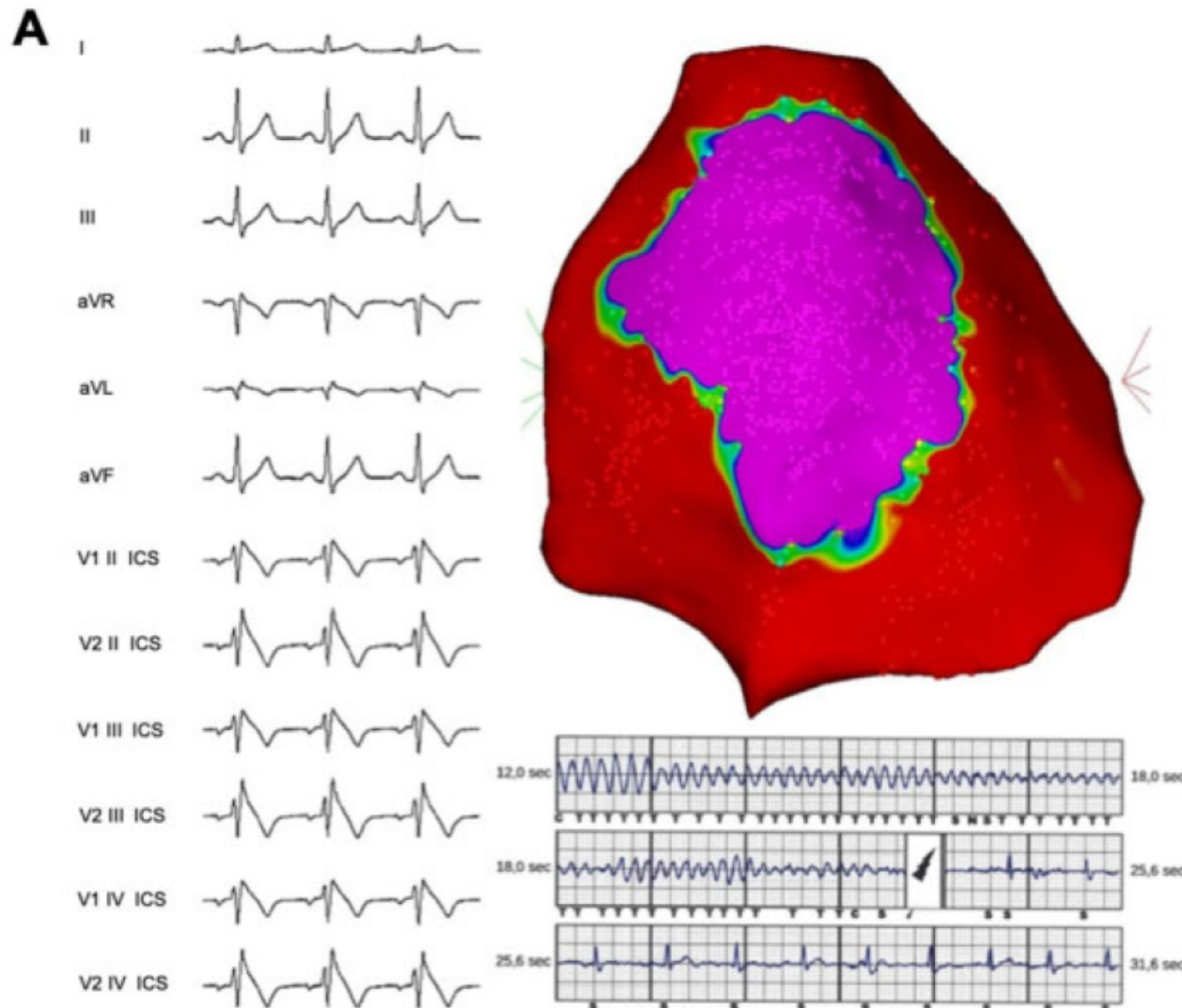
RM

LA:

Arr

Bas

Sub

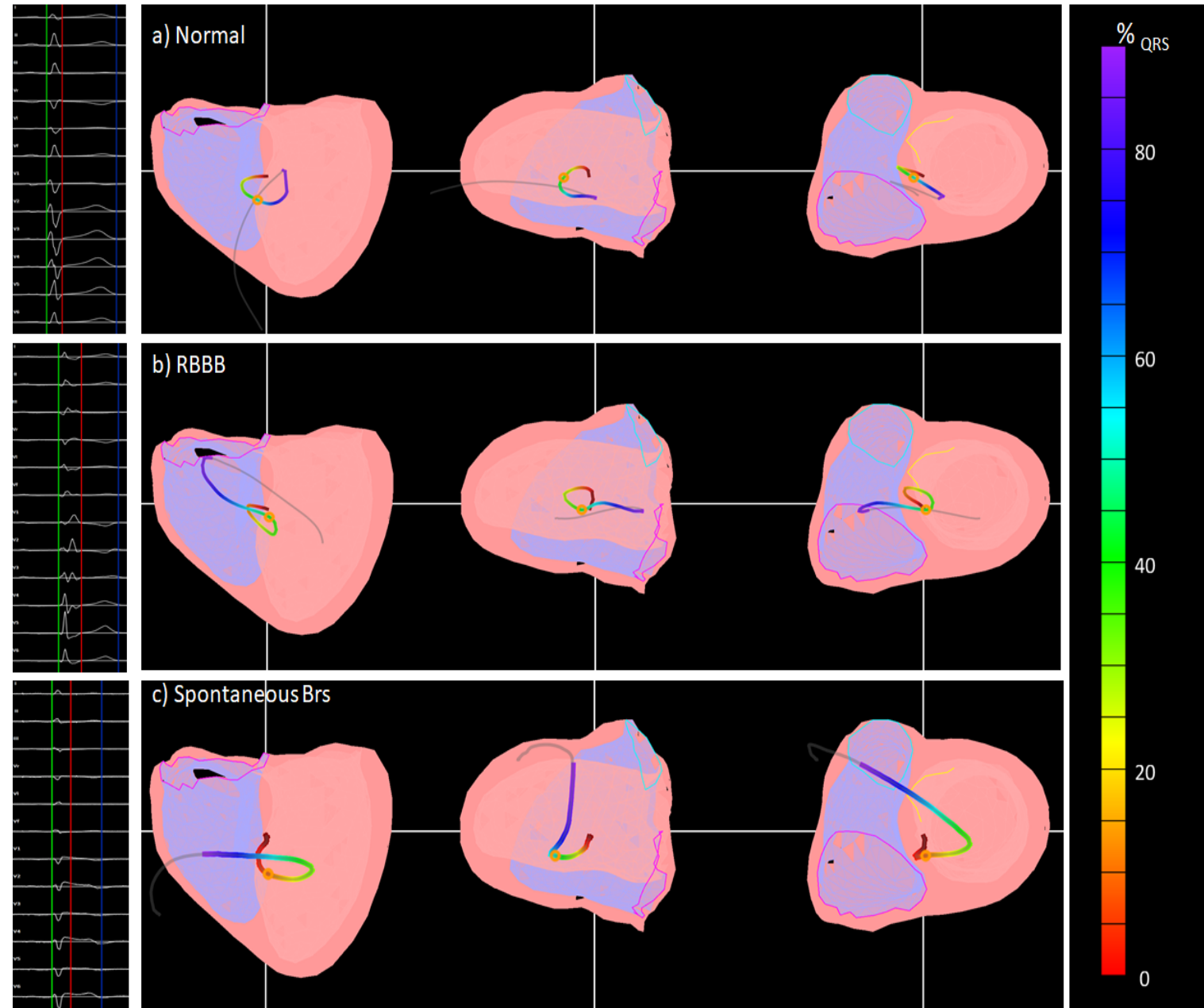


Baseline potential duration (ms) (mean ± SD)	108.2 ± 40.1	127.7 ± 46.0	101.6 ± 35.6	<0.001
Potential duration after ajmaline (ms) (mean ± SD)	202.6 ± 28.4	220.5 ± 31.5	196.7 ± 24.7	<0.001

CineECG in Brugada Type 1 versus Normal versus RBBB

The **terminal QRS** CineECG localization in RVOT, typical of BrS patients, was not present in normal controls and in RBBB patients.

