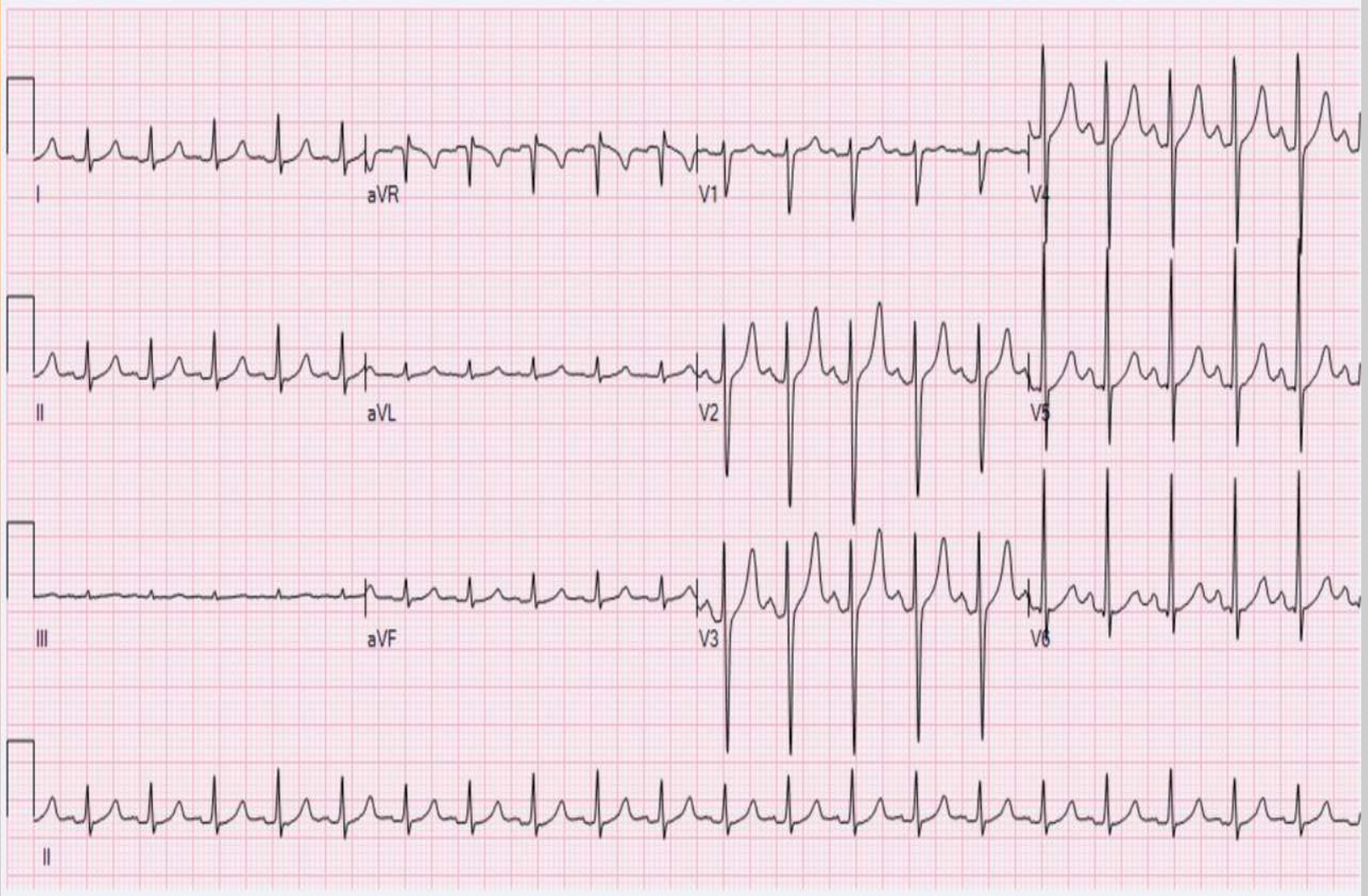
EKG Analysis

Arrhythmias

Dr E.FARAHANI cardiologist

Sinus tachycardia

- HR=100-180
- باورزش شدید ودرافرادجوان میتواندبیشترباشد
- حداکثر ضربان قلب باافزایش سن کاهش می یابد
- شروع وختم آریتمی تدریجی
- بخصوص درHR پایینPP تغییرجزیی درفاصله
- باماساژ کاروتید ووالسالواوواگ ریت آهسته ومجددا باقطع آن به ریت قبلی برمی گردد.هرچه سرعت بالاترپاسخ کمتر
- شکل وعرض موجP نرمال اماارتفاع آن بالا می رود
- تسریع فاز۴ دیاستولیک دپلاریزاسیون


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

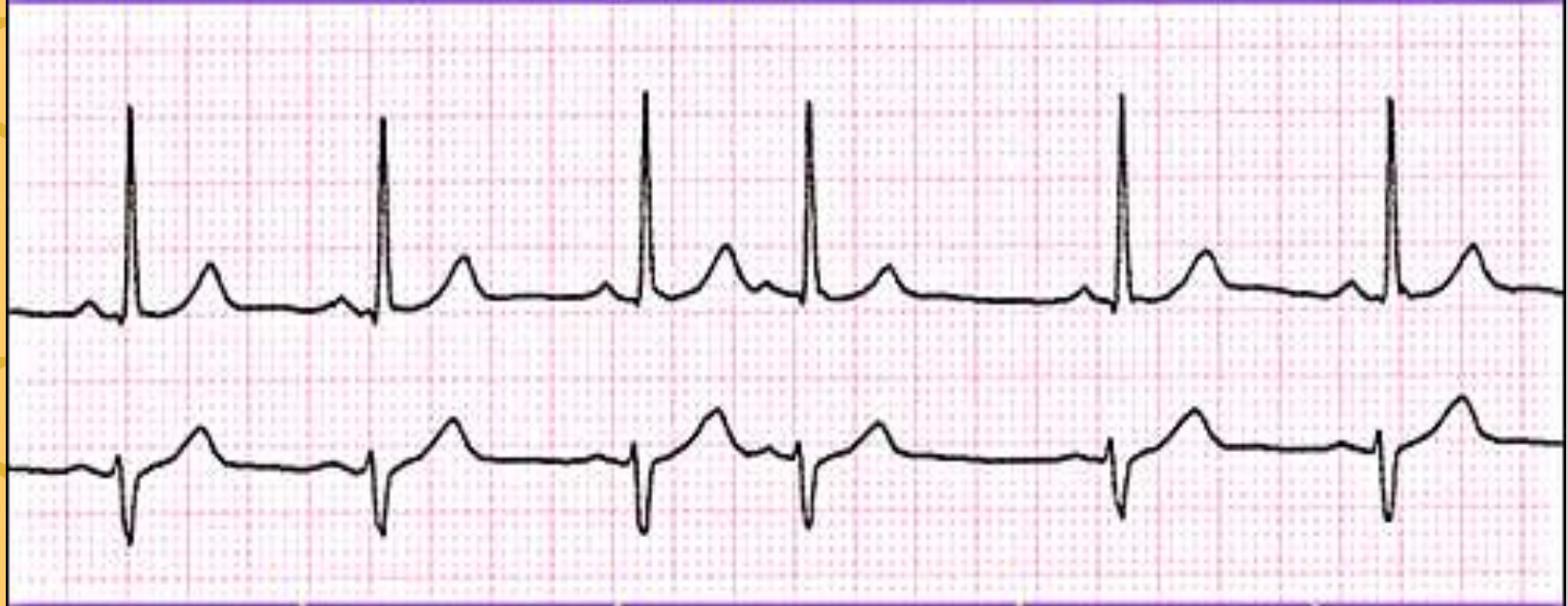
Inappropriate sinus tachycardia

- Persistent sinus tachycardia at **rest or with minimal exertion**
- Usually occurs in otherwise **healthy** people
- More common in **health care** personnel
- May result from a defect in either sympathetic or vagal control of sinus node automaticity or an abnormality of intrinsic heart rate
- No alchohol,tubaco,cafein,sympatomimetics
- CCB,BB,Ivabradine(IF blocker)
- S.N Abl

PAC

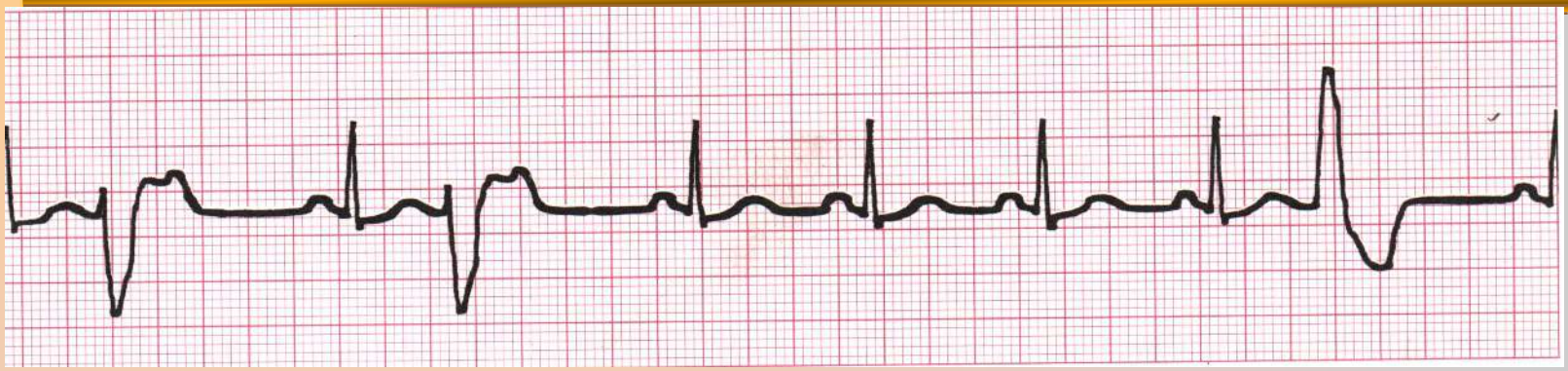
- P – در PAC داراي شكلي متفاوت از P سينوسي است (شكافدار- بي فاز يك يا منفي)
- QRS – معمولا" در PAC طبيعي است گاهي ممكنست وجود نداشته باشد و گاهي غير طبيعي و پهن است .
- P-R inteval= در PAC با فاصله P-R سينوسي تفاوت دارد گاهي كوتاه ، گاهي بلند و گاهي پهن است.
- منشاء:هرجا عمدتا بطن،افزايش با سن

Premature Atrial Contraction • Isolated PAC's: Occur Single



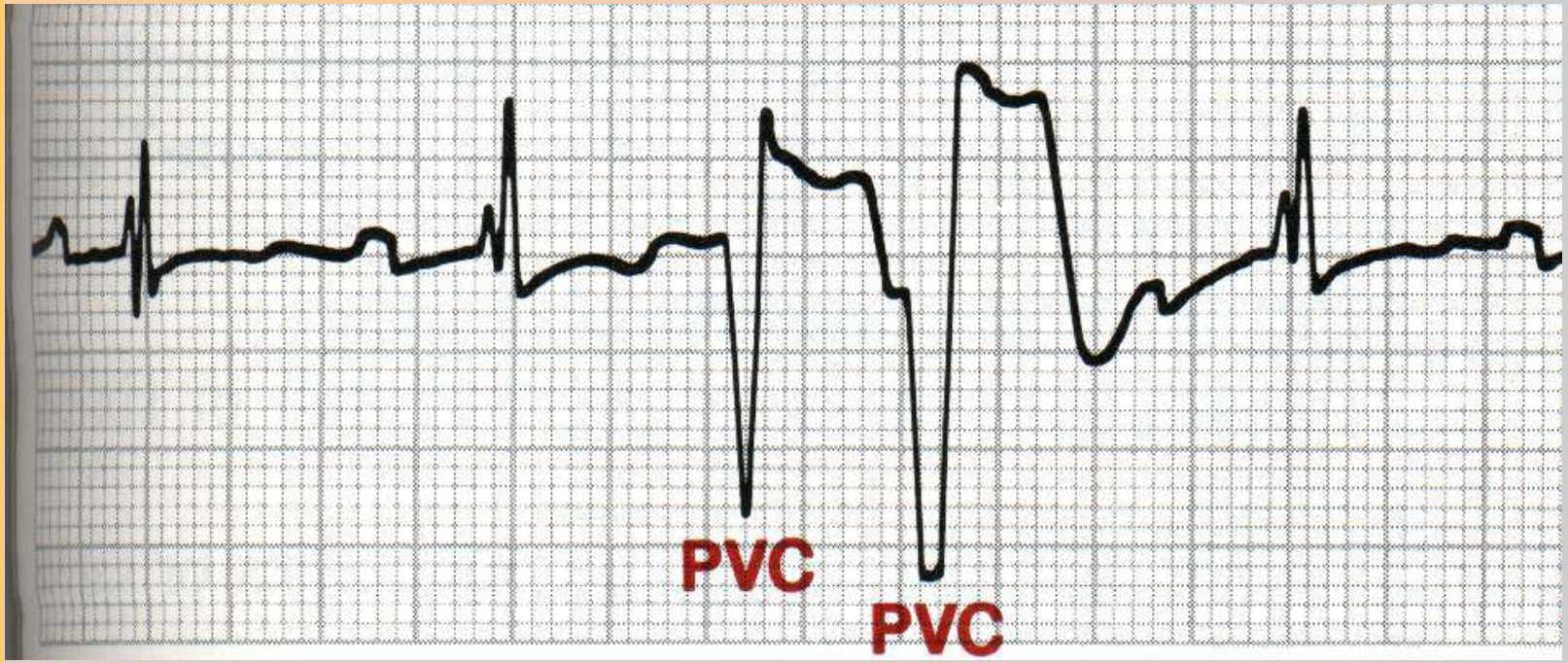
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
N/A	Irregular	Premature & abnormal or hidden	<.20	<.12

Premature Ventricular Contractions (PVCs)

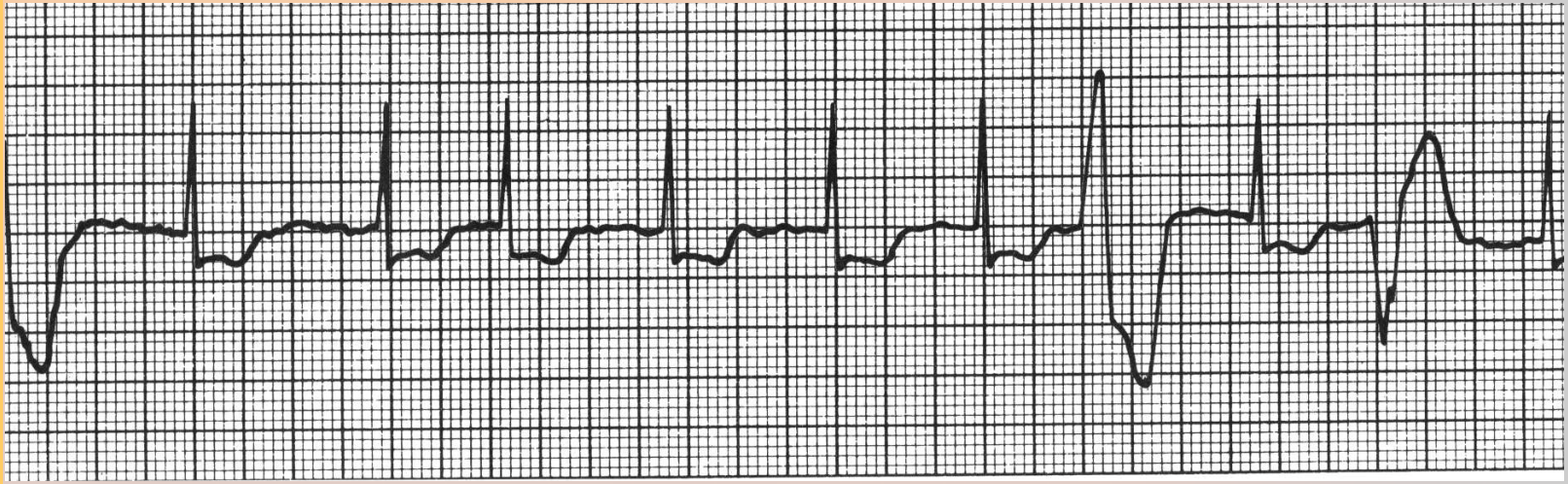


- Irritable focus causes ventricles to depolarize before the SA node fires
- Premature beat that has a wide QRS
  - QRS and T wave of a PVC usually point in opposite direction
- "Bad PVCs" – more than 6/minute, coupled, multifocal, and on or near the T wave of the previous sinus beat,>24% holter
- Suppressed by lidocaine,procainamide,inderal,Mg,1,2,3-1c exept post MI

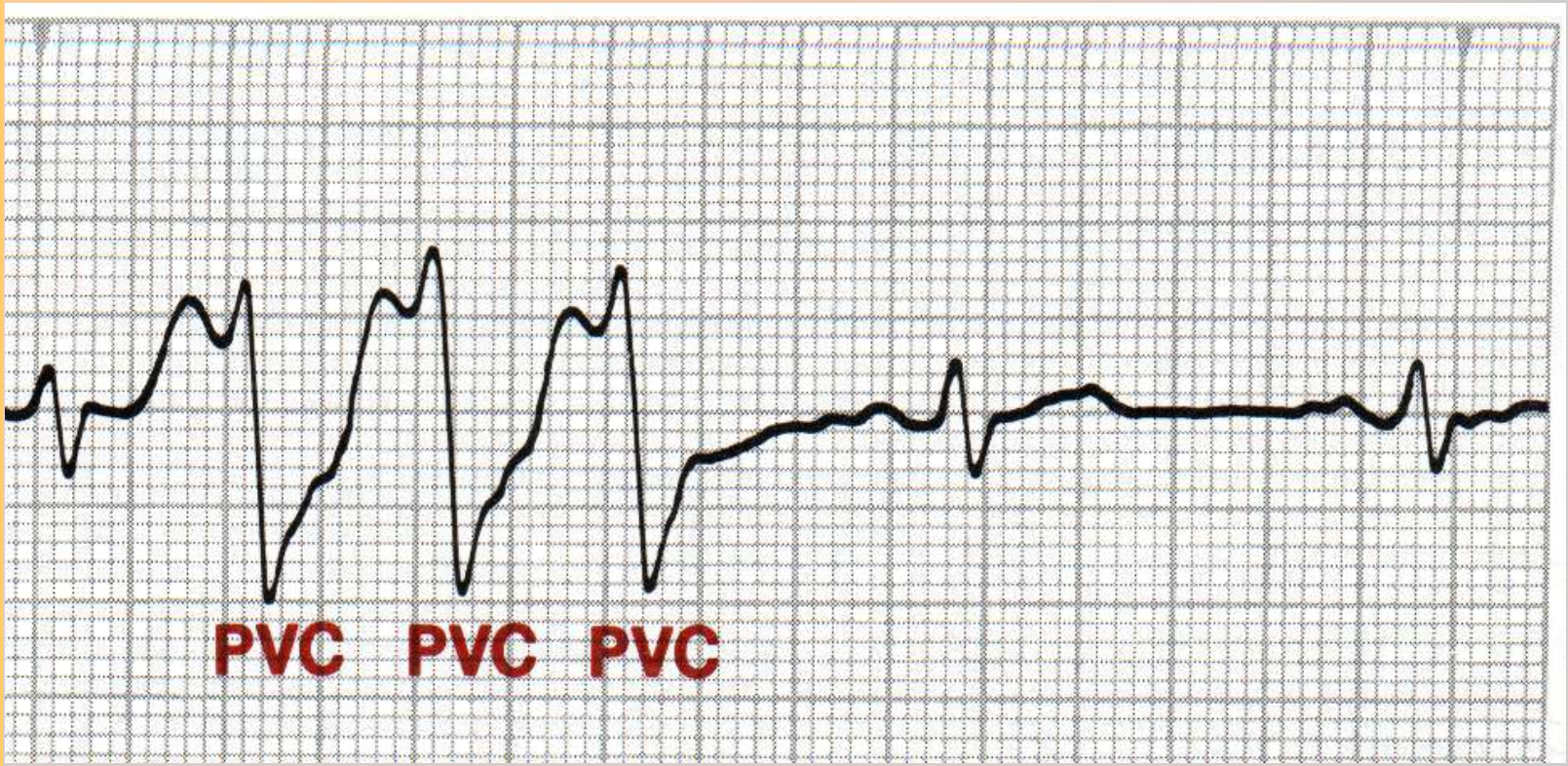
Coupled PVCs



Multifocal PVCs



R-on-T Phenomenon: May cause a run of PVCs or Vfib



SVT

- شروع با P زودرس
- Very short RP
- QRS like sinus rhythm
- AV association
- Stop or dec rate with vagal stimulus

AVNRT

- **HR:150-250**
- **شروع وختم ناگهانی**
- **Pseudo s inf- pseudo r v1**
- **اگرموج p دیده شود بسمت بالا وباریک**
- **شروعpacبا PR طولانی**
- **Short RP<50%RR**
- **Ablation95%**
- **10-50J Cardioversion**
- **Atrial or ventricular pacing**

- 1A,1C,III,DIG,ABL
- Adenosin; Inc K outward:dec atrial contractility,SAN,AVN

PSVT

余啟祥 (余啟祥 SHE, CHI HSIANG) ID:5909512 2014/03/05 17:22:02 Chang Gung Memorial Hospital KH

2013/10/07 (4月)
男性

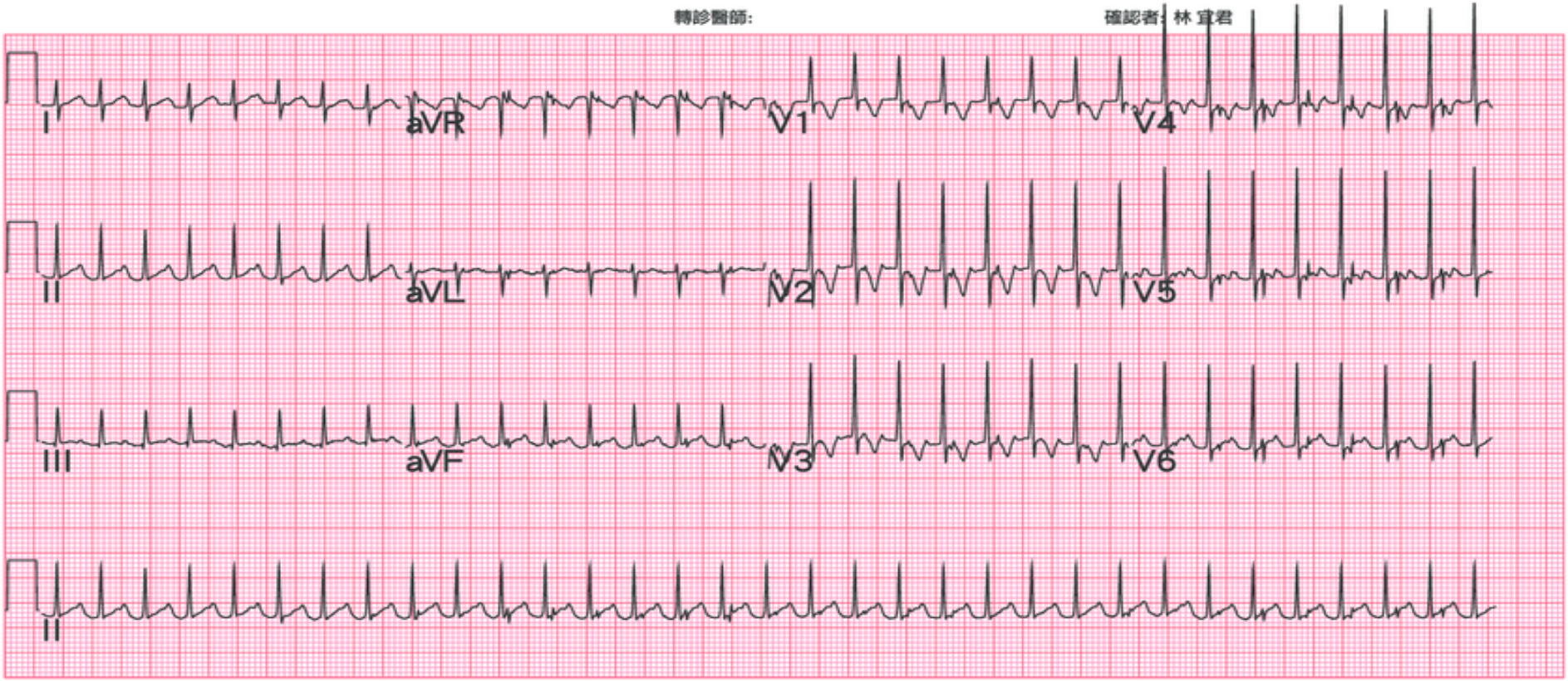
房號：
位置:10

Vent. rate	197	BPM
PR interval	88	ms
QRS 持續時間	218	ms
QT/QTc	214/387	ms
P-R-T 軸	* 81	0

