LE ARITMIE

Un mondo da inquadrare

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CASO CLINICO 1

- ♀, 82 aa
- Viene ricoverata dal PS in reparto medico con diagnosi di "Sincope"
- In anamnesi: ipertensione arteriosa, declino cognitivo lieve.
- EO: Obiettività toracoaddominale nei limiti, non deficit neurologici.
- TD: ramipril, cardioaspirin, lansoprazolo

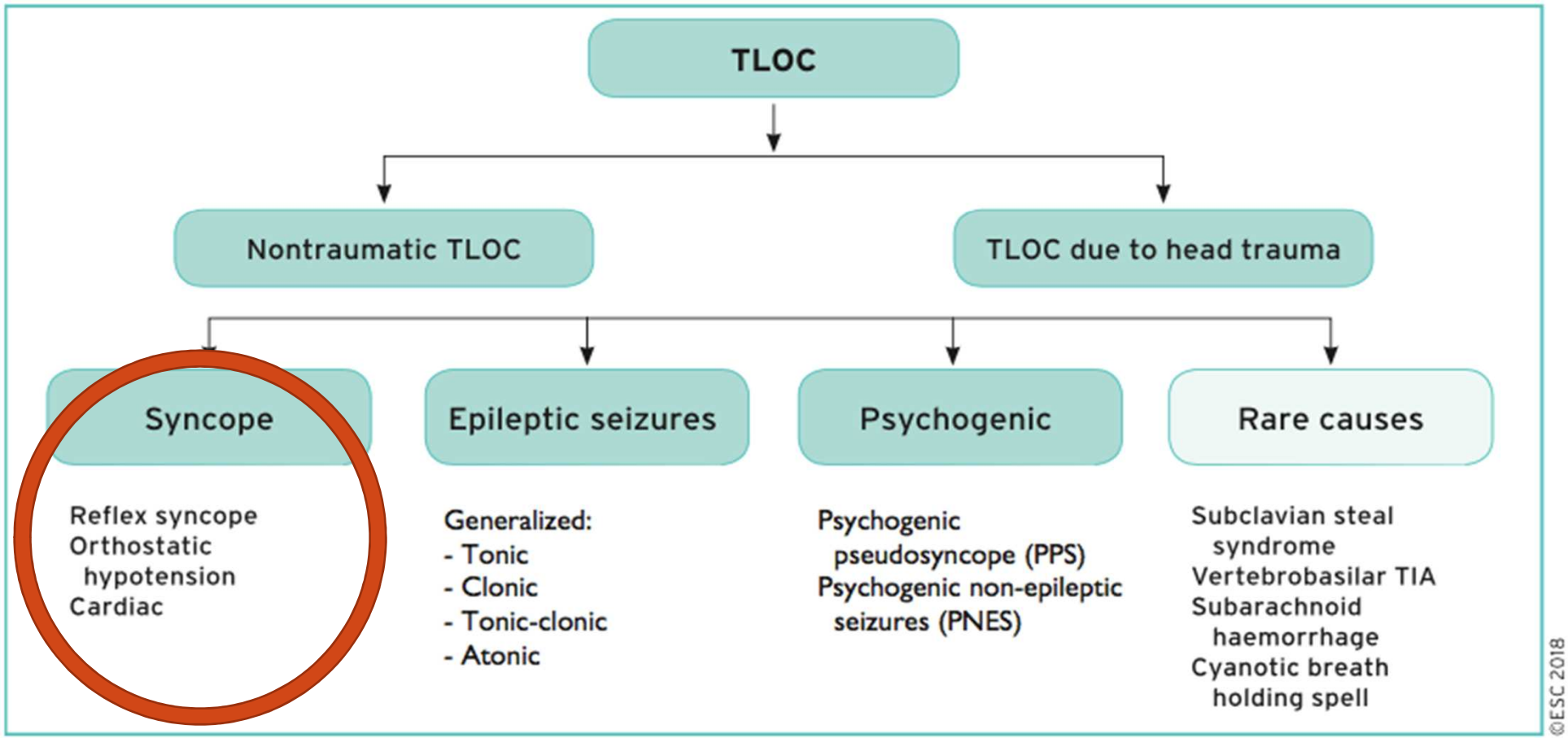
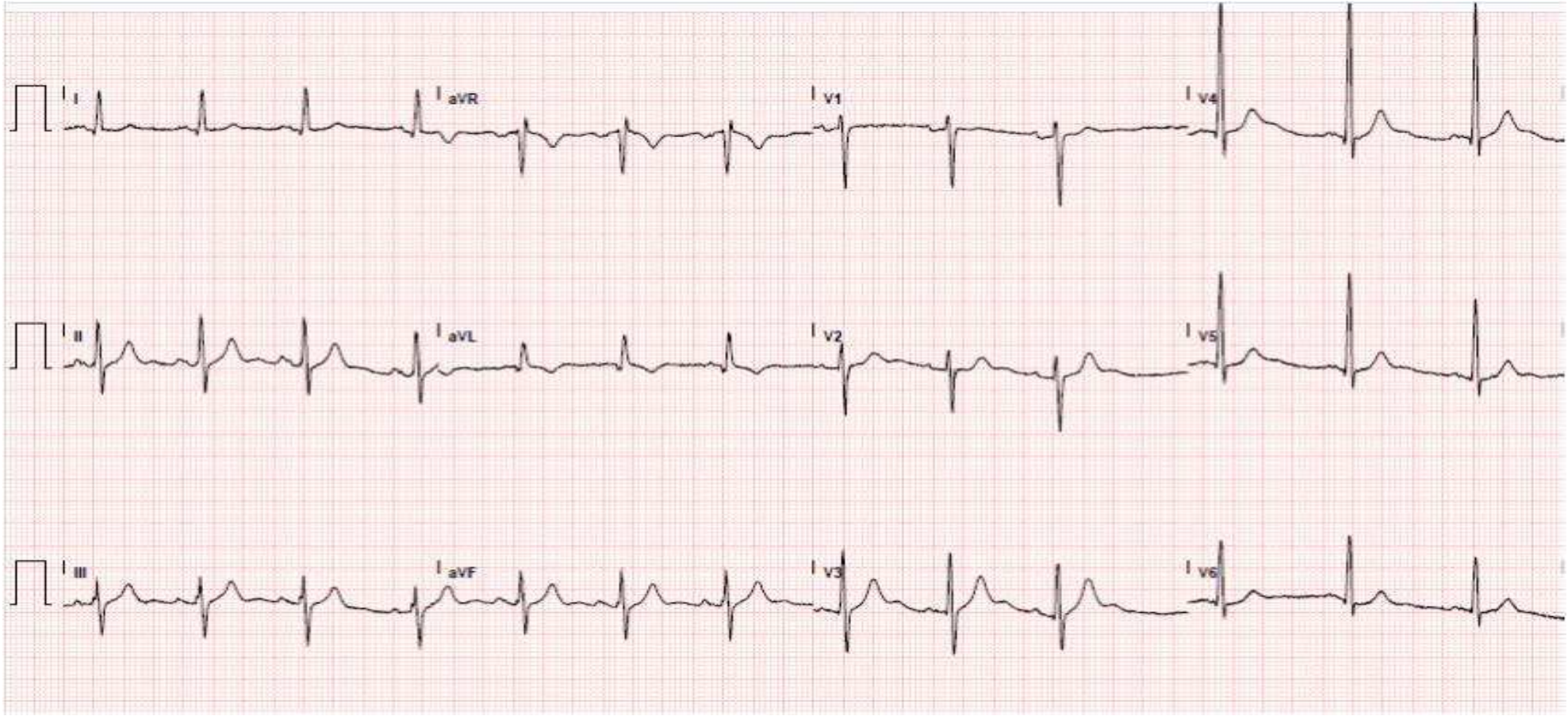


Figure 2 Syncope in the context of transient loss of consciousness. Non-traumatic transient loss of consciousness is classified into one of four groupings: syncope, epileptic seizures, psychogenic transient loss of consciousness, and a miscellaneous group of rare causes. This order represents their rate of occurrence. Combinations occur; e.g. non-traumatic transient loss of consciousness causes can cause falls with concussion, in which case transient loss of consciousness is both traumatic and non-traumatic. TIA = transient ischaemic attack; TLOC = transient loss of consciousness.

2018 ESC Guidelines for the diagnosis and treatment of syncope

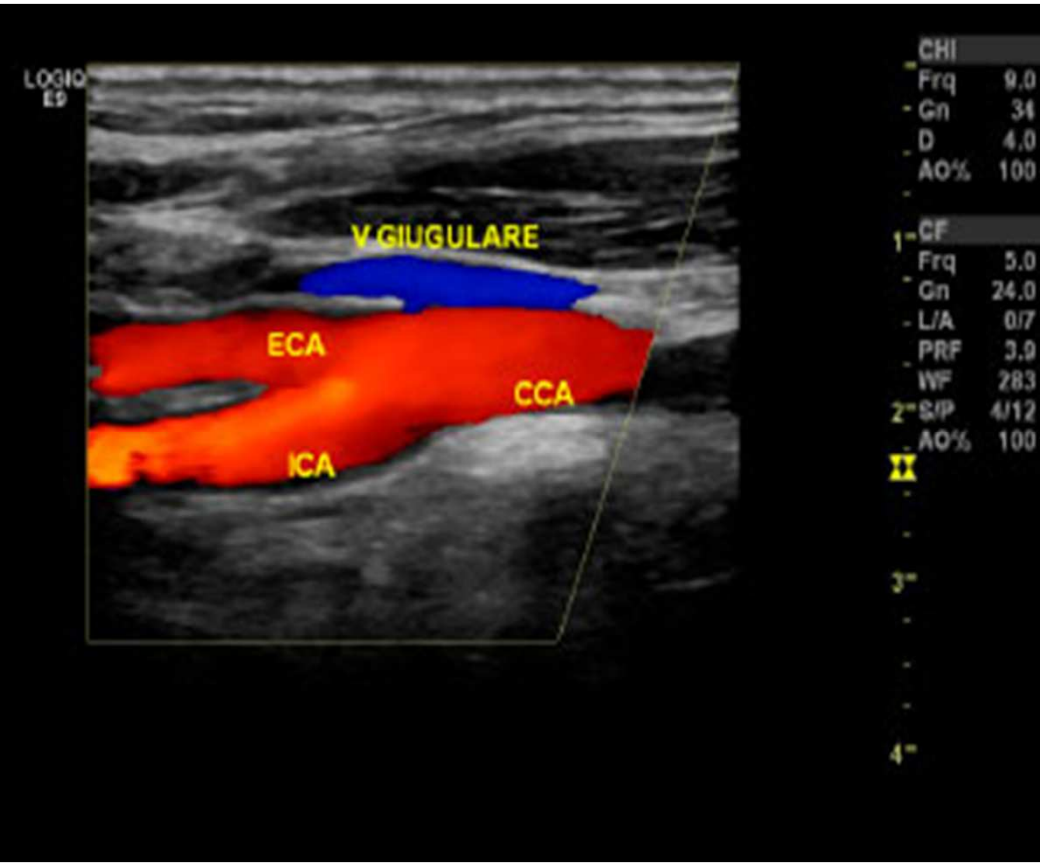
INDAGINI ESEGUITE (1)

- ECG: RS con FC 85 bpm, segni di IVS



INDAGINI ESEGUITE (2)

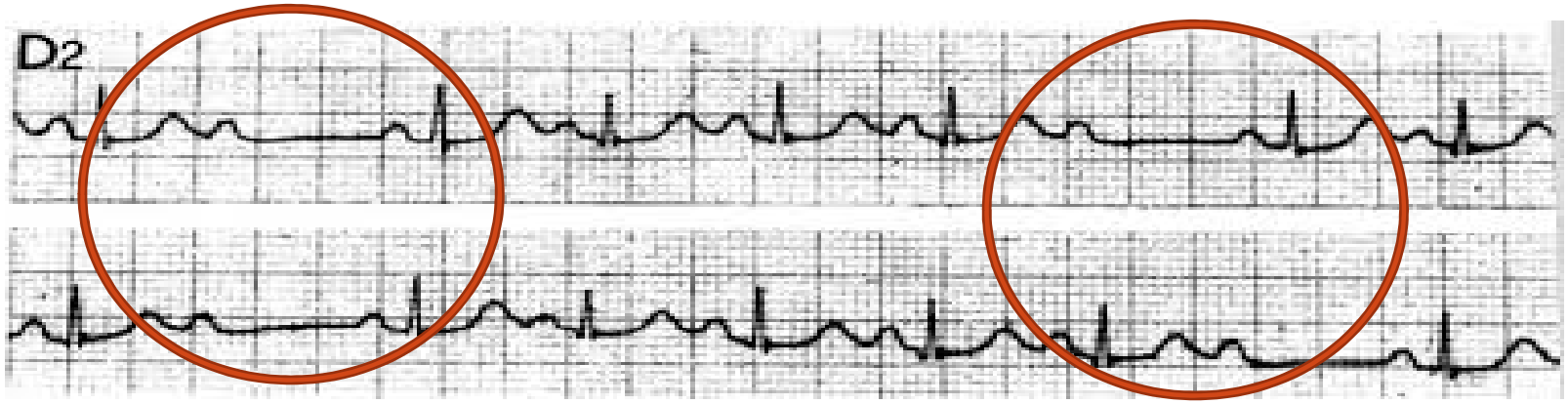
Ecocolordoppler TSA: Minimo ispessimento mio-intimale



- Prove clino-ortostatismo: negative

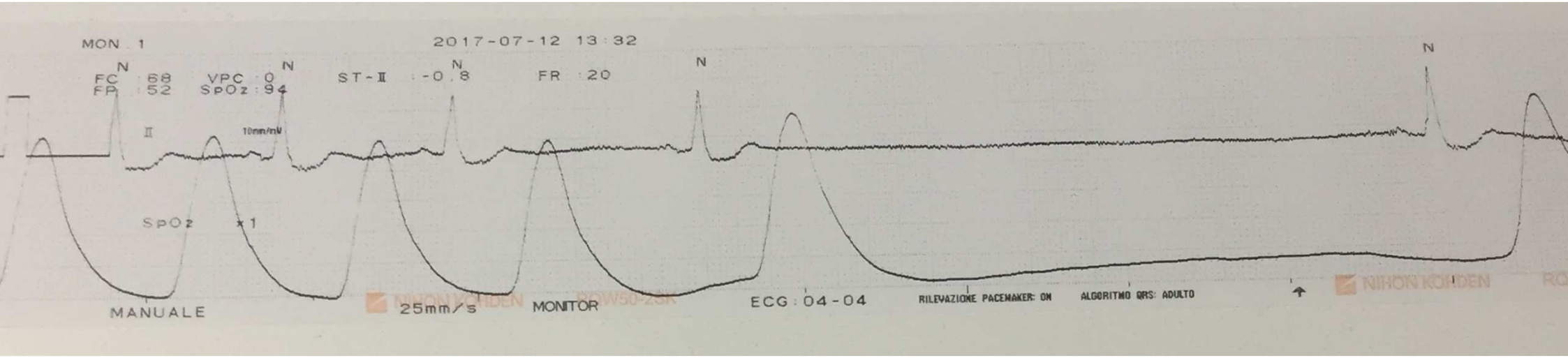
INDAGINI ESEGUITE (4)

- Holter-ECG: riscontro di numerose P bloccate



BAV II grado Mobitz 2? SSS?