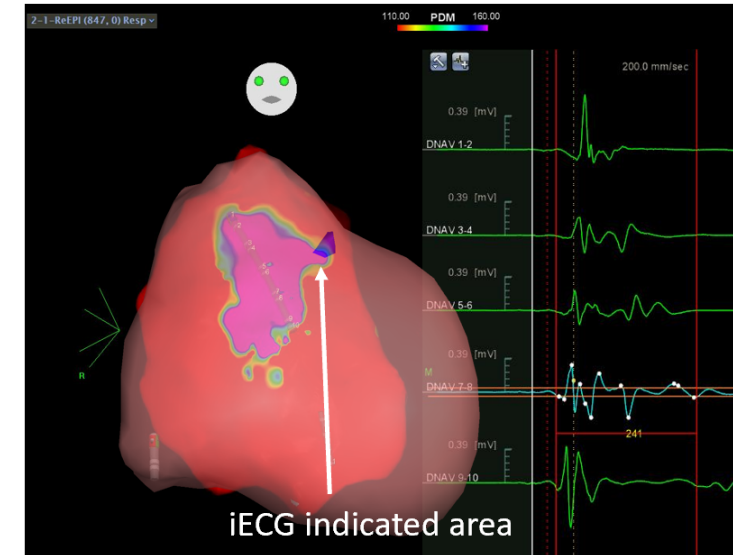
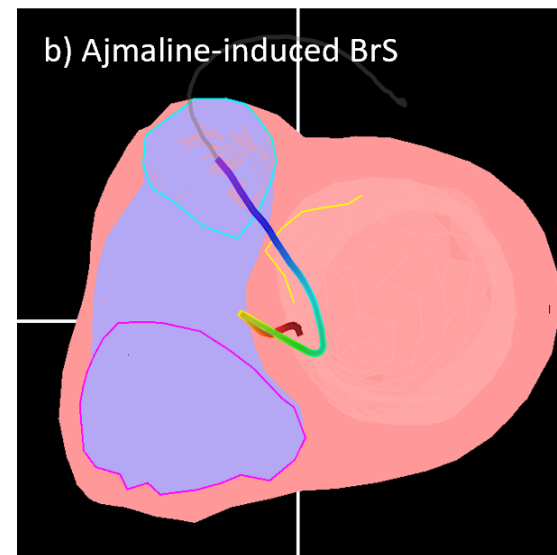
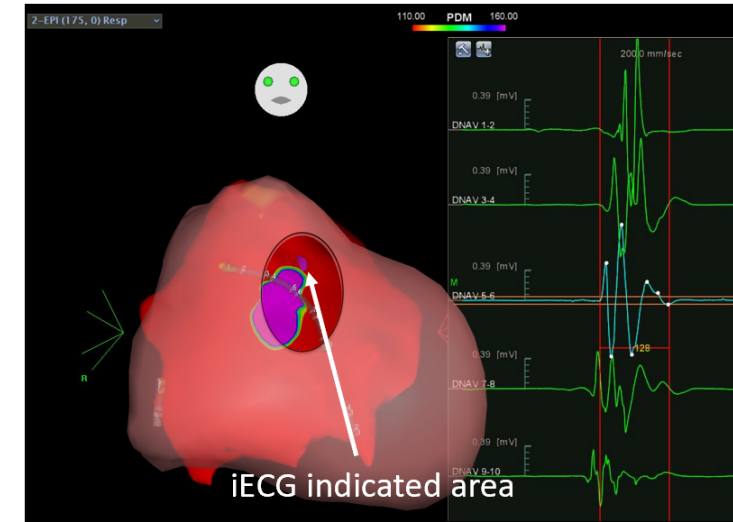
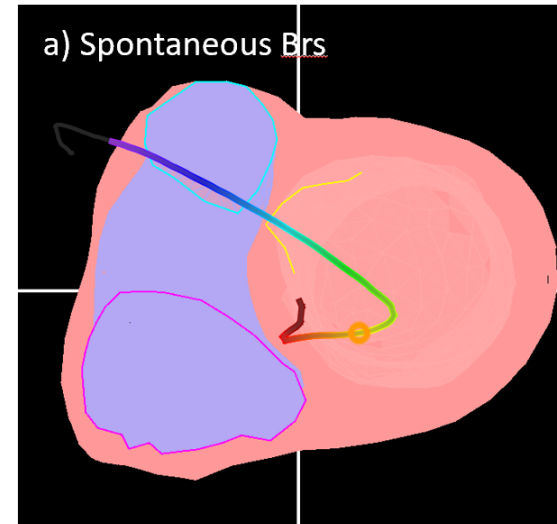
van Dam PM, Locati ET, et al. Novel CineECG Derived From Standard 12-Lead ECG Enables Right Ventricle Outflow Tract Localization of Electrical Substrate in Patients With Brugada Syndrome. *Circulation: Arrhythmia and Electrophysiology*. 2020;13:e008524.



Anatomical base of the CineECG terminal QRS localization

- The localization of **terminal QRS** activity in CineCG in BrS patients corresponds to the localization of the **arrhythmogenic substrate in RVOT**

van Dam PM, Locati ET, et al. Novel CineECG Derived From Standard 12-Lead ECG Enables Right Ventricle Outflow Tract Localization of Electrical Substrate in Patients With Brugada Syndrome. Circulation: Arrhythmia and Electrophysiology. 2020;13:e008524.



PAPER

Deep learning unmasks the ECG signature of Brugada Syndrome

Luke Melo,^a Giuseppe Ciconte,^b Ashton Christy,^a Gabriele Vicedomini,^b
Luigi Anastasia,^{c,*} Carlo Pappone^{b,d,*} and Edward Grant^{a,*}

^aDepartment of Chemistry, University of British Columbia, Vancouver, BC, V6T 1Z1, Canada, ^bArrhythmia and Electrophysiology Center, IRCCS Policlinico San Donato, Milan, Italy, ^cStem Cell Laboratory for Tissue Engineering, Università Vita-Salute San Raffaele, Milan, Italy and ^dDepartment of Cardiology, Università Vita-Salute San Raffaele, Milan, Italy.
*To whom correspondence should be addressed: anastasia.luigi@hsr.it; carlo.pappone@hsr.it; edgrant@chem.ubc.ca
FOR PUBLISHER ONLY Received on Day Month Year; accepted on Day Month Year

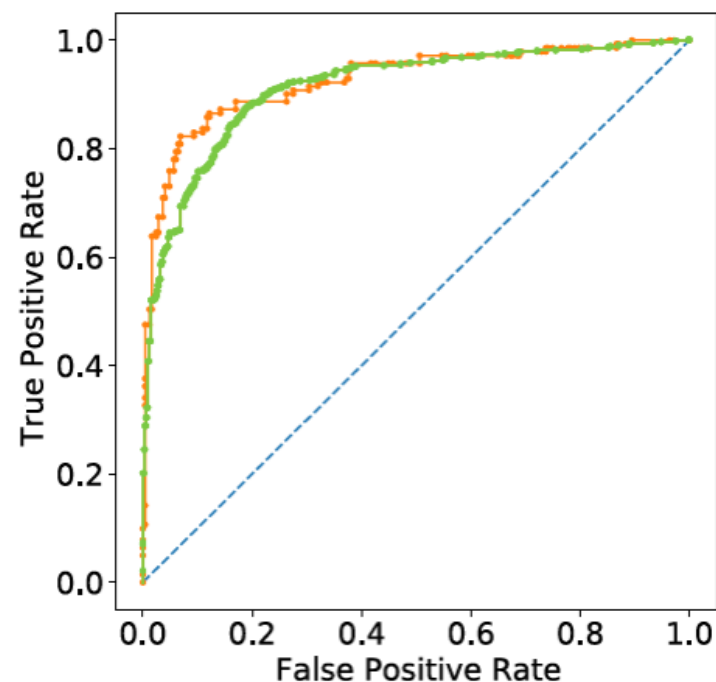


Fig. 4. Receiver-operator characteristic curve for the Claris (training, green) and Mortara (independent validation, orange) cohorts.