Pediatric ECG analysis
Supraventricular tachycardia

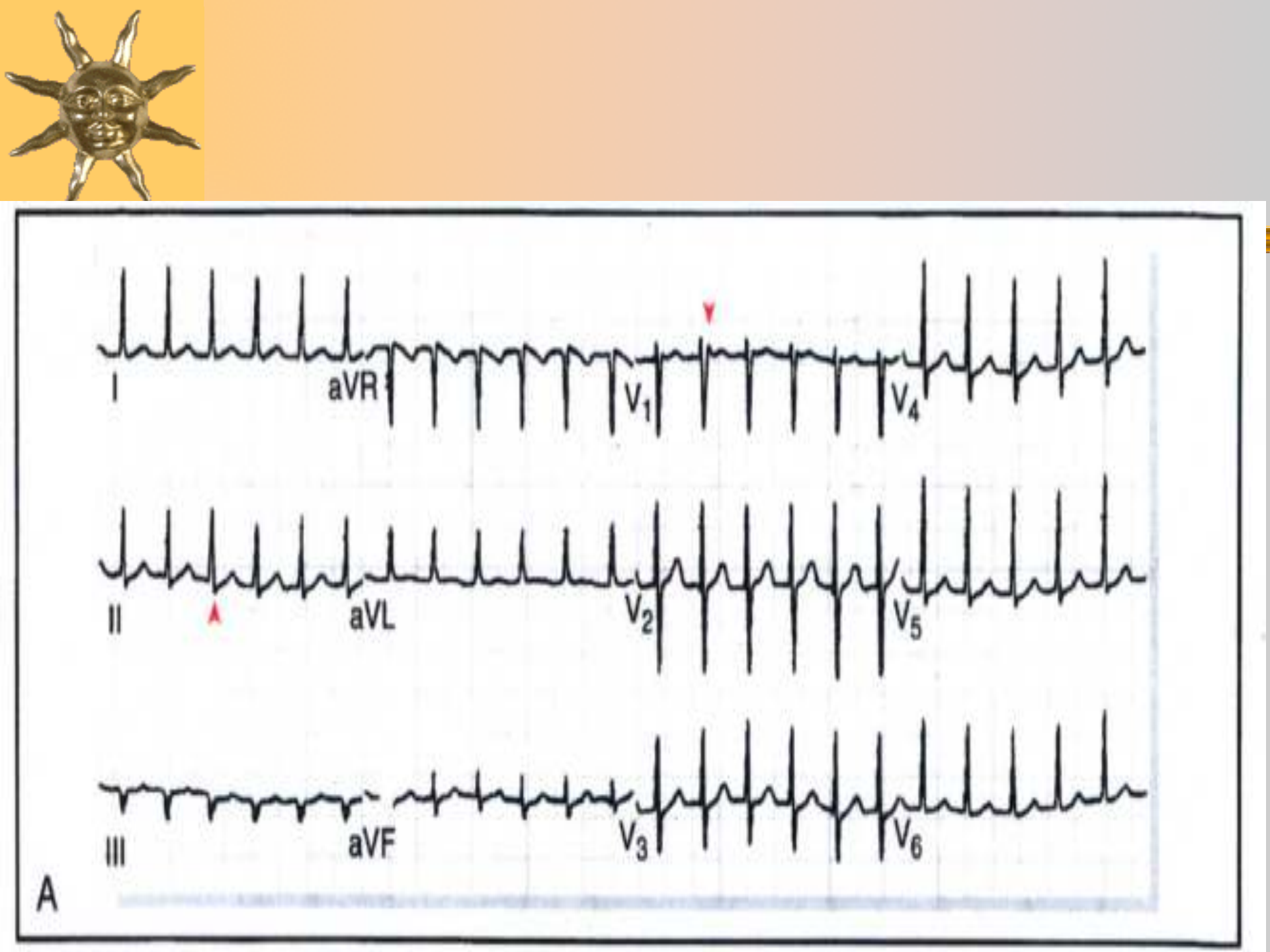
技術員：
檢查原因:

轉診醫師: 確認者: 林 宜君



25mm/s 10mm/mV 40Hz 8.0 SP2 12SL 237 CID: 1

EID:33 EDT: 17:38 2014/03/05 預約:


I
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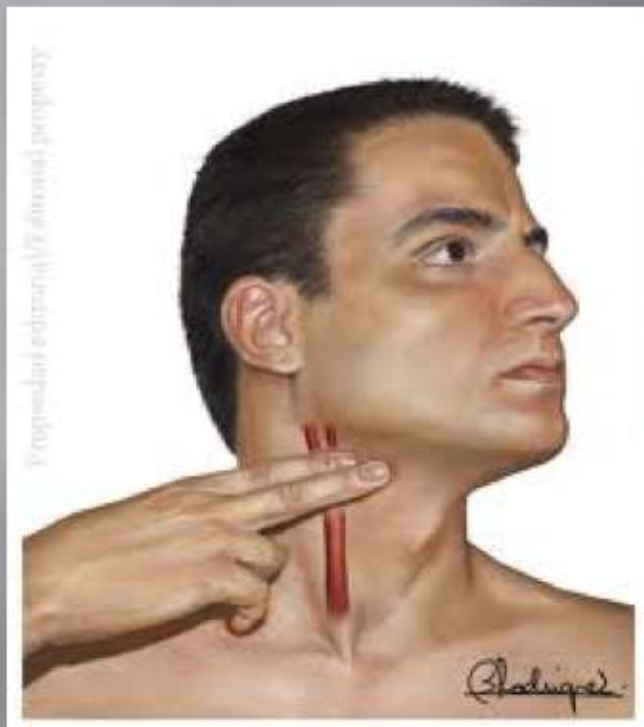
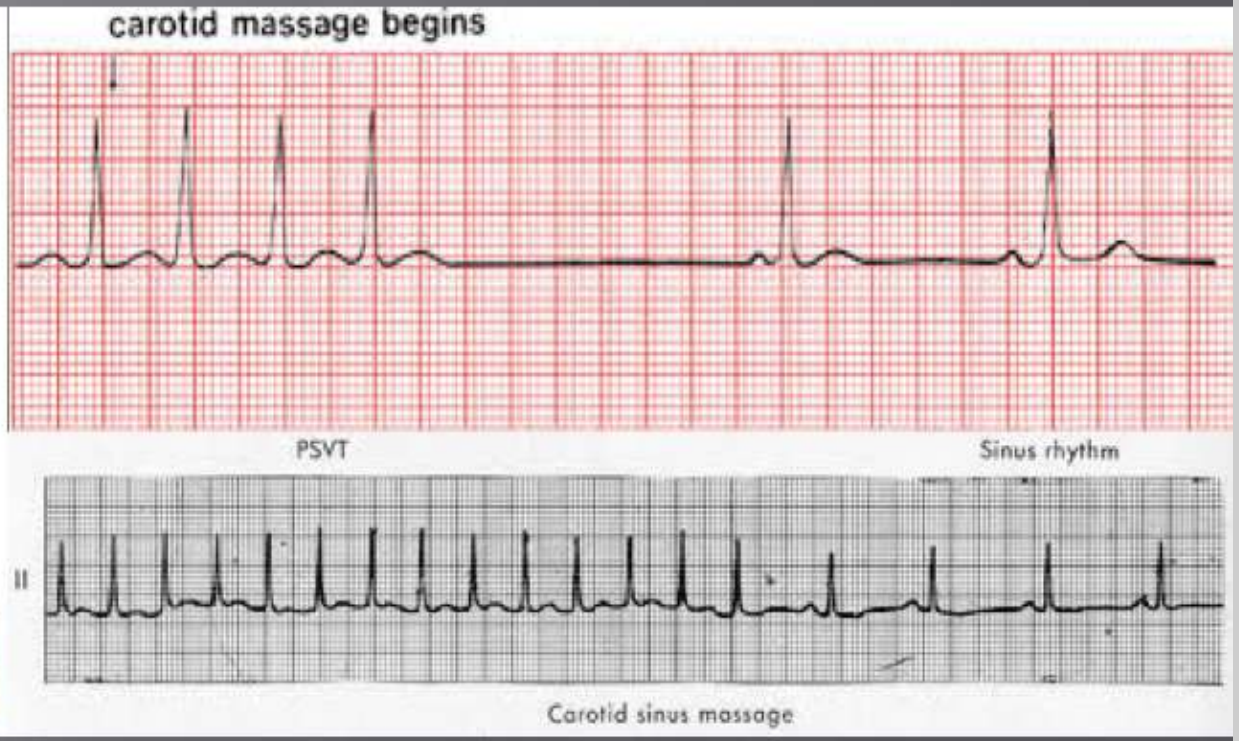
III
aVF
V3
V6
A

تحريك رفلكس واگ با ماساژ يا فشار روي سينوس كاروتيد يا باعث كندكردن موقت يا قطع ناگهاني تاكيكاردي حمله اي ميشود

Carotid Massage





: (Atrial Flutter)

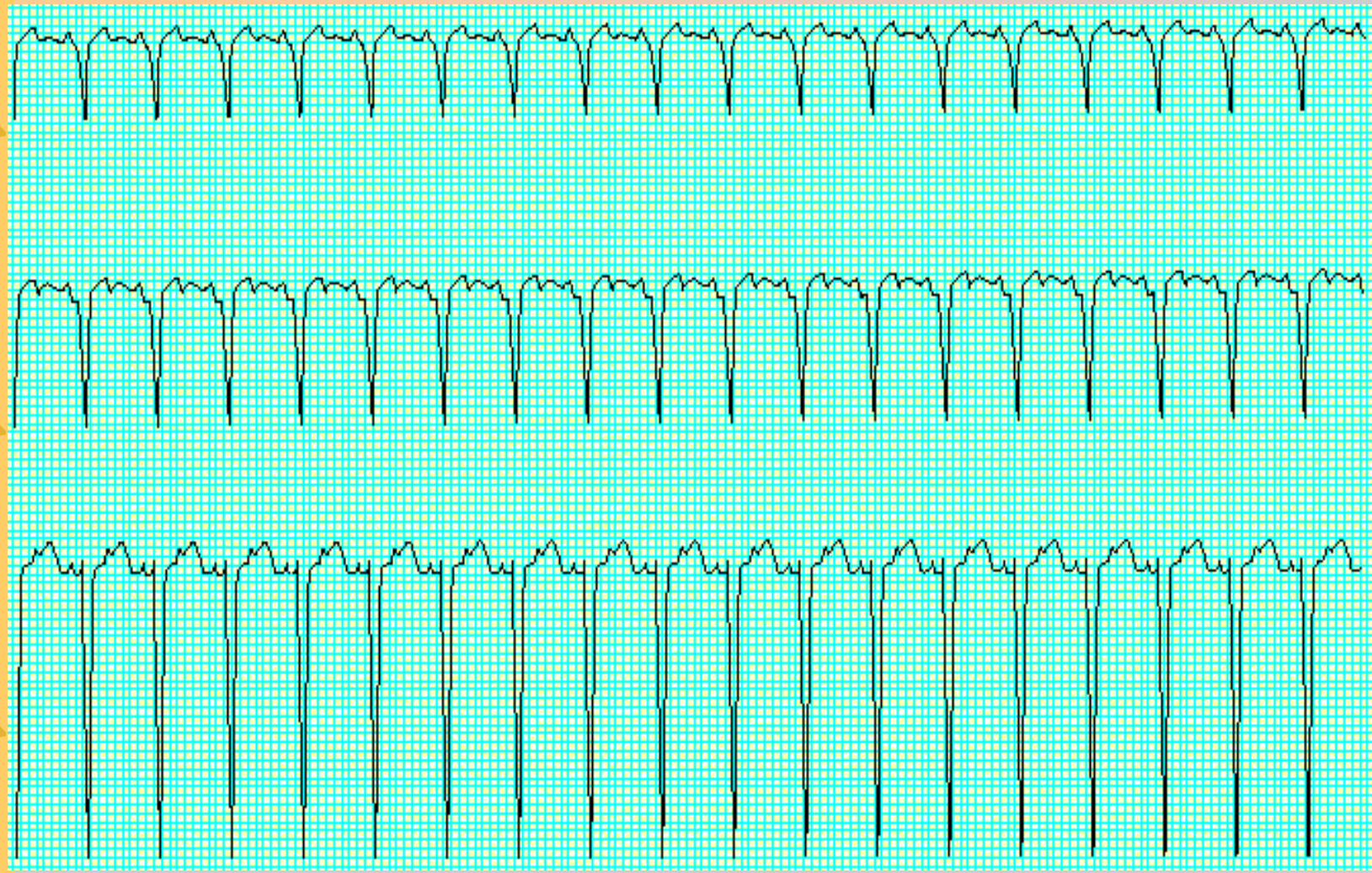
- **تعریف :** فلوتر دهلیزی به ضربانات سریع و منظم دهلیزها همراه با بلوک منظم برخی از ایمپالسها در گره دهلیزی بطنی اطلاق میشود که معمولا" ناشی از حرکت چرخشی منظم امواج در دهلیزها ناشی از بیماریهای قلبی است . فلوتر دهلیزی معمولا" بدنبال افزایش زمان هدایت در دهلیزها بر اثر بیماریهای قلبی, ریتم دهلیزی بسیار منظم است .
- ریتم بطنی ممکنست منظم یا بدلیل وجود طیف گسترده ای از انواع بلوکهای دهلیزی بطنی ، ریتم بطنی ممکنست نامنظم گردد . باماساژکاروتیدناگهان آهسته سپس مثل قبل

- Macroreentry
- Typic:sowtooth wave inf & v1
- Atypic:negative wave inf & v6
- Atrial activity of 240-320 with sawtooth pattern. Usually a 2:1 conduction pattern; if it is 3:1 or higher, there is AV node damage

Atrial Flutter

- Treatment is to slow AV node conduction with amiodarone, flecainide,propafenone or sotalol
- Ibutilide 60-90%
- Procainamide,Amiodaron
- DC cardiovert if <48 hours or unstable
- Ablation 90-100%(pathway within the atrium between the tricuspid and the IVC)
- Cardioversion 50j
- Rapid atrial pacing

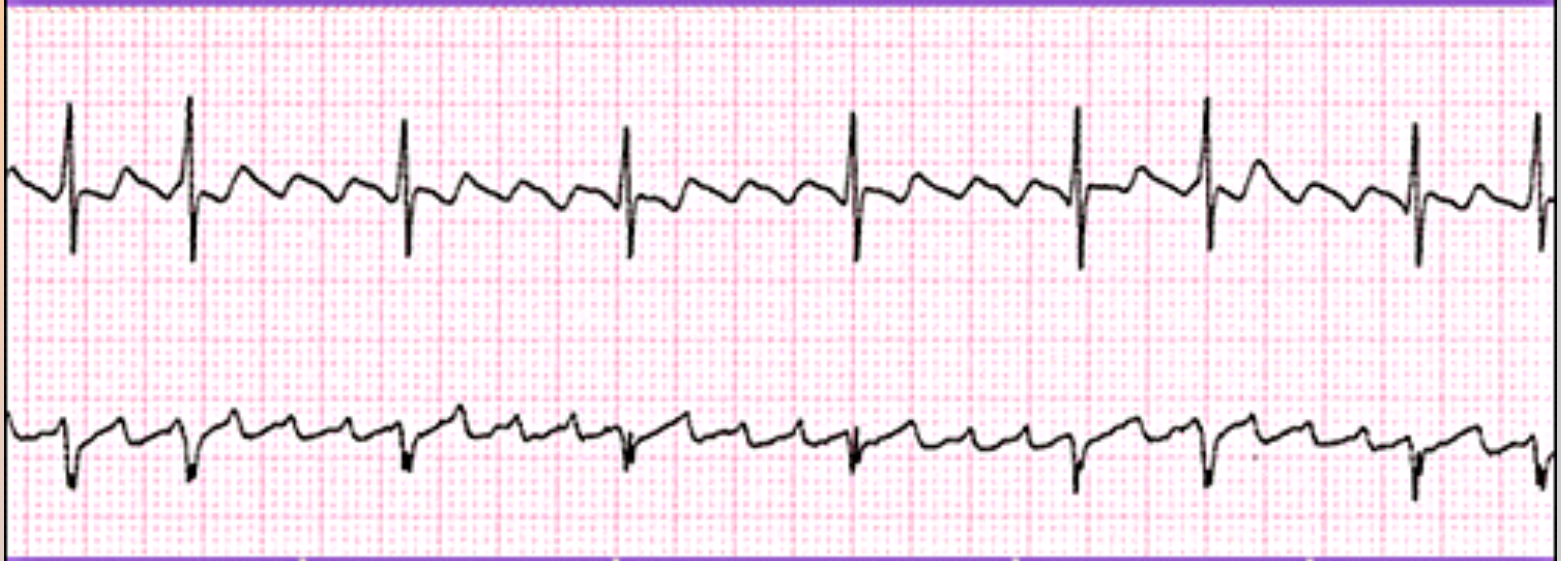
Atrial Flutter(250-350)



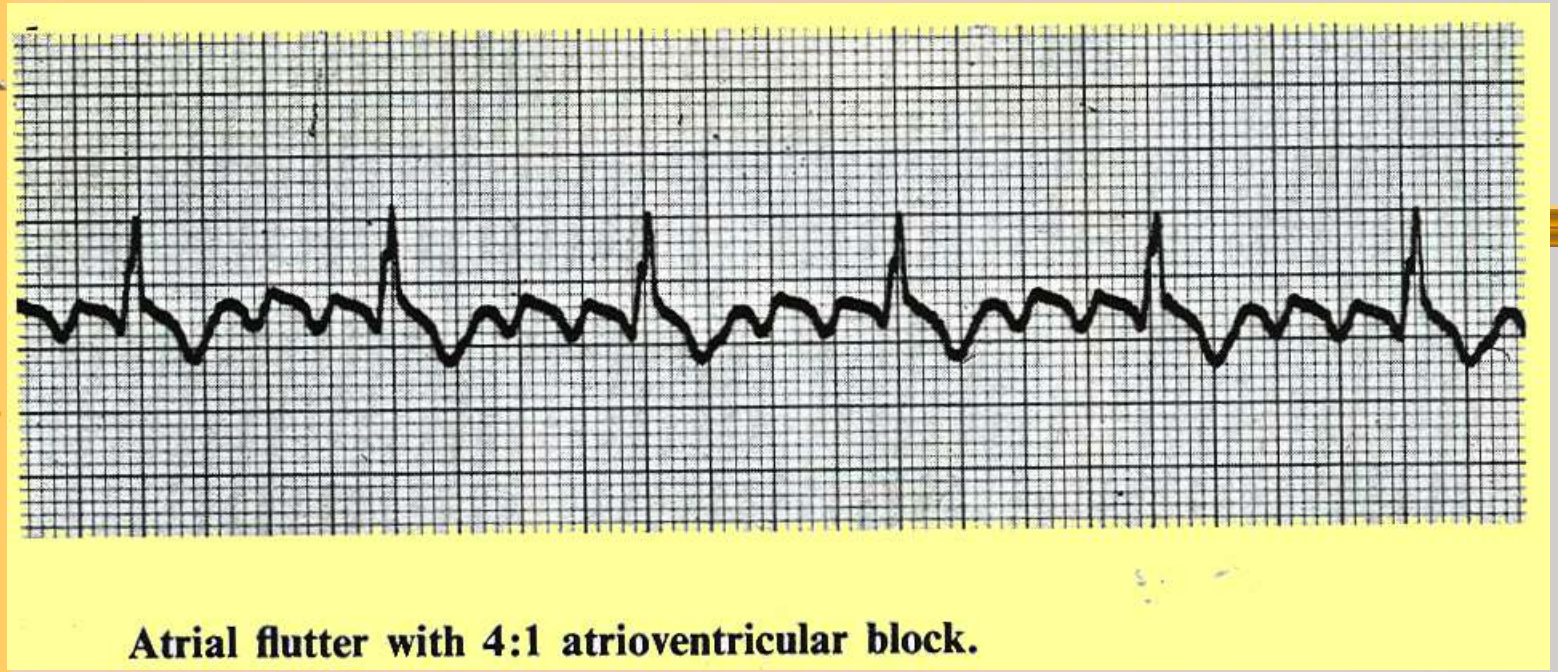
اتیولوژی :

- بیماریهای هایپر تانسیو قلبی
- – بیمار های آترواسکلروتیک قلبی
- – پریکاردیت کانستراکتیو
- – بیماریهای قلبی ریوی حاد
- – عفونت
- – هیپوکسی
- – استرس
- – انفارکتوس میوکارد
- – اختلالات تیروئید
- در مصرف داروهای قلبی مثل دیگوکسین
- – الکلیسم
- – بیماری روماتیسم قلبی
- سن بالا،ابلیشن قبلی
- – نارسایی احتقانی قلب و بیماریهایی که منجر به گشاد شدن دهلیزها میگردند . مثل : نقص دیواره دهلیز ، آمبولی ریوی ، بیماریهای دریچه های میترال و سه لتی و نارسایی مزمن قلبی ، رخ میدهد.

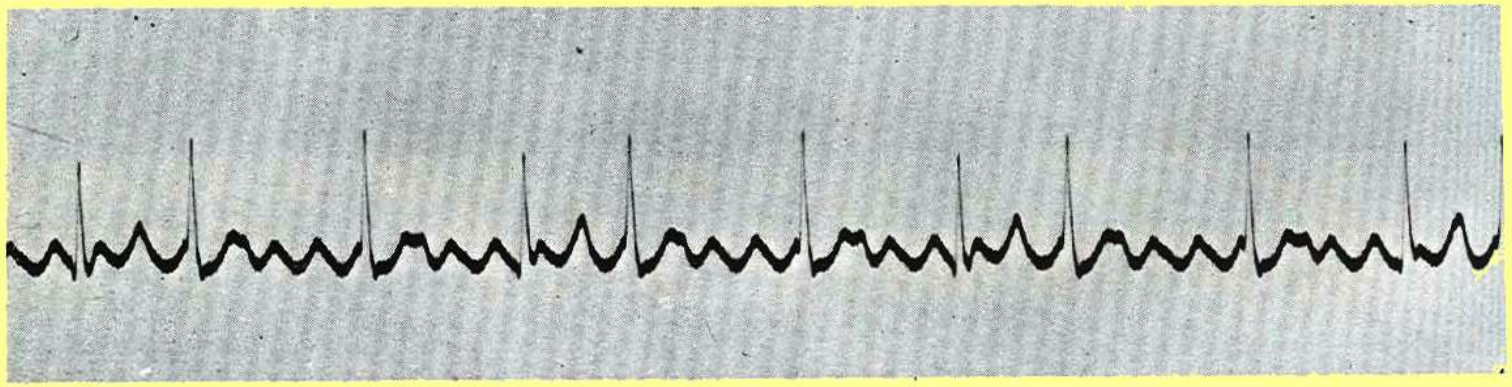
Atrial Flutter



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 220-430 bpm V: <300 bpm	Regular or variable	Sawtoothed appearance	N/A	<.12



Atrial flutter with 4:1 atrioventricular block.