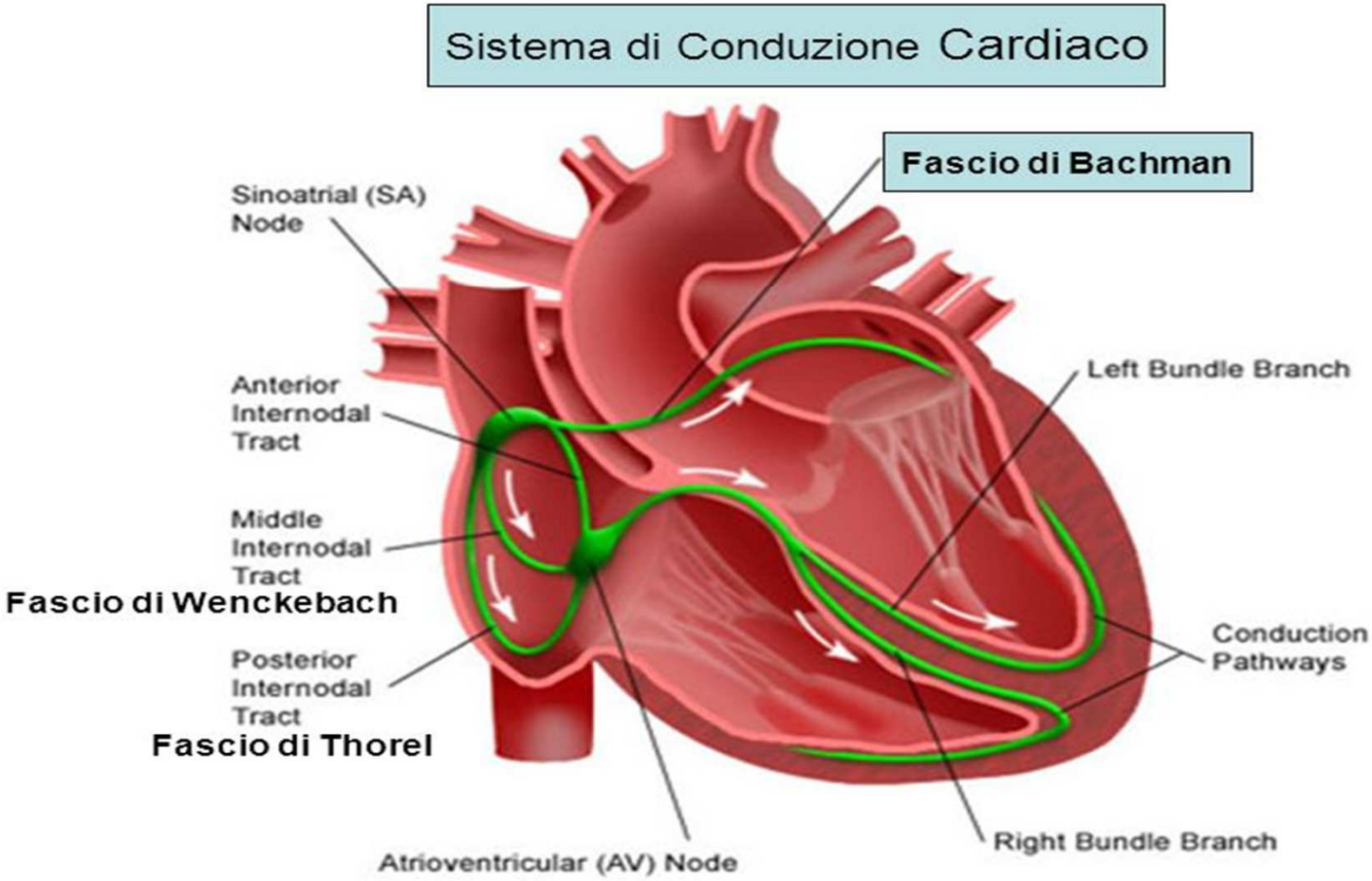
MASSAGGIO DEL SENO CAROTIDEO



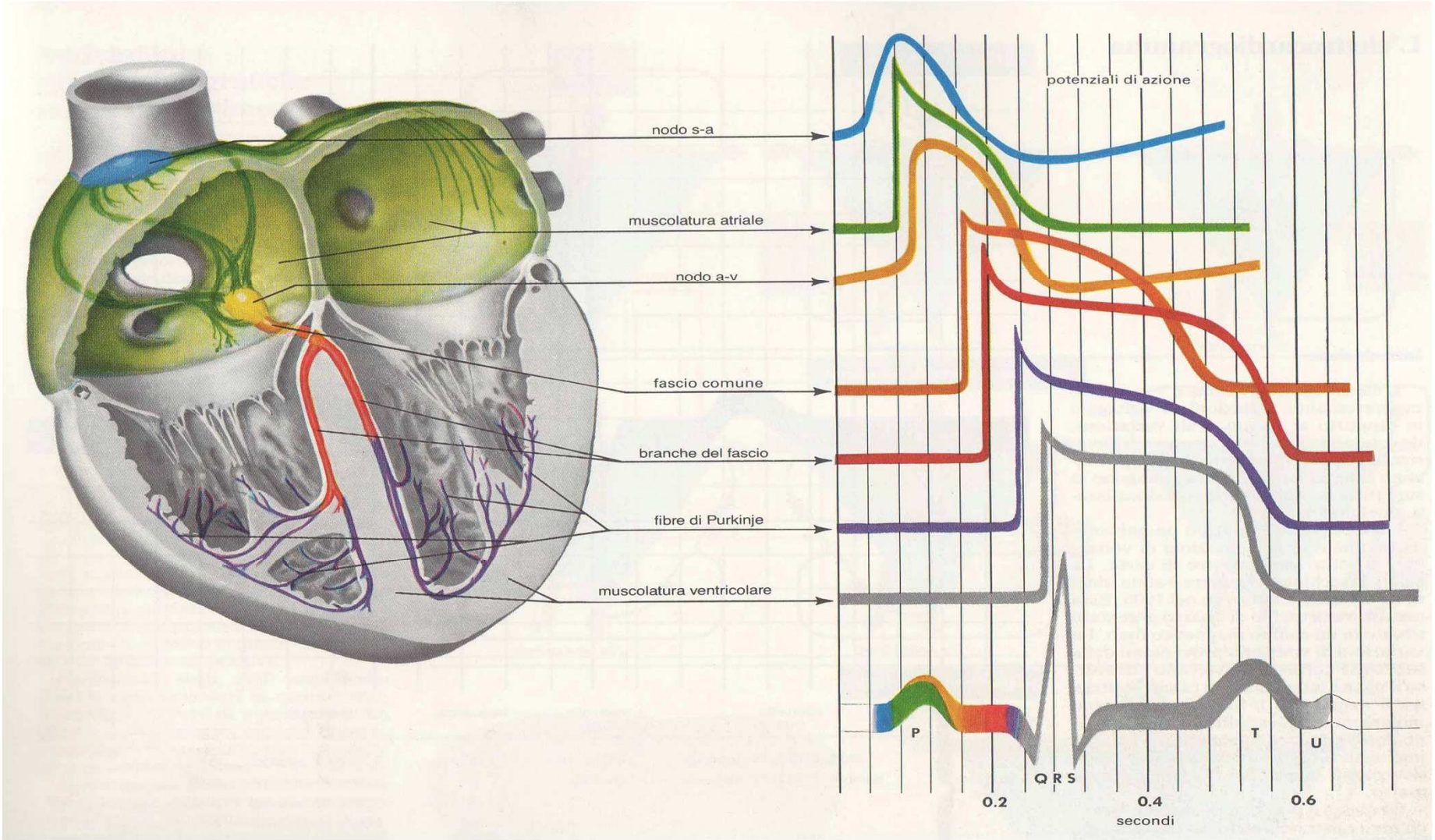
- Massaggio positivo per SSS. Paziente indirizzata ad impianto di PM

INQUADRAMENTO DELLE ARITMIE

PARTIAMO DAL PRINCIPIO

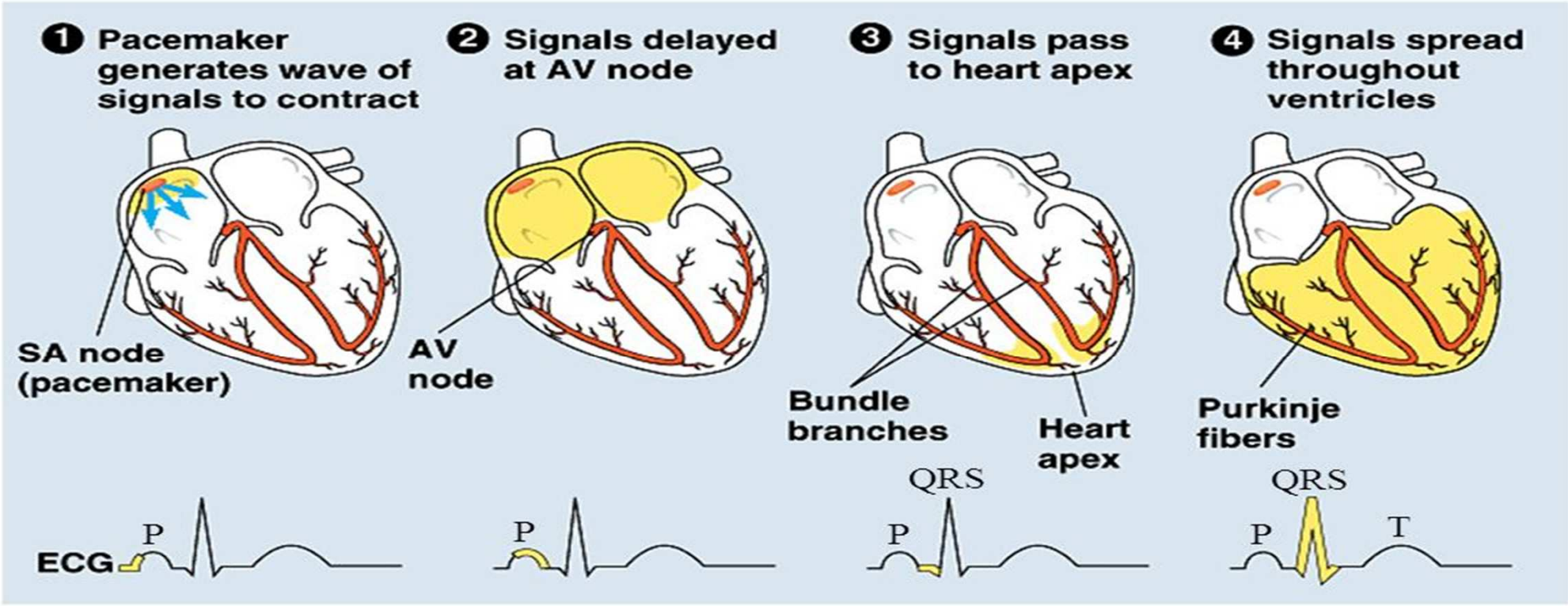


FORMAZIONE DELL'IMPULSO



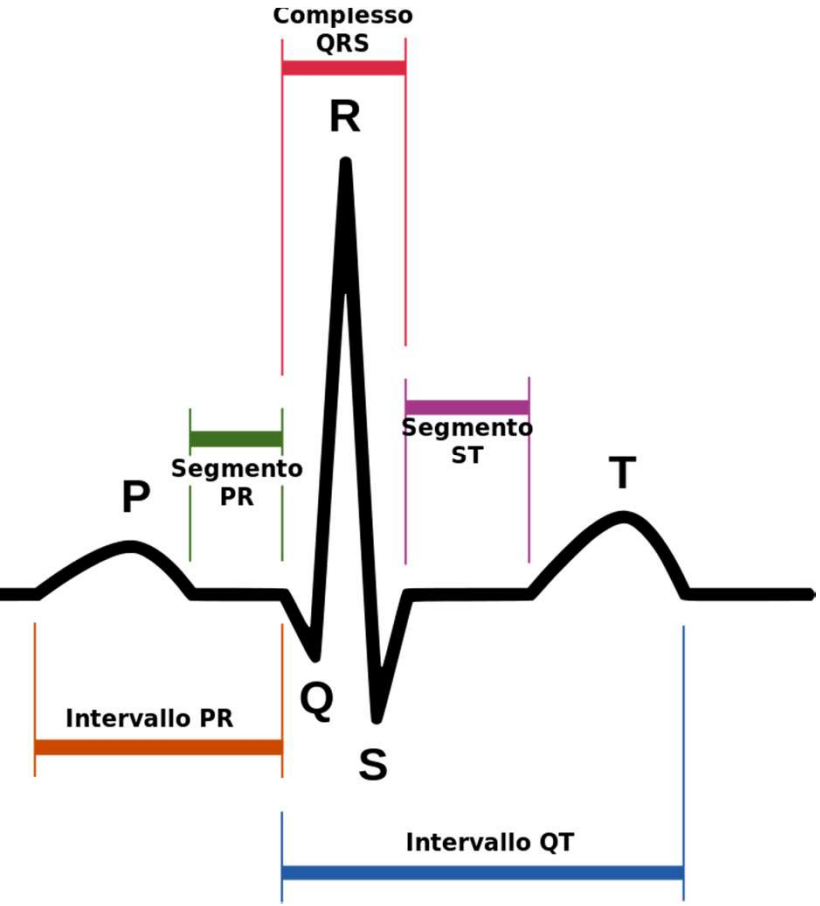
ATTIVAZIONE ELETTRICA DEL CUORE

La sequenza spaziale e temporale dell'attivazione elettrica del cuore è determinata da proprietà elettrofisiologiche (differenze nella velocità di conduzione del PA nei diversi tipi cellulari del cuore) e morfologiche (distribuzione del sistema di conduzione; presenza dello scheletro fibroso; orientamento dei fasci miocardici).

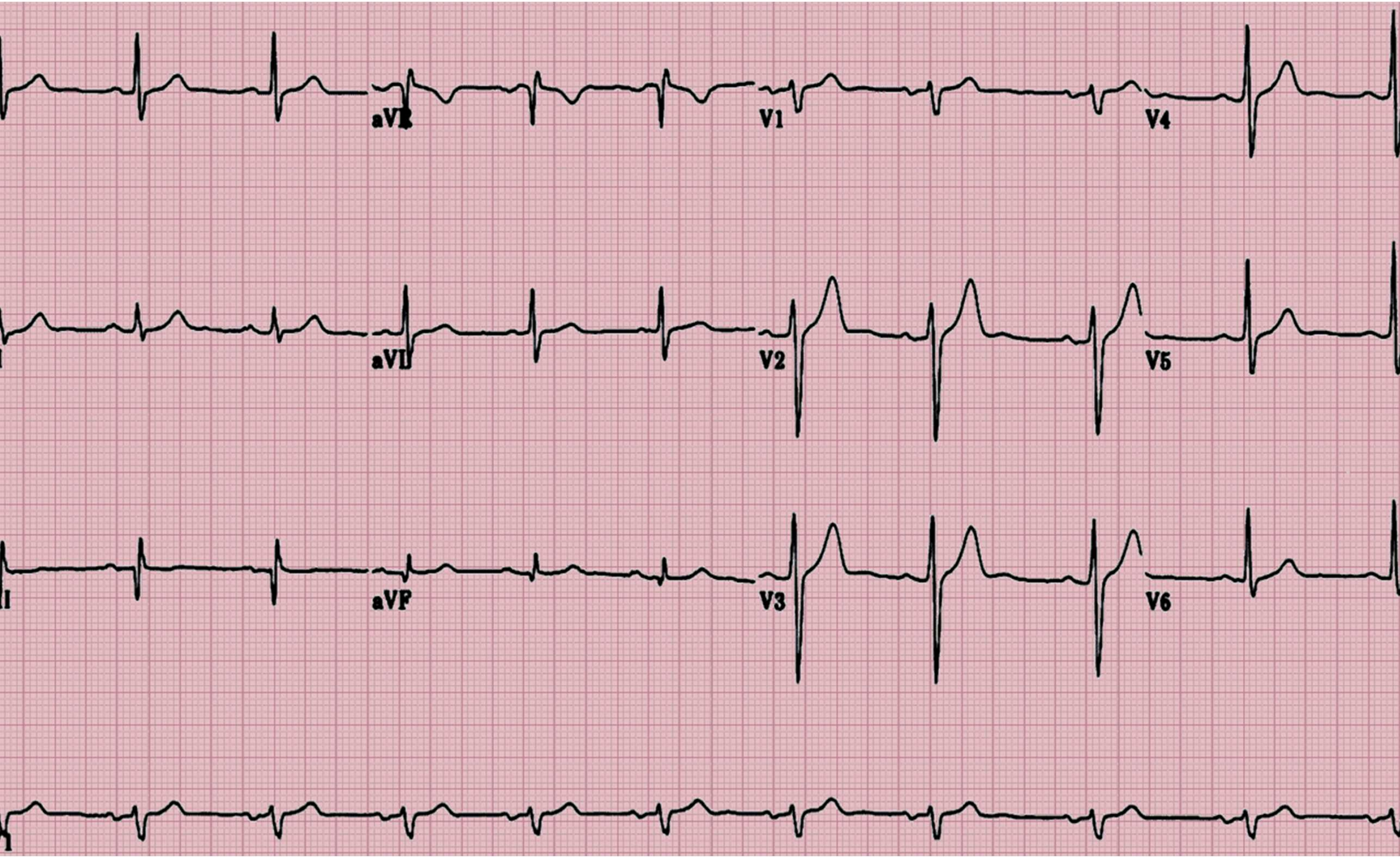


L'EFFETTO FINALE

MA SULL'ECG?



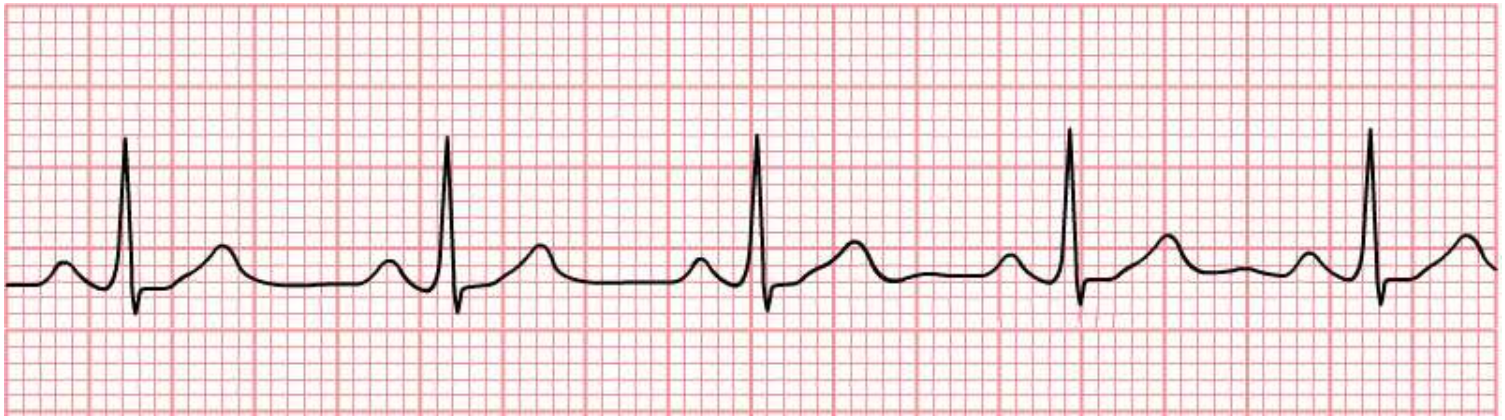
- Onda P → Depolarizzazione atriale
- Tratto PR → Passaggio impulso attraverso il NAV
- Complesso QRS → Depolarizzazione ventricolare
- Tratto ST → Periodo refrattario e inizio ripolarizzazione ventricolare
- Onda T → Ripolarizzazione ventricolare


aVR
V1
V4
aVL
V2
V5
aVF
V3
V6

COS'È IL RITMO SINUSALE?

- Ritmo fisiologico cardiaco

- Presenta alcune caratteristiche elettrocardiografiche:
  - P positiva in D1e D2
  - P negativa in aVR
  - FC tra 60-100 bpm (BS se FC<60 bpm, TS se >100 bpm)



ARITMIE

Cos'è un'aritmia?

Ogni situazione non classificabile come ritmo cardiaco normale, inteso come ritmo ad origine nel nodo del seno, regolare e con normale frequenza e conduzione

CLASSIFICAZIONE DELLE ARITMIE

- Aritmie ipocinetiche (Bradiaritmie)
- Aritmie ipercinetiche (Tachiaritmie)

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ARITMIE IPOCINETICHE

nomalie di conduzione (o **blocchi cardiaci**) consistono nel parziale o totale impedimento alla duzione dell'impulso lungo le normali vie fisiologiche.

conda della sede del blocco distinguiamo:

alattia del nodo del seno (SSS)

occo AV di 1° grado

occo AV di 2° grado

- ***di tipo I di Mobitz***
- ***di tipo II di Mobitz***

occo AV di 3° grado

occhi fascicolari

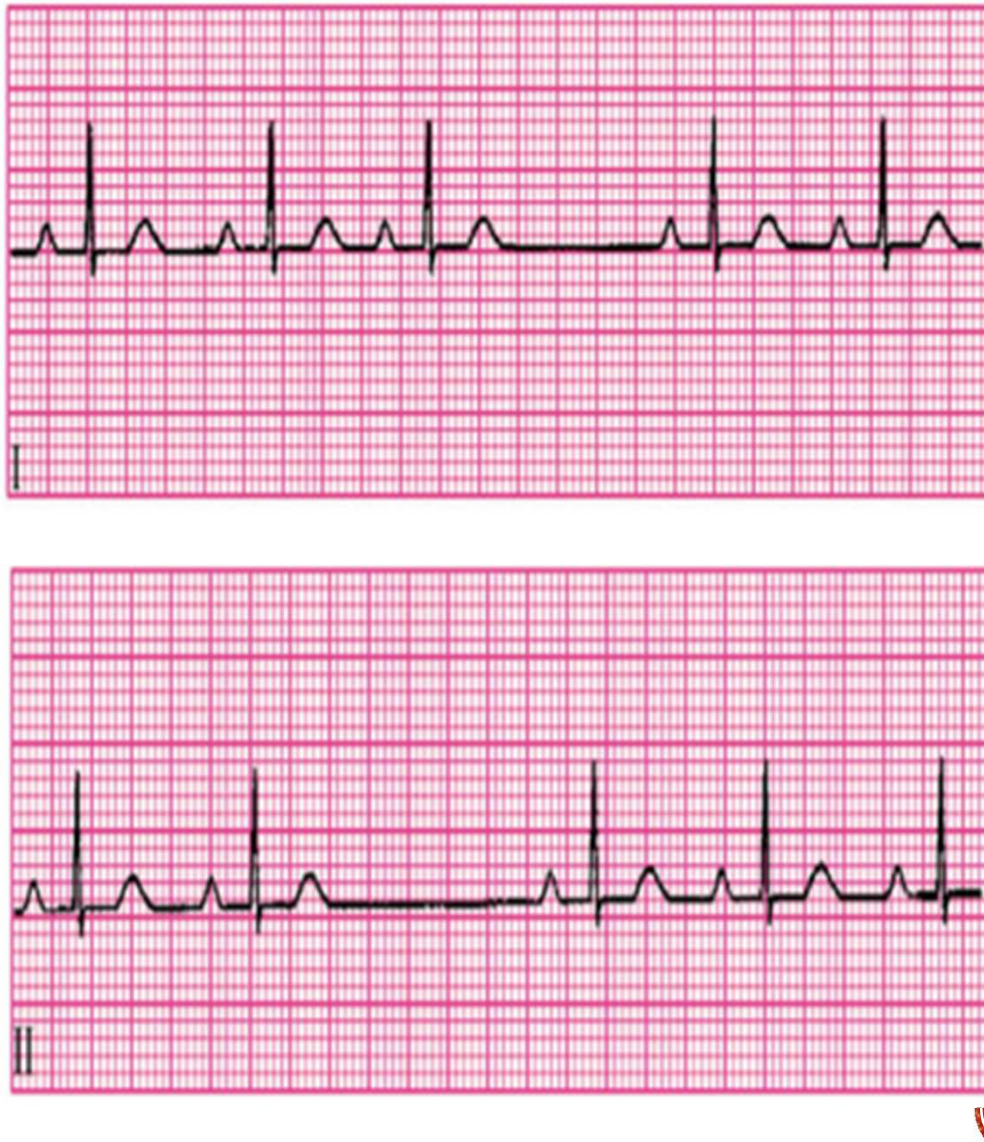
- ***Emiblocco anteriore sinistro***
- ***Emiblocco posteriore sinistro***