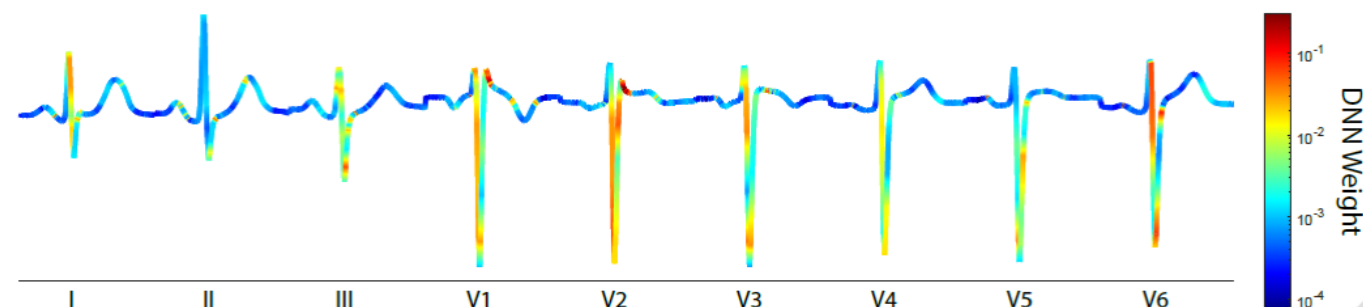
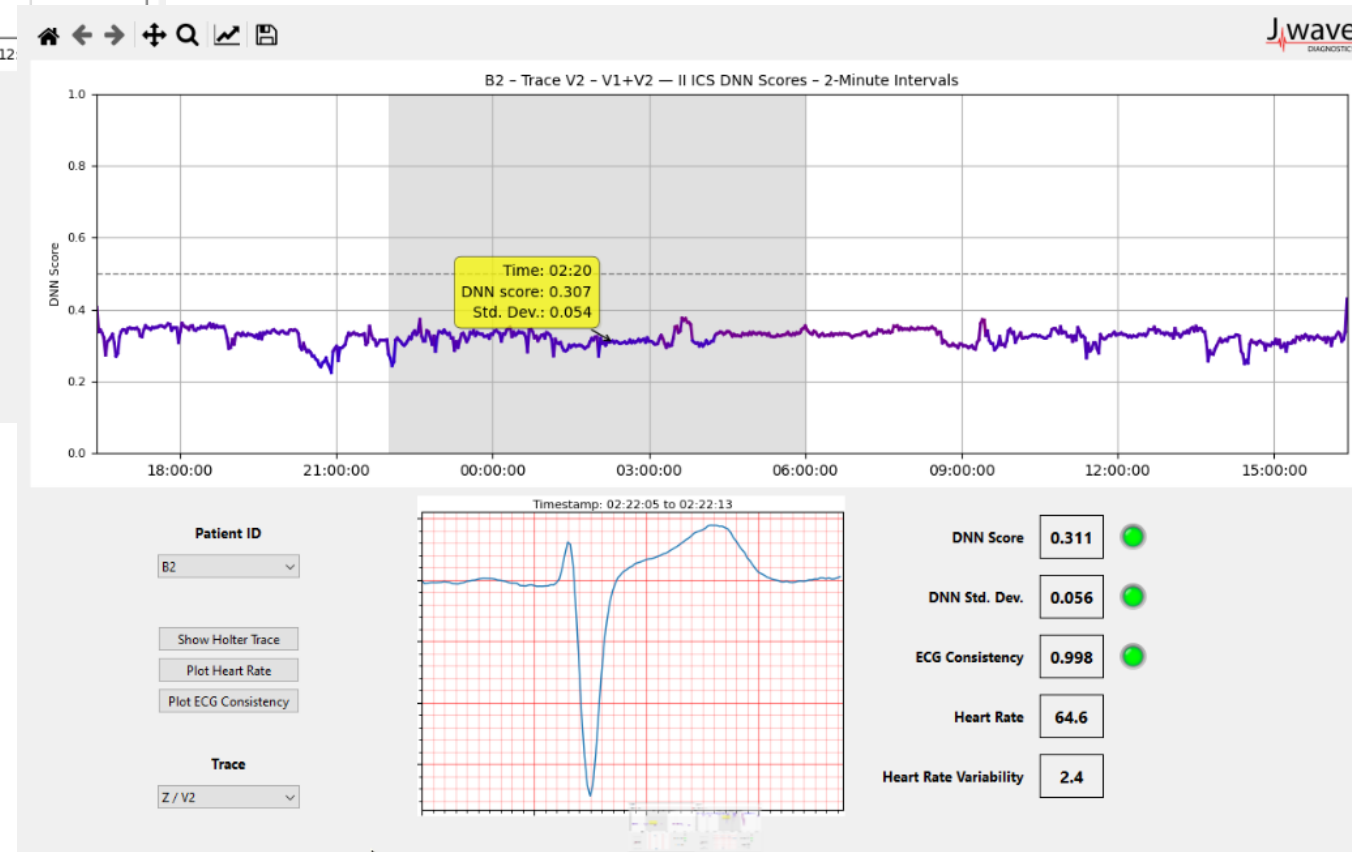


Fig. 5. DNN feature importance shown on a 9-lead fused ECG trace, representative of the DNN training data. The colormap displays the magnitude of the weights applied by the DNN when predicting ajmaline response. The weights range from 0.0001 to 0.3021; higher-weighted regions have a greater influence on the DNN model.

AI J Wave Algorithm Applied to 24-hr Holter Recordings



9-Second Pause in a Patient with Mild Positive Brugada Pattern During ILR Monitoring

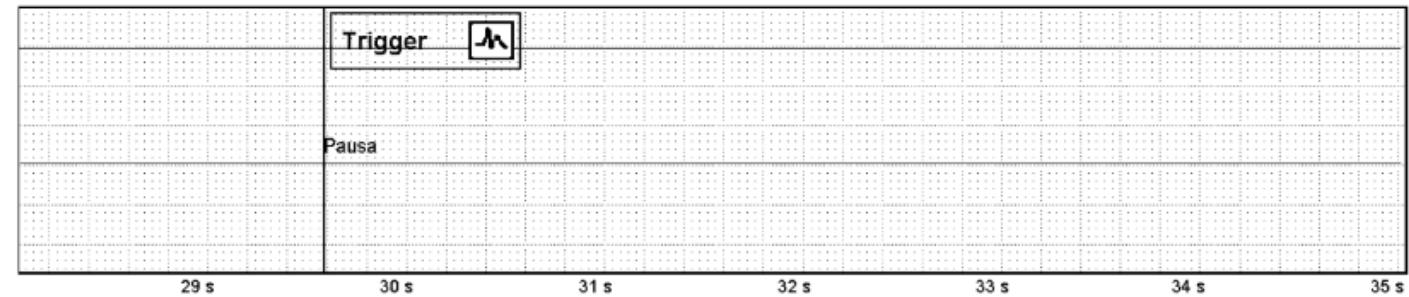
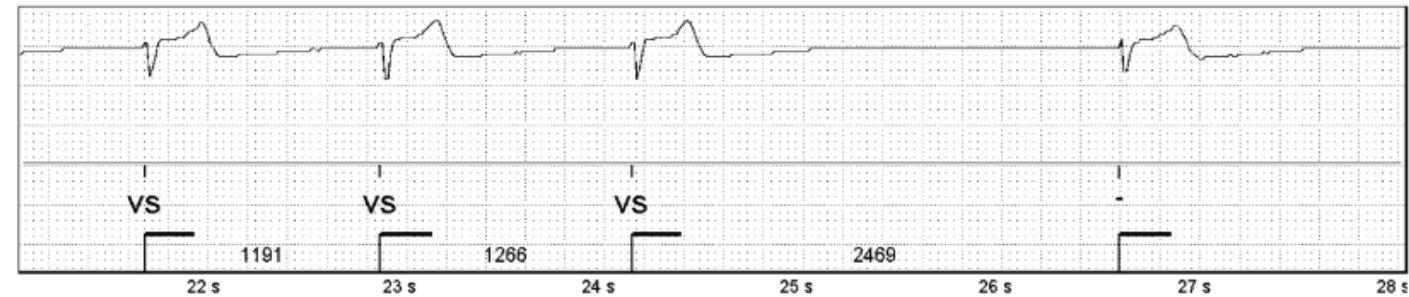
Episodio Pausa

Pagina 2 di 3

1: VEGM AutoGain (9.7 mm/mV)