Atrial flutter, 2:1 and 4:1 atrioventricular block producing paired beats.

درمان فلوتر دهليزي

- اگر بيمار دچار وضعيت ناپايدار هموديناميكي ،علائم كاهش خونرساني Hypo Perfusiem مثل اختلال وضعيت هوشياري، درد قفسه سينه يا ادم ريه باشد، اولين و فوري ترين اقدام درماني، شوك سينكرونايز مي باشد.

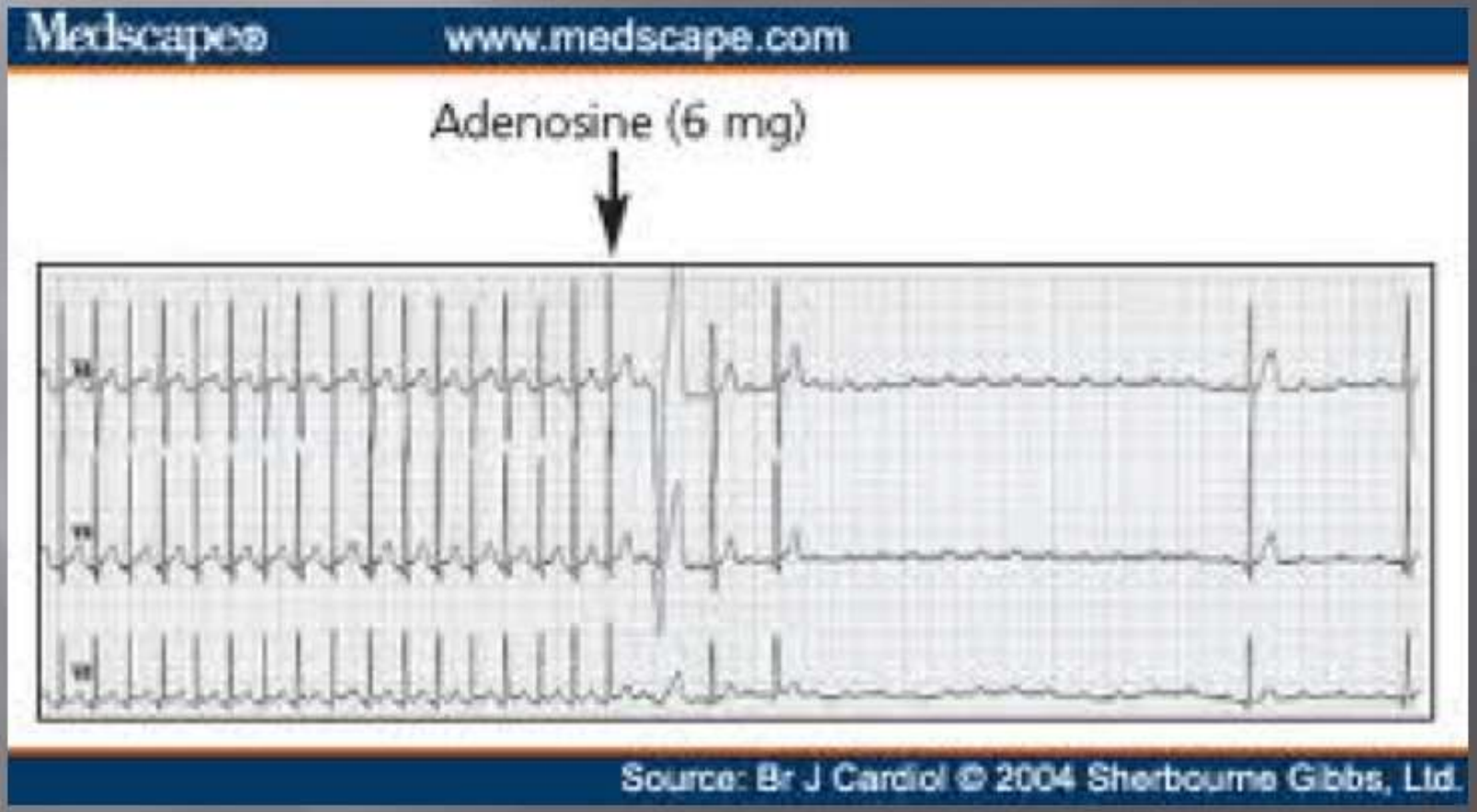
- ميزان انرژي شوك بايد در آغاز يا ولتاژ كم 50-25 ژول باشد .
- گاهي اوقات ممكن است فيبريلاسيون دهليزي بدنبال شوك الكتريكي رخ دهد كه در اين صورت بايد شوك دوم را با 100 ژول انرژي داد.

- شوك كاردیوورژن نبايد به دنبال مصرف دوز بالا ديگوكسين داده شود، زيرا خطر ايجاد ديس ريتمي هاي بطني به ويژه فيبريلاسيون بطني را به همراه دارد.

دارو درماني در فلوتر دهليزي

- داروهاي ضد آريتمي كلاس IA (كيندين، پروكائين آميد)، IC (فله كاينيد ، پروپافنون، يا كلاس III (سوتالول، آميوداروون) ممكن است ريتم فلوتر را به سينوسي تبديل كنند. اين داروها به ويژه بعد از انجام كارديوورژن، قادر به تامين ريتم سينوسي مي باشند.
- 1A,1C WITH BB,CCB(ONLY DEC ATRIAL NOT AV RATE)
- داروي جديد ضد آرايتمي كلاس III ، " ايبوتيليد" وريدي اغلب در تبديل ريتم فلوتر دهليزي (كه به تازگي رخ داده است) به ريتم سينوسي مورد استفاده قرار گرفته است.
- ديگوكسين، وراپاميل، ديليتازم، و بتابلوكرها مي توانند در كاهش سرعت ضربان بطني مفيد واقع گردند اما قادر به تبديل فلوتر دهليزي به ريتم سينوسي نمي باشند.آدنوزين ختم نميكندگاهی تبديل بهAF

Adenosin effect on AFL

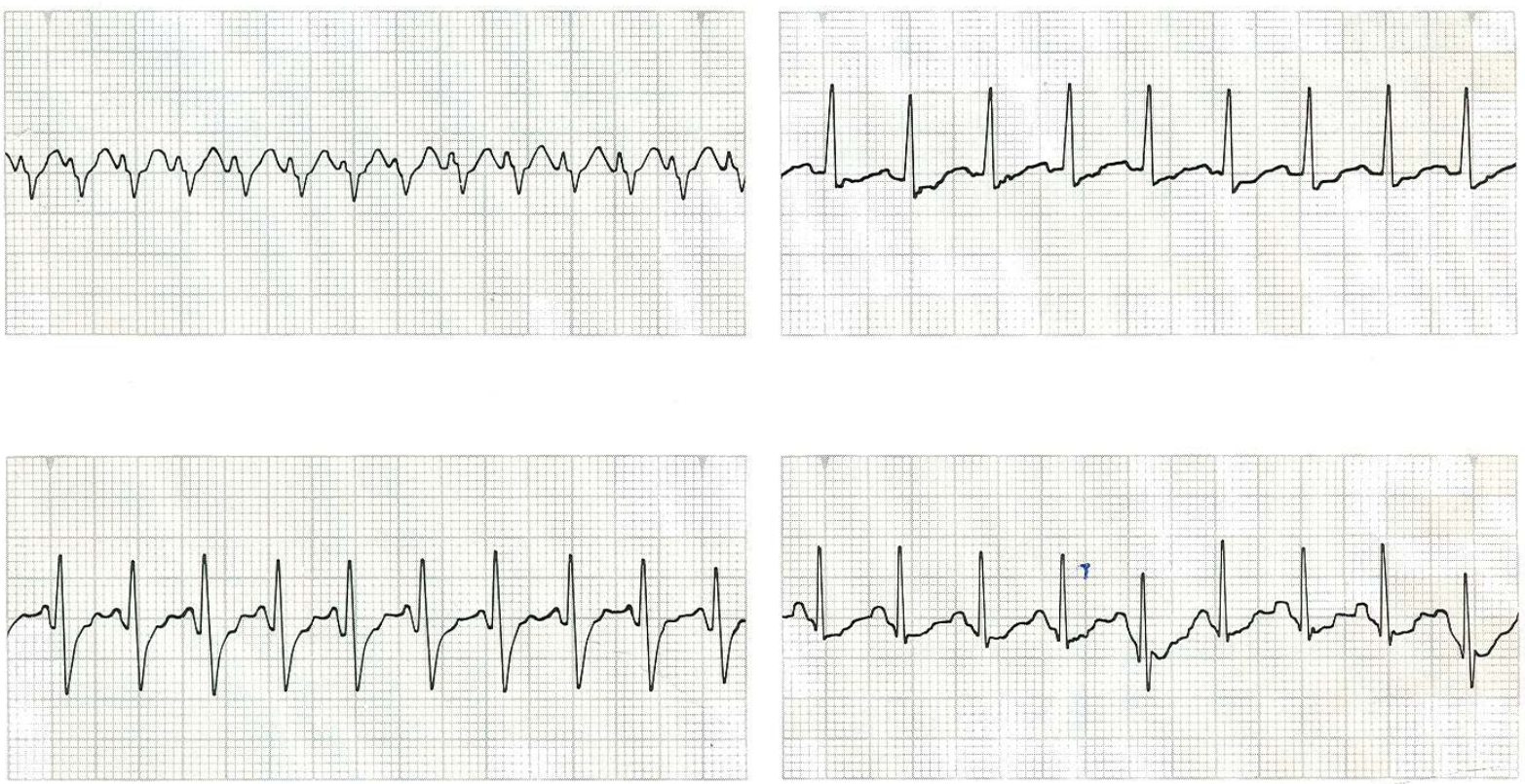


- **كنيدين، ديزوپيراميد و پروكائين آميد** تا زمانيكه پاسخ هاي بطني بيمار كاهش نيافته باشد، نبايد مورد استفاده قرار گيرند زيرا اثر واگوليتك و اثر كاهش ضربان دهليزي اين داروها باعث هدايت 1 به 1 دهليز به بطن مي گردد كه بسيار خطرناك است زيرا ضربان دهليزي مثلا از 300 به 200 ضربه در دقيقه كاهش يافته و گره دهليزي بطني فرصت مي يابد كه ايمپالس هاي دهليزي را بيشتر از زمان بلوك به بطن ها هدايت نمايد و در نتيجه ضربانات بطني افزايش مي يابند.

AT

- ***Rhythm*** = منظم
- ***Rate*** = 150 تا 250 ضربان در دقيقه
- ***موج P*** = در صورت مشخص بودن با موج P سينوسي تفاوت دارد . بر خلاف تاكيكاردي سينوسي ، تشخيص P مشكل است و گاهي ممكنست موج P روي موج T قبلي بيفتد .
- ***فاصله P-R*** = كوتاه و كمتر از 12/0 ثانيه ميباشد
- ***كمپلكس QRS*** = معمولا" طبيعي است مگر اينكه بر اثر ديپولاريزاسيون بطنها از راههاي غير طبيعي ، پهن گردد .
- Long RP
- AT+BLOCK COMMON
- **P V1 + or biphasic,1&AVL(-):LA**
- **RA:P1(+),V1(-,biphasic)**

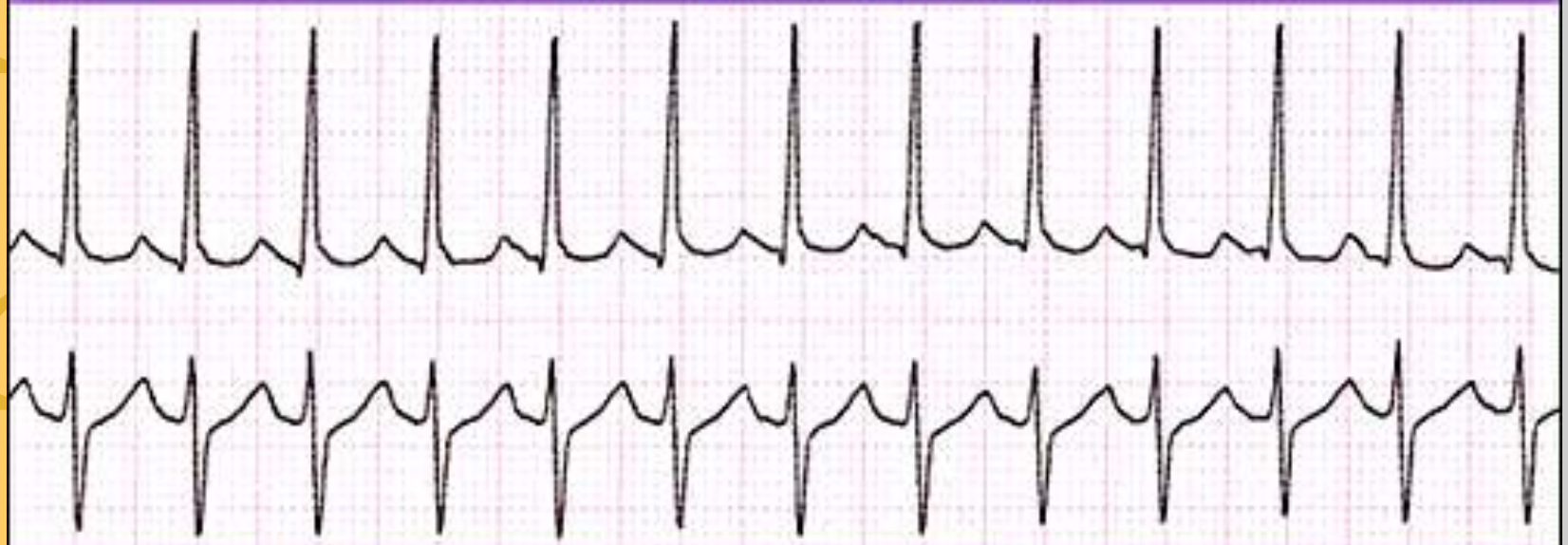
PAT



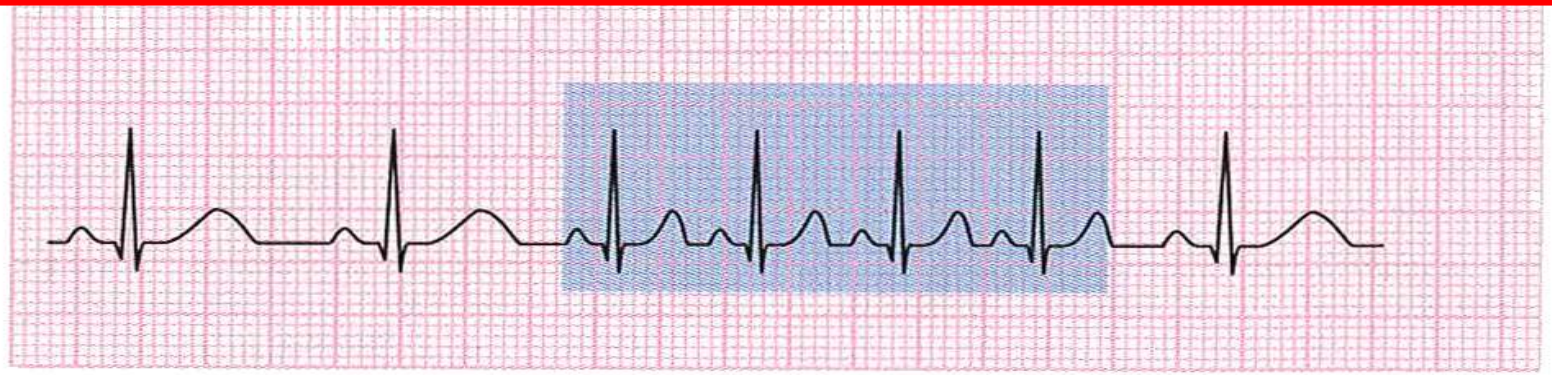
FAT

- : عارضه شايع افراد جوان ، يا افراديكه بطور مادرزادي مدار چرخشي دارند و يا خانمهاي جواني كه دچار اختلالات ارگانيكي قلبي ميباشند(نادر) يا افراد سالم بعلل نامعلوم نيز رخ ميدهد .
- ريتم قلبي ناگهان بر اثر هيجان ، تنباكو ، كافئين ، خستگي ، داروهاي سمپاتوميمتيك و يا الكل تغيير ميكند .
- After CHD surgery-Atrial abl-Dig toxocity-CAD,MI,HF,corpulmonal
- ضربانات سريع ممكنست موجب دردهاي آنژيني بعلت كاهش خون كرونري گردد . بازده قلب كاهش مي يابد و ممكنست منجر به CHF گردد . معمولا" بيمار اين ريتم را به مدت طولاني نميتواند تحمل كند.
- شروع وخاتمه تدريجی-شروع warm up
- AVN غيروابسته
- خط پايه درتمام ليدها
- ماساژ سينوس كاروتيد ياآدنوزين فقط كاهش HR معمولا ختم آريتمی نميدهد

Atrial Tachycardia



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
140-250 bpm	Regular	Abnormal P before each QRS (difficult to see)	<.20	<.12



Rate:	100–180 BPM
Regularity:	Regular
P wave:	Morphology of ectopic focus is different
P:QRS ratio:	1:1
PR interval:	Ectopic focus has a different interval
QRS width:	Normal, but can be aberrant at times
Grouping:	None
Dropped beats:	None

Putting it all together:

Ectopic atrial tachycardia occurs when an ectopic atrial focus fires more quickly than the underlying sinus rate. The P waves and PR intervals are different because the rhythm is caused by an ectopic atrial pacemaker (a pacemaker outside of the normal SA node). The episodes are usually not sustained for an extended period. Because of the accelerated rate, some ST- and T- wave abnormalities may be present transiently.

Figure Ectopic atrial tachycardia.

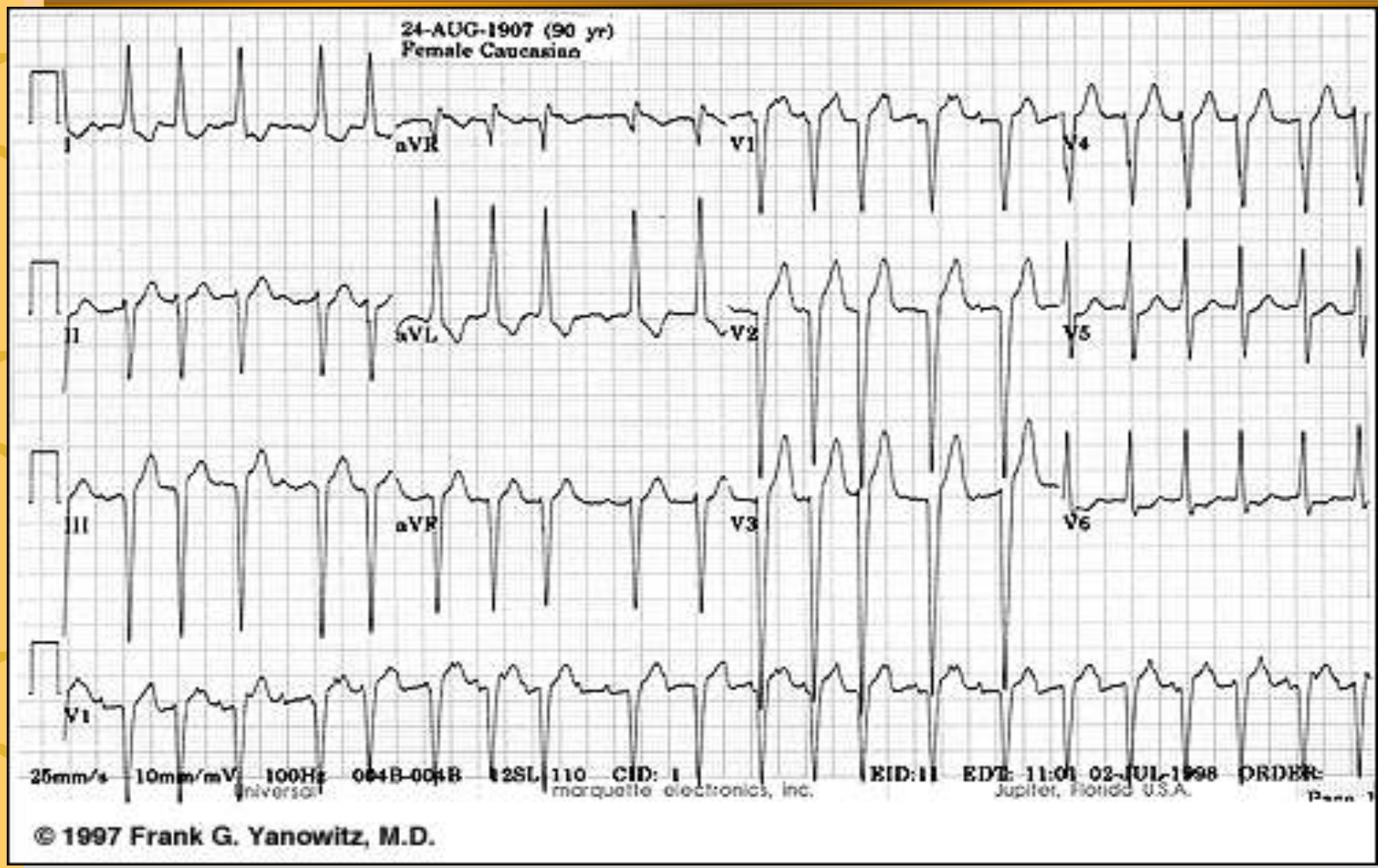
درمان PAT

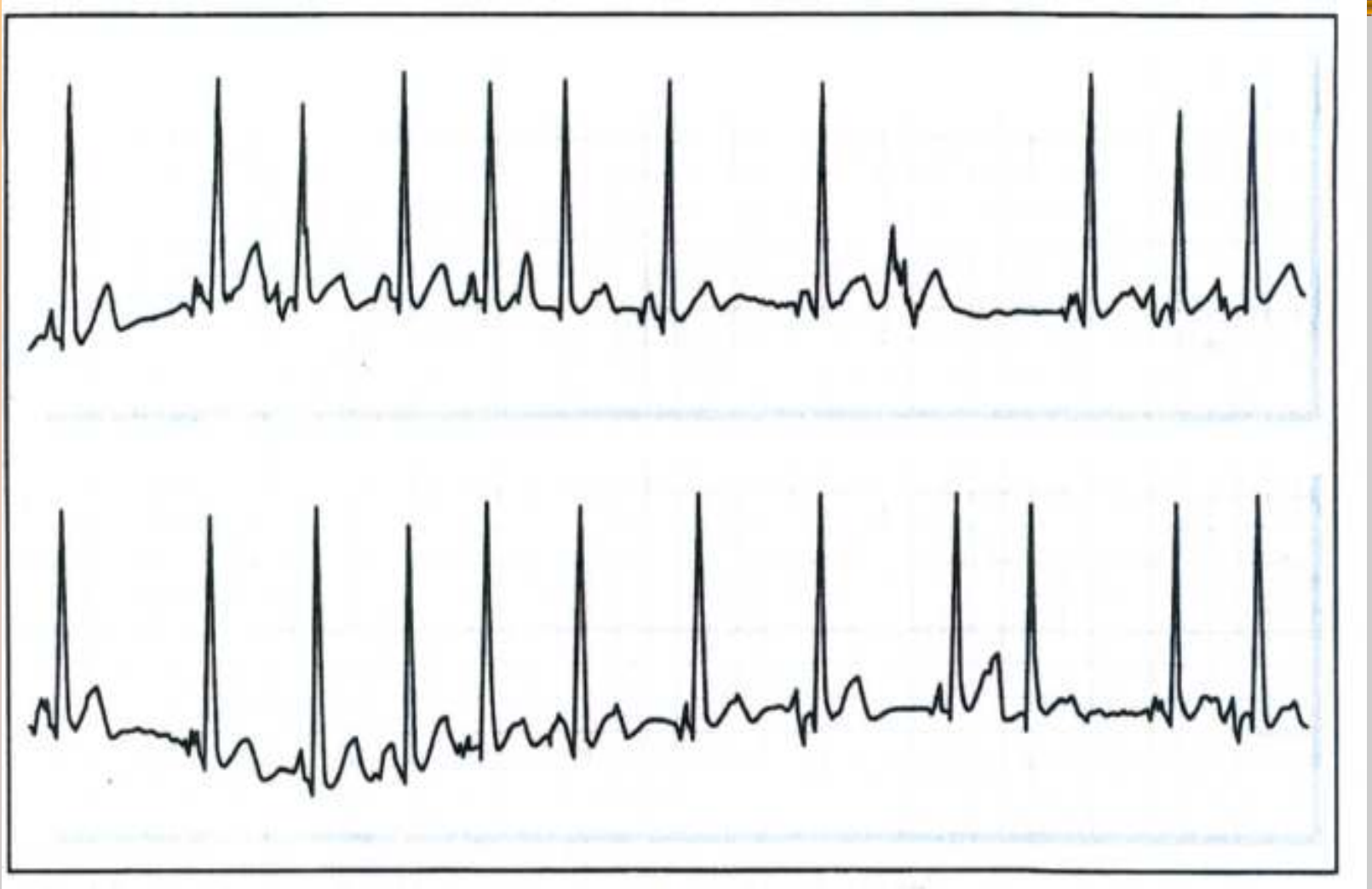
- درمان بسوي رفع علت و كاهش ضربان قلب معطوف ميباشد.
- در بسياري از بيماران PAT خود بخود و بدون درمان بهبود مي يابد و ممكنست حتي حس نكنند .
- تحريك عصب واگ ,ادنوزين بندرت بادوز بالاو گاهی فقط تشخیصی
- وراپامیل-بتابلوکر –دیگوگسین
- 1a,1c,3
- کنیدین –فلکائینید-پروپافنون-دیزوپیرامید-سوتالول-آمیوداروون
- شوك الكتريكي (Cardioversion) در reentry
- Abl:80% بخصوص بدون بیماری ساختمانی