occo di branca destra/sinistra

SSS

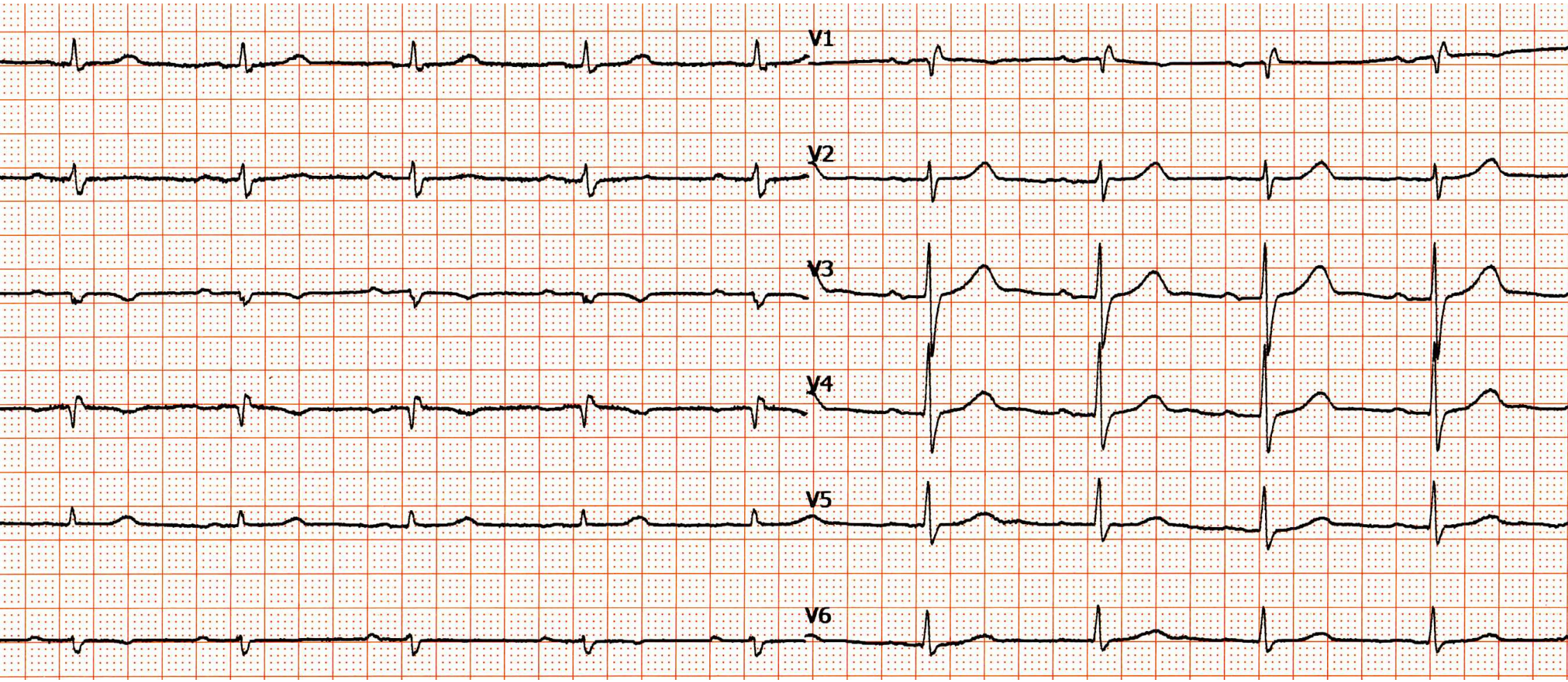
Caratterizzata dalla presenza di ***bradicardia sinusale***, ***arresto sinusale*** (mancata formazione dell'impulso nel nodo del seno) o **blocchi seno-atriali** (disturbi di conduzione dell'impulso, regolarmente formatosi nel nodo del seno, agli atri) spesso a carattere intermittente; a volte vi è alternanza tra fasi bradicardiche ed aritmie ipercinetiche sopraventricolari (***s. bradi-tachi***) poiché la bradicardia rende disomogenei i periodi refrattari e favorisce i fenomeni di rientro.

Talora la diagnosi differenziale tra le diverse forme può essere fatta solo con lo studio elettrofisiologico; si manifestano con la mancanza intermittente di una o più onde P (***pausa***).



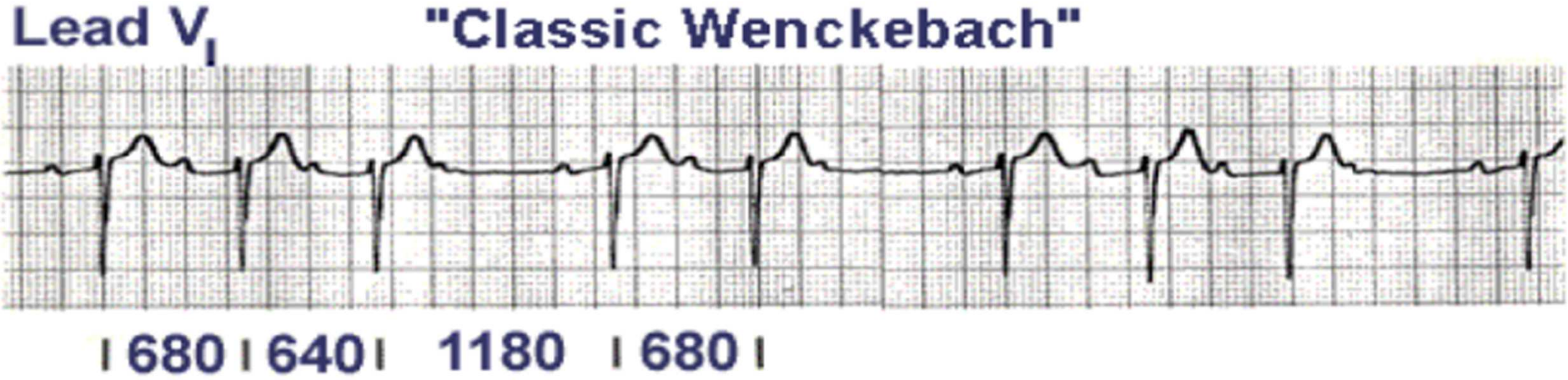
BLOCCHI ATRIOVENTRICOLARI

BAV I Grado: Allungamento del tratto PR oltre i limiti di norma (v.n. 120-200 ms)



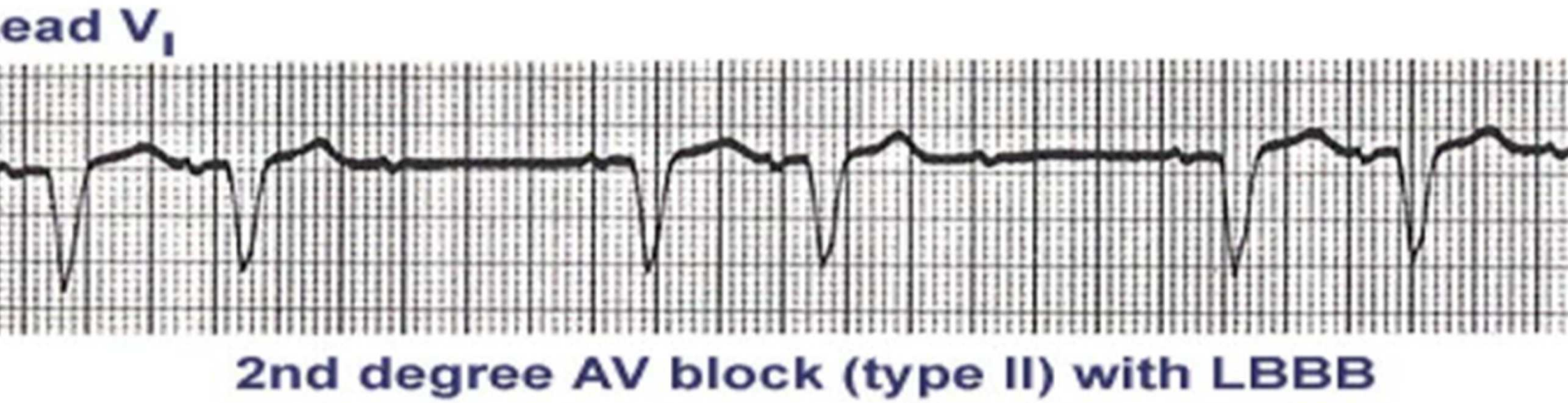
BLOCCHI ATRIOVENTRICOLARI

BAV II M1: Progressivo allungamento del PR finché una P non è seguita da QRS



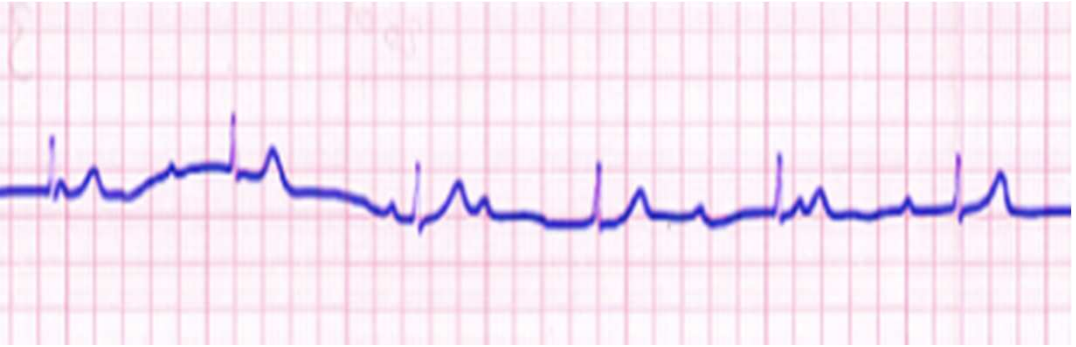
BLOCCHI ATRIOVENTRICOLARI

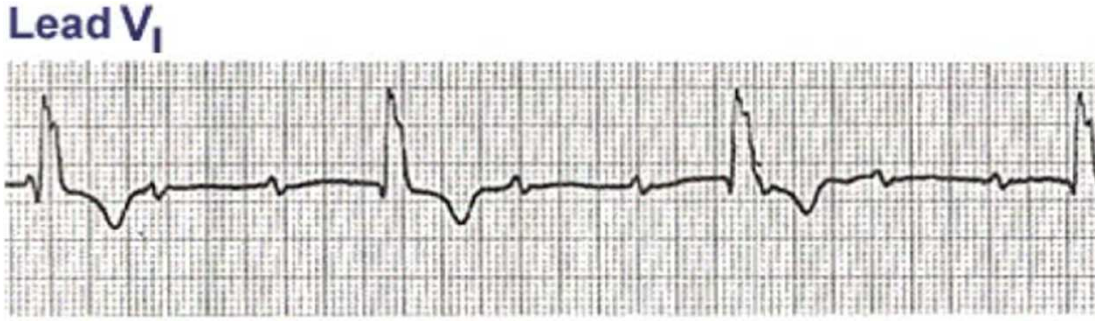
II M2: Improvvisa mancata conduzione di un impulso sinusale con un'onda P non seguita da QI



BLOCCHI ATRIOVENTRICOLARI

BAV III: Completa dissociazione AV





<u>Scappamento giunzionale</u>:
- QRS stretto (80-120 ms)
- FC 40-50 bpm

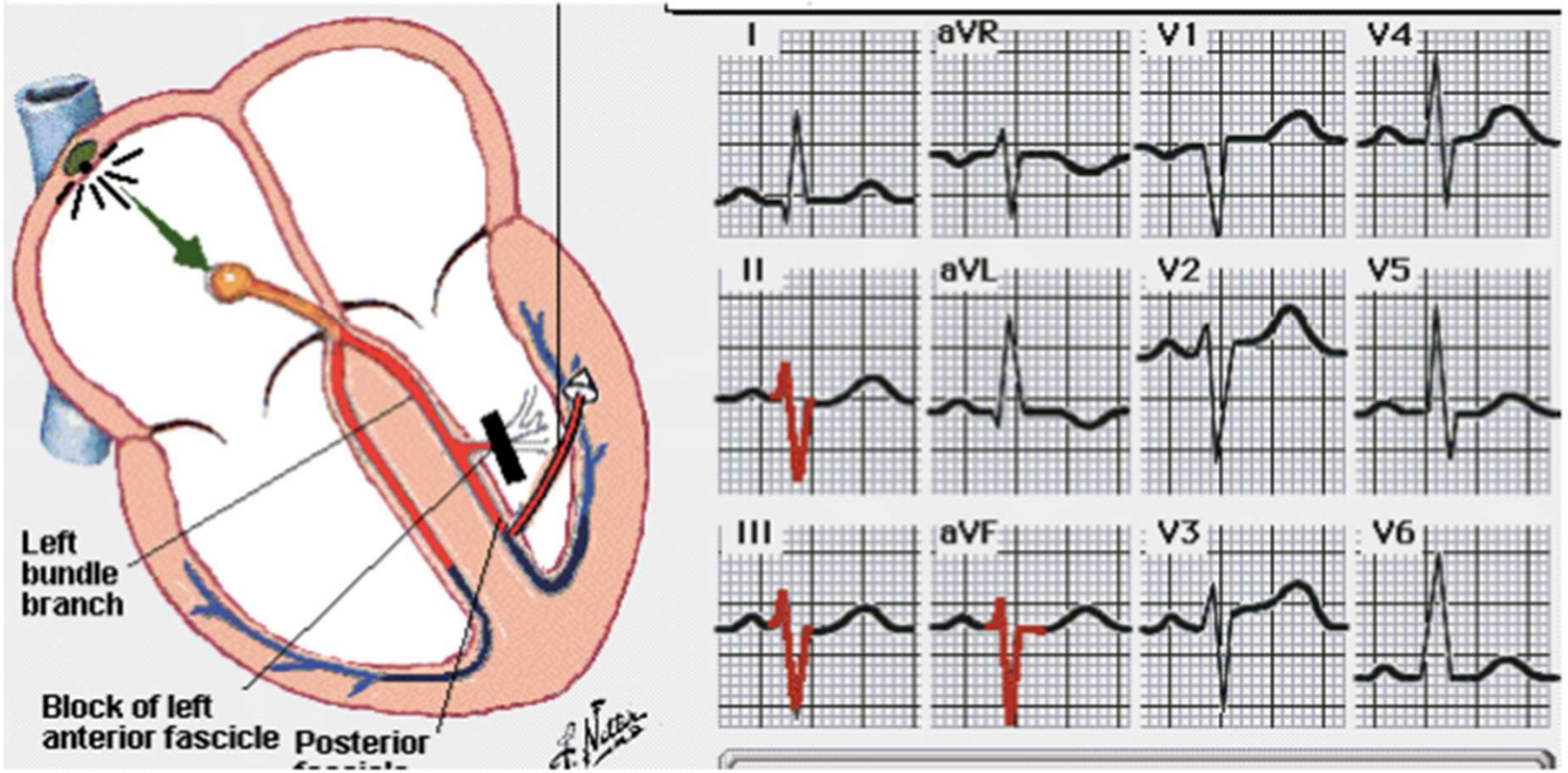
<u>Scappamento ventricolare</u>:
- QRS largo (>120 ms)
- FC 30-40 bpm

BLOCCHI FASCICOLARI

EAS

QRS allungato ma non prolungato, con deviazione assiale sinistra.

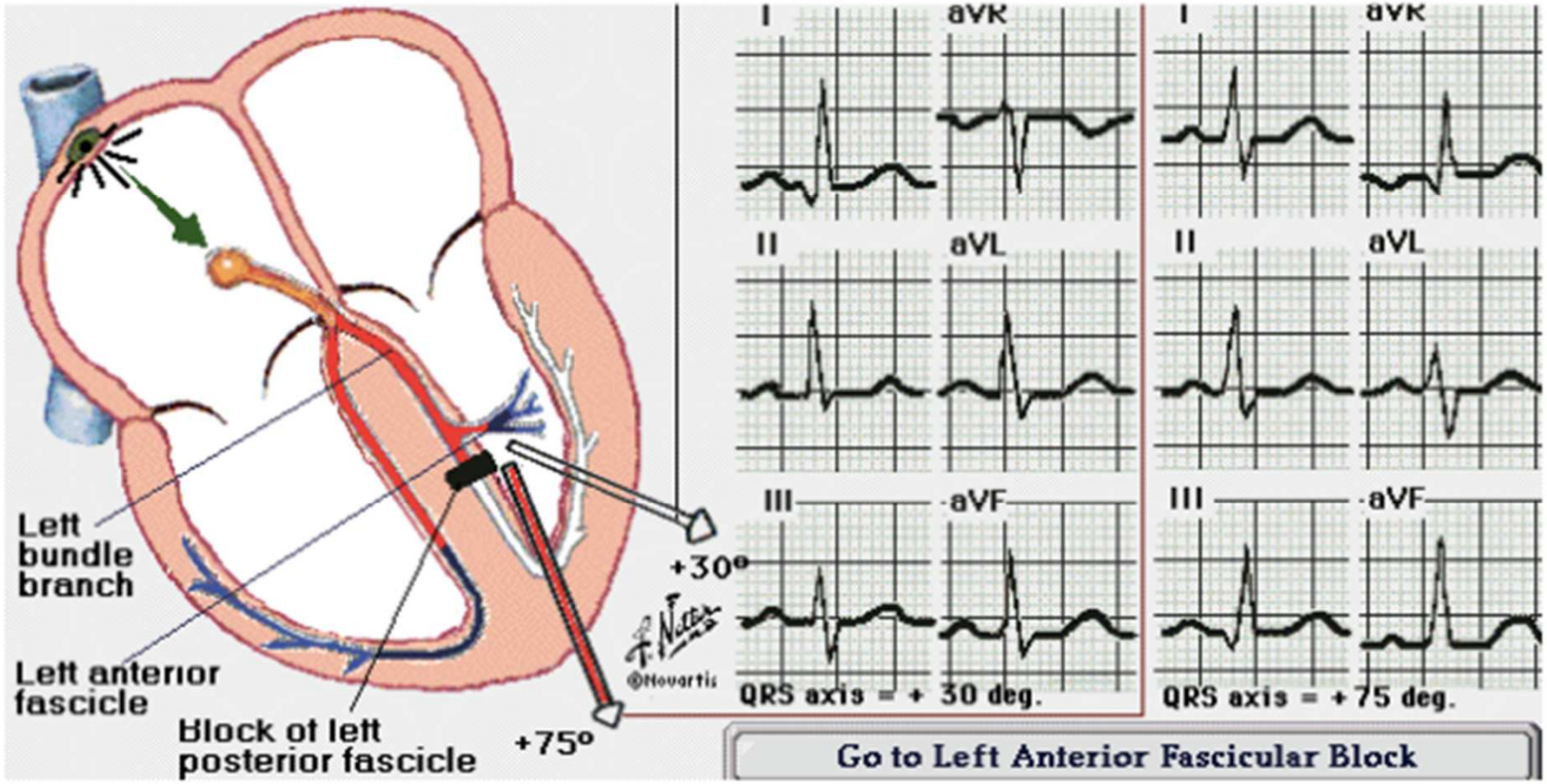
Dovuto a interruzione dellla branca anteriore