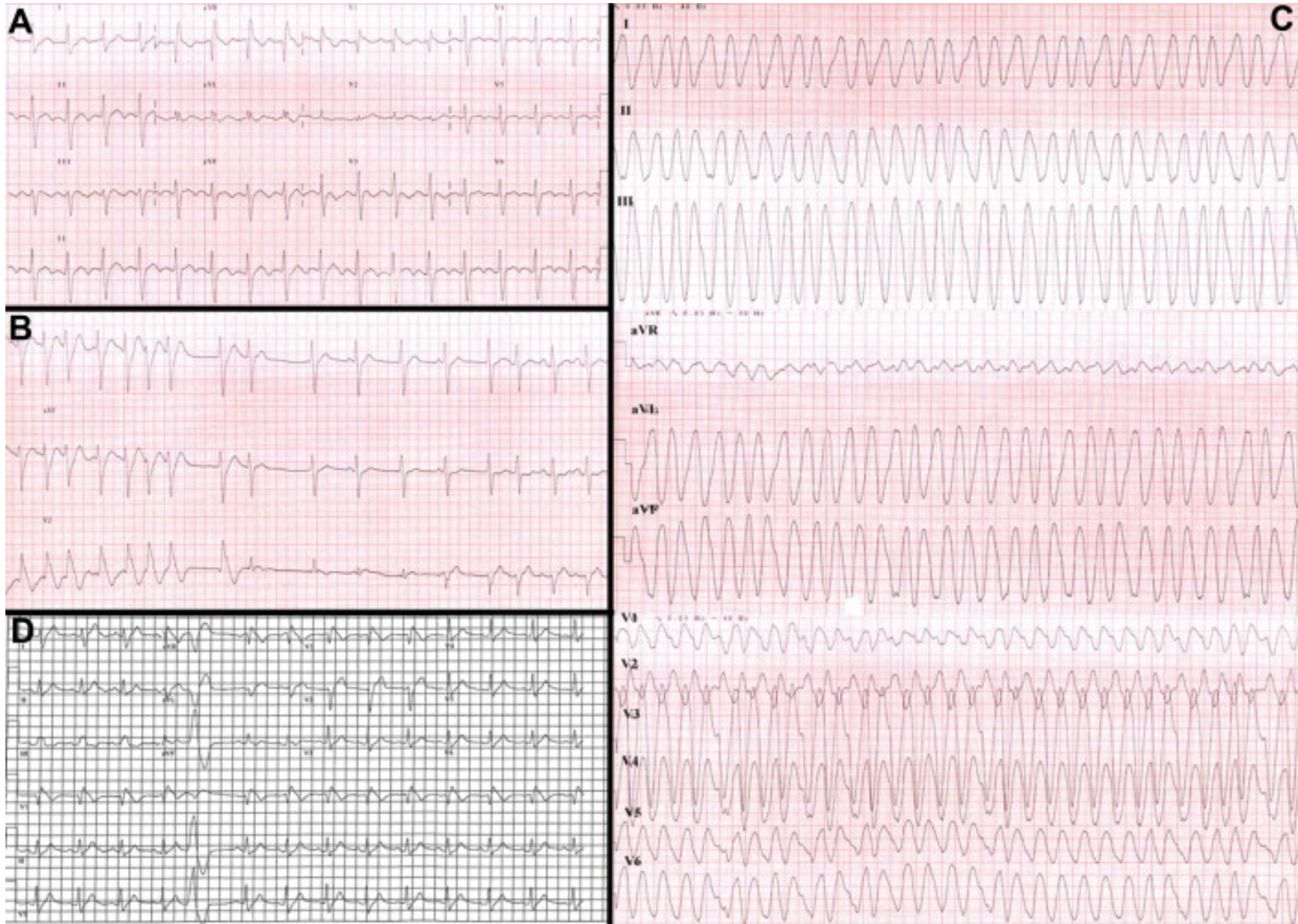
2: Marker

Velocità di scorrimento: 25 mm/s



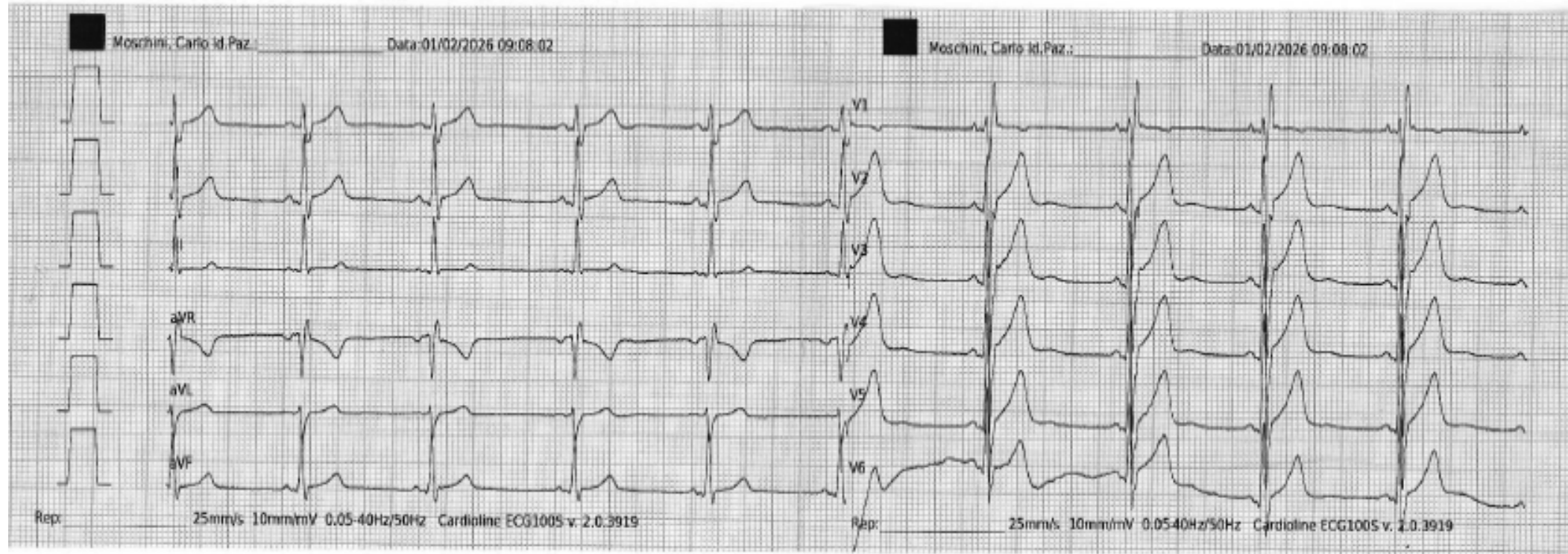
Male, 24 yrs, asymptomatic patient with occasional finding of BRS pattern on ECG

Atrial Fibrillation and Ventricular Fibrillation in a Patient with Brugada Syndrome

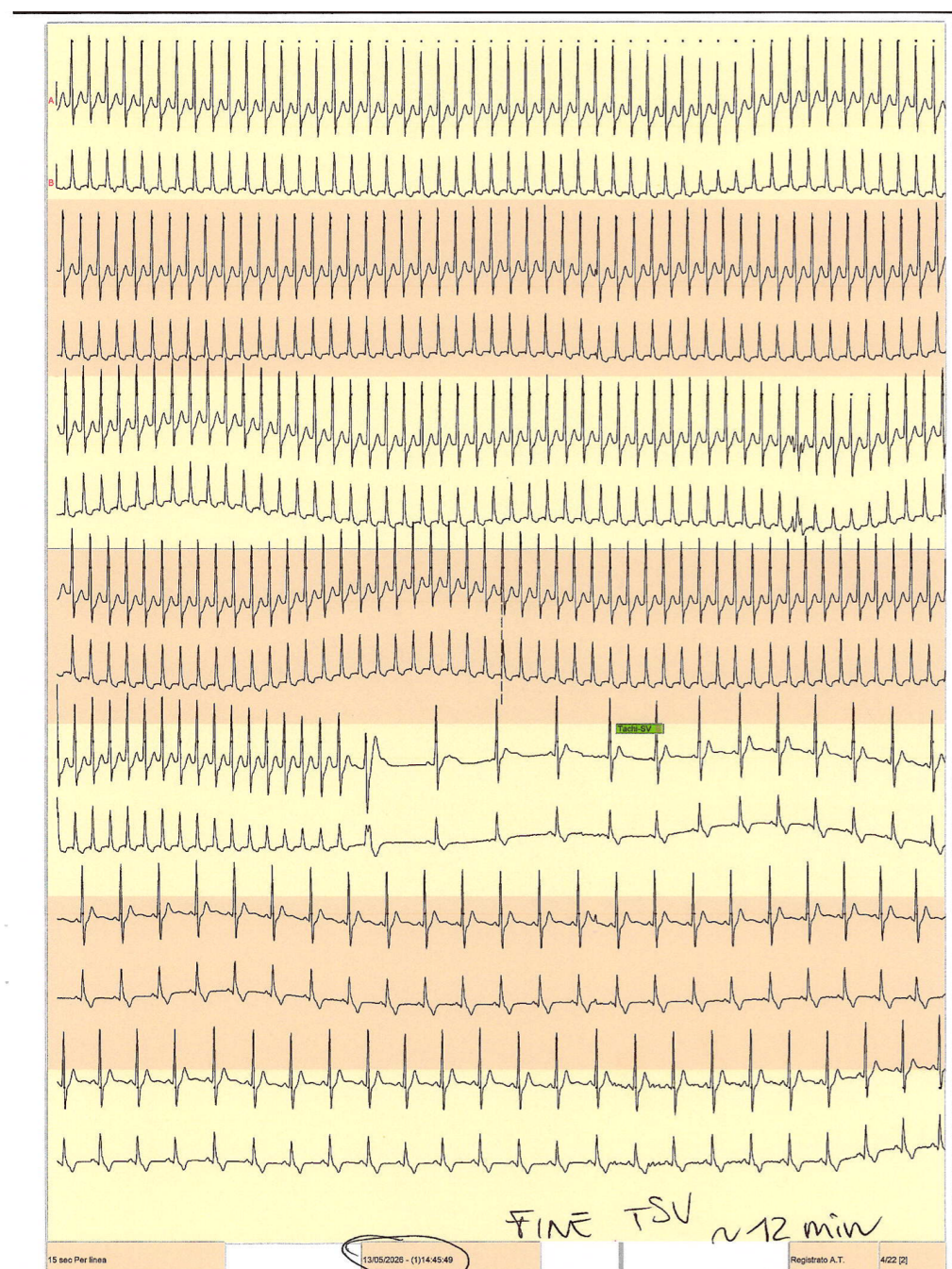
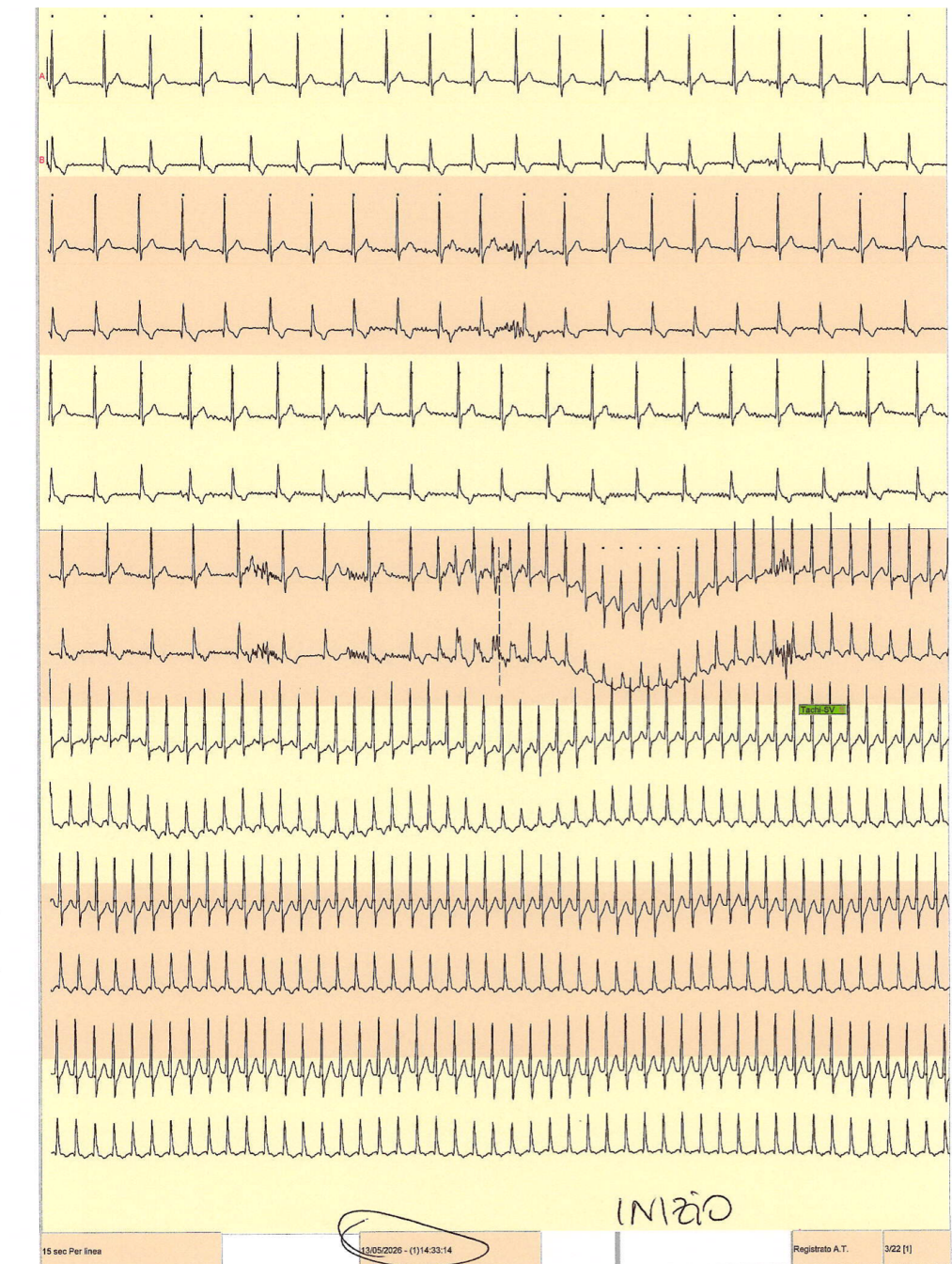