MAT

- **Automatic atrial rhythm from various different foci**
- **HR:100-130**
- **Irregular PP-Irregular PR-different P wave (3 forms),isoelectric line**
- **Seen in hypoxia, COPD, atrial stretch and local metabolic imbalance. Old age-CHF-dig-theophylin**
- **treat with oxygen and slow channel blocker like verapamil or diltiazem. amiodaron-k-Mg-abl**
- **Shock is not useful**

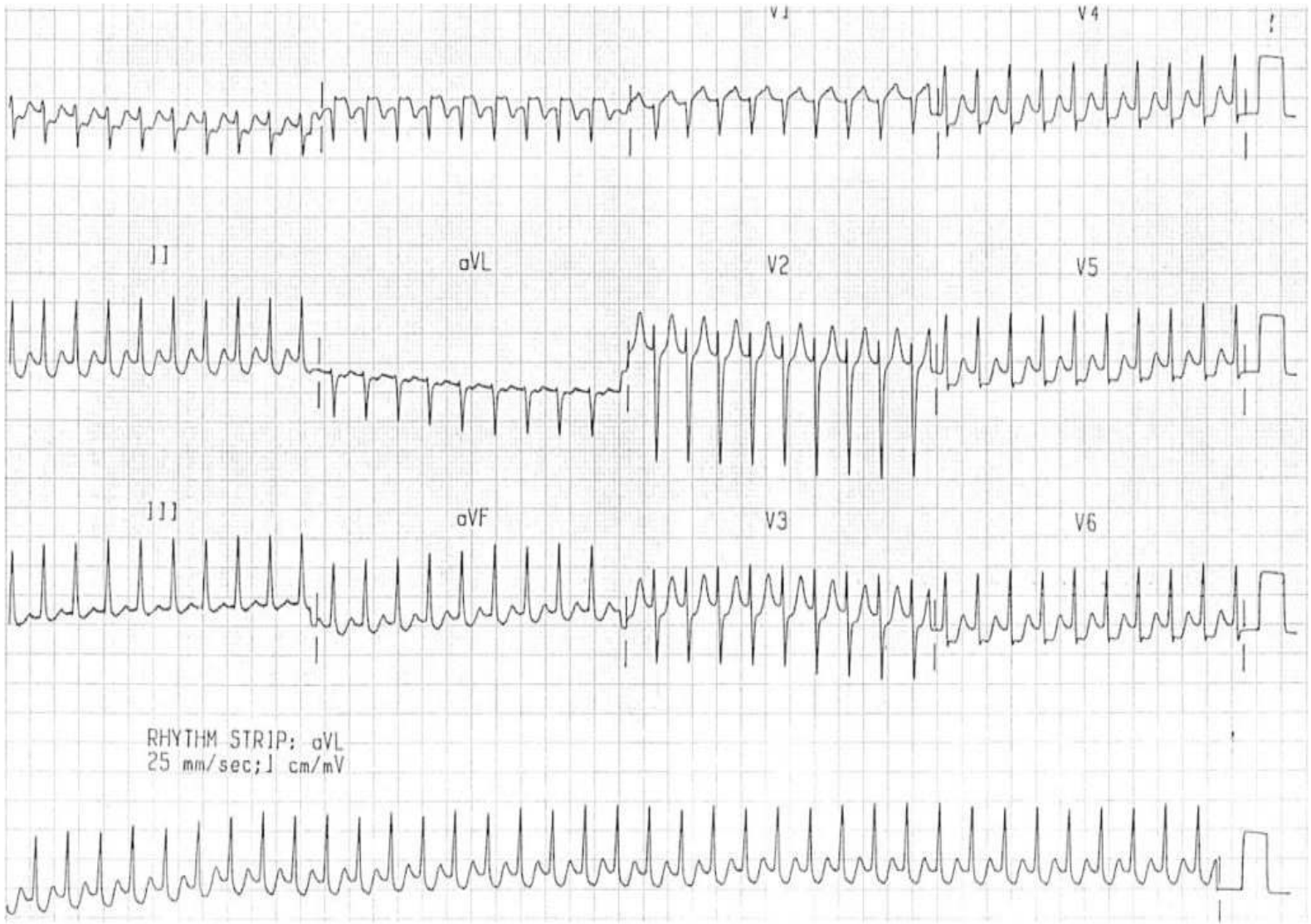
Multifocal Atrial Tachycardia





AVRT

- شروع وختم ناگهانی
- ۱۵۰-۲۵۰
- No discernible P-waves
- Regular,faster than AVN،NARROW QRS
- ST depression during tachycardia
- Negative invert p before or<30ms after QRS excluded AP
- اگرموقعی که هیس دررفرکتوری است PVC موجa دهلیزی بدهد راه فرعی اثبات می شود
- VA<50% RR
- بطن ودهلیز اجزا اصلی مدار آریتمی وبلوک درهریک قطع آریتمی


V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
RHYTHM STRIP: aVL
25 mm/sec; 1 cm/mV

WPW

- Ventricles receive partial signal normally and partially through accessory pathway
- Symptomatic tachycardia, short PR interval (<0.12), a delta wave and prolonged QRS (>0.12)، secondary ST-T change,normal terminal QRS
- EPS helps to identify the location of the accessory pathway

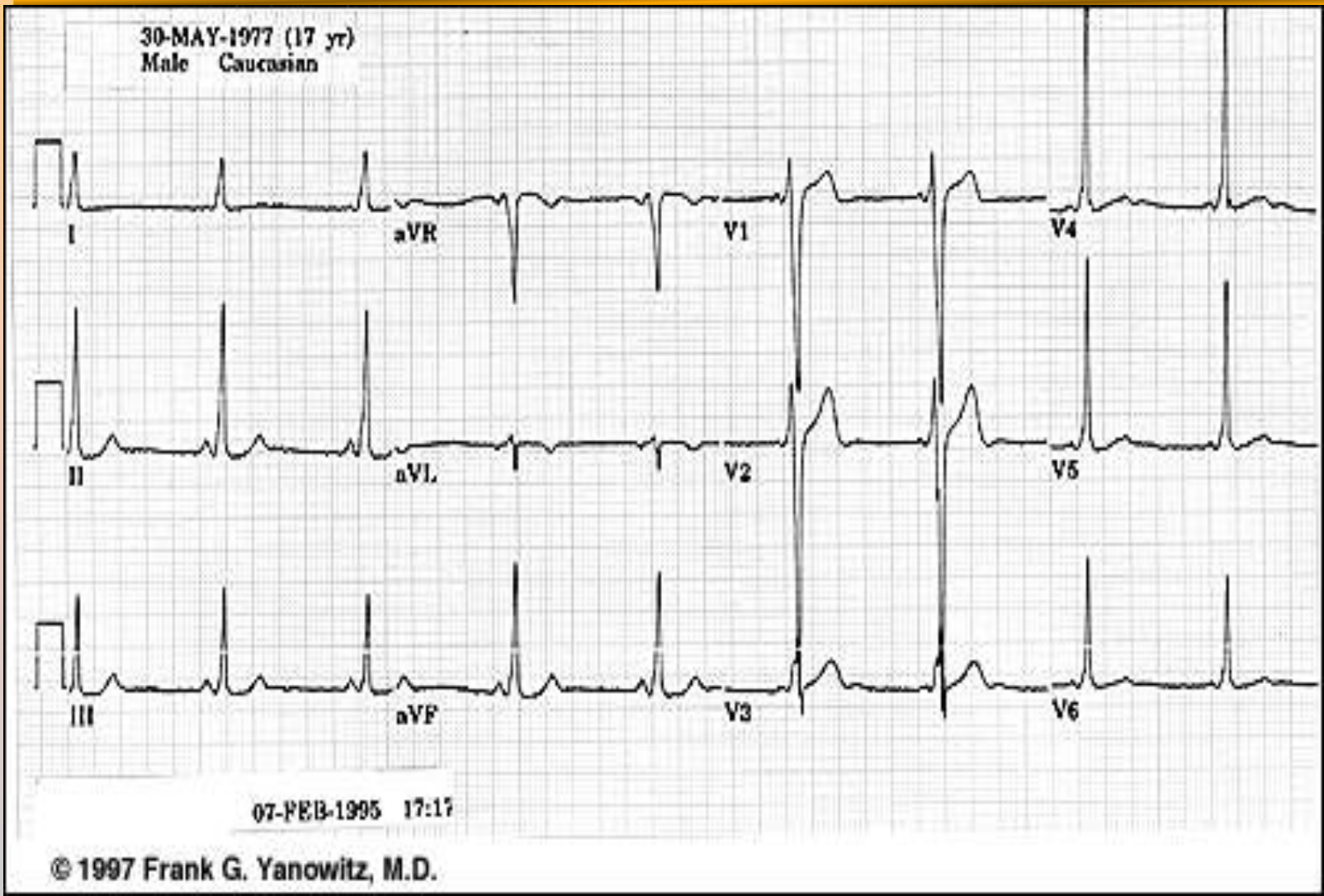
WPW

- Because WPW has both normal conduction through the AV node and accessory pathway conduction that bypasses the AV node, a-fib can happen via the accessory pathway
- Inhibition of the AV node will end up in worsening the a-fib because none of the signals are slowed down by the AV node before hitting the ventricle.

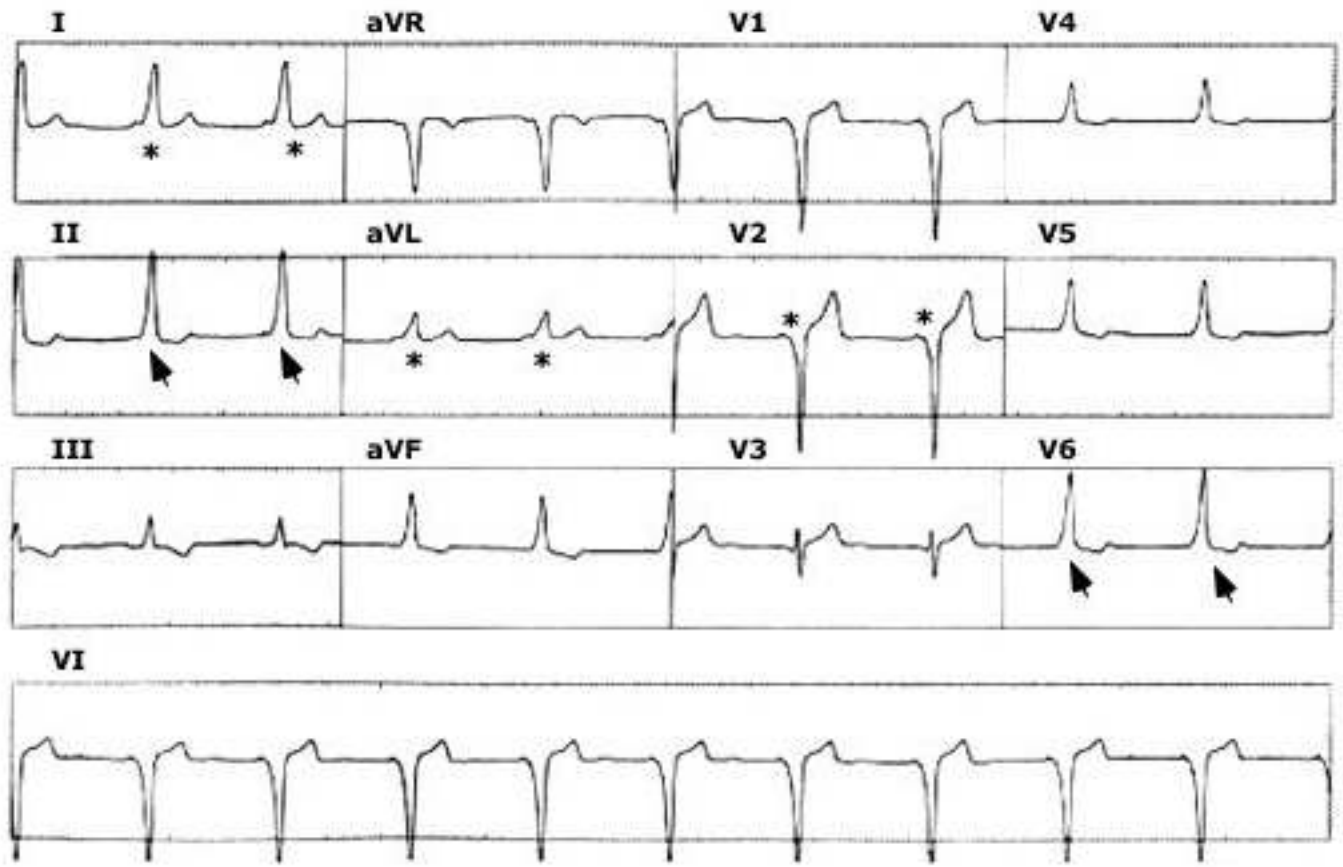
* Do not use any meds that will slow AV node conduction, ie digoxin, beta-blockers, adenosine or calcium channel blockers.

* The best choice is procainamide as it slows the accessory pathway. *If patient becomes hypotensive, cardiovert immediately!

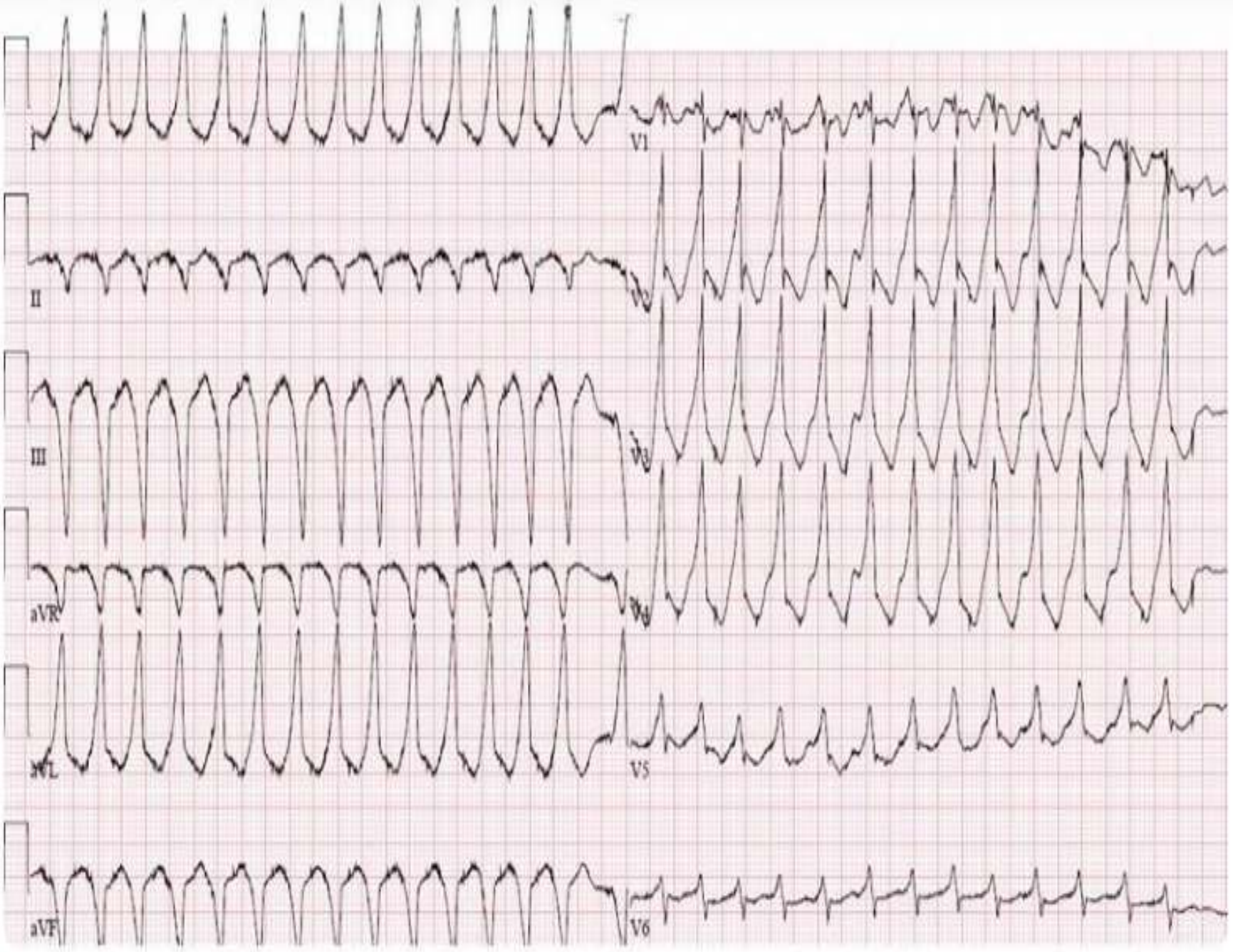
Wolf Parkinson White



ECG in Wolff-Parkinson-White



The 12 lead ECG shows the typical features of Wolff-Parkinson-White; the PR interval is short (*) and the QRS duration prolonged as a result of a delta wave (arrow), indicating ventricular preexcitation.
Courtesy of Martin Burke, DO.

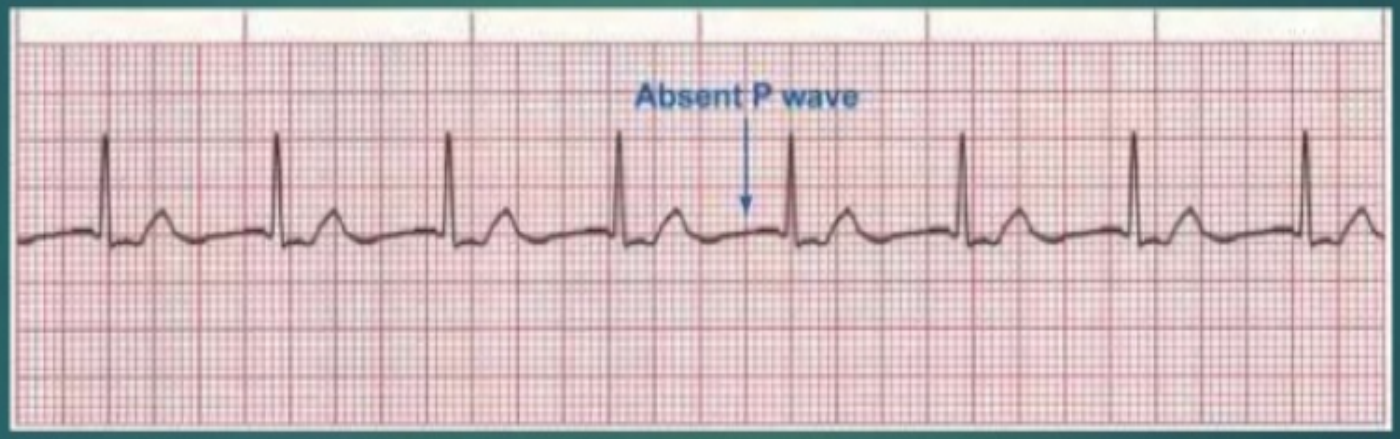

I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

Junctional tacycardia

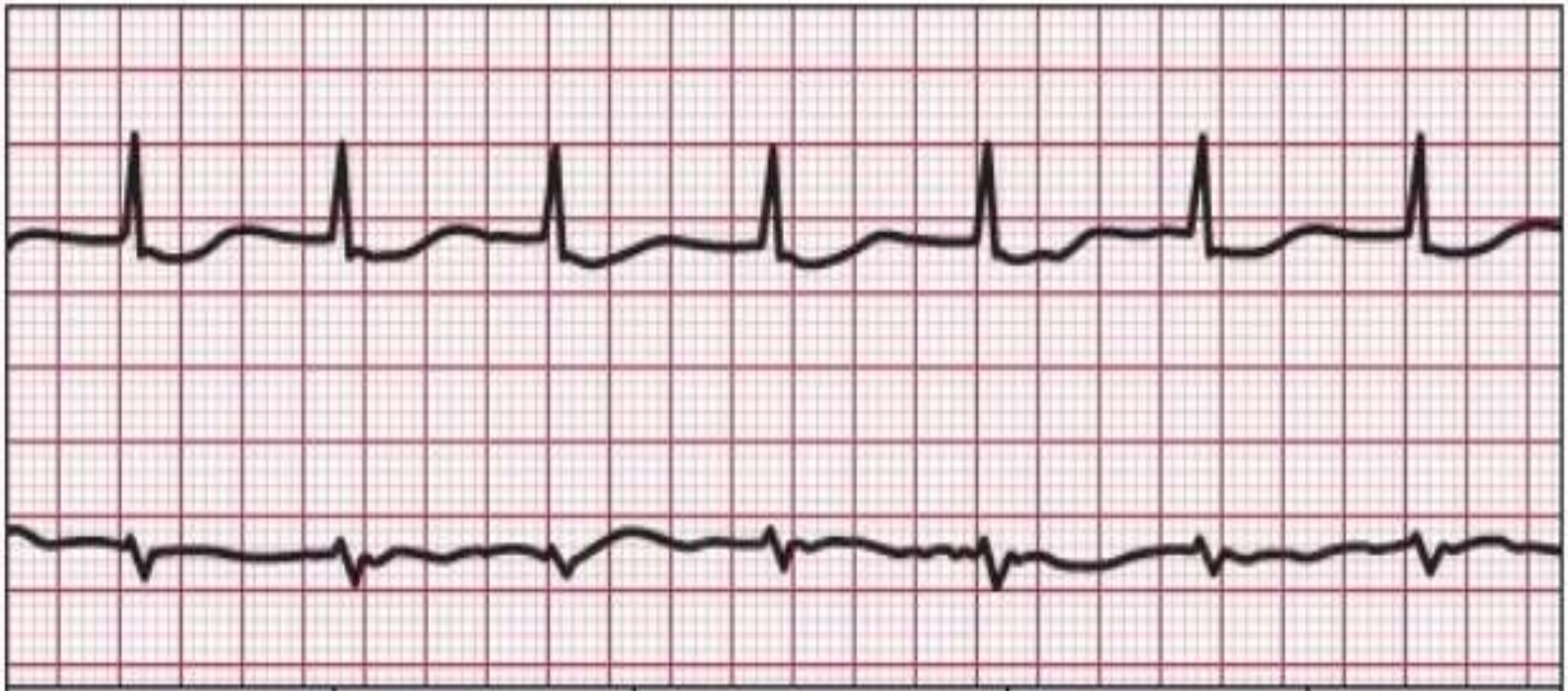
- JET(automaticity AVN),rare in adult,children(after CHD surgery),narrow QRS
- Accelerated:after PVC,gradually,no treatment,p like AVNRT