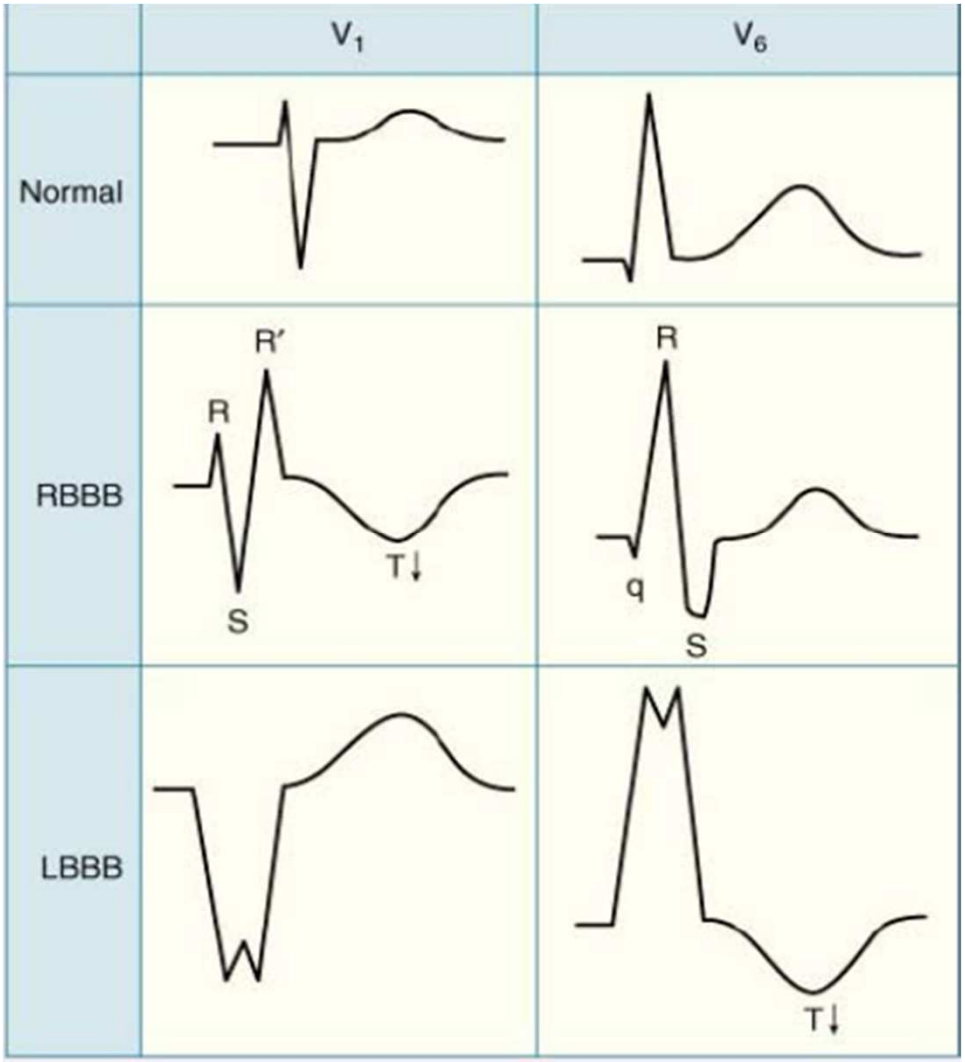
BLOCCHI FASCICOLARI

EPS

Difficile identificazione se non per allungamento del qrs con asse elettrico deviato in basso



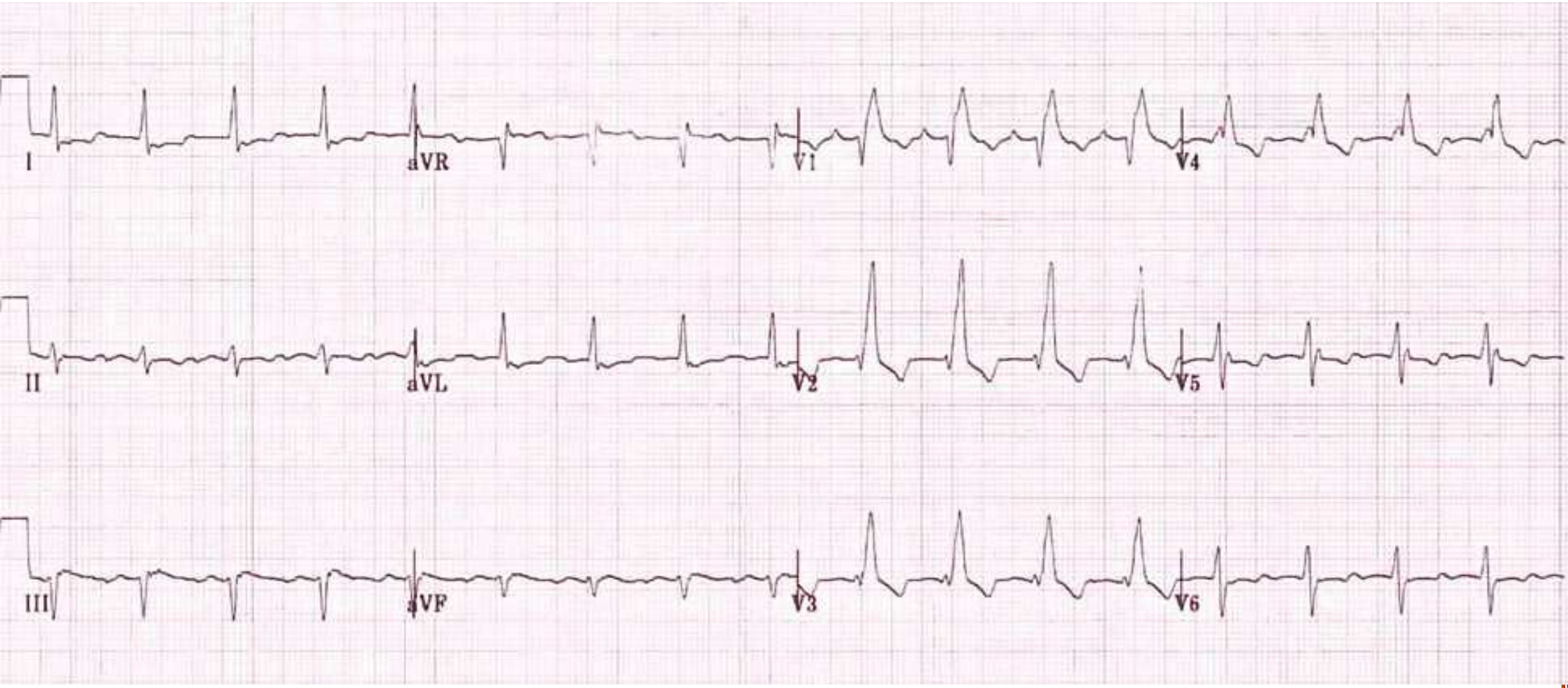
BLOCCHI DI BRANCA



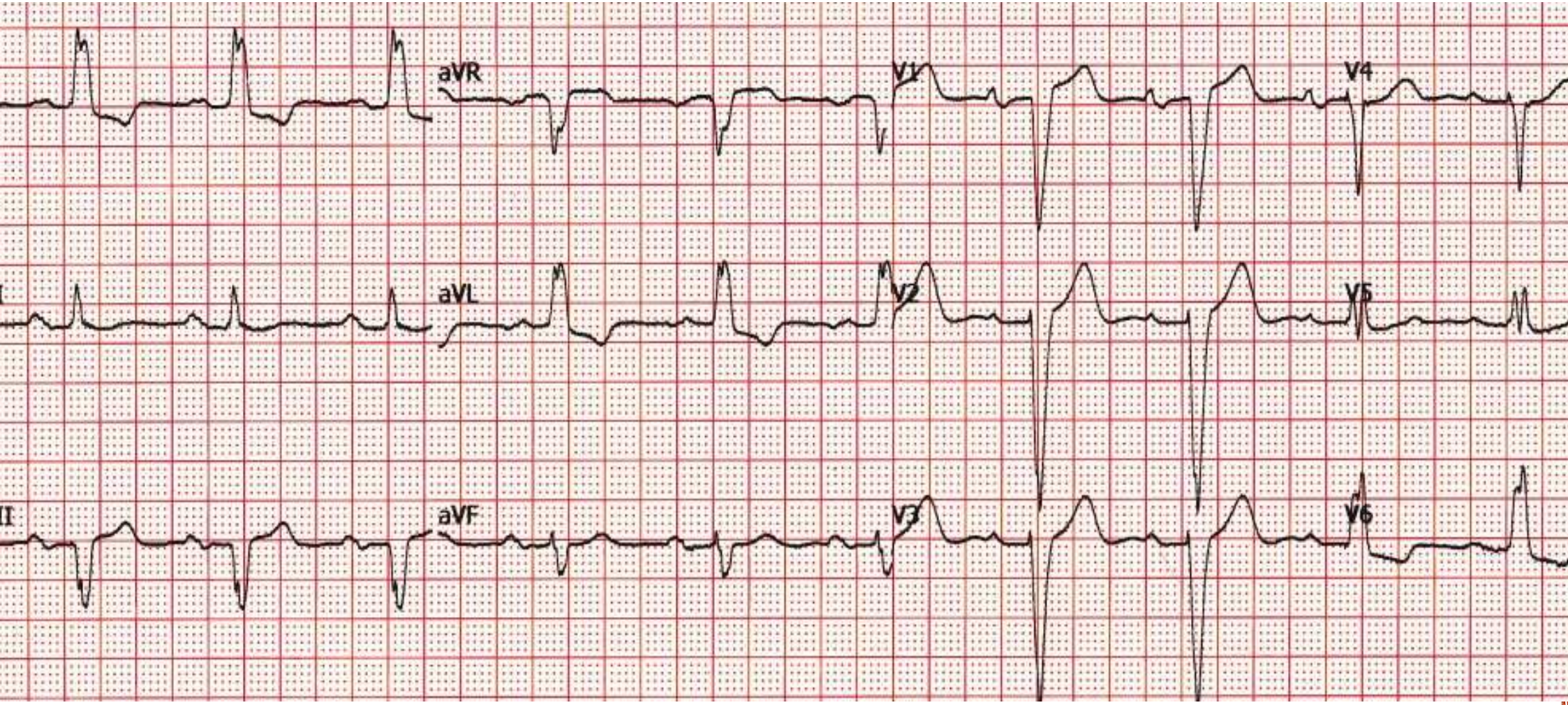
QRS >0.12 secondi
rSr' in V1-V3
onda S in V5-V6 alterazioni della ripolarizzazione

QRS >0.12 secondi
QRS negativo in V1-V2
QRS positivo in V5-V6 (bifido) assenza onde R in V1-V2
assenza onde Q in D1, aVL, V5, V6
alterazioni della ripolarizzazione

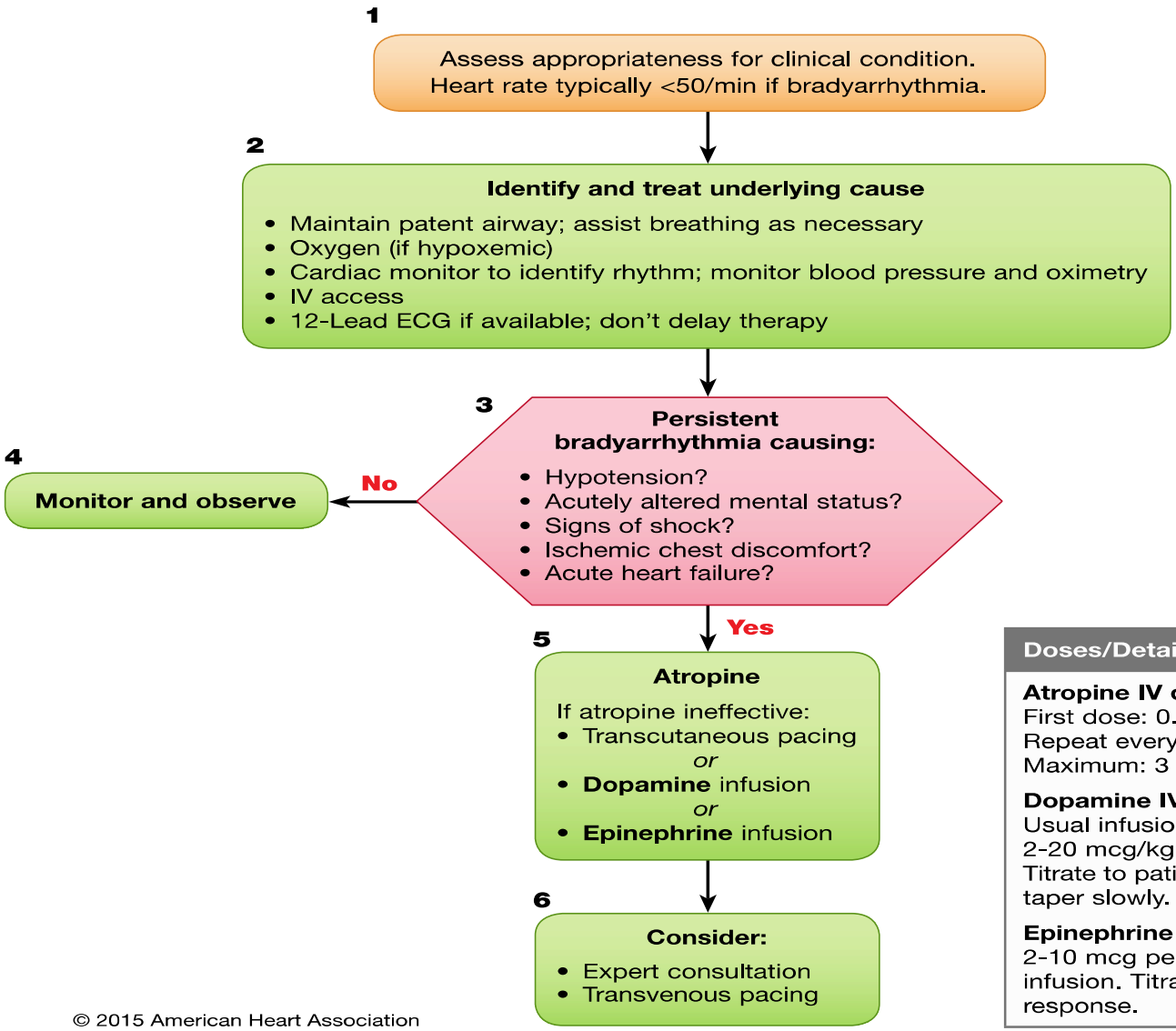
BBDX



BBSX



Adult Bradycardia With a Pulse Algorithm



Doses/Details

Atropine IV dose:
First dose: 0.5 mg bolus.
Repeat every 3-5 minutes.
Maximum: 3 mg.

Dopamine IV infusion:
Usual infusion rate is 2-20 mcg/kg per minute.
Titrate to patient response; taper slowly.

Epinephrine IV infusion:
2-10 mcg per minute infusion. Titrate to patient response.

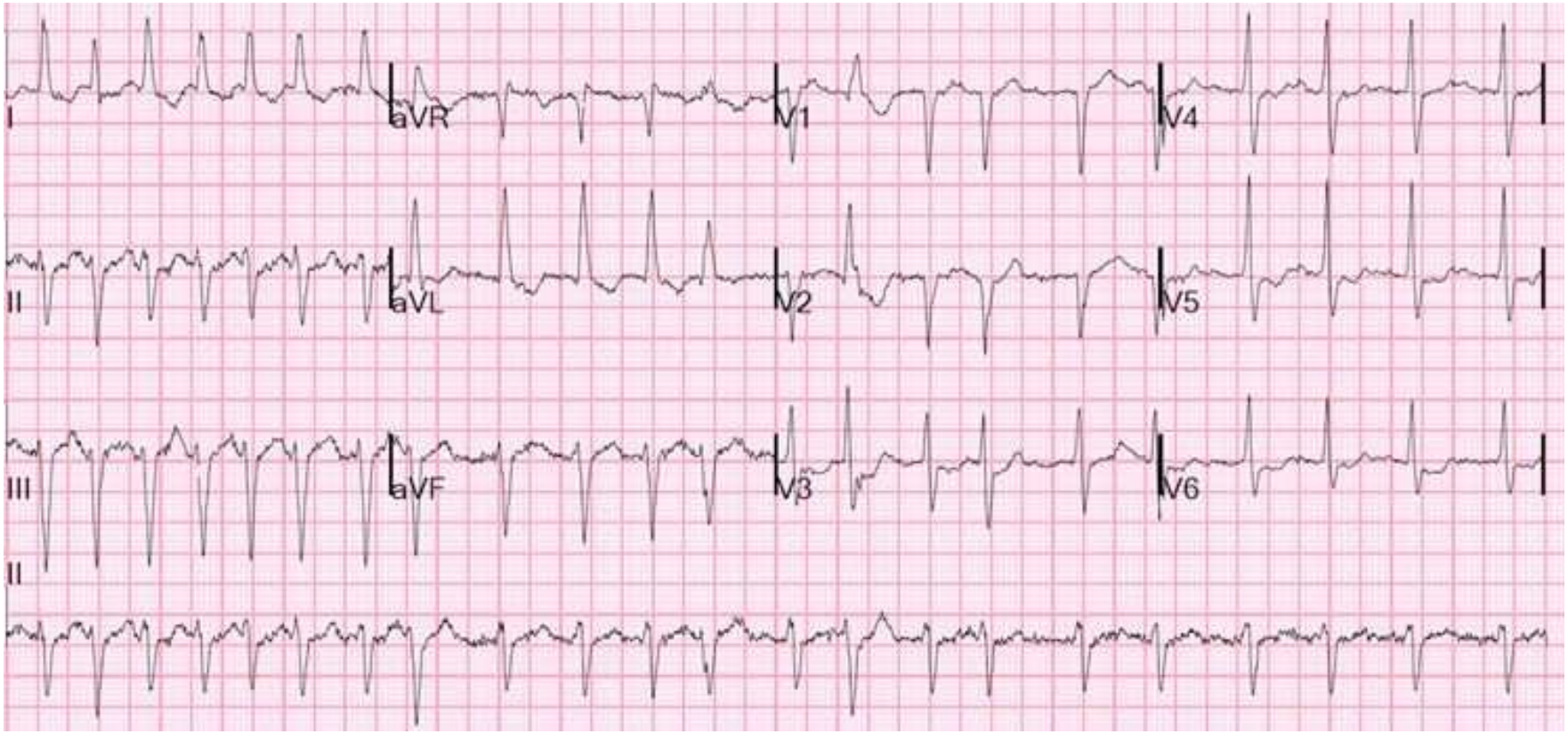
CASO CLINICO 2

- ♂, 56 aa
- Entra in PS per dispnea e cardiopalmo insorti da circa 30 minuti
- In anamnesi: cardiopatia ischemica post-infartuale, ipertensione arteriosa, ex fumatore
- All'arrivo: PA 95/55 mmHg, FC 139 bpm, SpO2 86% in aa

INDAGINI ESEGUITE

- Esami ematochimici: lieve incremento indici miocardiocitonecrosi e BNP
- Rx Torace: negativo
- ECG: Tachifibrillazione atriale con fcm 130 bpm

ECG



- Paziente rapidamente portato in shock room dove veniva eseguita CVE con beneficio

CLASSIFICAZIONE DELLE ARITMIE

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- Aritmie ipercinetiche (Tachiaritmie)

ARITMIE IPERCINETICHE

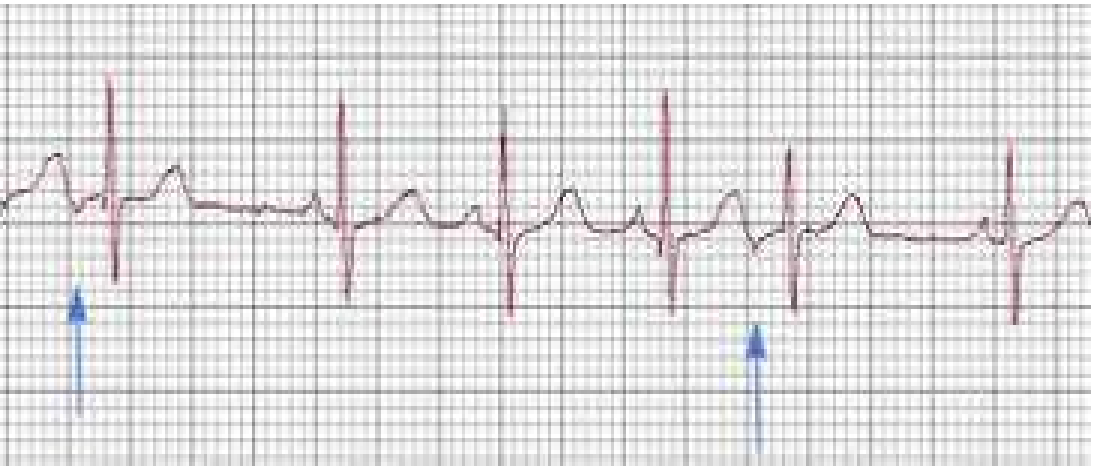
Sono alterazioni del ritmo cardiaco caratterizzate o da un aumento della frequenza sinusale *(**tachicardia sinusale**)* o dalla presenza di battiti anticipati rispetto al ritmo cardiaco di base ed ectopici cioè originanti in sede diversa dal nodo del seno. Tali sedi possono essere:

- Il miocardio atriale ed il tessuto di conduzione atrio-ventricolare fino al fascio di His *(**aritmie ipercinetiche sopraventricolari**)*
- Il tessuto di conduzione a livello del sistema hissiano ed il miocardio ventricolare *(**aritmie ipercinetiche ventricolari**)*

ARITMIE IPERCINETICHE SOPRAVENTRICOLARI

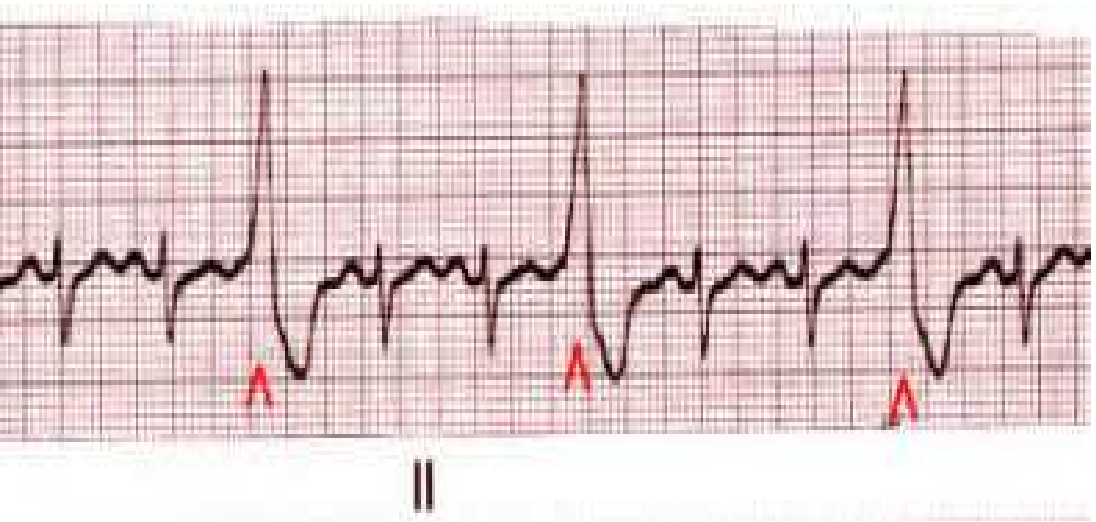
- **A localizzazione atriale**
  - Tachicardia sinusale
  - Tachicardia atriale
  - Flutter atriale
  - Fibrillazione atriale
- **A localizzazione giunzionale**
- **A localizzazione atrio-ventricolare**

EXTRASISTOLI



BESV

Battito ectopico ad origine sopraventricolare. Caratterizzato da QRS stretto e possibile P retrocondotta

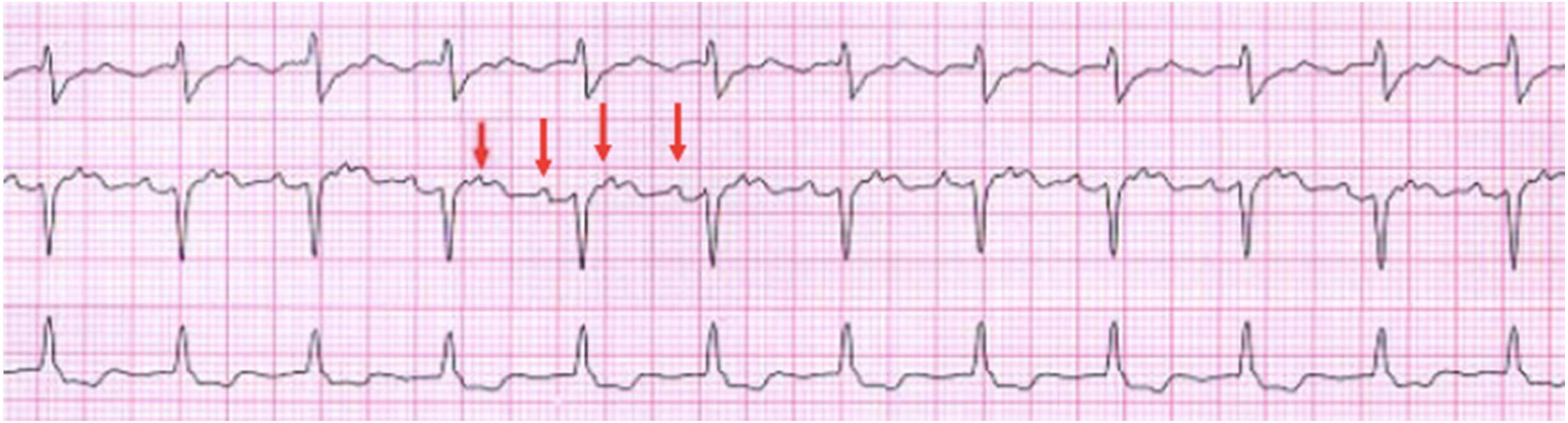


BEV

Battito ectopico ad origine ventricolare. Caratterizzato da QRS largo con morfologia totalmente differente dai normali QRS.

TACHICARDIA ATRIALE

- ***Serie di 3 o più onde P ectopiche di identica morfologia che si susseguono con una frequenza >100/min (130-210/min) con intervalli P-P costanti***
- ***Rapporto di conduzione AV variabile in funzione del blocco nodale (1:1, 2:1, etc.)***
- ***QRS stretti (larghi in presenza di blocco di branca preesistente o conduzione aberrante)***



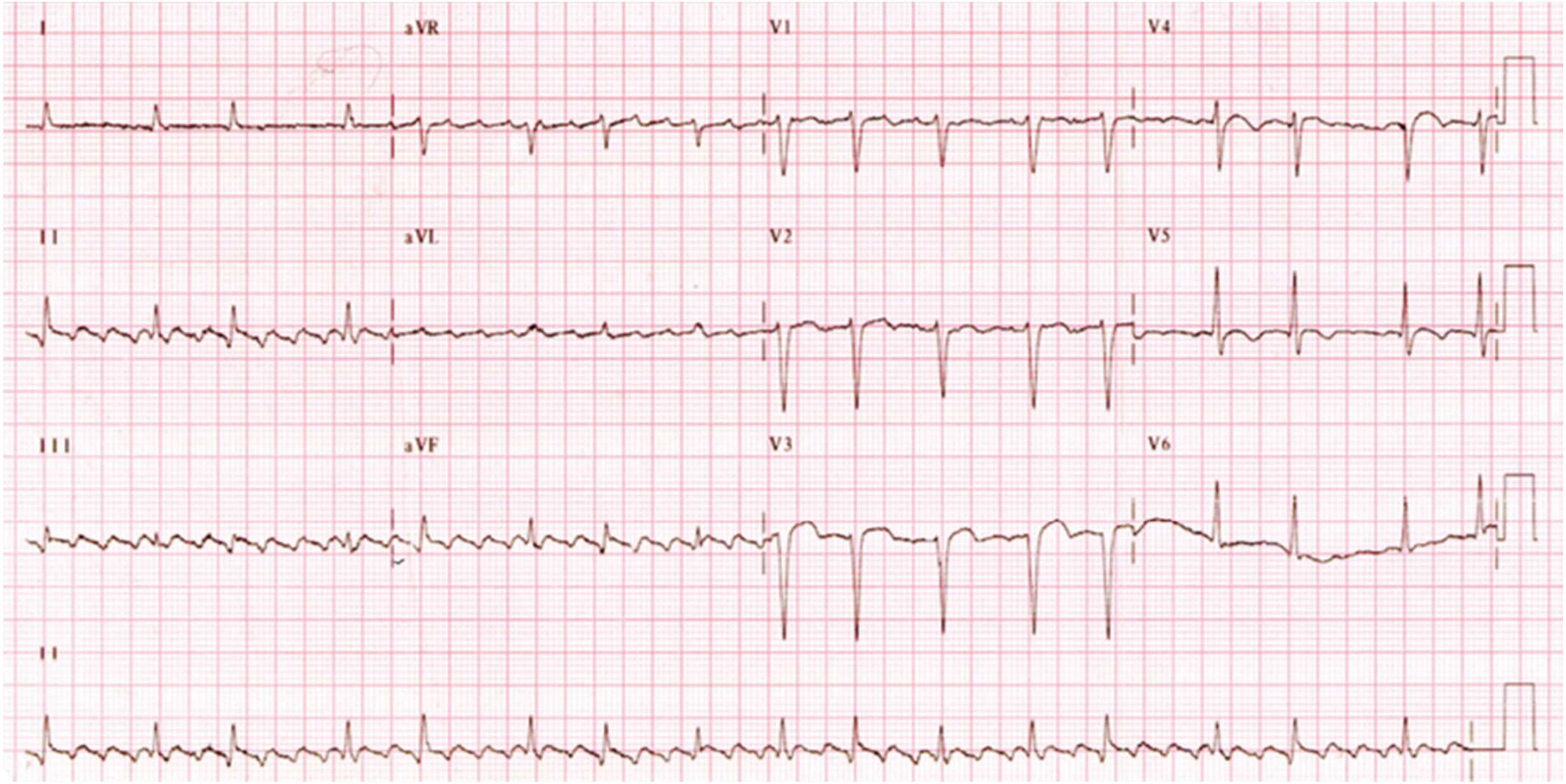
FLUTTER ATRIALE

'elettrogenesi del Flutter atriale riconosce un meccanismo di acrorientro localizzato nell'atrio destro

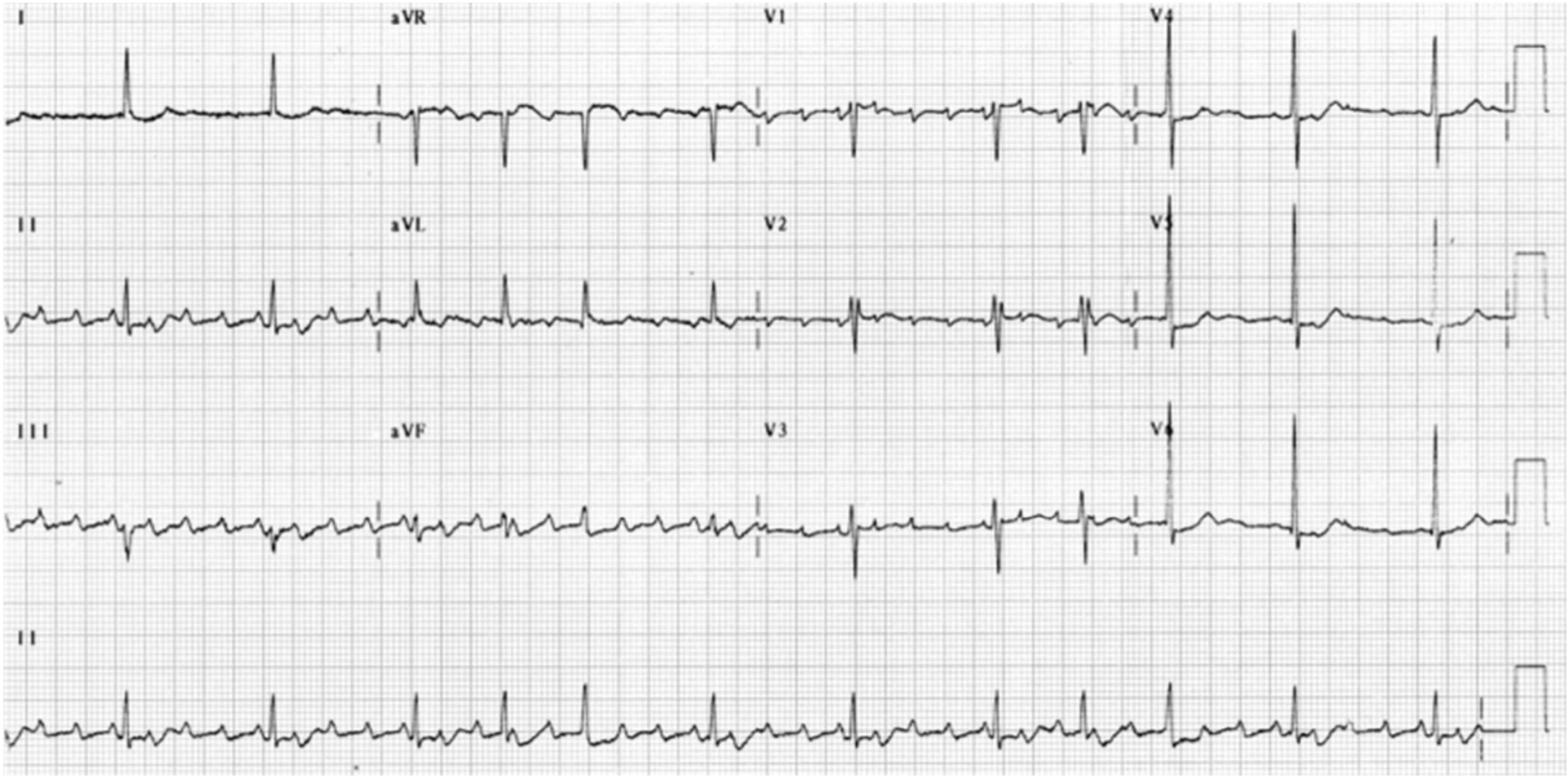
<u>Comune</u>: circola in senso antiorario attorno all'anello della tricuspide
→ *All'ecg onde atriali (onde F) a denti di sega (negative/positive)nelle derivazioni D2,D3,aVF senza linea isoelettrica interposta e branche asimmetriche*

<u>Non comune</u>: circola in senso orario attorno all'anello della tricuspide
→ *All'ecg onde atriali (onde F) a denti di sega ma hanno una prevalente positività nelle derivazioni D2,D3,aVF e branche simmetriche*

FLUTTER ATRIALE COMUNE

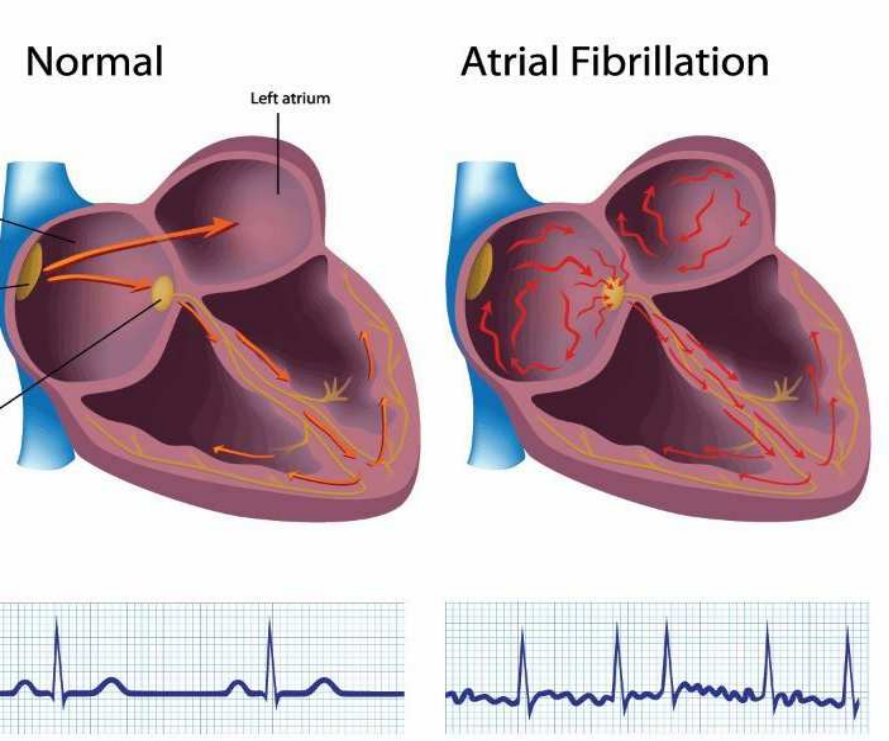


FLUTTER ATRIALE NON COMUNE



FIBRILLAZIONE ATRIALE

Tachiaritmia sopraventricolare caratterizzata da un'attivazione atriale scoordinata, irregolare e frammentaria, con la contemporanea presenza di più fronti d'onda che circolano in modo imprevedibile, così che la diffusione dell'impulso negli atri è continuamente variabile



Meccanismo di rientro multiplo e irregolare

Conseguente deterioramento della funzione meccanica atriale

2016 ESC Guidelines for the diagnosis and treatment of atrial fibrillation

European Heart Journal
doi:10.1093/eurheartj/ehw210

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

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

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

Endorsed by the European Stroke Organisation (ESO)

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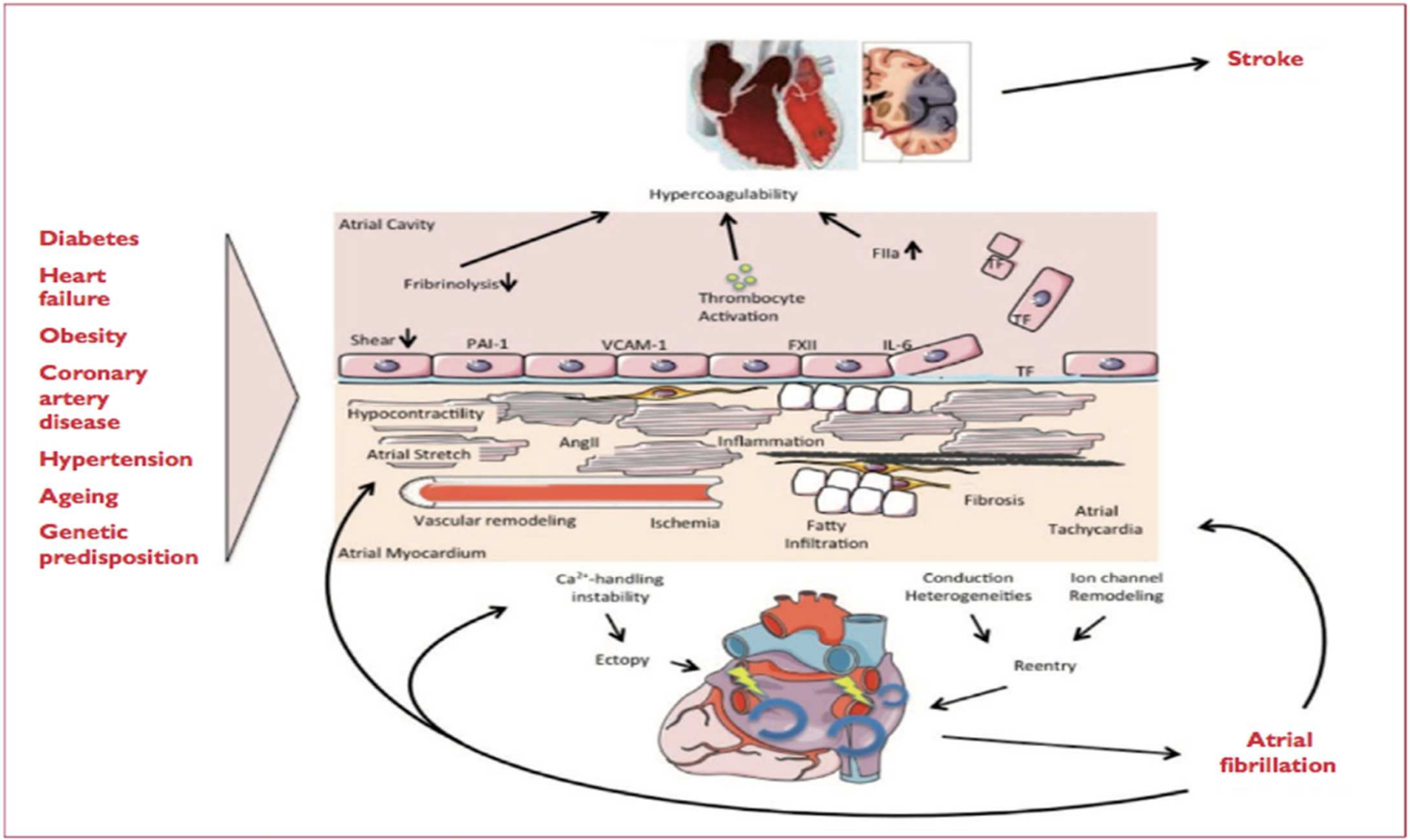
FISIOPATOLOGIA



AngII = angiotensin II; TF = tissue factor; FXII = factor XII; IL-6 = interleukin 6; PAI-1 = plasminogen activator inhibitor 1; VCAM-1 = vascular cell adhesion molecule 1.