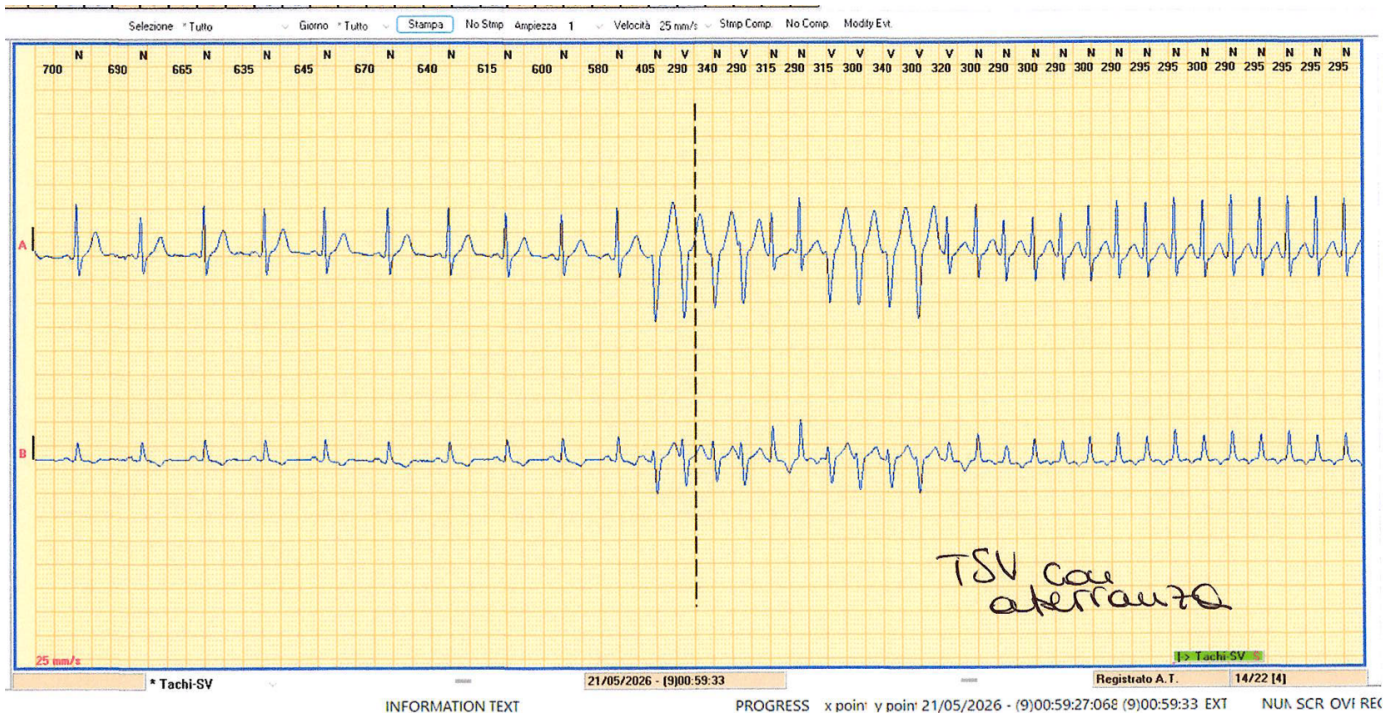
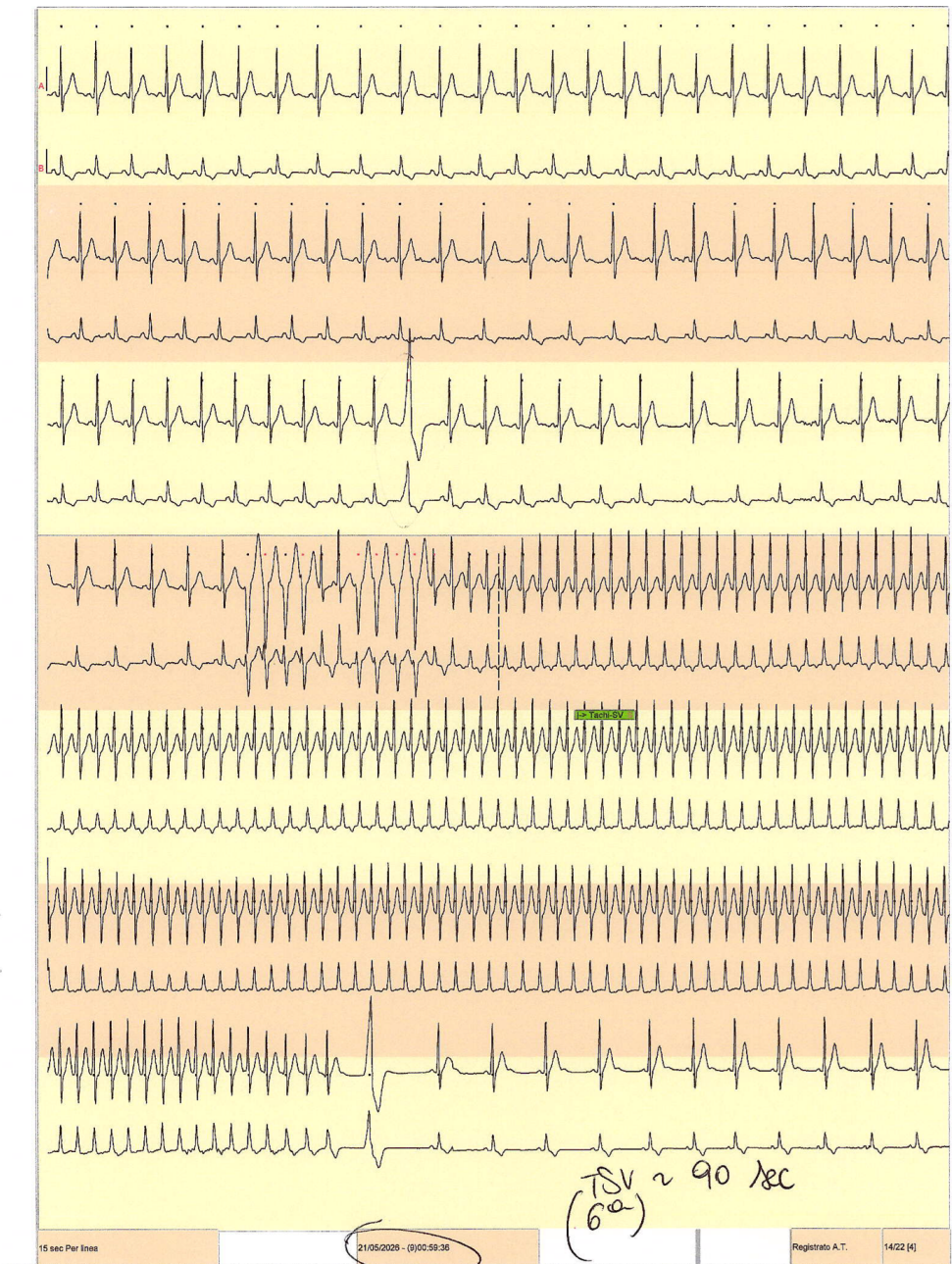
Tracings of a 22-year-old woman admitted to emergency department because of typical atrial flutter (A). After flecainide infusion, she developed a Brugada-like electrocardiographic pattern and later conversion to sinus rhythm (B), with subsequent degeneration in ventricular fibrillation (C). (D) Electrocardiogram after infusion of ajmaline (1 mg/kg), compatible with typical BS type 1 pattern.