ACCELERATED JUNCTIONAL RHYTHM

- **Rate:** 61–100 bpm **Rhythm:** Regular
- **P Waves:** Absent, inverted, buried, or retrograde
- **PR Interval:** None, short, or retrograde
- **QRS:** Normal



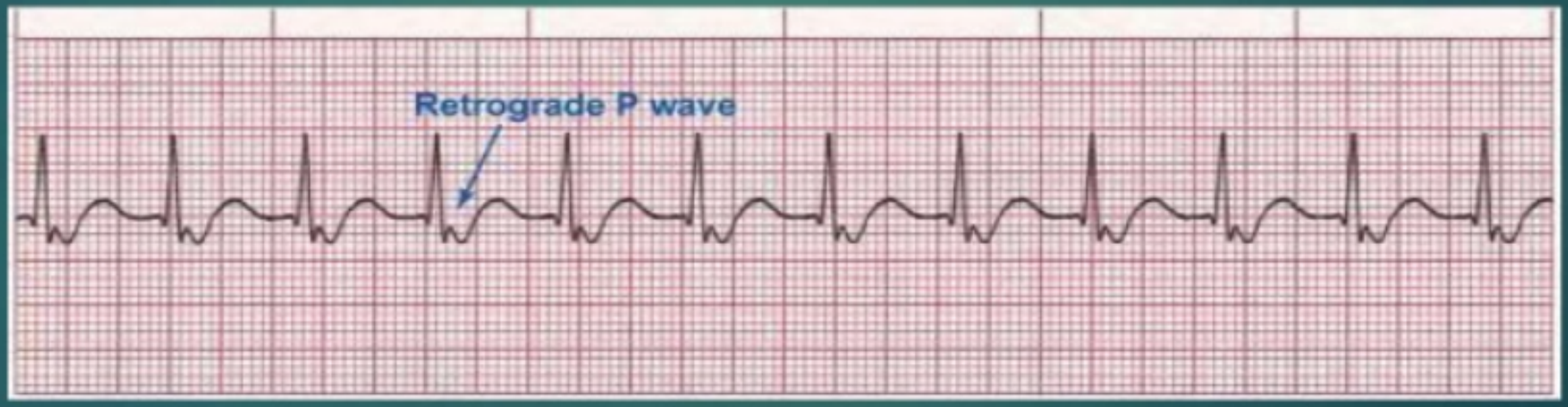
Accelerated Junctional Rhythm



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
60-100 bpm	Regular	Inverted, absent or after QRS	<.12	<.12

JUNCTIONAL TACHYCARDIA

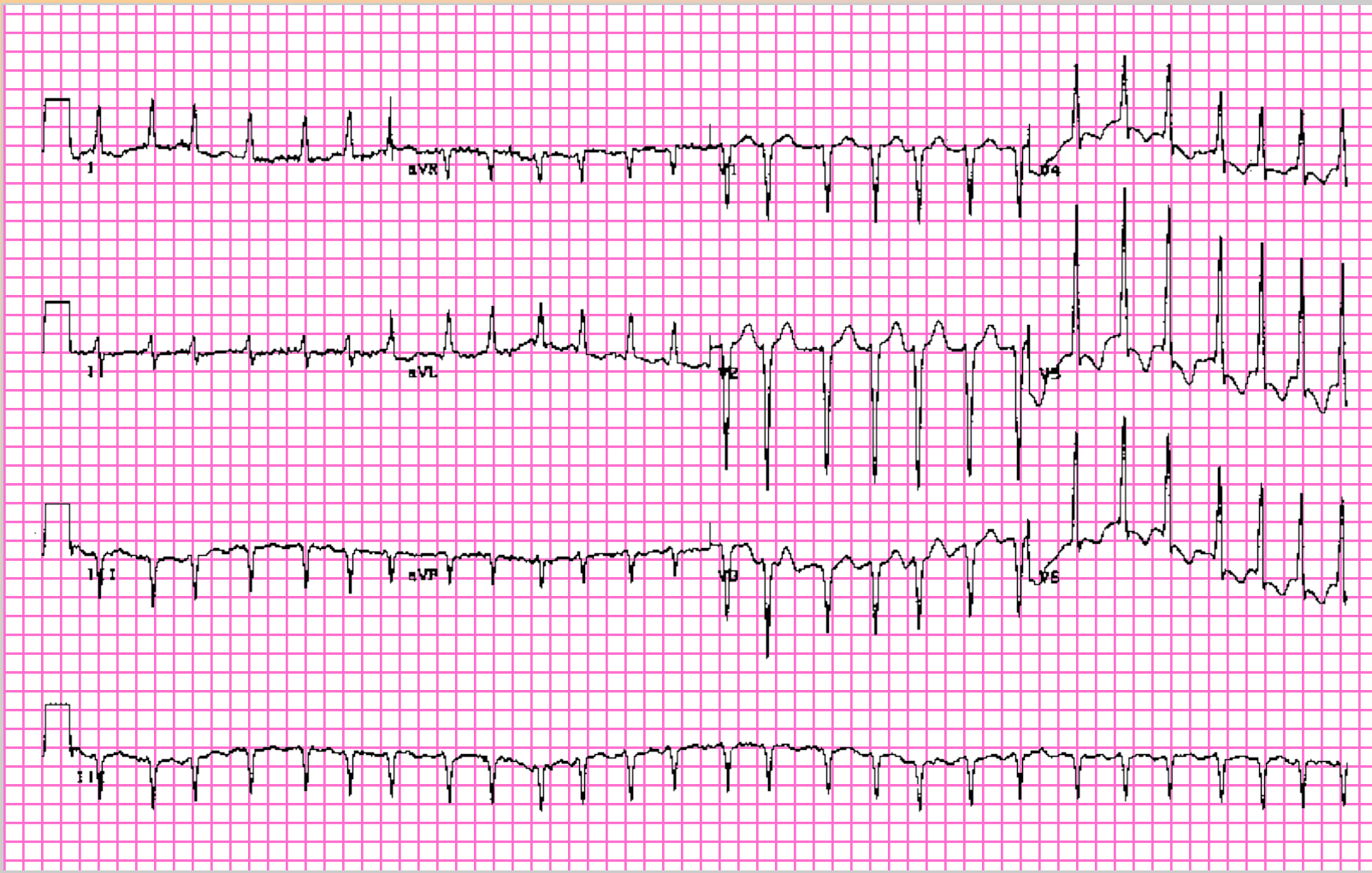
- **Rate:** 101–180 bpm **Rhythm:** Regular
- **P Waves:** Absent, inverted, buried, or retrograde
- **PR Interval:** None, short, or retrograde
- **QRS:** Normal



AF

- F wave 300-600
- Automaticity,triggered activity,microreentry
- Paroxysmal <7days
- Persistant>7days
- Longstanding>1y
- Permanent>1y+resistant to cardioversion
- Can be due to HTN, cardiomyopathy, valvular heart desease, sick sinus, WPW, thyrotoxicosis

Atrial Fibrillation



Rate control

- Rest(60-80),mild activity(90-115),severe(120-160)
- Beta-blockers
  - Continuation after CABG may prevent a-fib
  - Good for hyperthyroid or post-MI patients with a-fib
  - Carvedilol decreases mortality in patients with CHF
  - Esmolol is good for acute management
- Digoxin actually increases vagal tone, thus indirectly slowing AV node conduction. But it is used essentially only in patients with LV dysfunction because it's inotropic.

Rate control

- Calcium Channel Blockers
  - Nondihydropyridines (verapamil or dilitiazem) block AV node conduction but also have negative inotropy, so don't use in CHF.
- Adenosine is too short acting to be of any use in a-fib
- Last choice is AV node ablation and permanent pacing

Rhythm control

- Rhythm control does not decrease thromboembolic risk and may be proarrhythmic
  - Class 1A (quinidine, procainamide, disopyramide) slows conduction through HIS can cause TDP. They also enhance AVNconduction, so they should be used only **after rate is controlled**
  - Class 1B (lidocaine, mexilitine, tocainide) are **useless** for a-fib
  - **Class 1C (propafenone, and flecainide) slow conduction through HIS are good first choice. Lone AF-mild SHD**
- **Amiodarone** is good if patient is post-MI or has systolic dysfunction.

Cardioversion for A-Fib

- Cardiovert if symptomatic
- Patients with a-fib for more than 2 days should be receive 3 weeks of anticoagulation before electrical cardioversion. (95%success)
- Give anticoagulant for 4 weeks after cardioversion
- NOACS 1.5-2H before

chemical

- Drug:no effect in AF>7D
- AF<2-3D
  - Ibutilide(EF>35%)=60-70%
  - Amiodarone=40-50%
  - Procainamide=30-40%

Anticoagulation Rules for A-Fib

- Everybody who has RHD,prosthetic valves, thyrotoxicosis,RF, LAE ,should be anticoagulated
- CHA2DS2-VASC(>=2)
- >75 y give WARFARIN but keep INR 2-2.5 due to increased risk of bleed

HAS-BLED

- HTN
- Abnl RENAL-LIVER
- Stroke
- Abnl INR
- 75Y
- Multi drug(NSAID-ASA)
- Coagulopathy
- Alcohol
- >=5:12%/Y bleeding

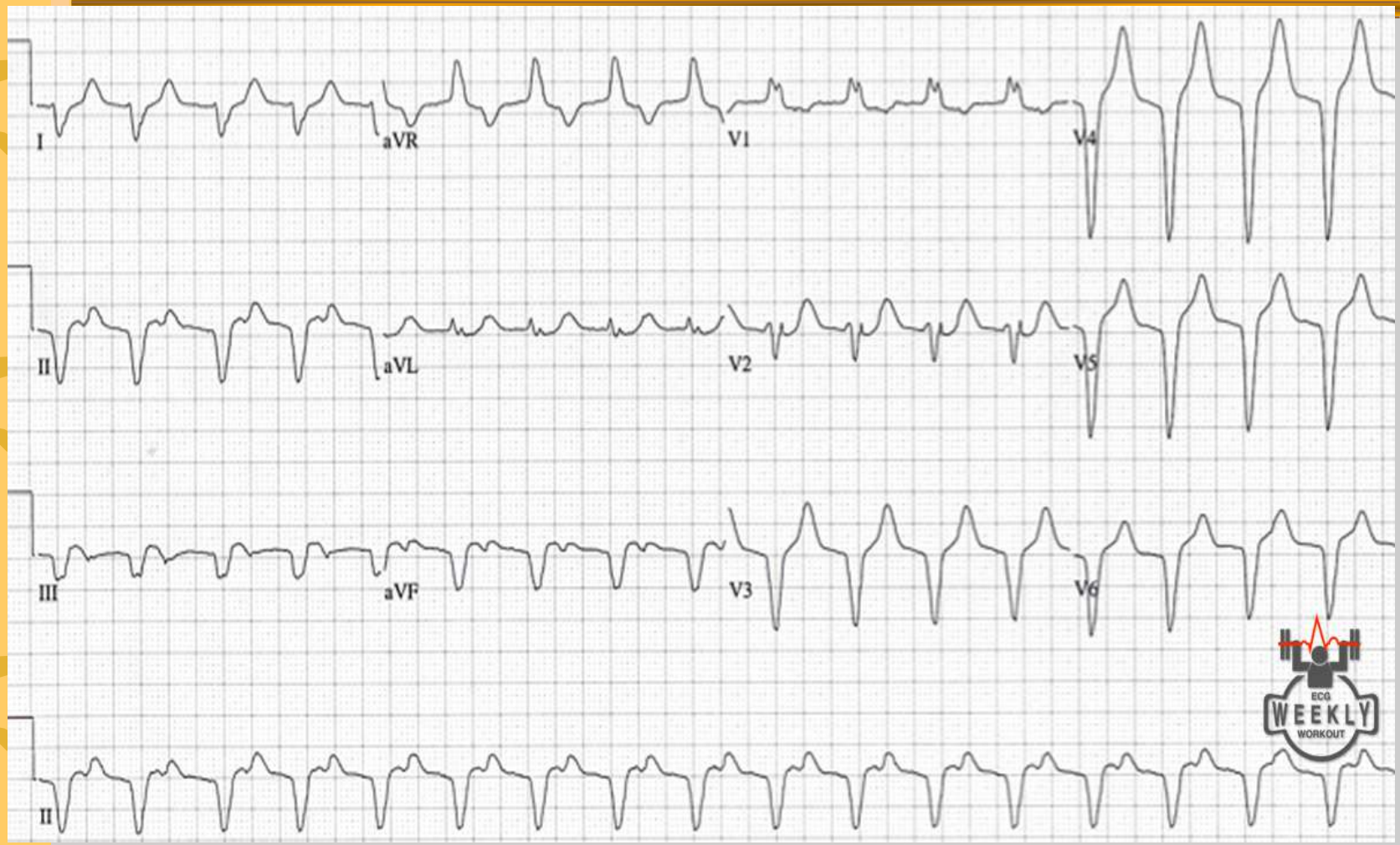
Arrhythmia Presentation

VENTRICULAR ARRHYTHMIAS - LEVELS OF SYMPTOMS

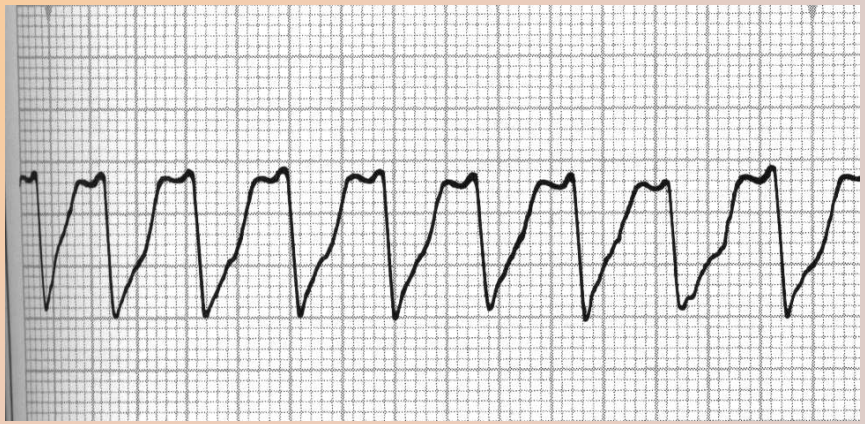
SYMPTOM-FREE	→	UNAWARE OF RHYTHM
MINIMAL SYMPTOMS	→	PALPITATIONS
LIFESTYLE LIMITING	→	LIGHTHEADEDNESS
HEMODYNAMIC EFFECTS	→	SYNCOPE
LIFE-THREATENING	→	CARDIAC ARREST

AIVR

- HR=60-110
- Fusion beatآریتمی وانتهای ابتدا
- بعلت HR پایین capture beat شایع
- Automaticity-گذراومتناوب-پروگنوزوهمودینامیک معمولا بی تغییر
- درقلب بیماروشکل ساختاری(سکته قلبی ومسمومیت بادیگوگسین-احیا-رپرفیوژن)
- معمولا به VTسرعت بالا تبدیل نمیشود
- شروع وختم آریتمی هم تدریجی


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
ECG
WEEKLY
WORKOUT

Vtach: 3 or more PVCs in a row

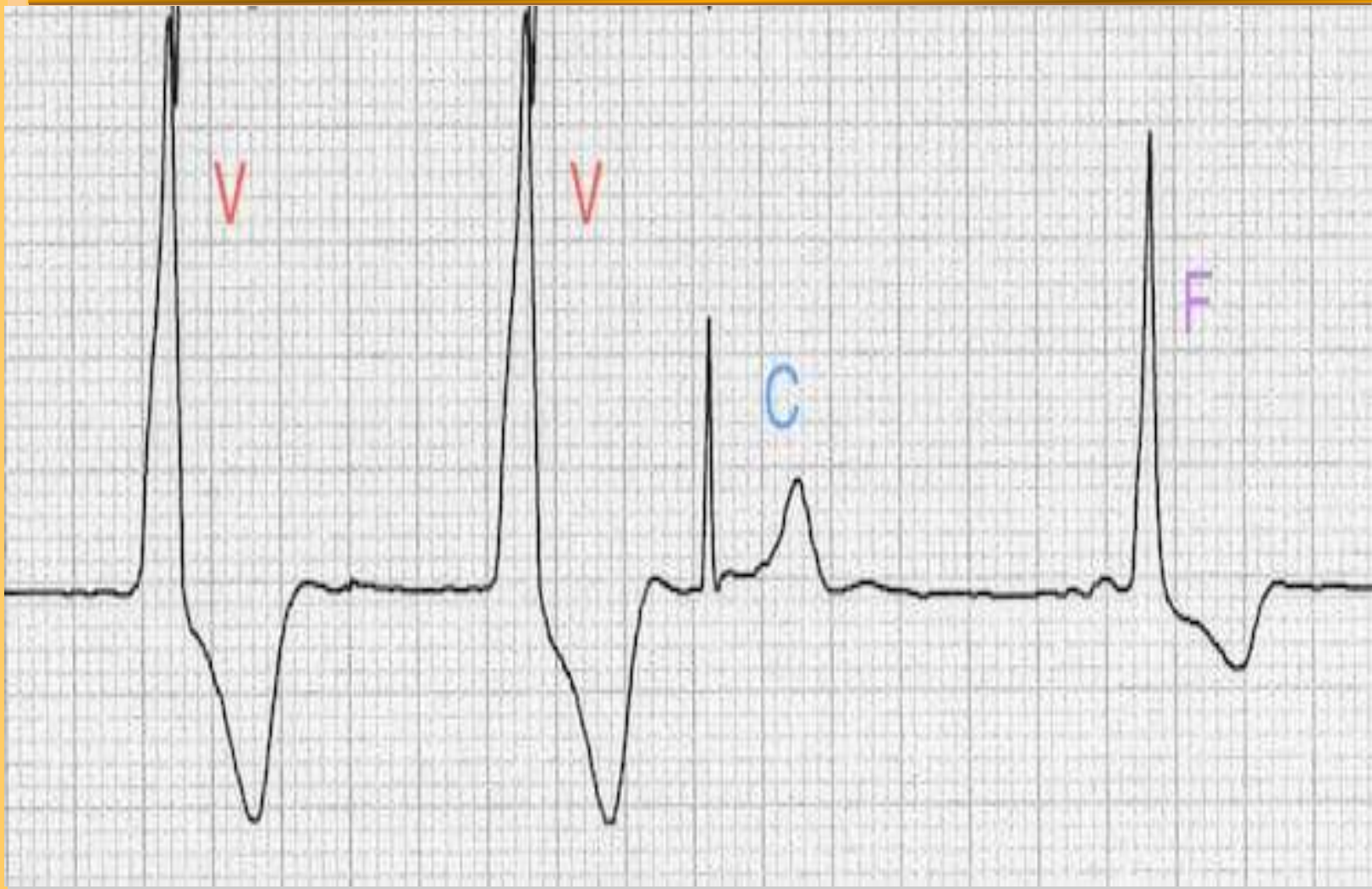


- Wide QRS with a regular pattern and a rate of 150-200
- Slow VT=70-120
- sustainVT>30s:Patient will usually lose consciousness
- help to have patient cough if they are still conscious

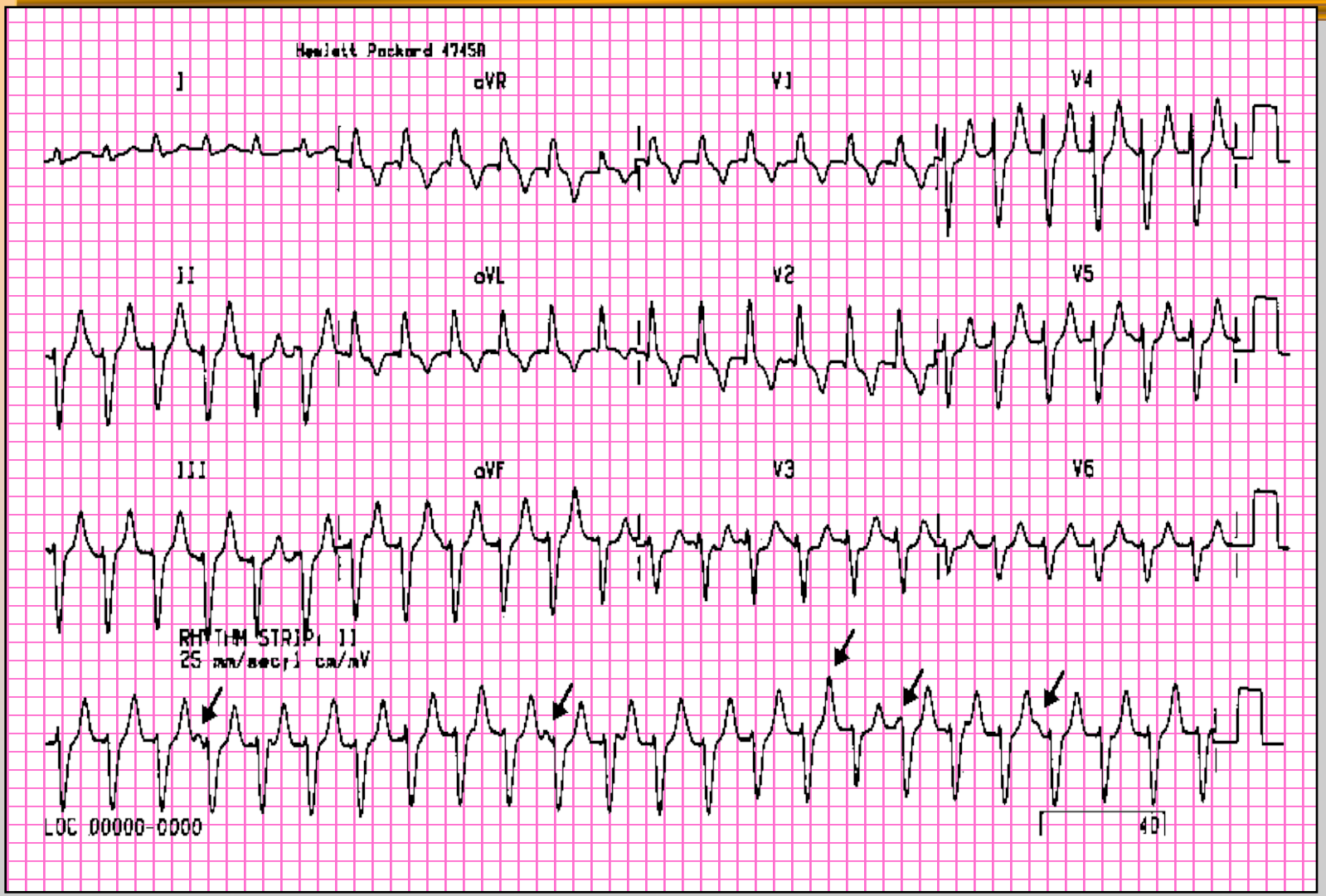
VT

- TA-automaticity
- AV dissociation
- Fusion beat:like VT but narrower
- Capture beat:reverse VT
- QRS>140
- R>R' V1,QS V6
- Concordance
- R+ AVR