SCREENENG E CLASSIFICAZIONE

Recommendations for screening for atrial fibrillation

Recommendations	Class[a]	Level[b]	Ref[c]
Opportunistic screening for AF is recommended by pulse taking or ECG rhythm strip in patients >65 years of age.	I	B	130, 134, 155
In patients with TIA or ischaemic stroke, screening for AF is recommended by short-term ECG recording followed by continuous ECG monitoring for at least 72 hours.	I	B	27, 127
It is recommended to interrogate pacemakers and ICDs on a regular basis for atrial high rate episodes (AHRE). Patients with AHRE should undergo further ECG monitoring to document AF before initiating AF therapy.	I	B	141, 156
In stroke patients, additional ECG monitoring by long-term non-invasive ECG monitors or implanted loop recorders should be considered to document silent atrial fibrillation.	IIa	B	18, 128
Systematic ECG screening may be considered to detect AF in patients aged >75 years, or those at high stroke risk.	IIb	B	130, 135, 157

AF = atrial fibrillation; AHRE = atrial high rate episodes; ECG = electrocardiogram; ICD = implantable cardioverter defibrillator; TIA = transient ischaemic attack.
[a]Class of recommendation.
[b]Level of evidence.
[c]Reference(s) supporting recommendations.

Table 5 Patterns of atrial fibrillation

AF pattern	Definition
First diagnosed AF	AF that has not been diagnosed before, irrespective of the duration of the arrhythmia or the presence and severity of AF-related symptoms.
Paroxysmal AF	Self-terminating, in most cases within 48 hours. Some AF paroxysms may continue for up to 7 days.[a] AF episodes that are cardioverted within 7 days should be considered paroxysmal.[a]
Persistent AF	AF that lasts longer than 7 days, including episodes that are terminated by cardioversion, either with drugs or by direct current cardioversion, after 7 days or more.
Long-standing persistent AF	Continuous AF lasting for ≥1 year when it is decided to adopt a rhythm control strategy.
Permanent AF	AF that is accepted by the patient (and physician). Hence, rhythm control interventions are, by definition, not pursued in patients with permanent AF. Should a rhythm control strategy be adopted, the arrhythmia would be re-classified as 'long-standing persistent AF'.

AF = atrial fibrillation.
[a]The distinction between paroxysmal and persistent AF is often not made correctly without access to long-term monitoring.[163] Hence, this classification alone is often insufficient to select specific therapies. If both persistent and paroxysmal episodes are present, the predominant pattern should guide the classification.

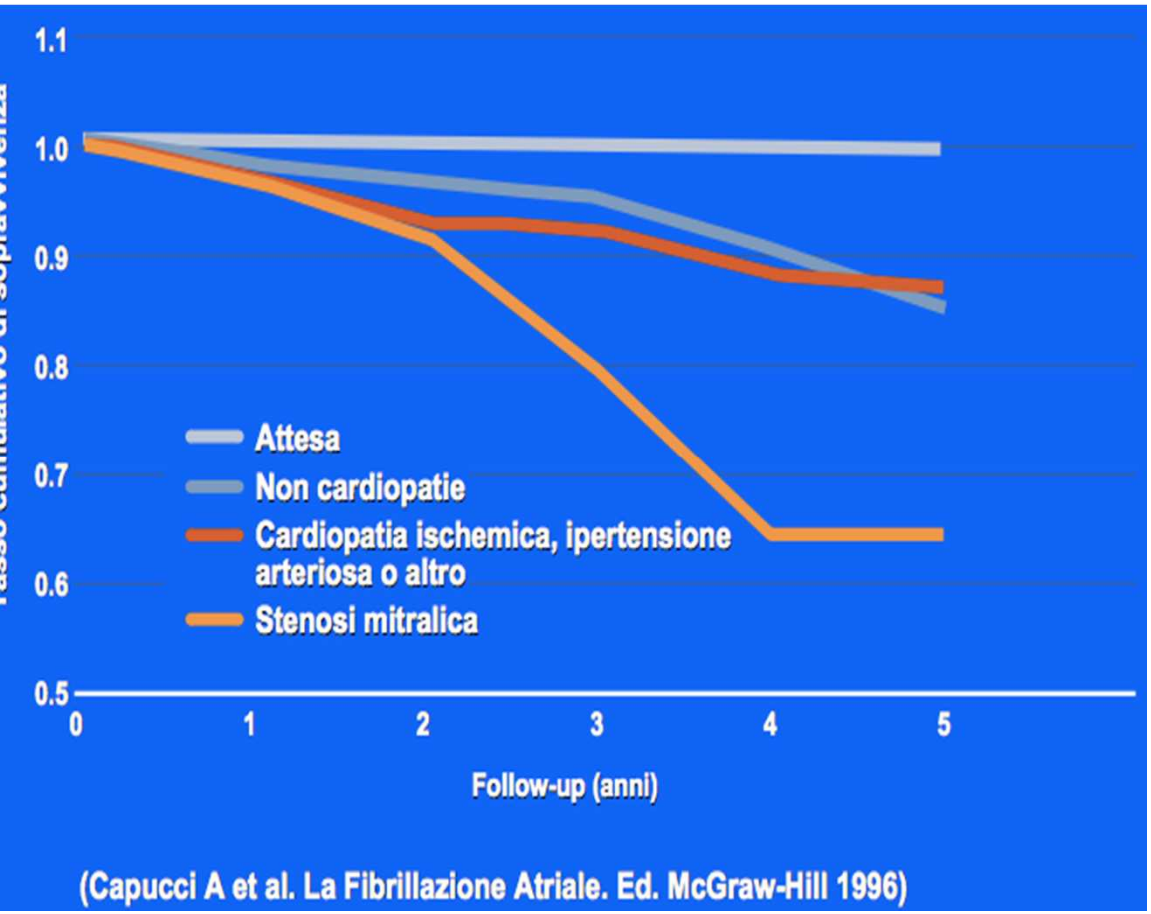
TIPOLOGIE CLINICHE

Table 6 **Clinical types of atrial fibrillation**[a]

AF type	Clinical presentation	Possible pathophysiology
AF secondary to structural heart disease	AF in patients with LV systolic or diastolic dysfunction, long-standing hypertension with LVH, and/or other structural heart disease. The onset of AF in these patients is a common cause of hospitalization and a predictor of poor outcome.	Increased atrial pressure and atrial structural remodelling, together with activation of the sympathetic and renin-angiotensin system.
Focal AF	Patients with repetitive atrial runs and frequent, short episodes of paroxysmal atrial fibrillation. Often highly symptomatic, younger patients with distinguishable atrial waves (coarse AF), atrial ectopy, and/or atrial tachycardia deteriorating in AF.	Localized triggers, in most cases originating from the pulmonary veins, initiate AF. AF due to one or a few re-entrant drivers is also considered to be part of this type of AF.
Polygenic AF	AF in carriers of common gene variants that have been associated with early onset AF.	Currently under study. The presence of selected gene variants may also influence treatment outcomes.
Postoperative AF	New onset of AF (usually self-terminating) after major (typically cardiac) surgery in patients who were in sinus rhythm before surgery and had no prior history of AF.	Acute factors: inflammation, atrial oxidative stress, high sympathetic tone, electrolyte changes, and volume overload, possibly interacting with a pre-existing substrate.
AF in patients with mitral stenosis or prosthetic heart valves	AF in patients with mitral stenosis, after mitral valve surgery and in some cases other valvular disease.	Left atrial pressure (stenosis) and volume (regurgitation) load are the main drivers of atrial enlargement and structural atrial remodelling in these patients.
AF in athletes	Usually paroxysmal, related to duration and intensity of training.	Increased vagal tone and atrial volume.
Monogenic AF	AF in patients with inherited cardiomyopathies, including channelopathies.	The arrhythmogenic mechanisms responsible for sudden death are likely to contribute to the occurrence of AF in these patients.

Clinical types of AF are modified from the report on the 4th AFNET/EHRA consensus conference[76,a]. AF = atrial fibrillation; AFNET = German Competence NETwork on Atrial Fibrillation; EHRA = European Heart Rhythm Association; LV = left ventricular; LVH = left ventricular hypertrophy. It is recognized that these types of AF will overlap in clinical practice, and that their impact for management needs to be evaluated systematically.

MORTALITÀ PER I PAZIENTI CON FA



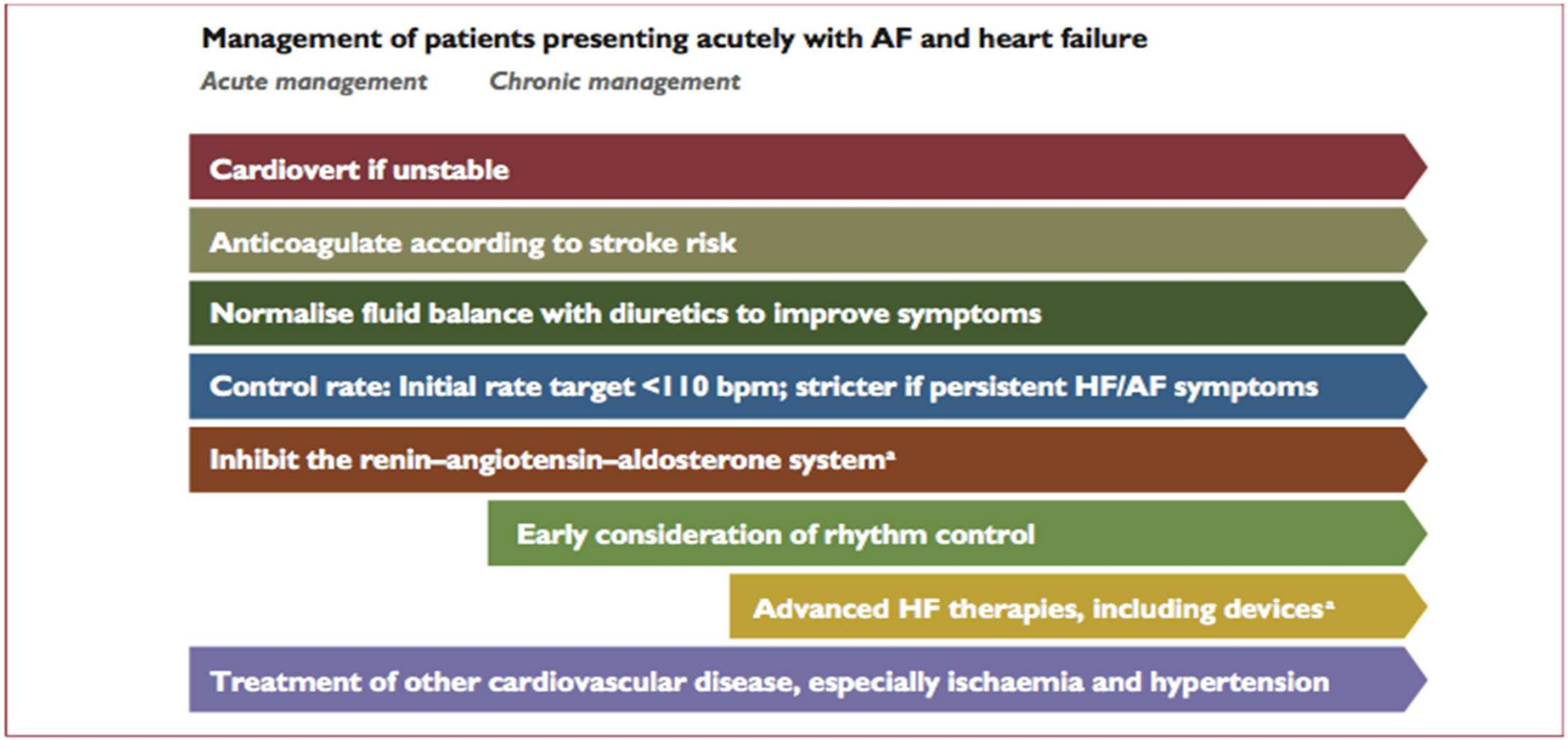
(Capucci A et al. La Fibrillazione Atriale. Ed. McGraw-Hill 1996)

- **Soggetti con FA isolata:** nessun aumento di mortalità

- **Soggetti con cardiopatia e/o fattori di risch**
mortalità +50% tra gli uomini e +90% nelle don
rispetto a quanto osservato nella popolazione generale

FA causa sostanzialmente morbidità. Non è ancora dimostrato se tale aument di mortalità sia dovuto alla FA di per sé oppure ad una condizione clinica sottostante più grave o fattori di rischi associati.

GESTIONE CLINICA



ACE = angiotensin-converting enzyme; AF = atrial fibrillation; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor neprilysin inhibition; bpm = beats per minute; HF = heart failure.
[a]In patients with heart failure and reduced ejection fraction. Also consider combined ARNI in patients able to tolerate an ACE inhibitor or ARB with ongoing symptoms.
*Adapted from Kotecha and Piccini.[218]

Figure 4 Initial management of newly diagnosed concomitant heart failure and AF. Adapted from Kotecha and Piccini.[218]

SINTOMATOLOGIA

La FA può essere **asintomatica** o **sintomatica:**

- **Cardiopalmo**
- **Dolore toracico**
- **Dispnea**
- **Astenia**
- **Sincope** (soprattutto se associata a disfunzione del nodo del seno o a ostruzione emodinamica come nella stenosi valvolare aortica, nella cardiomiopatia ipertrofica o nella patologia cerebrovascolare)
- **Poliuria**

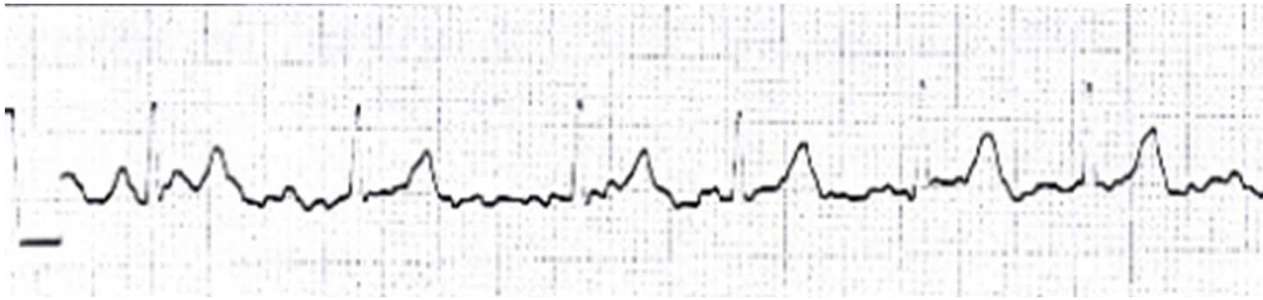
I sintomi variano con la frequenza cardiaca, con lo stato funzionale sottostante, con la durata della FA e con la percezione individuale del paziente

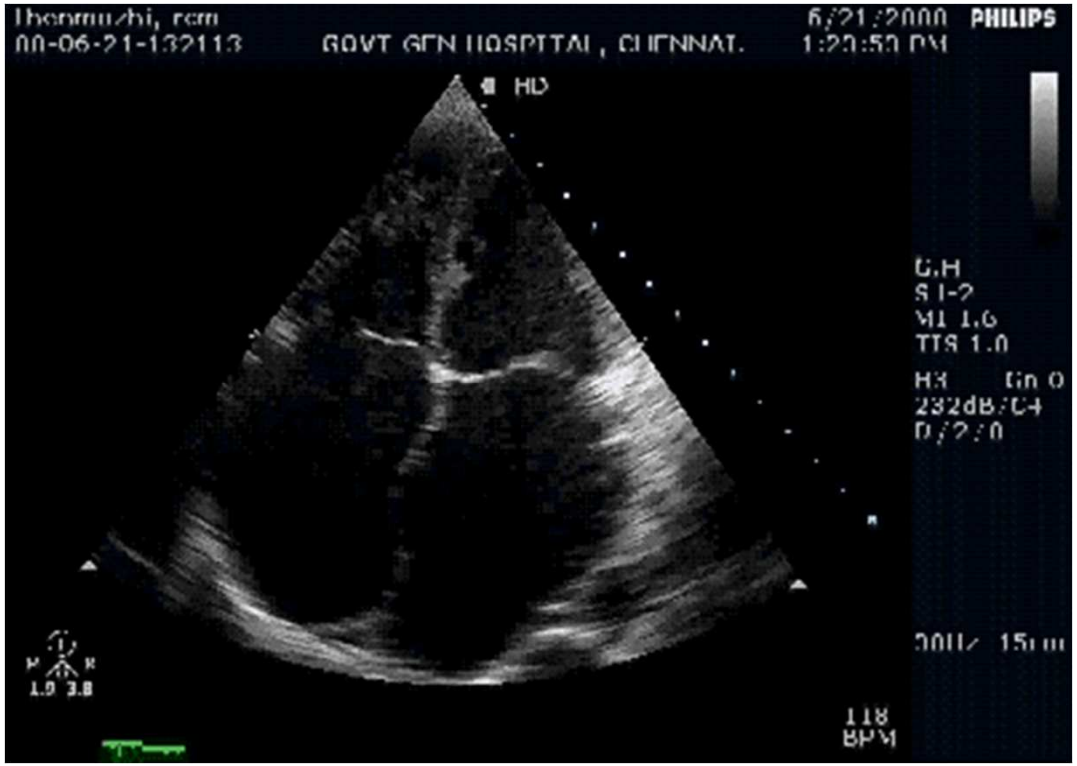
DIAGNOSI

commendations for diagnostic workup of atrial rillation patients

Recommendations	Class[a]	Level[b]	Ref[c]
ECG documentation is required to establish the diagnosis of AF.	I	B	349
A full cardiovascular evaluation, including an accurate history, careful clinical examination, and assessment of concomitant conditions, is recommended in all AF patients.	I	C	
Transthoracic echocardiography is recommended in all AF patients to guide management.	I	C	339
Long-term ECG monitoring should be considered in selected patients to assess the adequacy of rate control in symptomatic patients and to relate symptoms with AF episodes.	IIa	C	

= atrial fibrillation; ECG = electrocardiogram.
ss of recommendation.
vel of evidence.
ference(s) supporting recommendations.