Un tracciato basale apparentemente banale, un Holter prolungato molto complesso



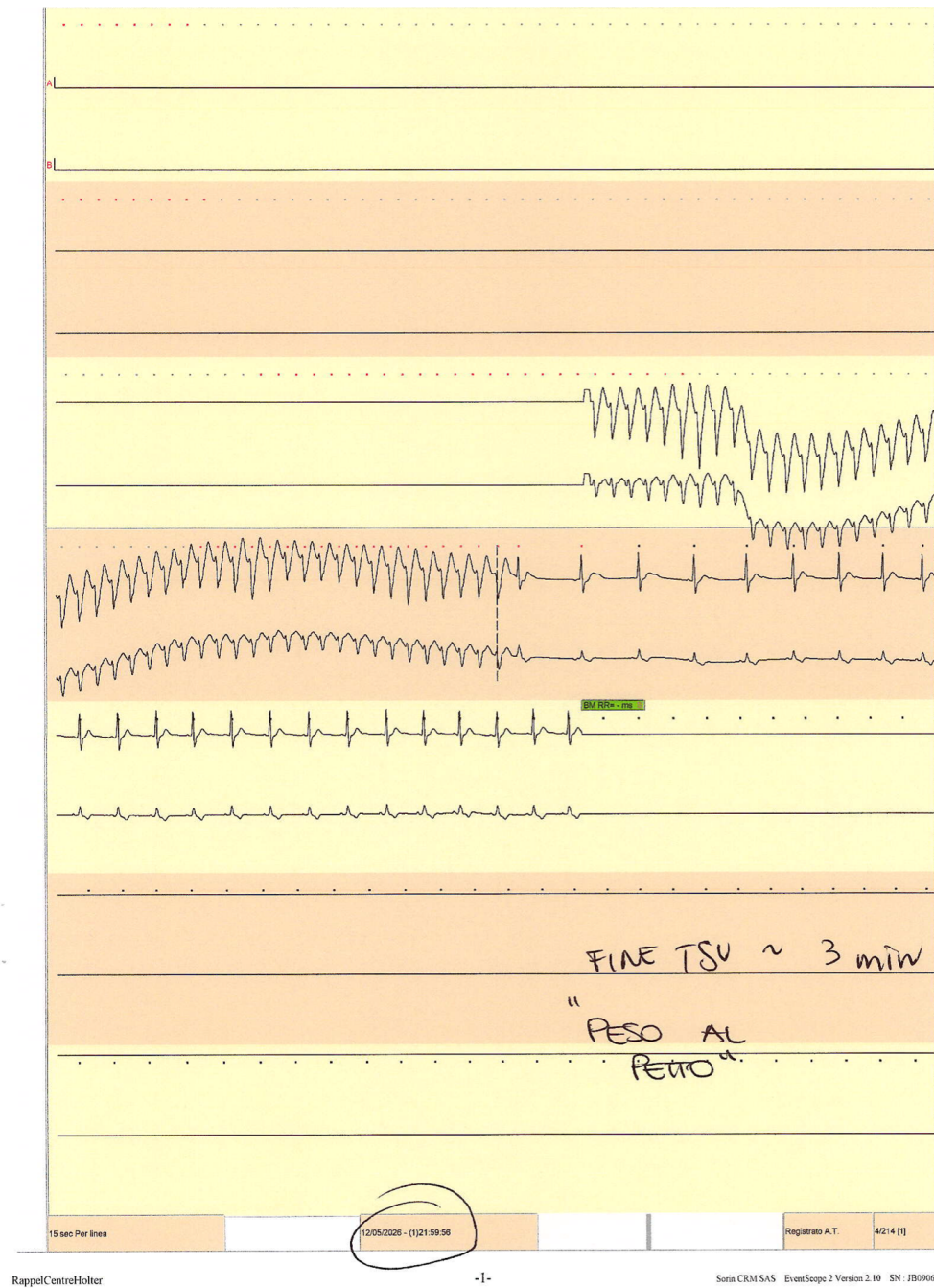
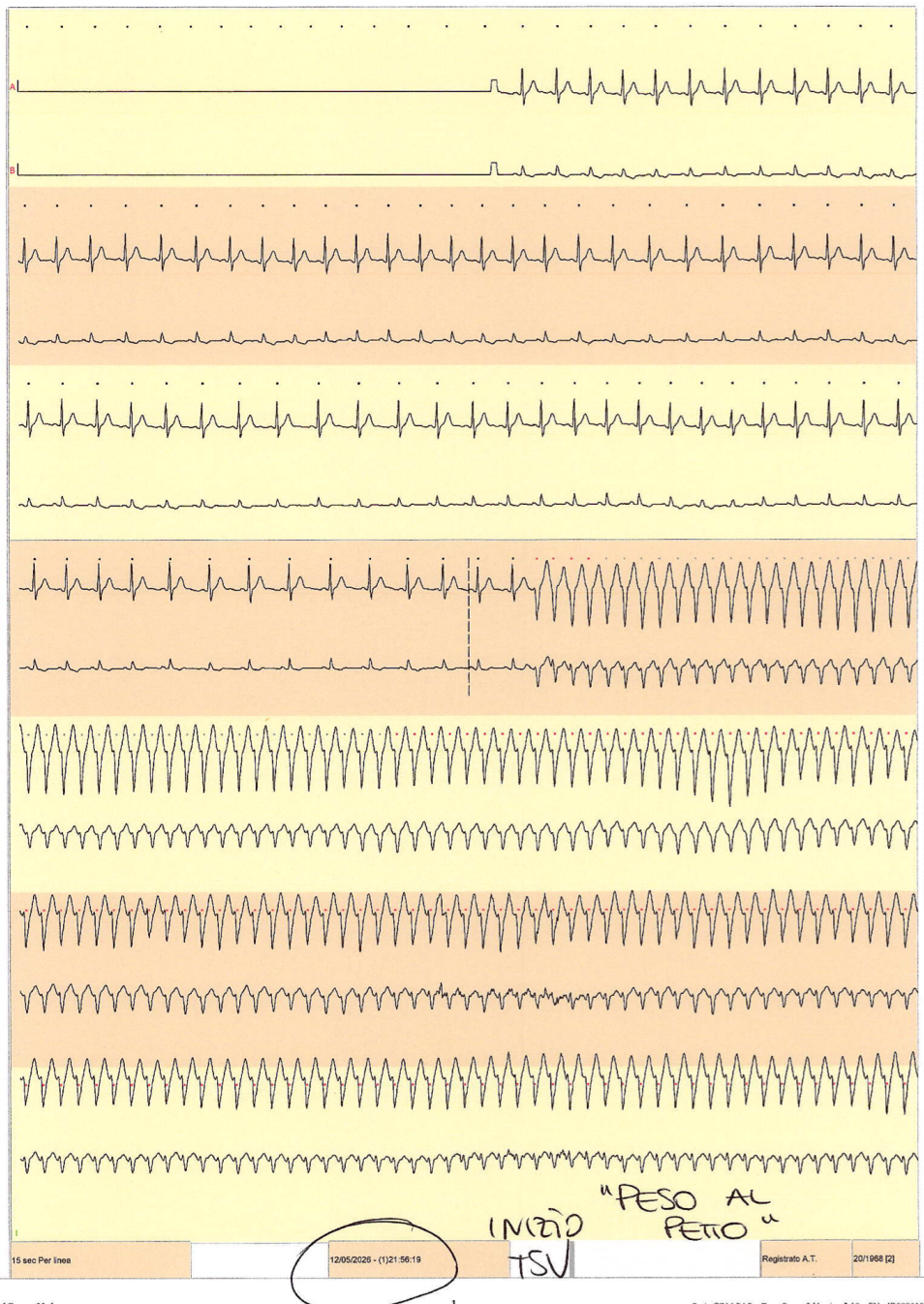
CM, 18 anni, storia di frequenti palpitazioni