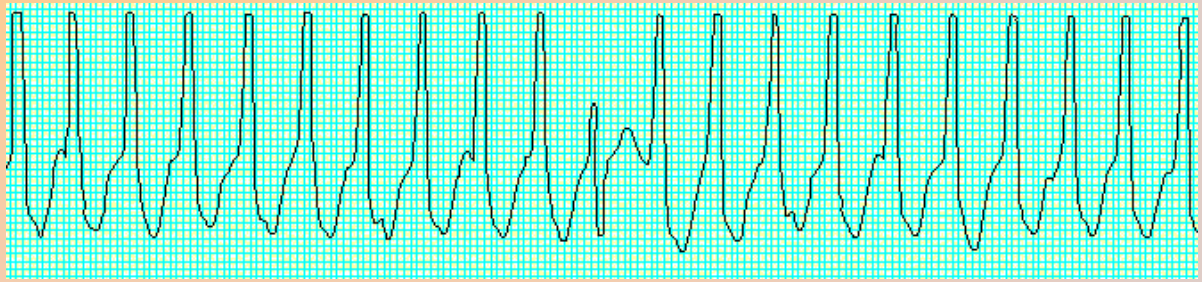
Capture beat:reverse VT
FUSION:LIKE VT



Ventricular Tachycardia



Ventricular Tachycardia

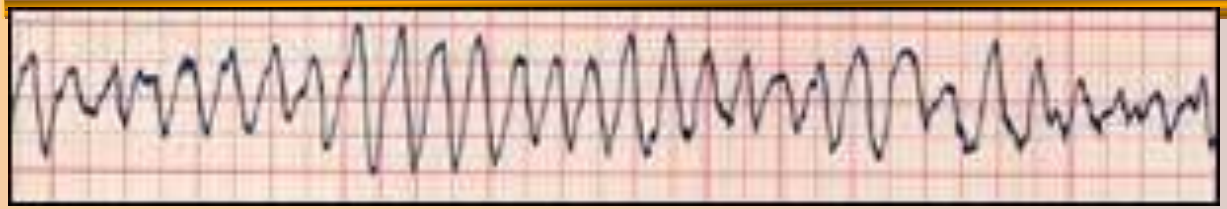


- Impulse is initiated from the ventricle itself
- Wide QRS, Rate is 140-250
- If unstable DC cardiovert
- If not, IV Amiodarone and/or lidocaine(very fast) \ procainamide(no RF/HF)

VT

- LBBB:RAD,
- V1 is more negative thanv6,
- Rv1>40ms,
- qR,QS V6,
- R-nadir S>60 V1
- Wide QRS>14Oms,LAD
- IF RS(R-nadir S=>100ms)
- Concordance
- RBBB(R>R'),V1mono or biphasic
- first part reverse sinus,
- rS or QS V6

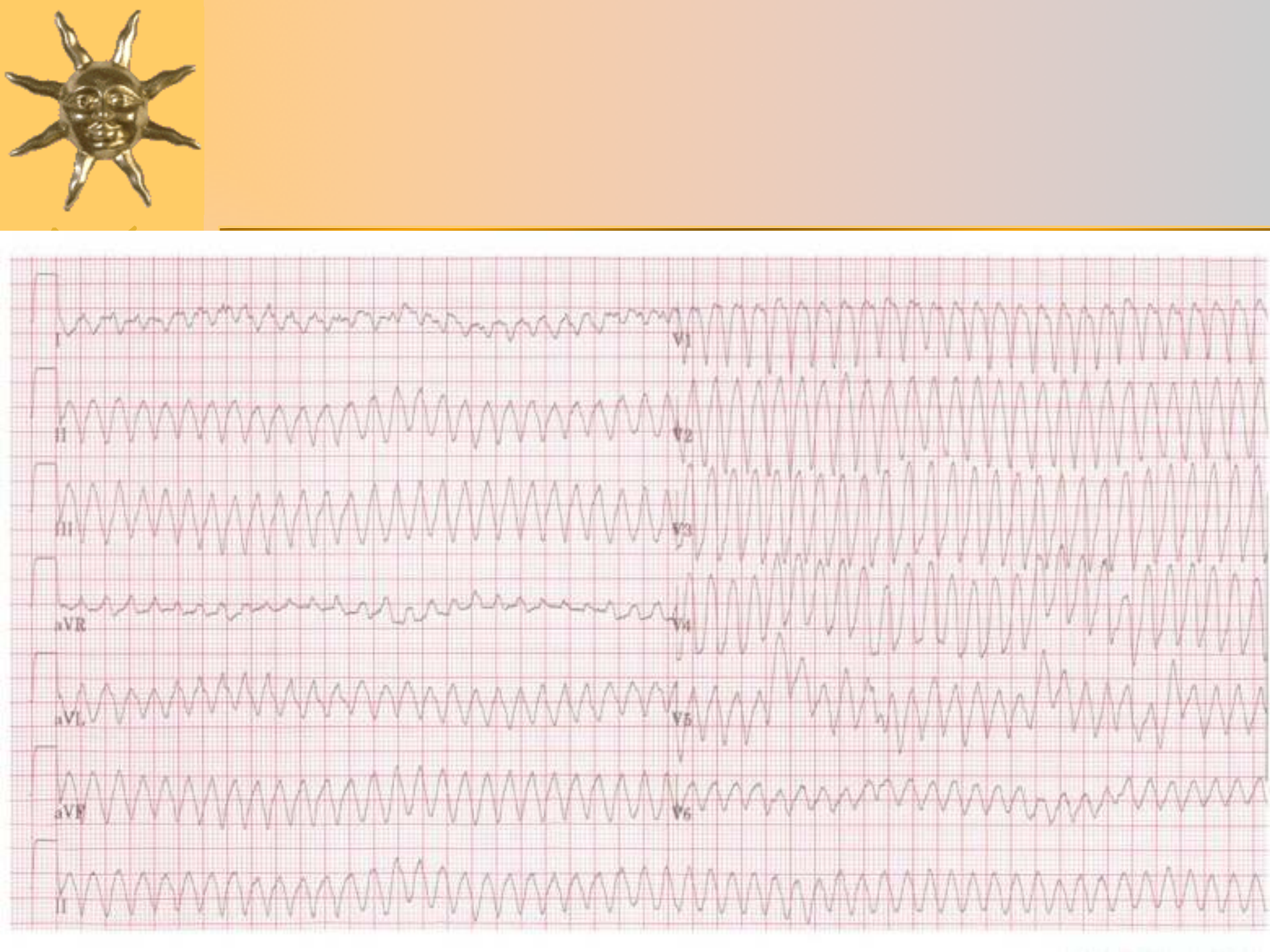
Torsades de Pointes



- "Twisting of the points" is usually caused by medication (quinidine, disopyramide, sotalol, TCA), hypokalemia or bradycardia especially after MI
- HR=200-250
- Has prolonged QT interval
- Begin wieh EAD,Continued with reentry
- Acute: Remove offending medication. Shorten the QT interval with **magnesium, lidocaine,phenytion isoproterenol, or temporary overdrive pacing(1B)**
- Chronic: may need pacemaker/ICD, amiodarone, beta-blockers

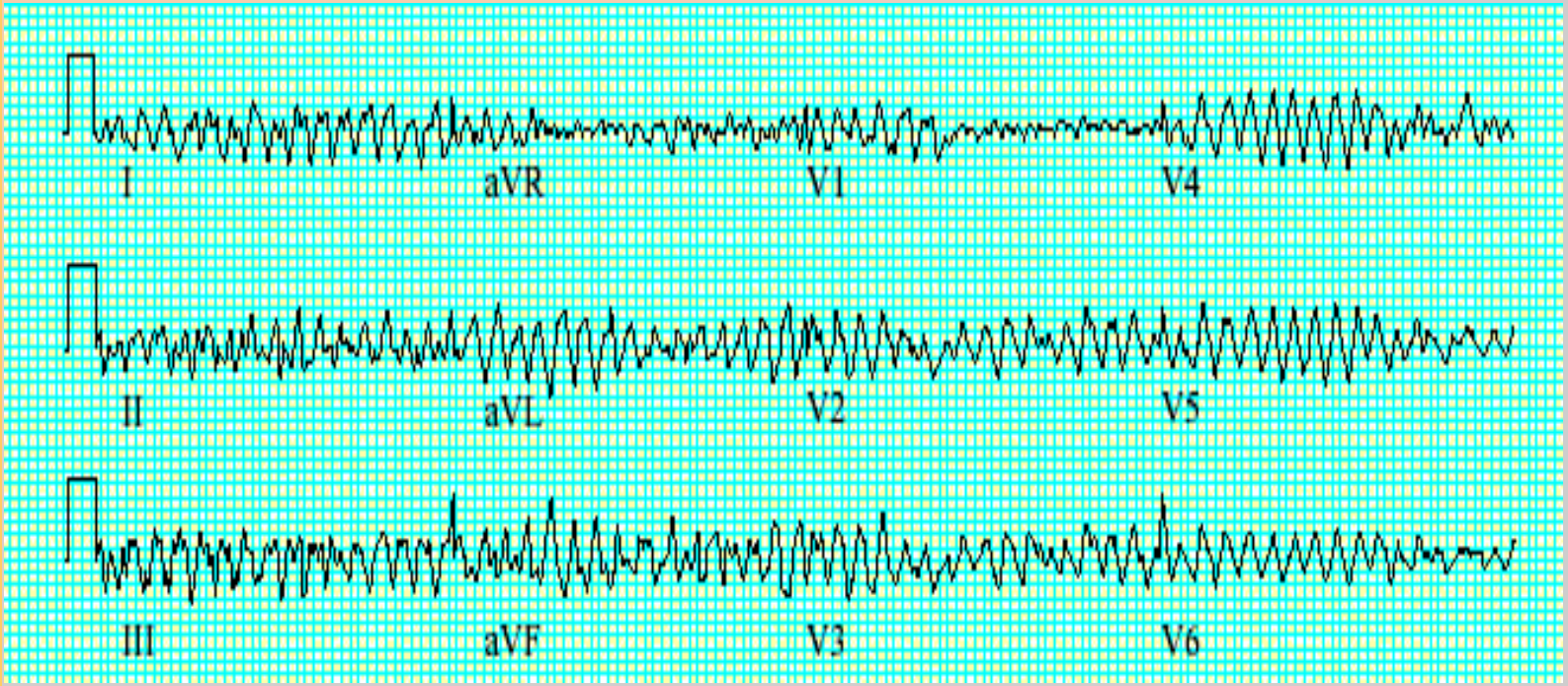
VFL

- HR=150-300
- نوسانات منظم بزرگ
- Ventricular flutter is mostly caused by re-entry with a frequency of 300 bpm. The ECG shows a typical sinusoidal pattern. During ventricular flutter the ventricles depolarize in a circular pattern, which prevents good function. Most often this results in a minimal cardiac output and subsequent ischemia. Often deteriorates into Ventricular Fibrillation.


I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
II

VF

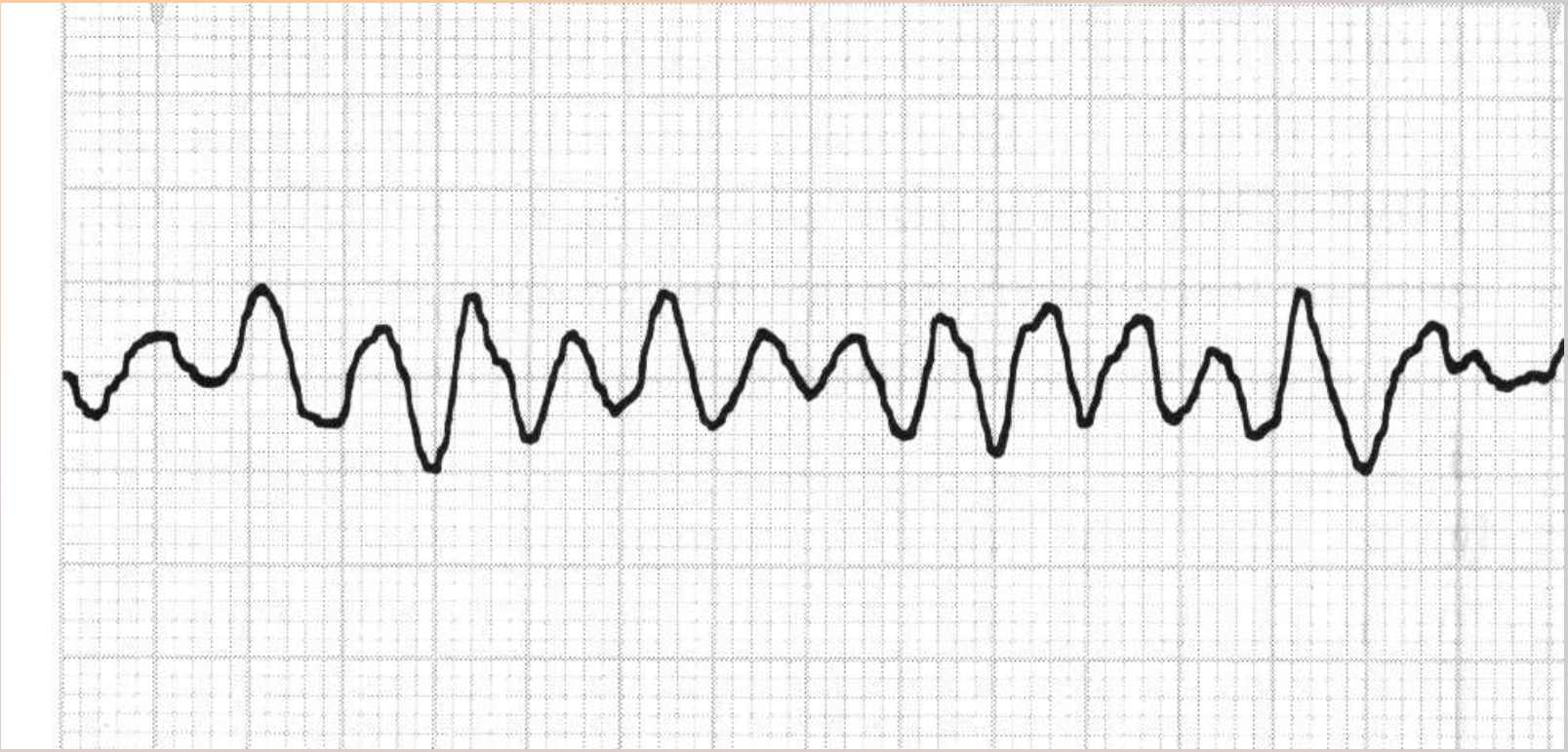
- نوسانات نامنظم باشکل وآمپلیتود متغیر
- در ۵-۳ دقیقه بیمار رامی کشد



Ventricular Fibrillation

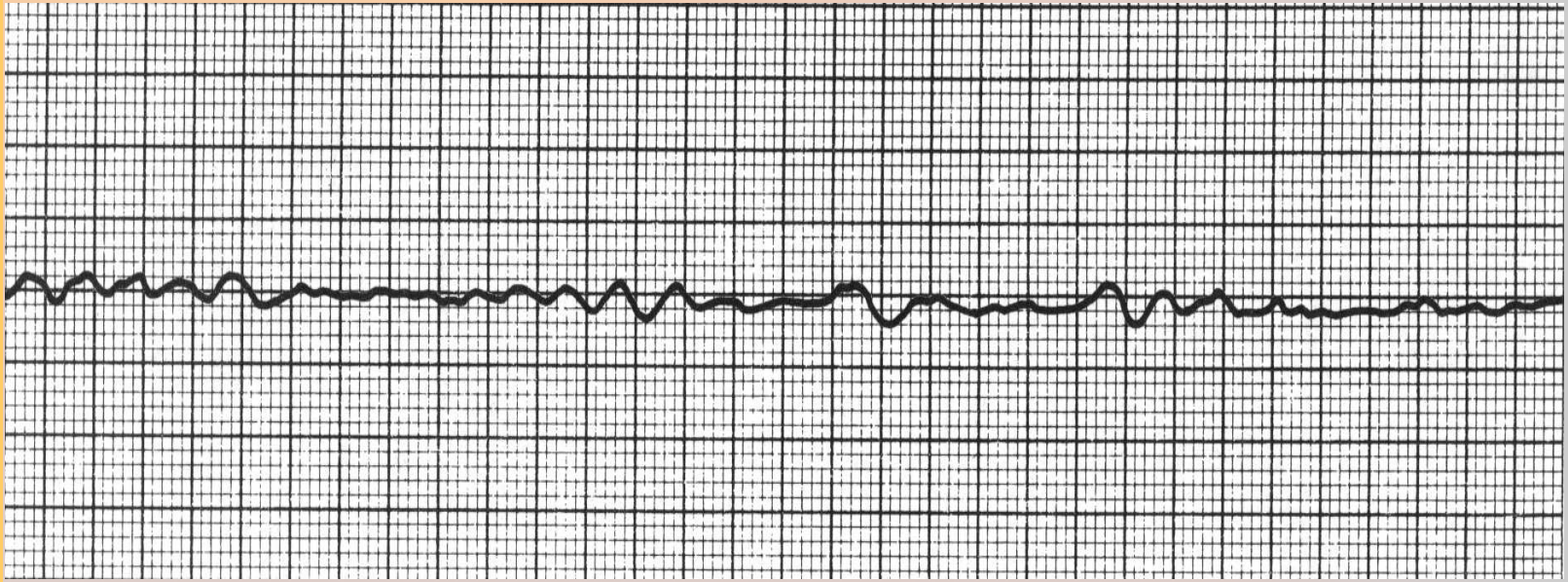
- Many ectopic foci firing at the same time
- There is no regular pattern as in Vtach
- *No effective cardiac output!*
- *Requires CPR and DC shock>200j async*
- Most common in acute MI, also drug overdose, anesthesia, hypothermia & electric shock
- Absence of ventricular complexes
- Usually terminal event
- Use Amiodarone if refractory to DCCV.

Vfib



This is “coarse” vfib

Vfib



This is “fine” vfib<0.2MV

Types Of Cardioversion "chemical" or "electrical".

Chemical cardioversion: antiarrhythmia

It can take several minutes to days for a successful cardioversion .

Electrical cardioversion :synchronized

- electrical shock.
- IV access
  - Airway management equipment
  - Sedative drugs (midazolam and fentanyl)
  - Cardioversion monitoring device

Also recommended in the following:

- Supraventricular tachycardia due to re-entry
- Atrial fibrillation
- Atrial flutter
- Atrial tachycardia
- Monomorphic VT with pulses

Contraindications

- **Digoxin toxicity**
- **Sinus tachycardia**
- **MAT**

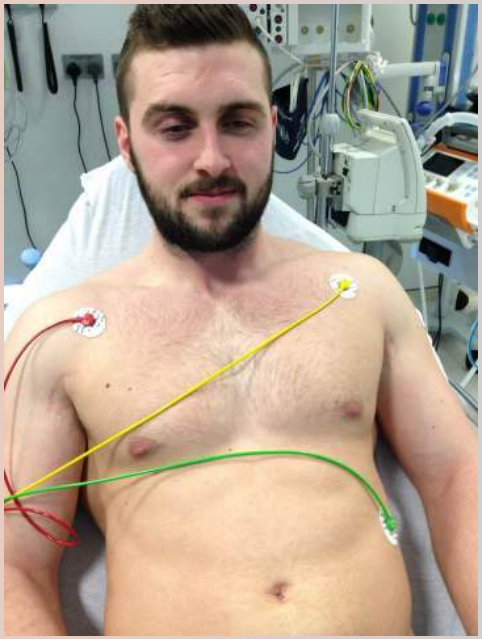
Preparing for a Cardioversion

- Do not eat or drink for at least **eight** hours
- Do not apply any lotions or ointments to chest
- Stop **digoxin before 48** hours prior the procedure.