GESTIONE CLINICA

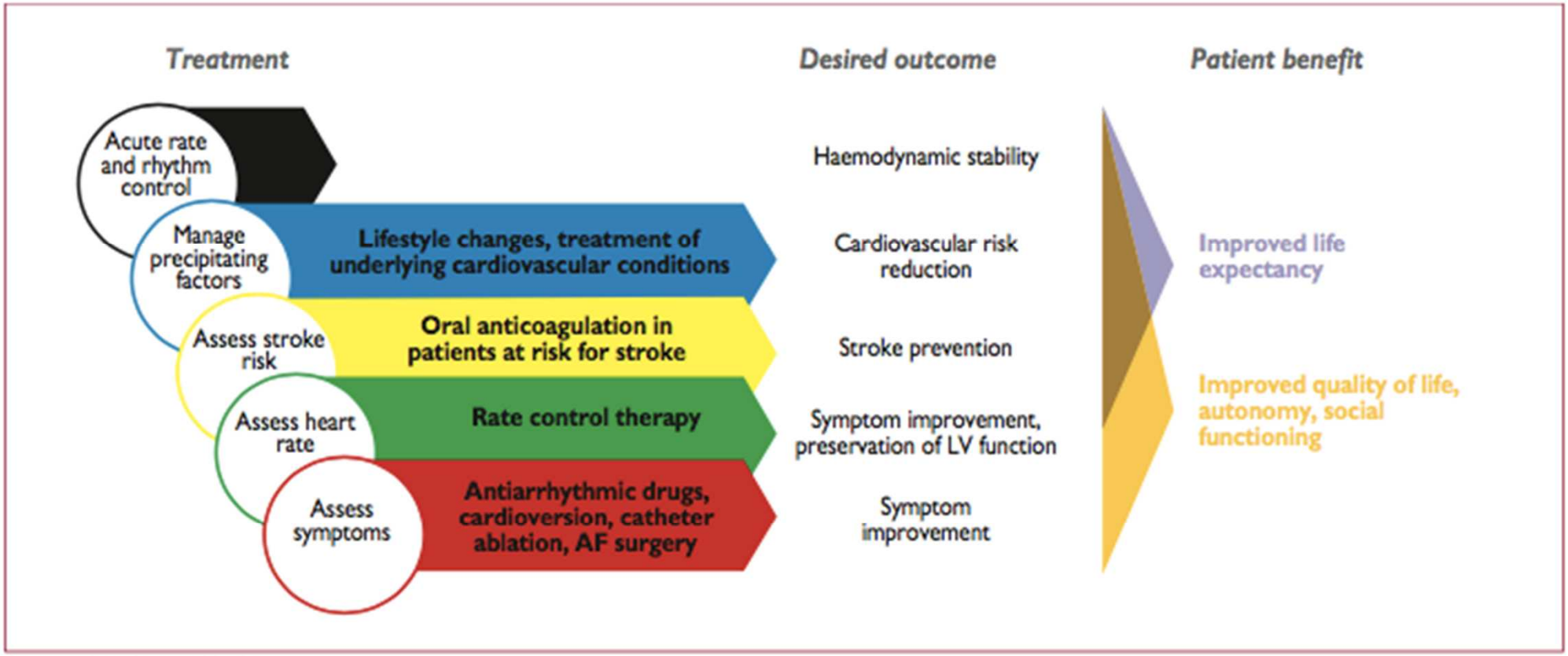


Figure 5 Acute and chronic management of atrial fibrillation patients, desired cardiovascular outcomes, and patient benefits. Adapted from the report on the 4th AFNET/EHRA consensus conference.[76]

VALUTARE IL RISCHIO CARDIOEMBOLICO

CHA_2DS_2-VASc	Score	HAS-BLED	Score
Congestive heart failure/LV dysfunction	1	Hyper sion i.e. uncontrolled	1
Hypertension	1	Abnormal al/liver functi	1 or 2
Aged ≥75 years	2	Stroke	1
Diabetes mellitus	1	Bleeding tenden redisposition	1
Stroke/TIA/TE	2	Labile INR	1
Vascular disease [prior MI, PAD, or aortic plaque]	1	Age (e.g. >65	1
Aged 65-74 years	1	Drugs (e concomitant as or NSAID or alcohol	1
Sex category [i.e. female gender]	1		
Maximum score	**9**		**9**

I pazienti con **CHA_2DS_2-VASc score >2** sono ad alto rischio cardioembolico e vanno quindi trattati con **terapia anticoagulante** (livello di evidenza **I A**)

PREVENZIONE CARDIOEMBOLICA

Recommendations for stroke prevention in patients with atrial fibrillation

Recommendations	Class[a]	Level[b]	Ref[c]
Oral anticoagulation therapy to prevent thromboembolism is recommended for all male AF patients with a CHA_2DS_2-VASc score of 2 or more.	I	A	38, 318–321, 354, 404
Oral anticoagulation therapy to prevent thromboembolism is recommended in all female AF patients with a CHA_2DS_2-VASc score of 3 or more.	I	A	38, 318–321, 354, 404
Oral anticoagulation therapy to prevent thromboembolism should be considered in male AF patients with a CHA_2DS_2-VASc score of 1, considering individual characteristics and patient preferences.	IIa	B	371, 375–377
Oral anticoagulation therapy to prevent thromboembolism should be considered in female AF patients with a CHA_2DS_2-VASc score of 2, considering individual characteristics and patient preferences.	IIa	B	371, 376, 377
Vitamin K antagonist therapy (INR 2.0–3.0 or higher) is recommended for stroke prevention in AF patients with moderate-to-severe mitral stenosis or mechanical heart valves.	I	B	274, 435–440
When oral anticoagulation is initiated in a patient with AF who is eligible for a NOAC (apixaban, dabigatran, edoxaban, or rivaroxaban), a NOAC is recommended in preference to a Vitamin K antagonist.	I	A	39, 318–321, 404
When patients are treated with a vitamin K antagonist, time in therapeutic range (TTR) should be kept as high as possible and closely monitored.	I	A	395, 432, 441-444
AF patients already on treatment with a vitamin K antagonist may be considered for NOAC treatment if TTR is not well controlled despite good adherence, or if patient preference without contra-indications to NOAC (e.g. prosthetic valve).	IIb	A	39, 318, 319, 404, 408
Combinations of oral anticoagulants and platelet inhibitors increase bleeding risk and should be avoided in AF patients without another indication for platelet inhibition.	III (harm)	B	429, 445
In male or female AF patients without additional stroke risk factors, anticoagulant or antiplatelet therapy is not recommended for stroke prevention.	III (harm)	B	368, 371, 376, 377
Antiplatelet monotherapy is not recommended for stroke prevention in AF patients, regardless of stroke risk.	III (harm)	A	38, 429, 430
NOACs (apixaban, dabigatran, edoxaban, and rivaroxaban) are not recommended in patients with mechanical heart valves (Level of evidence B) or moderate-to-severe mitral stenosis (Level of evidence C).	III (harm)	B C	318–321, 400, 404

STRATEGIE TERAPEUTICHE

Mantenere in pazienti in ritmo sinusale **(Rhythm control)**

Vantaggi
- Migliore funzione cardiaca
- Migliore qualità della vita
- Migliore prevenzione degli eventi tromboembolici

Lasciare i pazienti in fibrillazione atriale ma controllare la frequenza **(Rate control)**

Vantaggi
- Assenza di effetti secondari da terapia farmacologica
- Minore necessità di cardioversioni elettriche ripetute

- **Tachicardia da rientro nel nodo A-V (TR-NAV)**

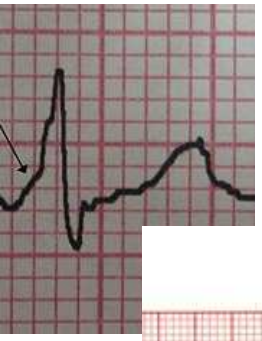
– "Slow-fast"
– "Fast-slow"
– "Slow-slow"

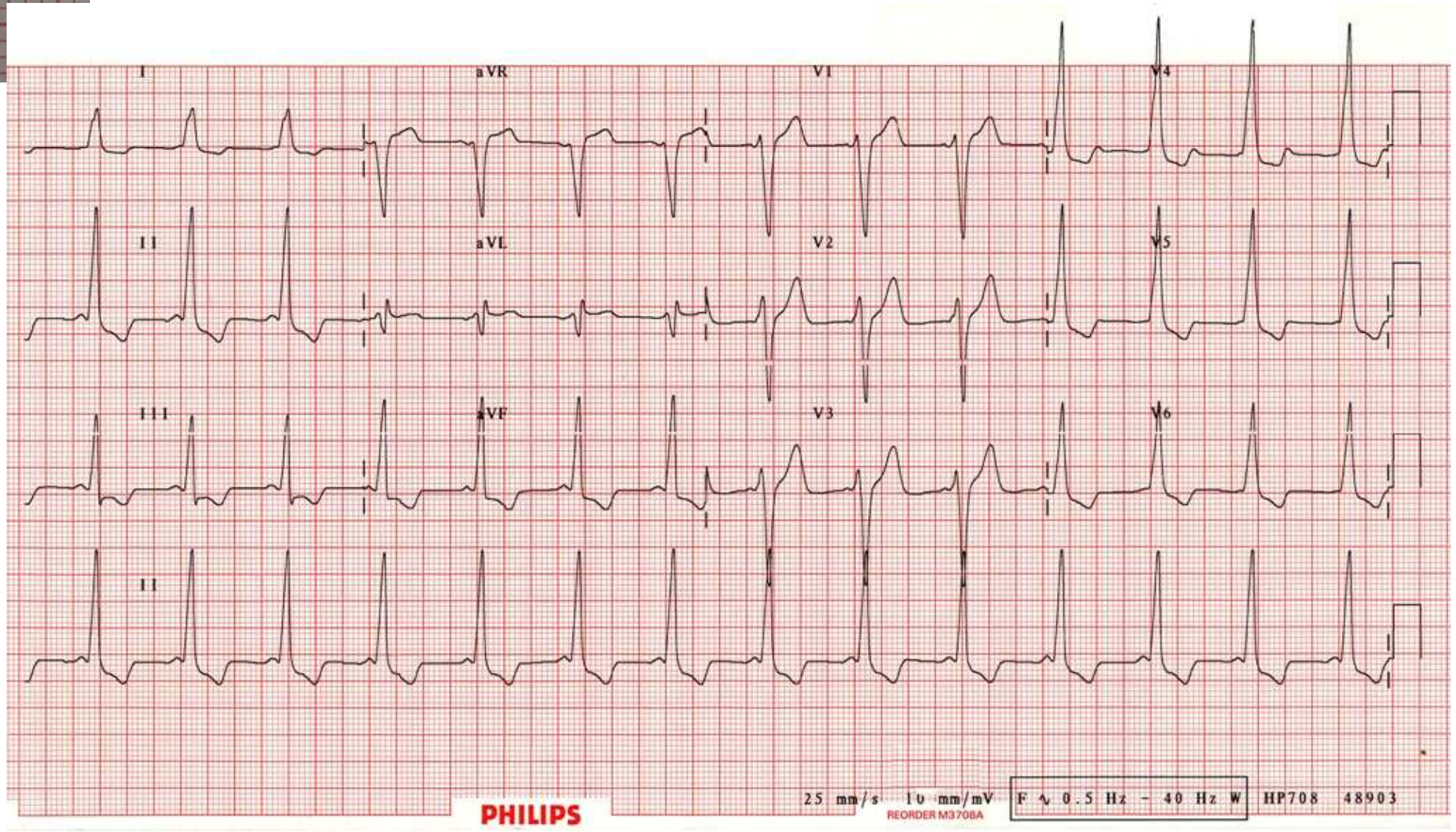
- **Tachicardia da rientro A-V (TRAV)**

– Da via accessoria manifesta (WPW) ortodromica
– Da via accessoria manifesta (WPW) antidromica
– Da via accessoria occulta
– Da via accessoria a conduzione lenta (tipo Coumel)
– Da fibre atrio-fascicolari o nodo-fascicolari (tipo Mahaim)

- **Tachicardia automatica giunzionale (TAG)**

WPW

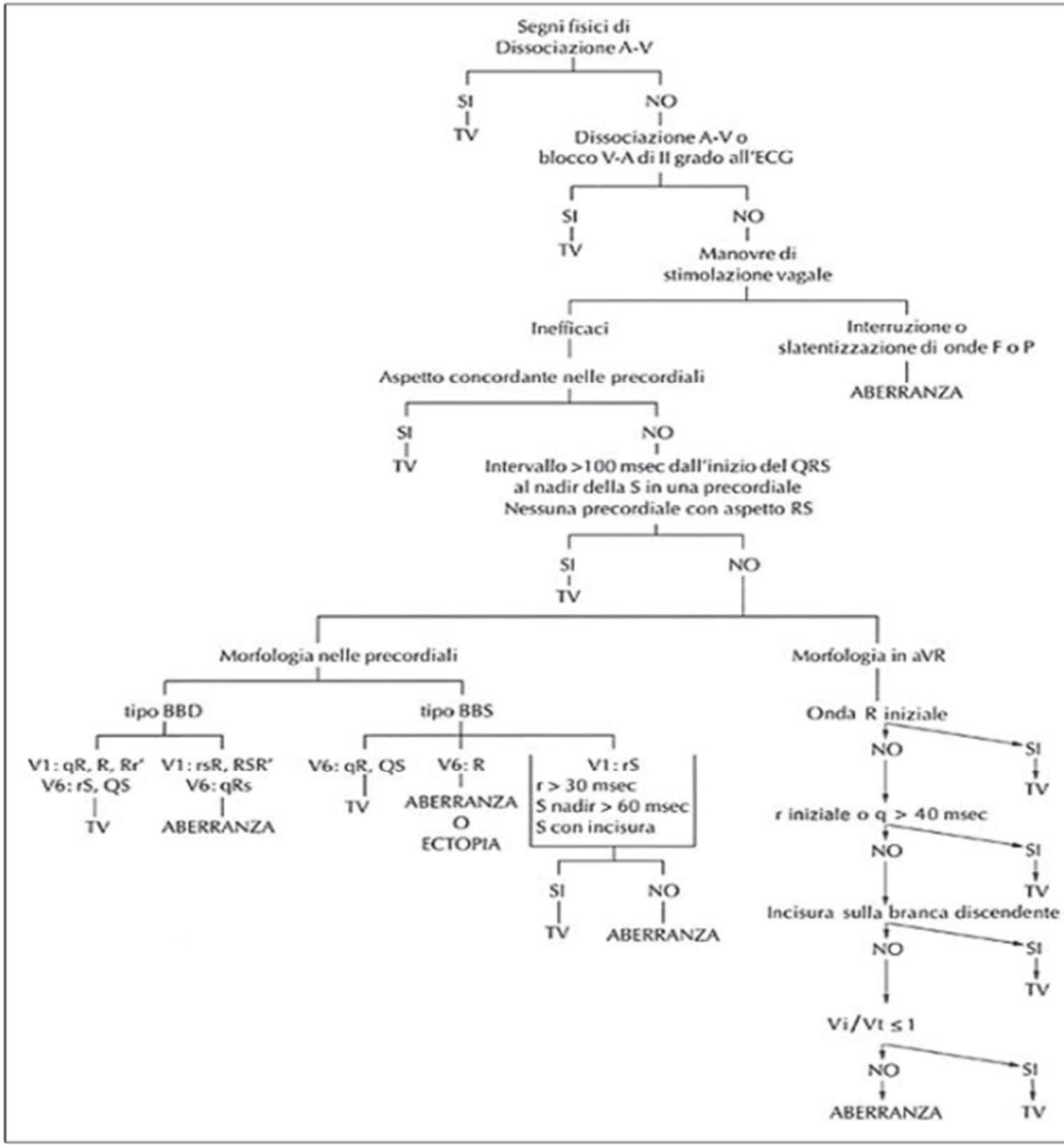



I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
25 mm/s 10 mm/mV
F ∿ 0.5 Hz - 40 Hz W
HP708 48903
PHILIPS
REORDER M3708A

ARITMIE IPERCINETICHE VENTRICOLARI

- Ritmo idioventricolare accelerato (RIVA)
- Tachicardia ventricolare
- Torsioni di punta
- Fibrillazione ventricolare

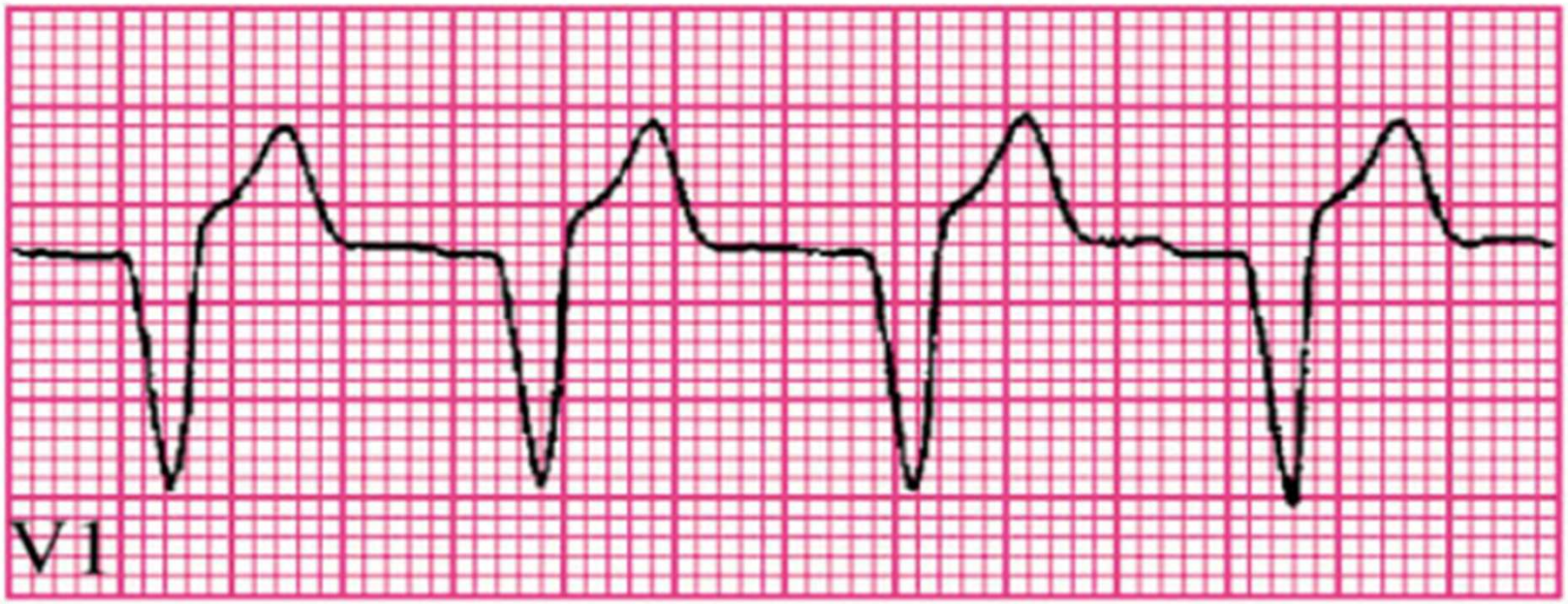
ALGORITMO PER LA VALUTAZIONE DELLE TACHICARDIE A QRS LARGO



t(et al - Tachicardia a QRS larghi – G It Cardiol 2010

RITMO IDIOVENTRICOLARE ACCELERATO

E' un ritmo ventricolare a QRS largo a frequenza 60- 100/m' in cui le onde P possono essere assenti o presenti (per retroconduzione dai ventricoli oppure dissociate dai complessi QRS). Si può osservare durante ischemia coronarica, riperfusione o tossicità digitalica.