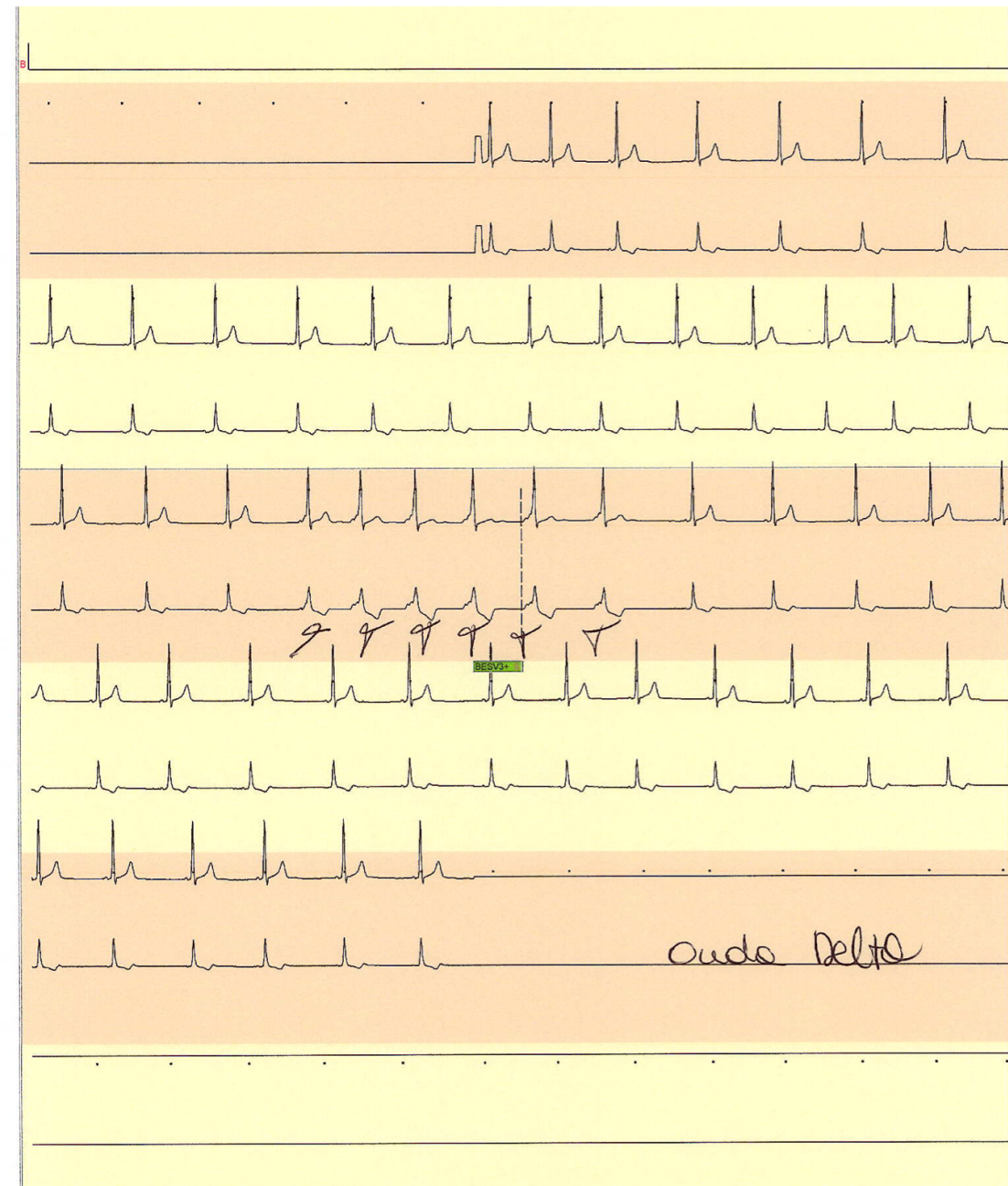


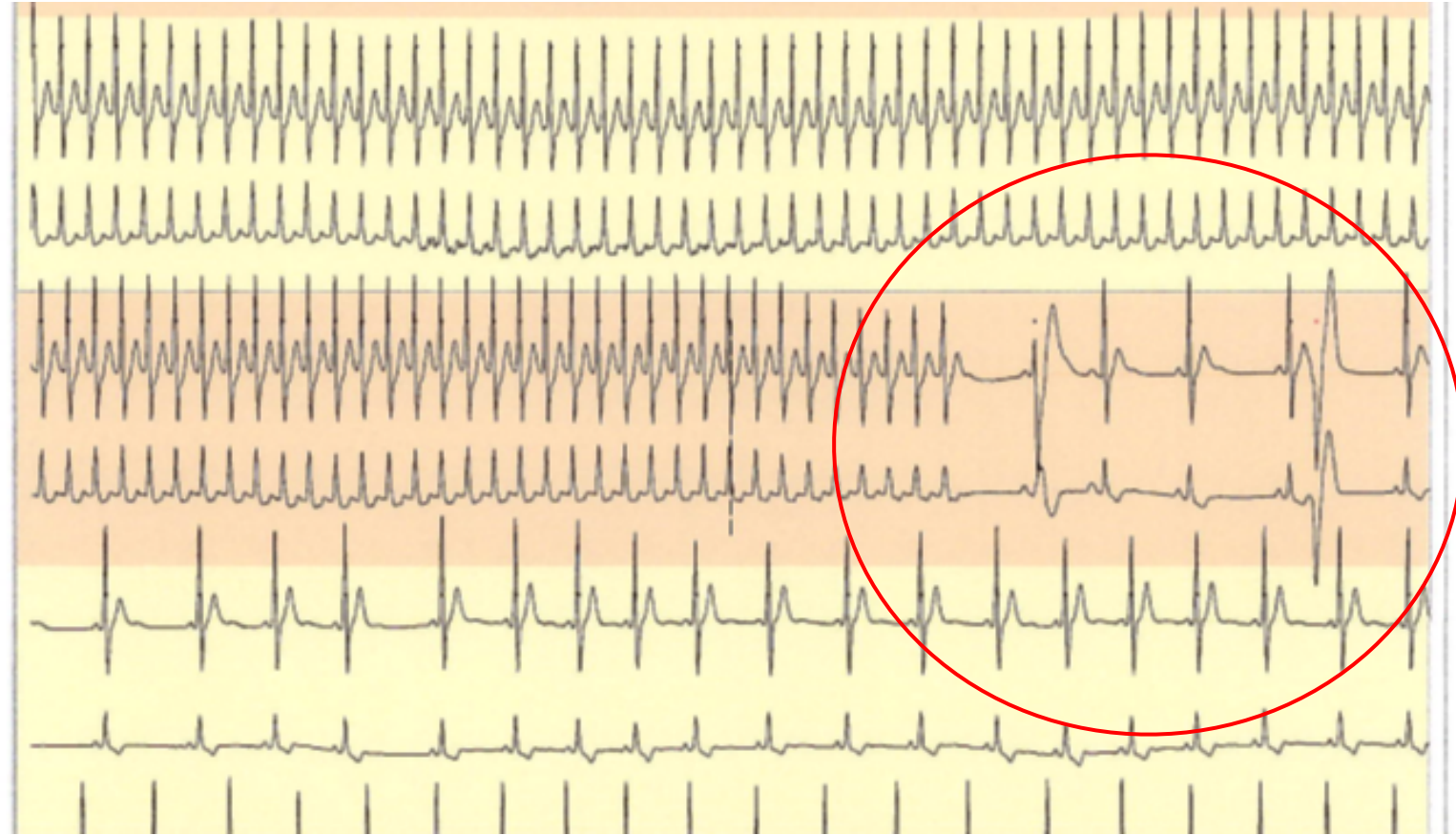
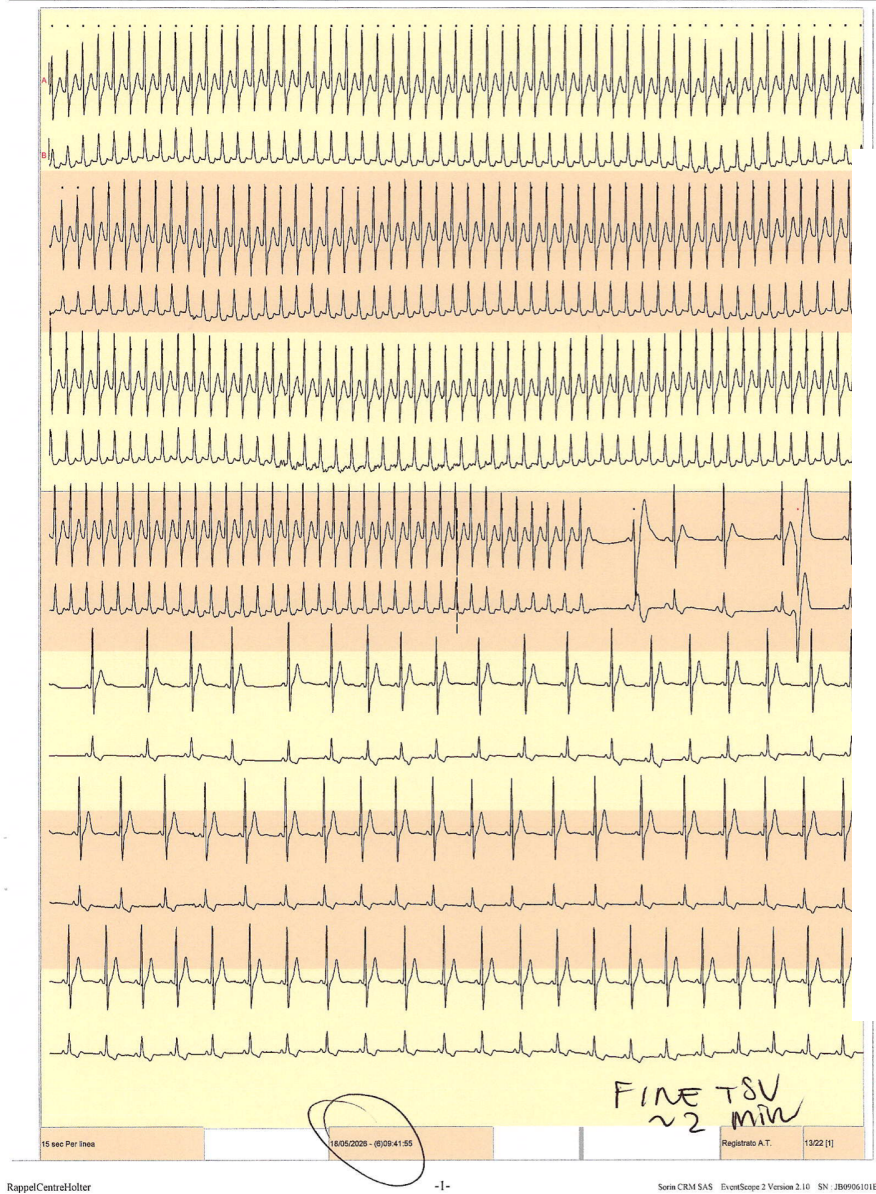