Step 1

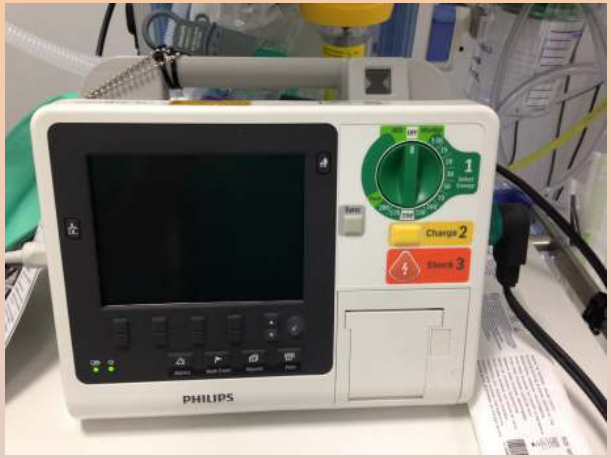
- Apply heart tracing leads

Leads: Red, Amber, Green (Traffic lights)



Step 2

- Turn the Cardioversion machine on



Off

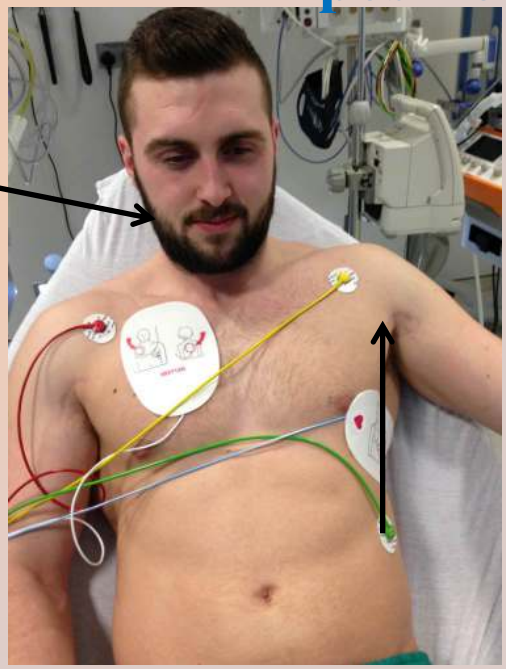
Twist green nozzle clockwise to activate

Step 3

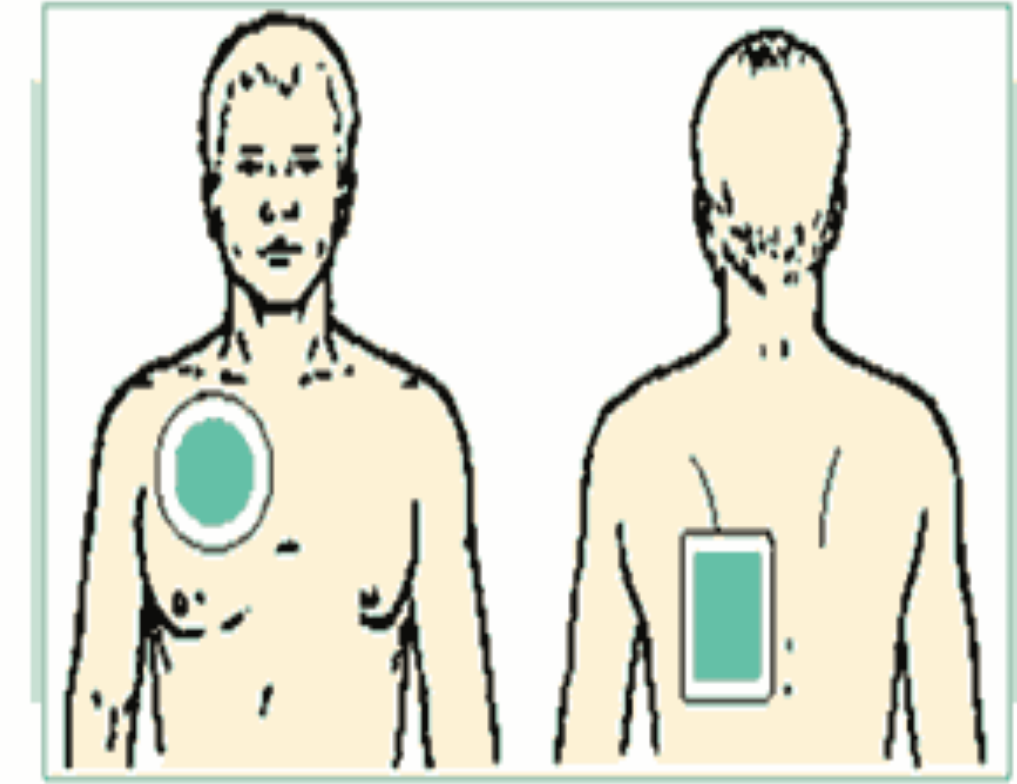
- Place on the pads

Anterolateral pad positioning

First pad: 2nd or 3rd intercostal space, Right Sternal edge



Second pad: 4th or 5th Intercostal space , Midaxillary line

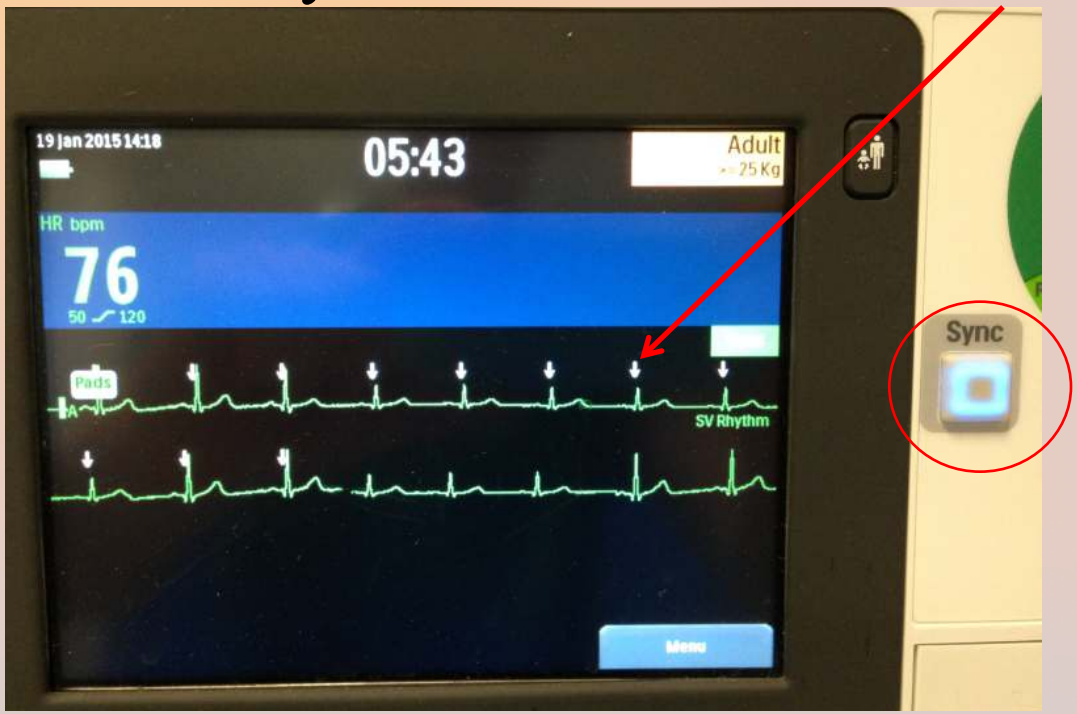


Step 4

- Check heart tracing is being picked up

Step 5

- Press Sync to locate R waves



R waves indicated by arrows

Step 6

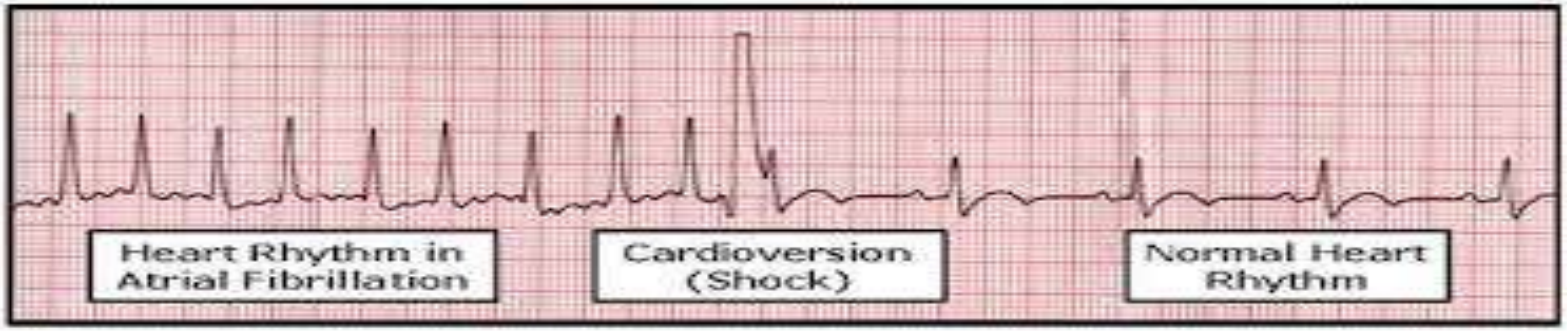
- Set the voltage as per guidelines on the condition presenting with the patient

Step 7

- **<u>WARN</u>** EVERYBODY AROUND THE BED YOU ARE ABOUT TO PRESS CHARGE AND SHOCK
- Everyone should step back from the patient and the bed

Step 8

- Press Charge
- Then Shock



- Watch the monitor and see if the Rhythm has returned to normal,If not, this may need to be repeated
- **Complications:uncommon**
- Worsening of the arrhythmias.
- Blood clots that can cause a stroke or other organ
- damage, bruising, burning or pain where the paddles were used.

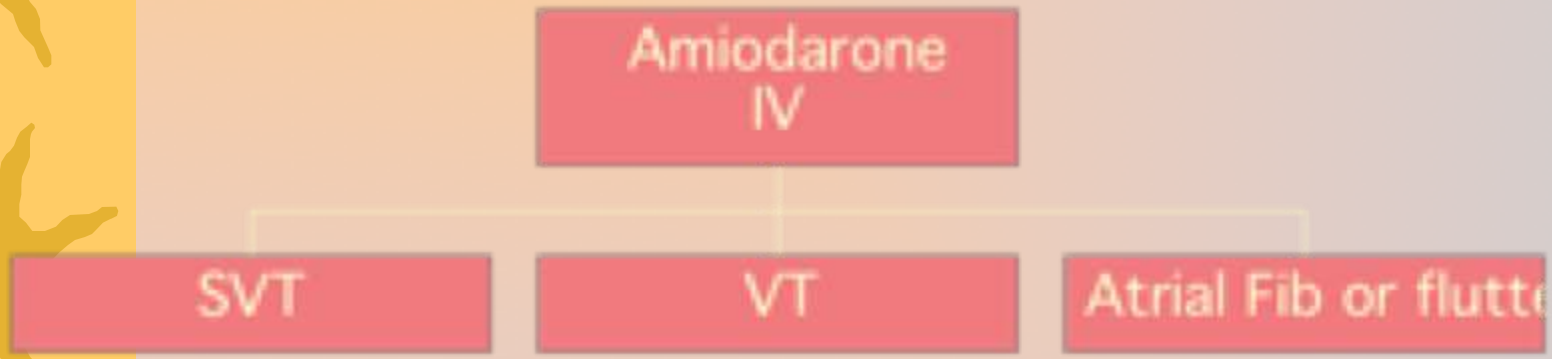
Defibrillation

medical technique used to counter the onset of **VF** ,and pulseless **ventricular tachycardia**,

Classification of Anti-arrhythmics

Class	Action	Examples	Side Effects
1A	Fast sodium channel blocker varies depolarization and action potential duration	Quinidine, procainamide, disopyramide	Class: nausea, vomiting Quinidine: hemolytic anemia, thrombocytopenia, tinnitus Procainamide: lupus
1B		Lidocaine, Mexiletine	Lidocaine: dizziness, confusion, seizures, coma Mexiletine: tremor, ataxia, rash
1C		Flecainide, Propafenone	Flecainide: pro-arrhythmia, nausea, dizzyness
2	beta-blockers ↓SA node & ↓AV node conduction	Propranolol, metoprolol	Class: CHF, bronchospasm, bradycardia, hypotension
3	Prolong action potential by blocking K+ channels	Amiodarone, sotalol	Amiodarone: hepatitis, pulmonary fibrosis, thyroid disorders, peripheral neuropathy Sotalol: bronchospasm
4	calcium channel blockers ↓AV node conduction	Verapamil, dilitiazem	Class: AV block, hypotension, bradycardia, constipation

When in doubt…Amiodarone



Is more effective in high HR
Low TDP risk

Magnesium indications.

- 1. **TDP** from any reason.
- 2. Arrhythmias in a patient with known **hypomagnesaemia.**
- 3. Consider its use in acute ischaemia to prevent early ventricular arrhythmias.
- 4. Digoxin induced arrhythmias.

bradicardia

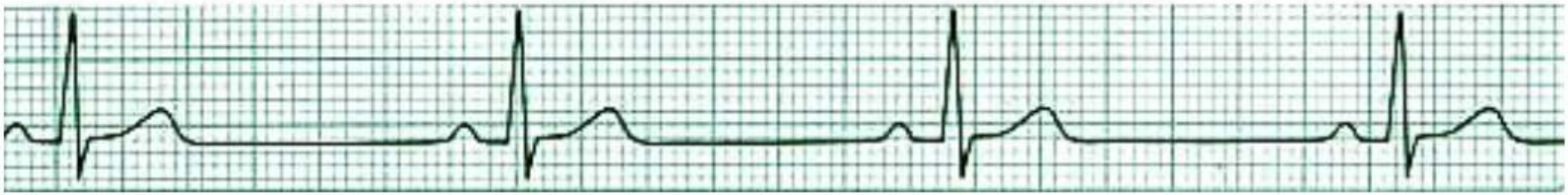
- Sinus node dysfunction * sick sinus syndrome
  - Sinus bradycardia
  - Sino-atrial block (SA block)
  - Sinus pause, Sinus arrest
  - Tachycardia bradycardia syndrome

- Atrio-Ventricular block (AV block)
  - 1st degree AV block
  - 2nd degree AV block (Mobitz type I, II)
  - 3rd degree AV block (complete AV block)

Sinus bradycardia

- Sinus rhythm at a rate less than 60bpm
- excessive vagal- decreased sympathetic tone - anatomic changes in sinus node
- Frequently occurs in healthy young adults,particularly well-trained athletes
- Sinus arrhythmia often coexists

Sinus bradycardia



Heart Rate	Rhythm	P Wave	PR interval (s)	QRS (s)
<60 bpm	Regular	Present before each QRS, identical	Normal, consistent (0.12 to 0.20)	Normal (< 0.12)

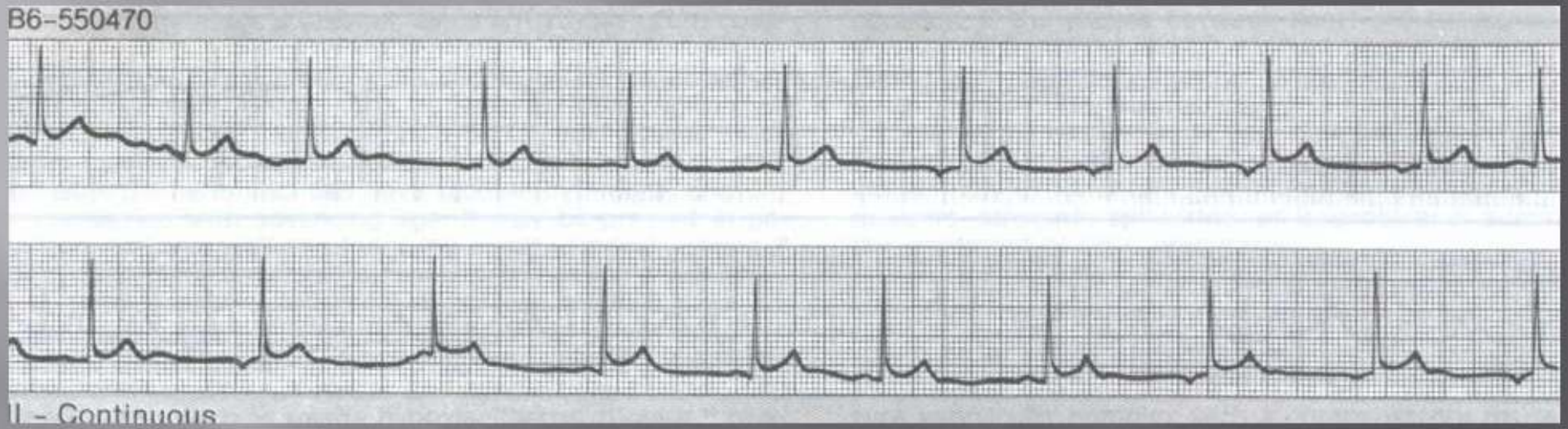
Inappropriate sinus bradycardia
Chronotropic Incompetence

- HR < 60 that doesn't increase appropriately with exercise
- Usually defined as failure to attain 80% of maximal age predicted HR (MAHR) on exercise testing
- MAHR = 220-Age

Wandering pacemaker

- Passive transfer of dominant pacemaker focus from sinus node to latent pacemakers in other atrial sites or AV junctional tissue
- Occurs in a gradual fashion over the duration of several beats
- Cyclic increasing RR,dec PR
- Normal variations,athletes

Wandering Pacemaker



Sinus Arrhythmia

- Phasic variation in sinus CL
- Maximum -minimumsinusCL>120msec
- (max-min/min>10%)
- PR>200
- May be considered the most common form
- Of arrhythmia
- Respiratory form is a normal event-dig toxicity
- Common in the young with slower HR or enhanced vagal tone

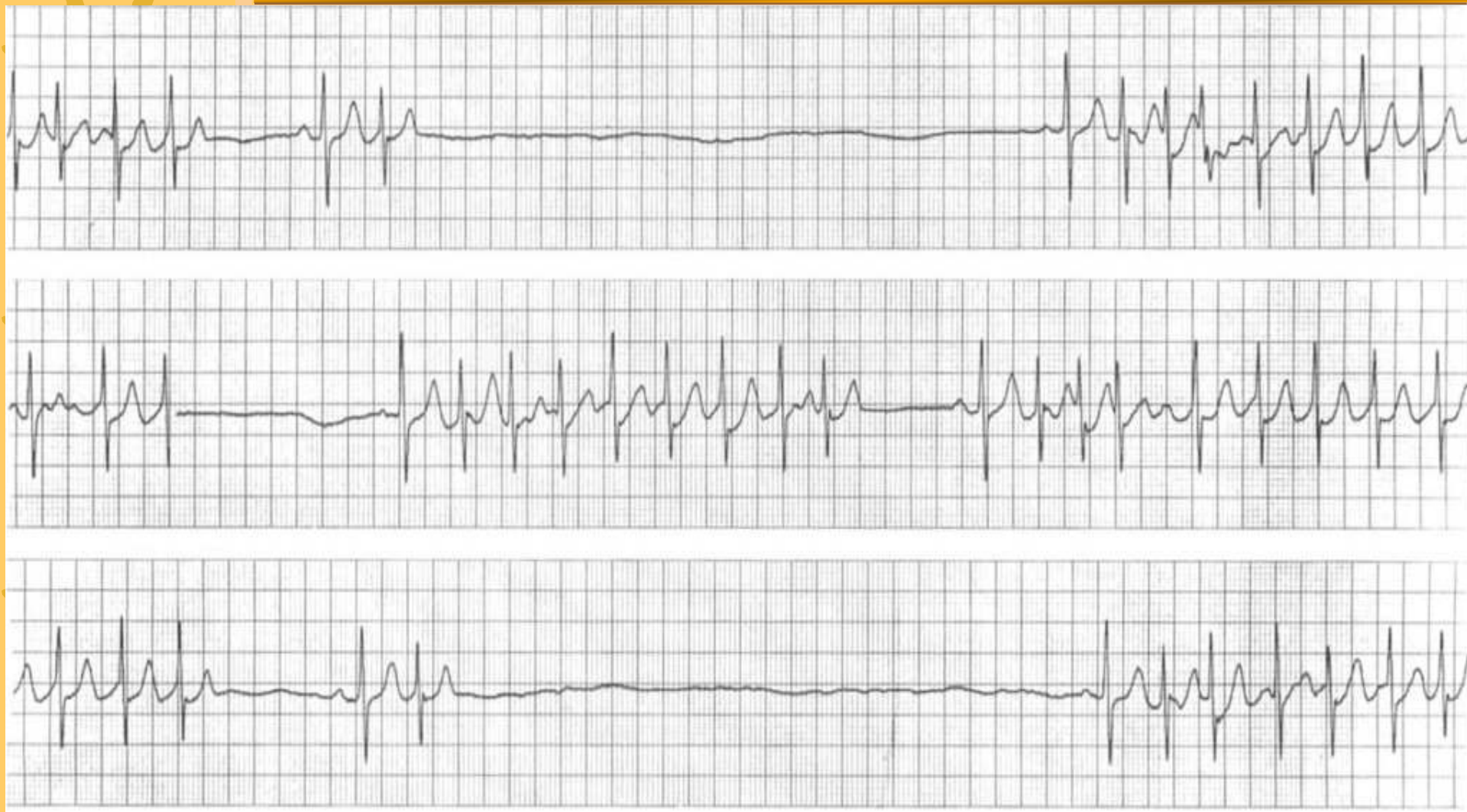
SAN DISEASE

- EXTRINSIC:hypo T,sleep apnea,hypotermia,hypoxia,IICP,endotracheal suction(vagus stimulation)
- INTRINSIC:CAD,pericarditis,myocardiris,RHD, amyloidosis,iatrogenic,HTN,DM,VHD,CMP, genetic(sss)

Sick Sinus Syndrome

- Persistant spontaneous S.B
- SA exit block or sinus arrest
- AVN disease+SAN disease
- Repetitive tachy-brady
- High incidence of AF
- Conduction problem with no junctional escape during sinus pause
- Diagnose with ECG or Holter. If inconclusive, need electrophysiologic testing.
- If asymptomatic, leave alone. If symptomatic, needs pacemaker.

Sick Sinus Syndrome



- The sinus impulse is blocked within the SA junction (between SA node and atrial myocardium)

- 3 types of SA block

1. First-degree
2. Second-degree : type I, type II
3. Third-degree

S Denotes discharge of impulse from sinoatrial node

Normal sinoatrial conduction

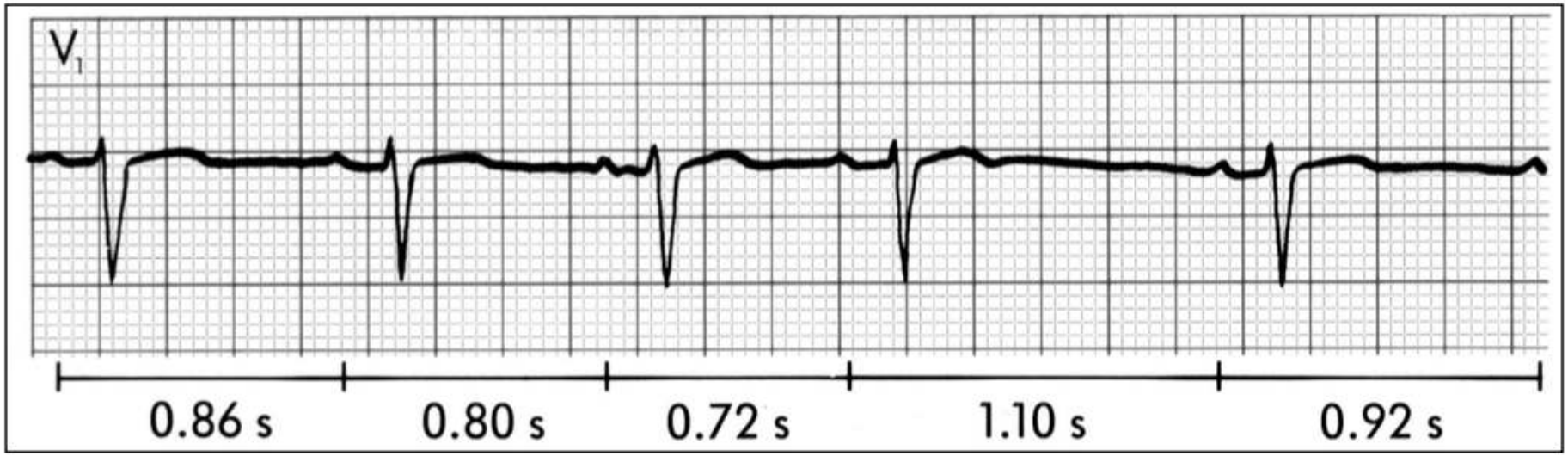
First-degree sinoatrial block

Note the delay from discharge of sinus impulse to beginning of atrial depolarization (P-wave). The delay is constant. Because the activity of the sinoatrial node is not noticeable on the ECG, the ECG will appear fully normal. First-degree sinoatrial block can therefore not be diagnosed with surface ECG (invasive examination is necessary).