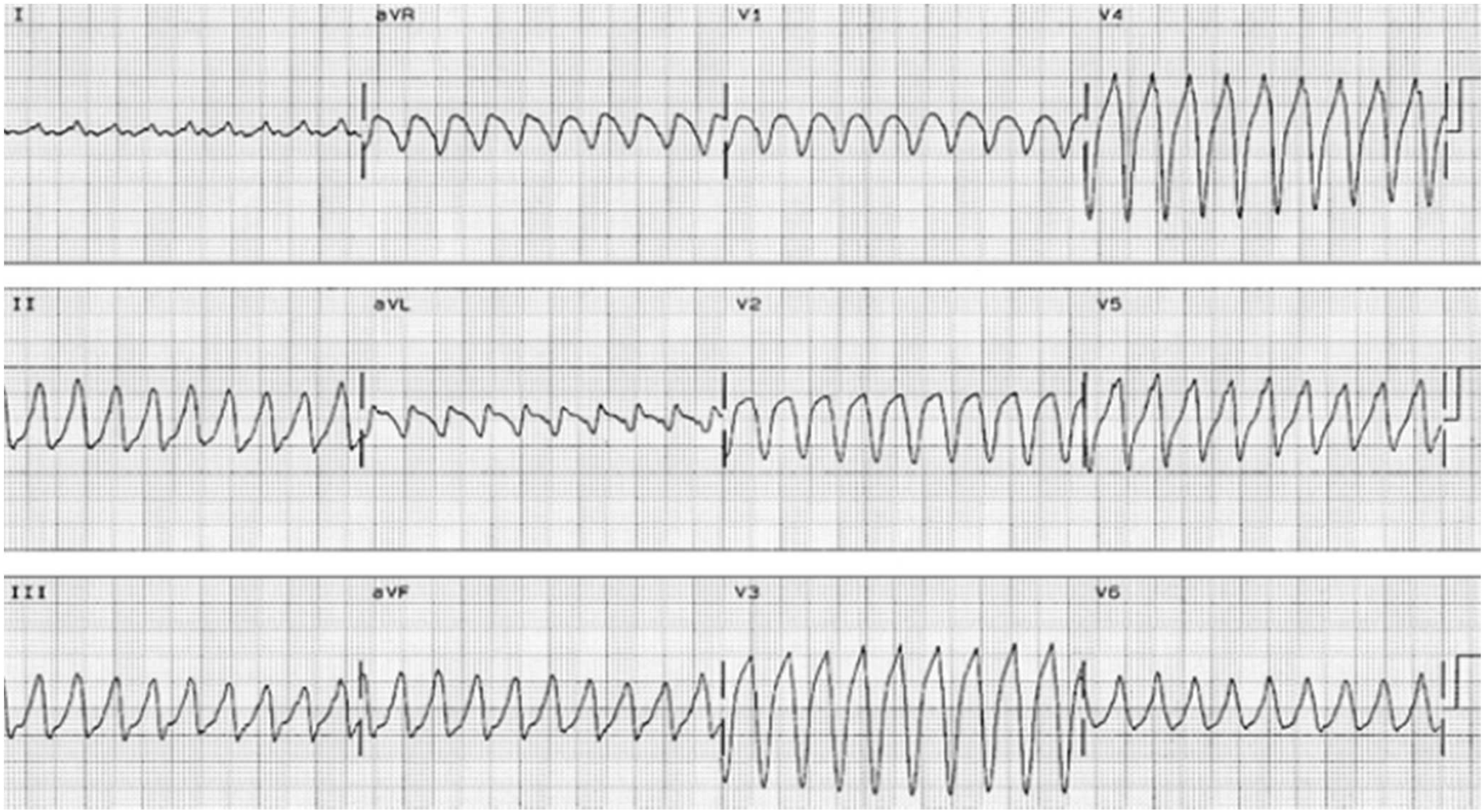
TACHICARDIA VENTRICOLARE

La **tachicardia ventricolare** (**TV**) è una successione di ***almeno 3 battiti ectopici ventricolari consecutivi a FC 100 - 250/min***; può essere:

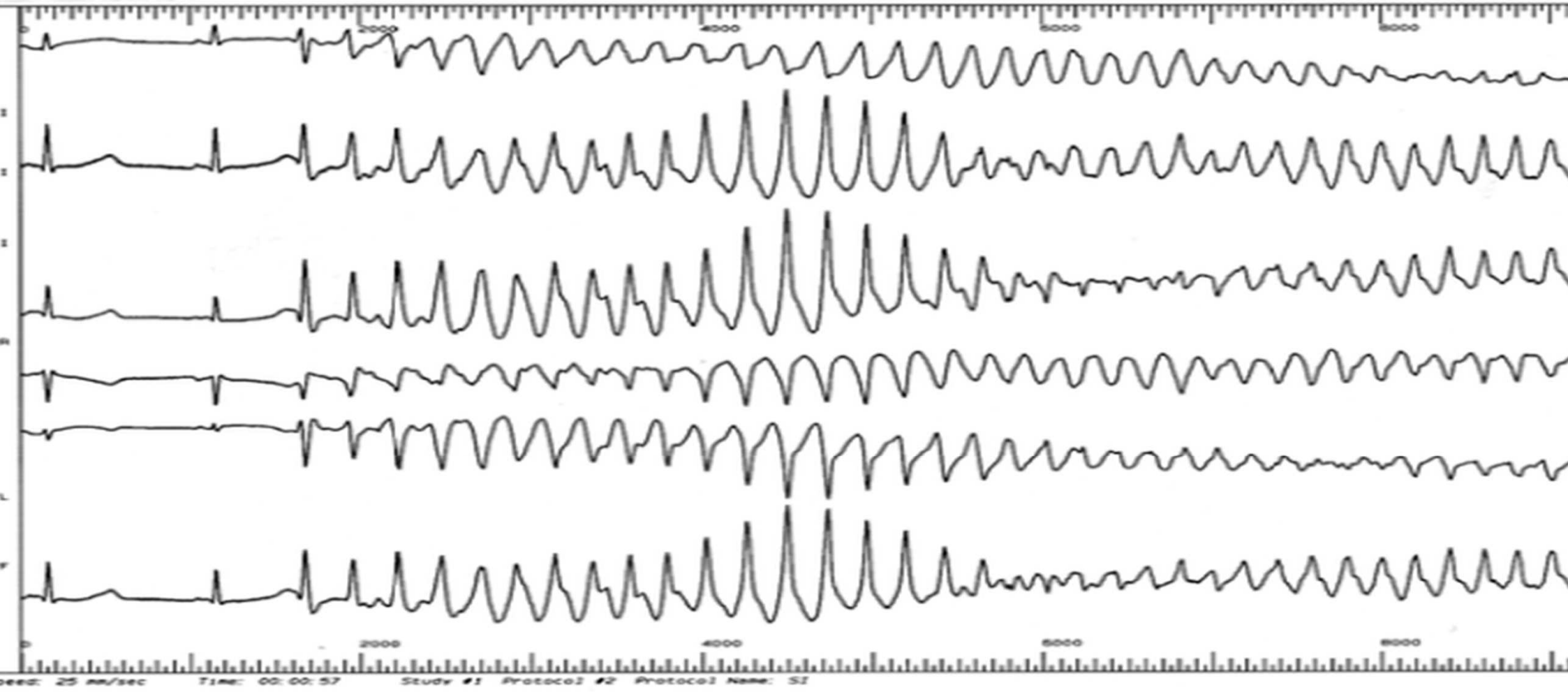
- **sostenuta** (durata > 30 sec e/o necessità di intervento terapeutico per compromissione emodinamica);
- **non sostenuta** (durata < 30 sec)
- **monomorfa** se la morfologia del QRS è costante.
- **polimorfa** se la morfologia del QRS è variabile;
- **con polso**;
- **senza polso**.

Può originare da meccanismi di ***rientro***, ma anche da un ***esaltato automatismo*** delle cellule del Purkinje o da "***triggered activity***" (post-potenziali precoci o tardivi).

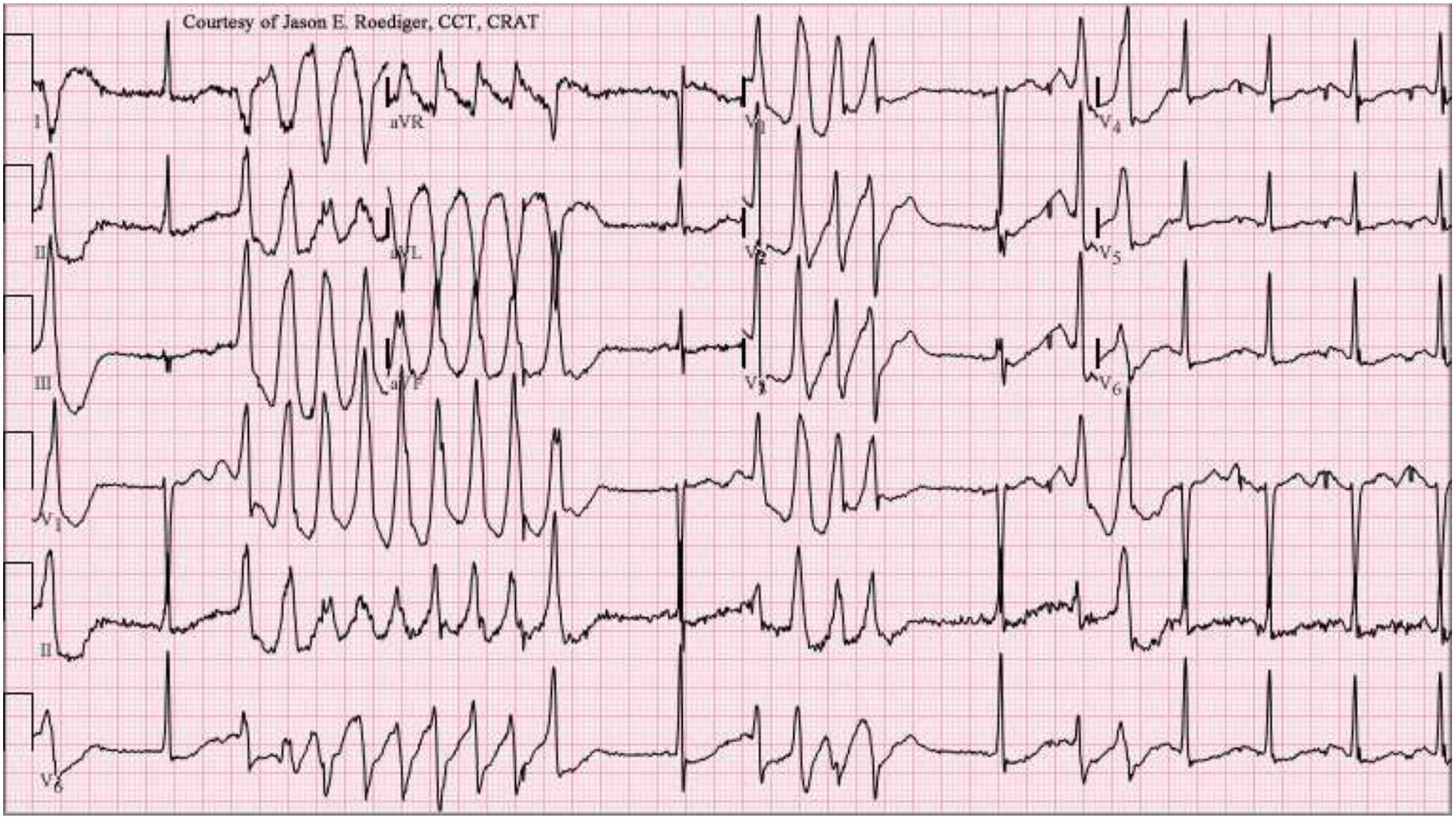
TV MONOMORFA



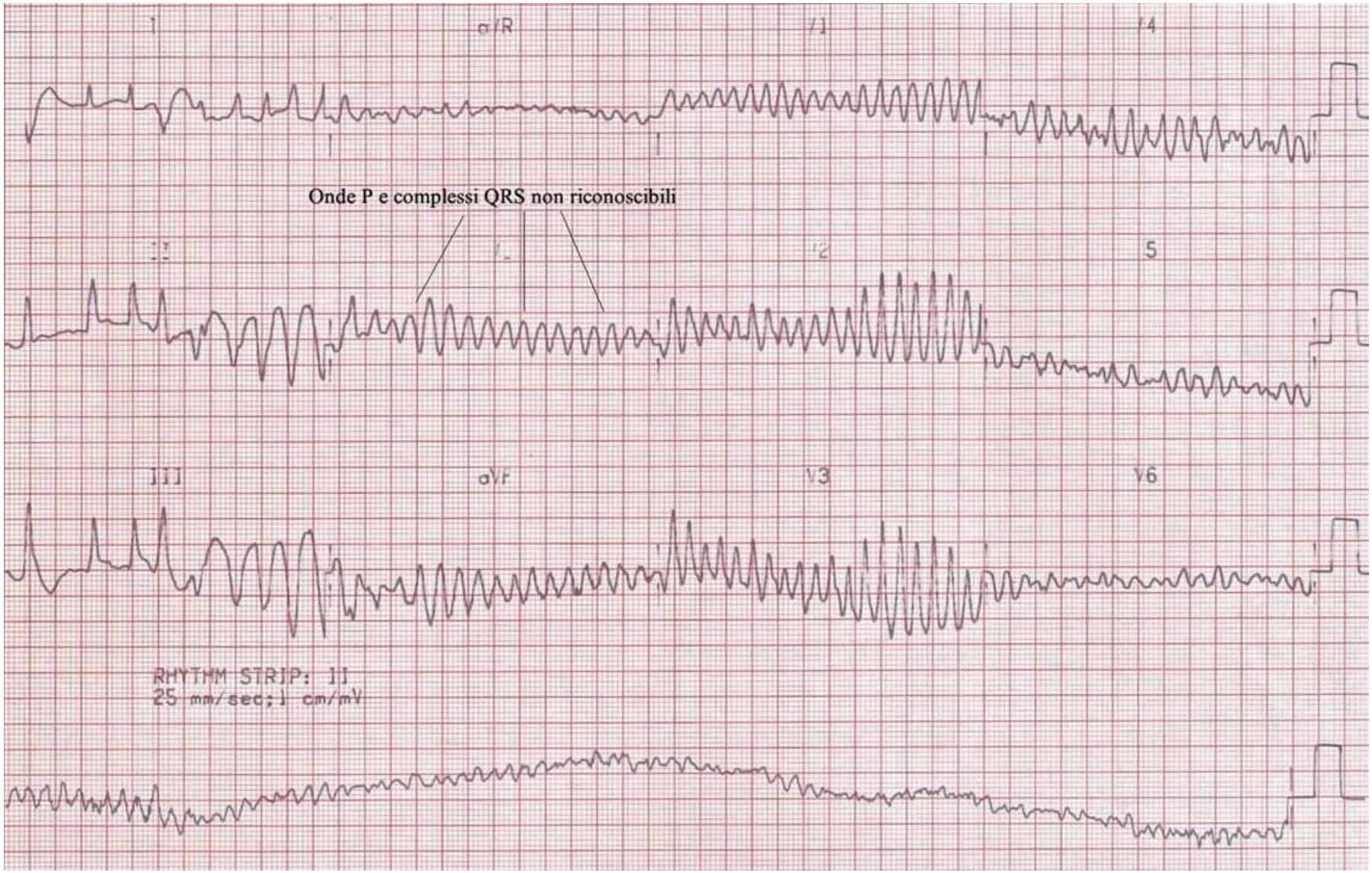
TV POLIMORFA



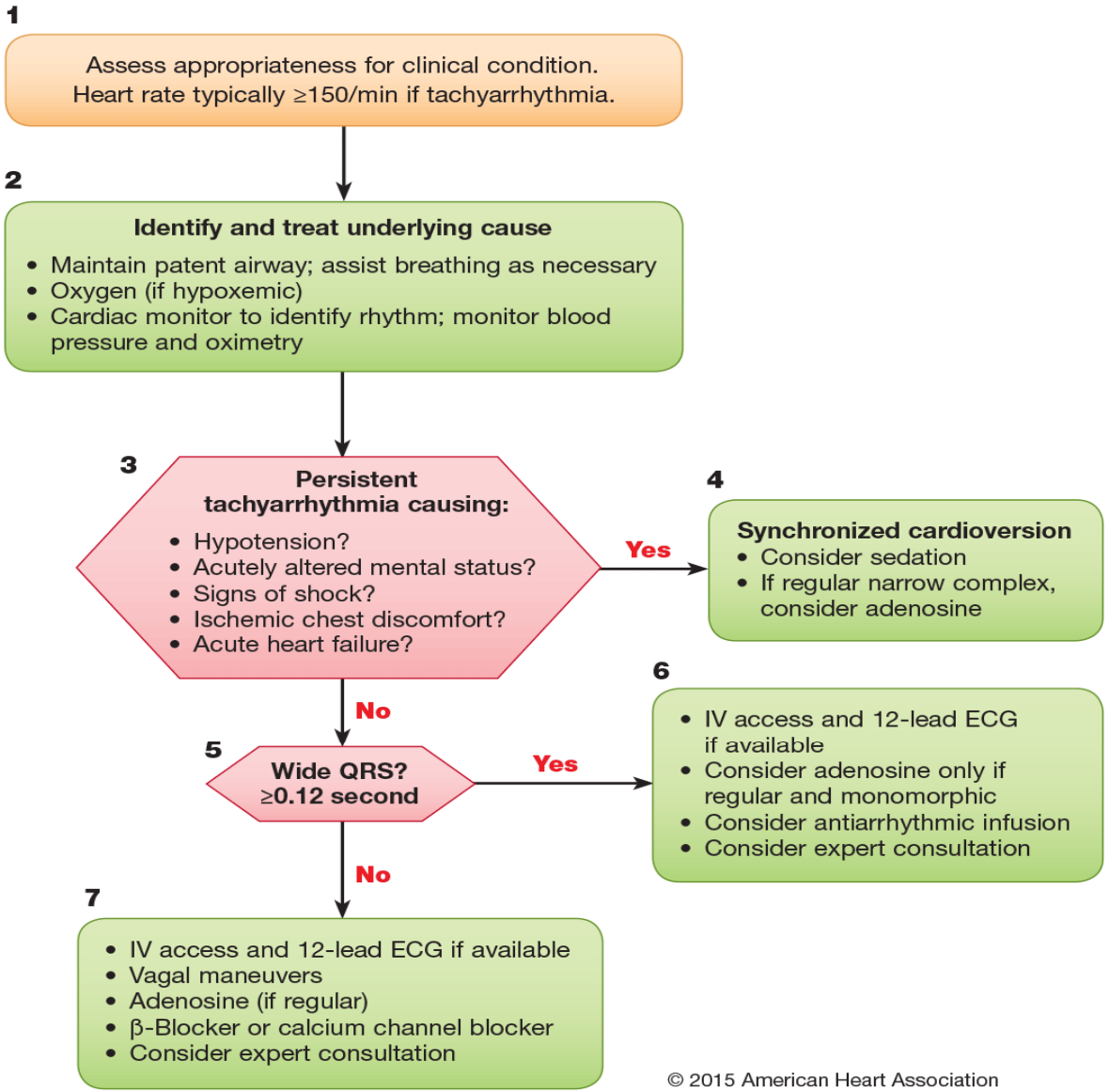
TORISIONE DI PUNTA



FIBRILLAZIONE VENTRICOLARE



Adult Tachycardia With a Pulse Algorithm



Doses/Details

Synchronized cardioversion:
Initial recommended doses:
- Narrow regular: 50-100 J
- Narrow irregular: 120-200 J biphasic or 200 J monophasic
- Wide regular: 100 J
- Wide irregular: defibrillation dose (*not* synchronized)

Adenosine IV dose:
First dose: 6 mg rapid IV push; follow with NS flush.
Second dose: 12 mg if required.

Antiarrhythmic Infusions for Stable Wide-QRS Tachycardia

Procainamide IV dose:
20-50 mg/min until arrhythmia suppressed, hypotension ensues, QRS duration increases >50%, or maximum dose 17 mg/kg given.
Maintenance infusion: 1-4 mg/min.
Avoid if prolonged QT or CHF.

Amiodarone IV dose:
First dose: 150 mg over 10 minutes.
Repeat as needed if VT recurs.
Follow by maintenance infusion of 1 mg/min for first 6 hours.

Sotalol IV dose:
100 mg (1.5 mg/kg) over 5 minutes.
Avoid if prolonged QT.

GRAZIE PER L'ATTENZIONE!!