INFORMATION TEXT

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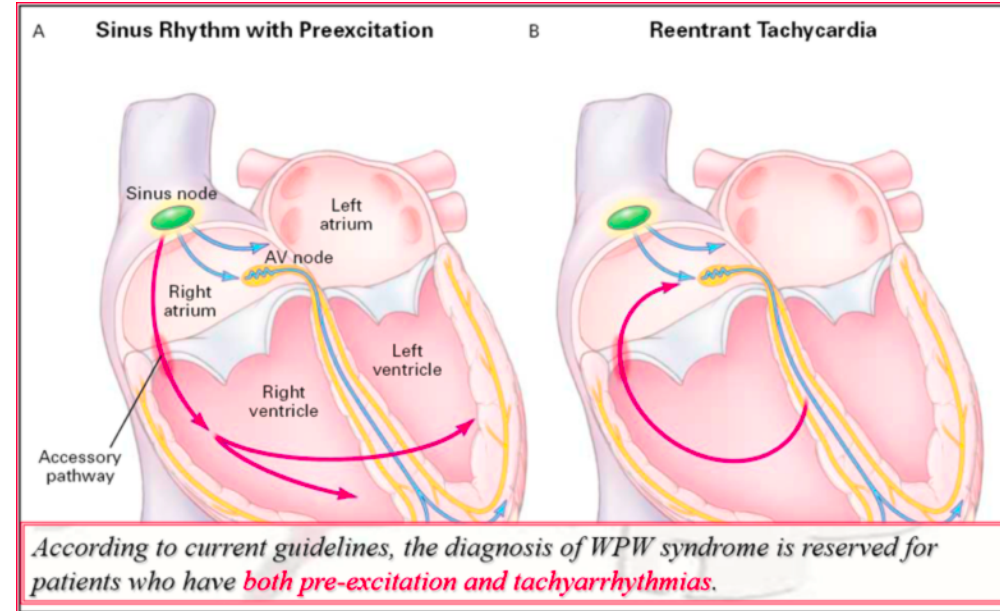
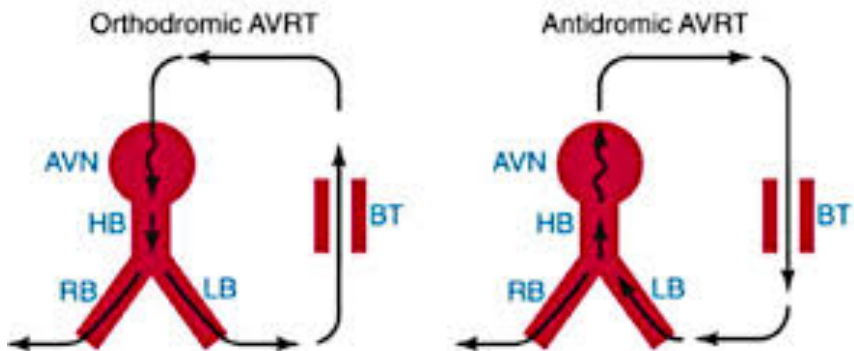




Alterazioni della conduzione

Il QRS si allarga quando:

- L'attività proviene dal tessuto ventricolare miocardico alterato
- Il sistema His-Purkinje è bloccato
- Esiste una via di conduzione antidromica.



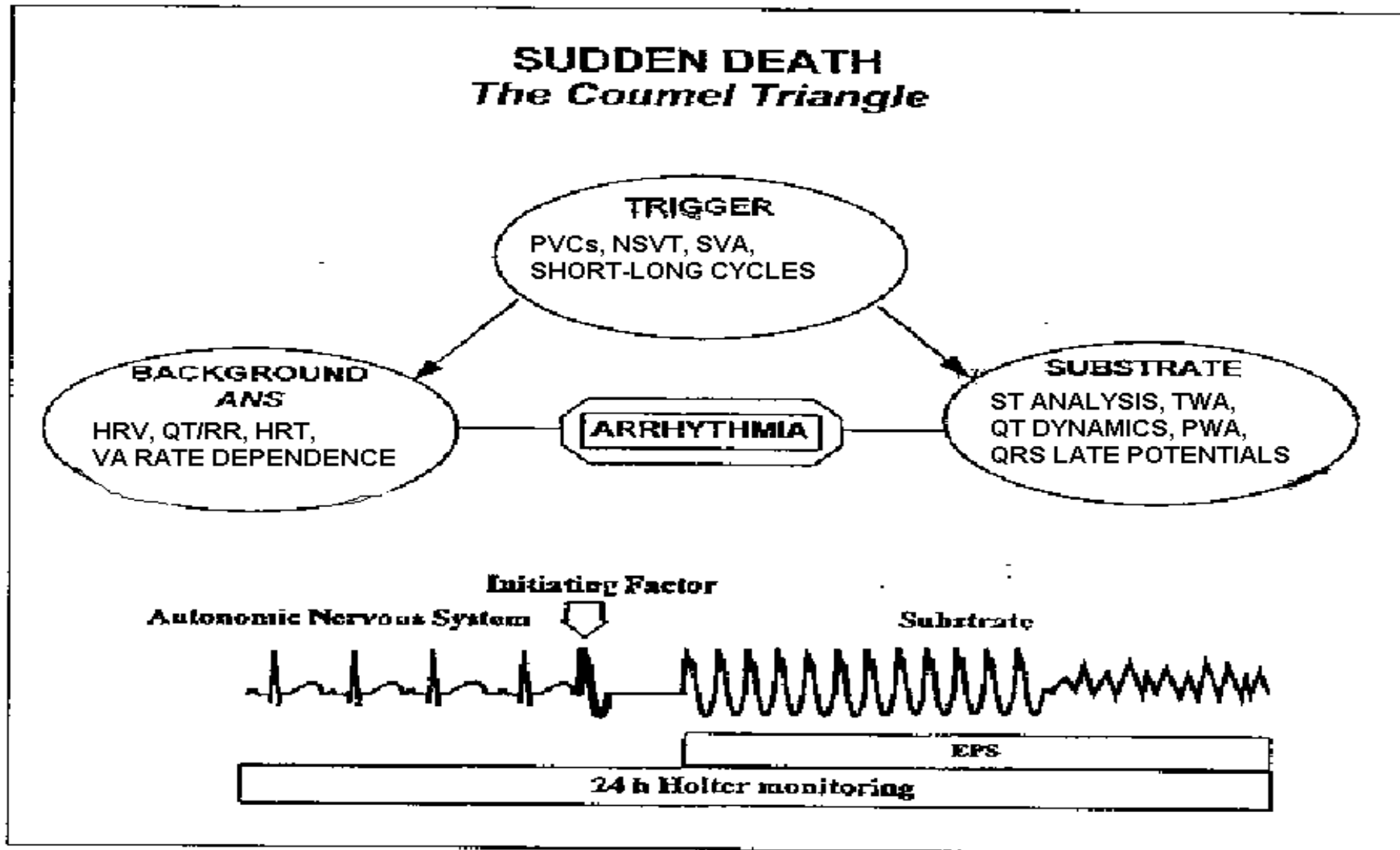
Conduzione Antidromico (Rara - ~10% dei casi):

L'impulso scende rapidamente (anterogrado) dagli atri ai ventricoli attraverso la **via accessoria**, e risale (retrogrado) ai ventricoli tramite il **nodo AV**.

Risultato ECG: Complesso QRS largo e bizzarro, con preeccitazione totale, spesso difficile da distinguere da una tachicardia ventricolare

Ambulatory ECG as Noninvasive Electrophysiologic Test

The Coumel Sudden Death Triangle



Grazie per l'attenzione!