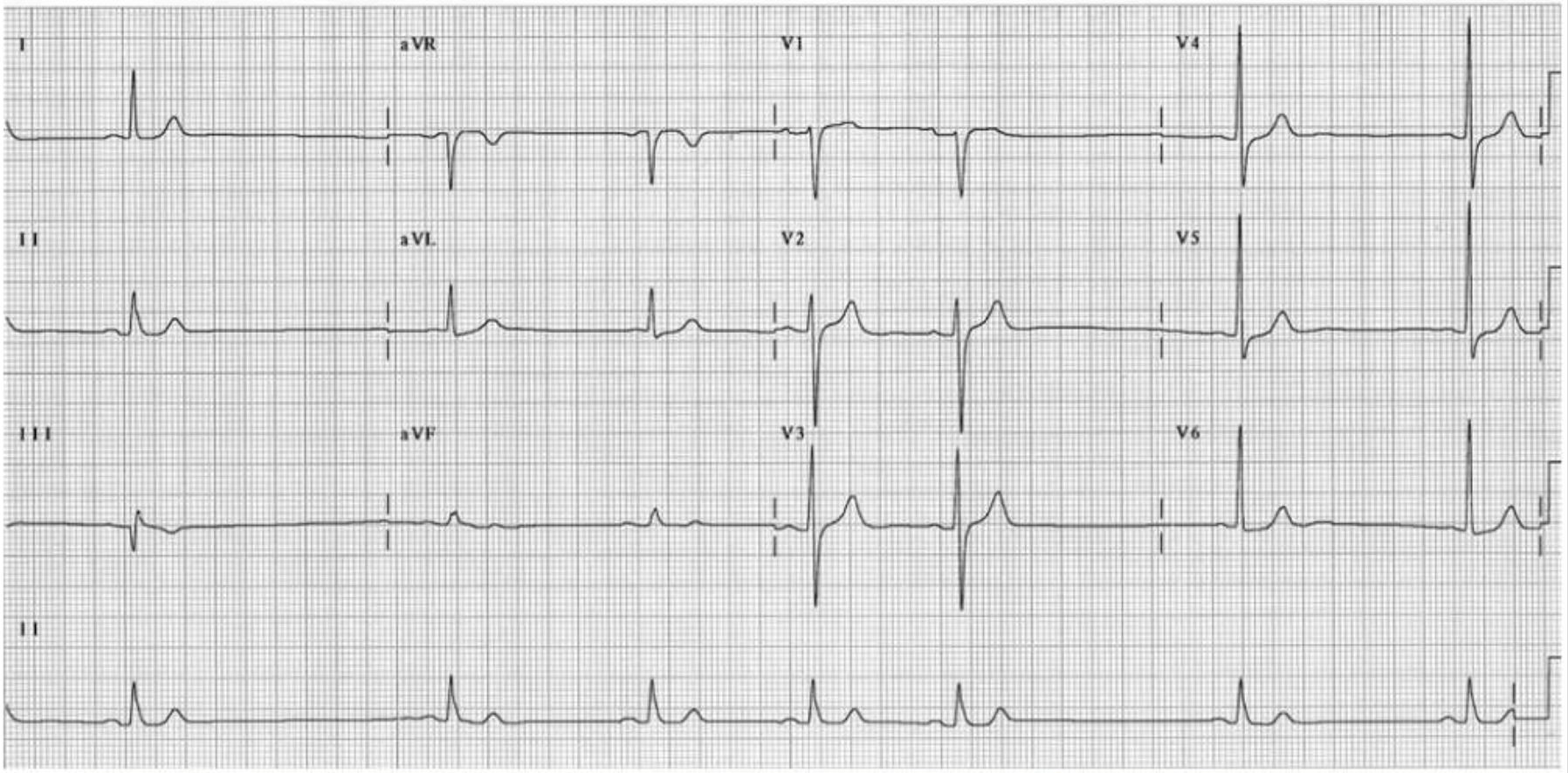
Figure 1. Upper tracing shows normal impulse conduction from the sinoatrial node to the atria. Atrial activation commences almost immediately after discharge of the impulse in the sinoatrial node. Lower tracing shows first-degree sinoatrial block, in which the time interval from impulse discharge to atrial activation is prolonged and this cannot be discerned on the surface ECG. As seen here, the rhythm is still regular and all complexes appear normal.

2nd degree Sinoatrial block, type I

- ✓ PP cycle becomes progressively shorter
- ✓ No P waves & QRS complexes
- ✓ Pause is less than twice the preceding PP cycle

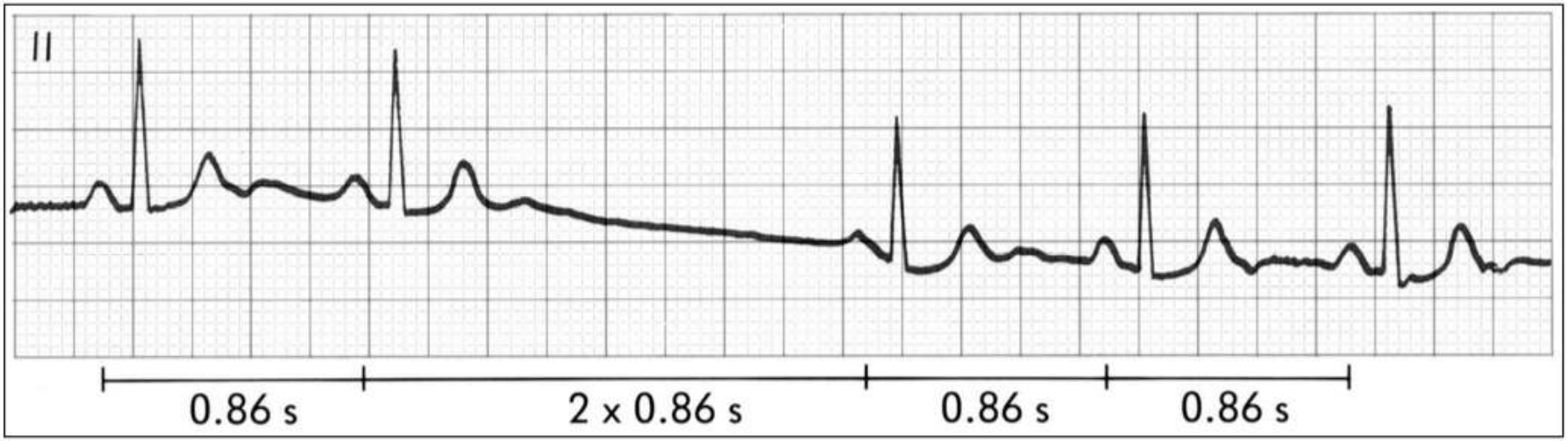


2nd degree Sinoatrial block, type I

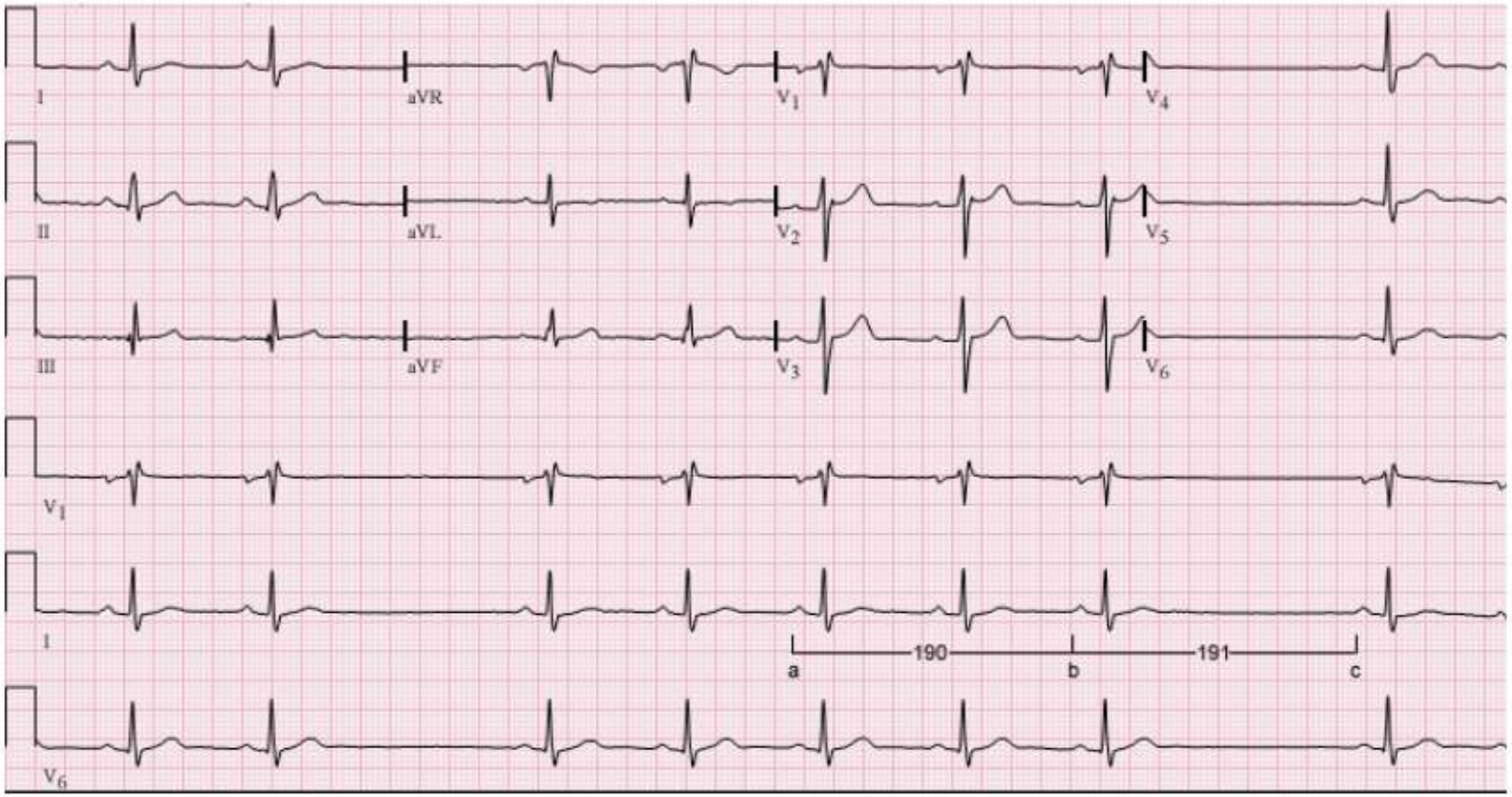


2nd degree Sinoatrial block, type II

- ✓ PP cycle is constant
- ✓ No P waves & QRS complexes
- ✓ Pause is twice the preceding PP cycle



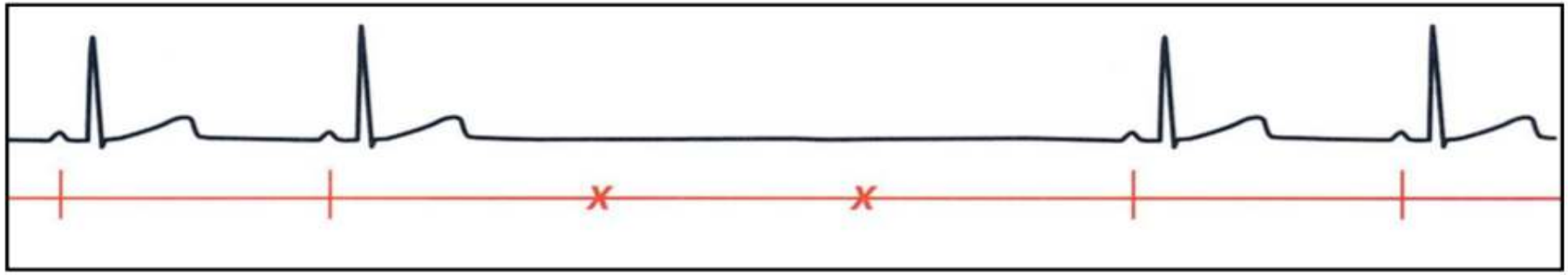
2nd degree Sinoatrial block, type II



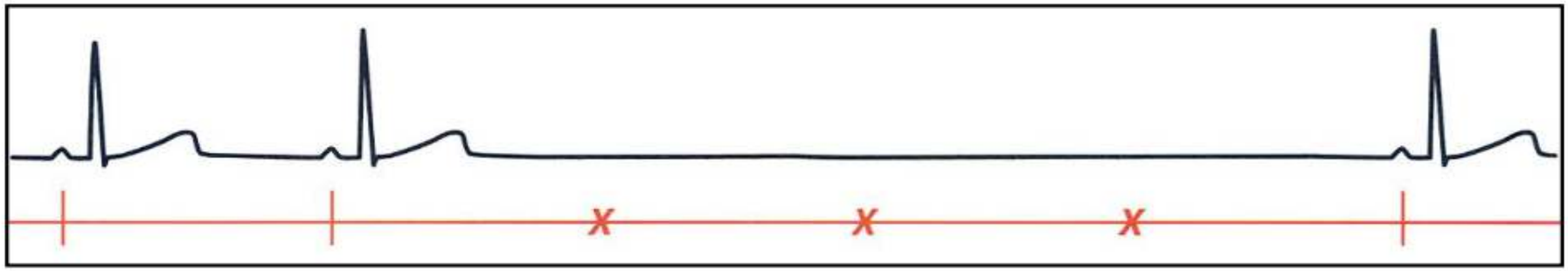
Third degree SA exit block

- There is a complete absence of P waves.
- Failure to conduct impulses to RA
- The onset of 3rd degree SA block may produce long sinus pauses or sinus arrest (may lead to fatal asystole).
- Rhythm may be maintained by a junctional escape rhythm.
- is indistinguishable from sinus arrest due to pacemaker cell failure. It can only be diagnosed with a sinus node electrode during EPS

- Multiple of the PP interval



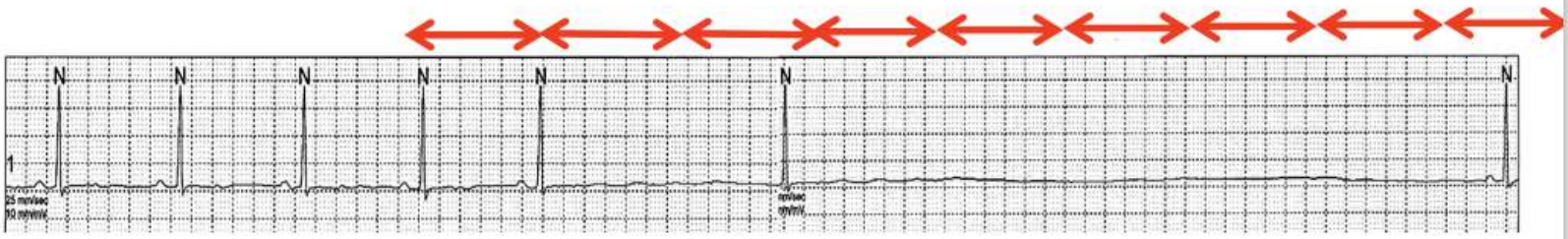
SA block 3 times the normal PP interval



SA block 4 times the normal PP interval

Sinus arrest:failure to form impulse

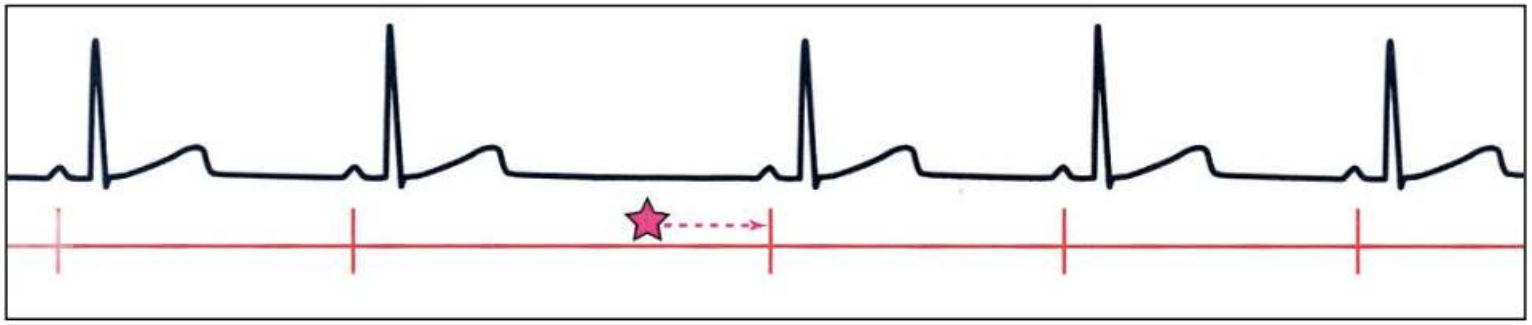
Sinus pause, arrest



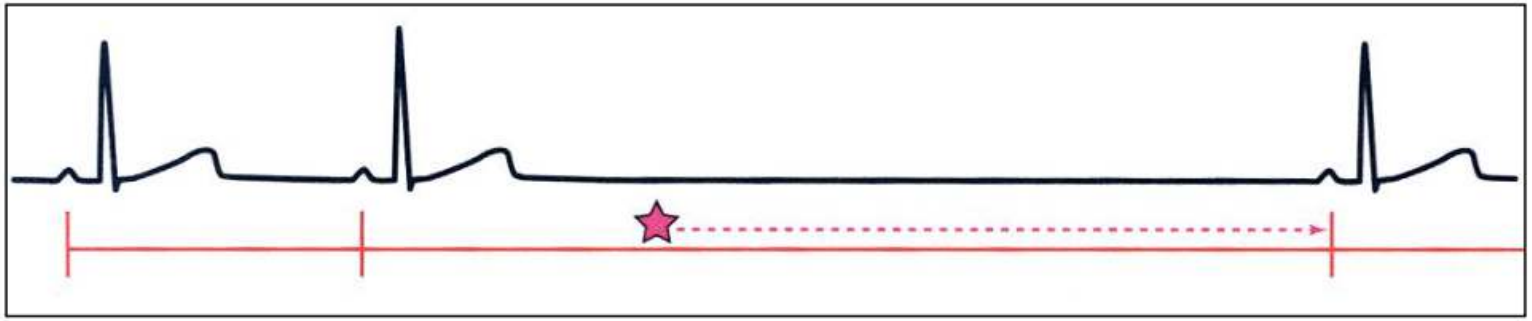
Heart Rate	Rhythm	P Wave	PR interval (s)	QRS (s)
N/A	irregular	Each QRS identical. New rhythm begins after a pause. The P to P interval is disturbed.	Normal (0.12 to 0.20)	Normal (< 0.12)

- The P-P interval during the pause is **not a multiple** of the P-P interval of the underlying rhythm

Sinus pause, arrest

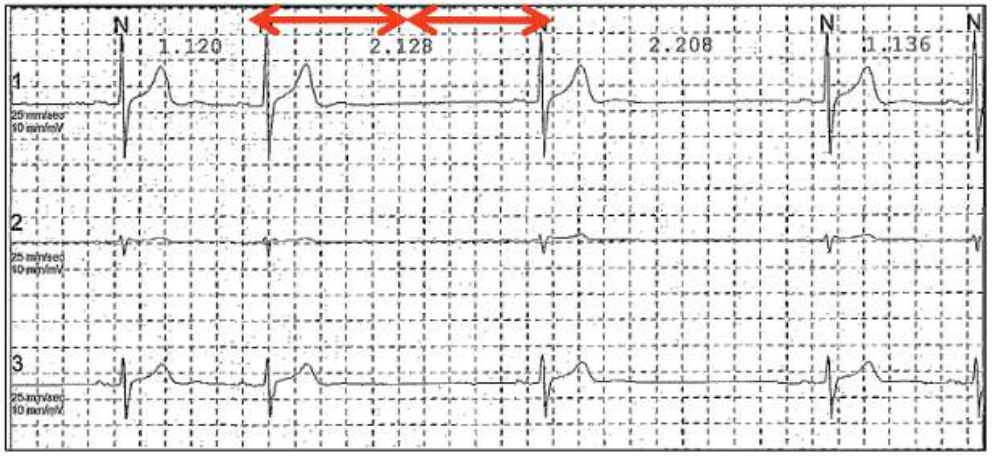


Sinus pause is not a multiple of the PP interval



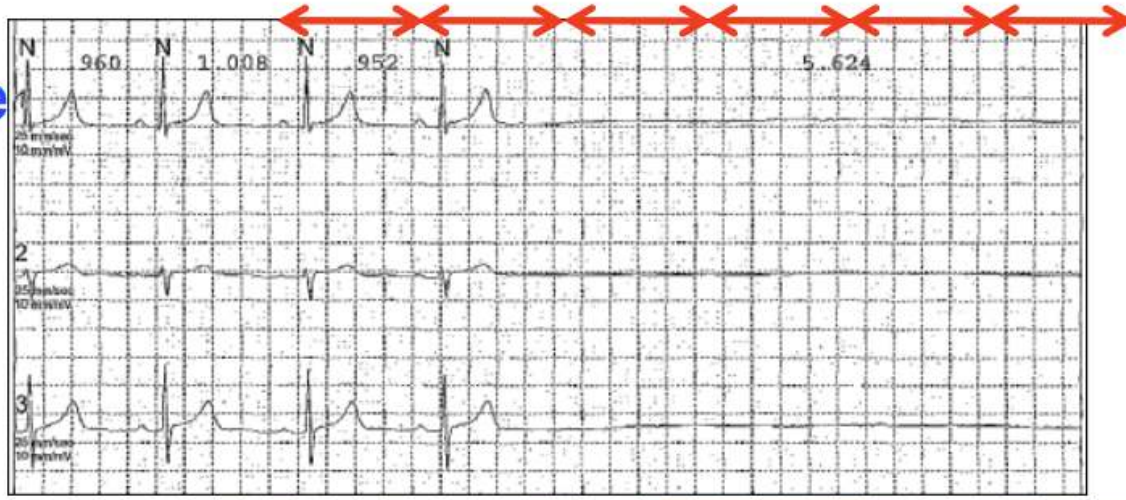
Sinus arrest is longer than a sinus pause
and is not a multiple of the PP interval

Sinus arrest with junctional escape beat

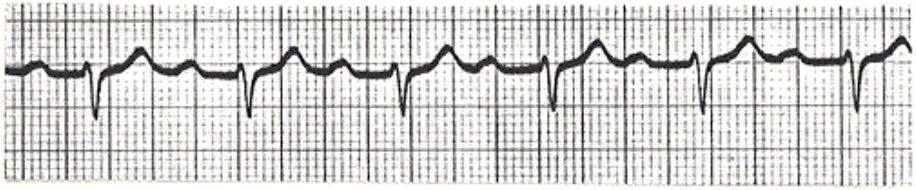


Sinus pause

Long sinus pause /sinus arrest



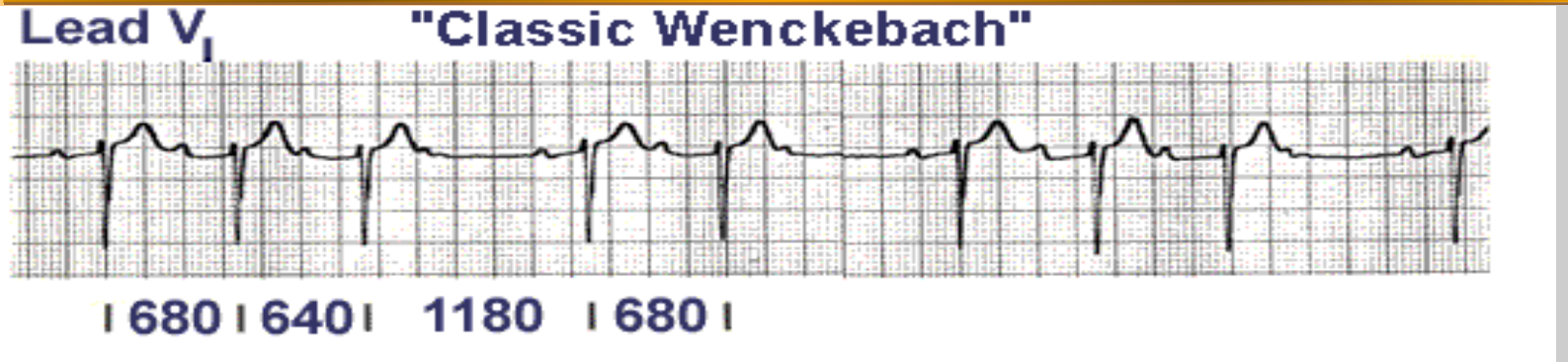
First Degree AV Block



1st degree AV block (PR = 280 ms)

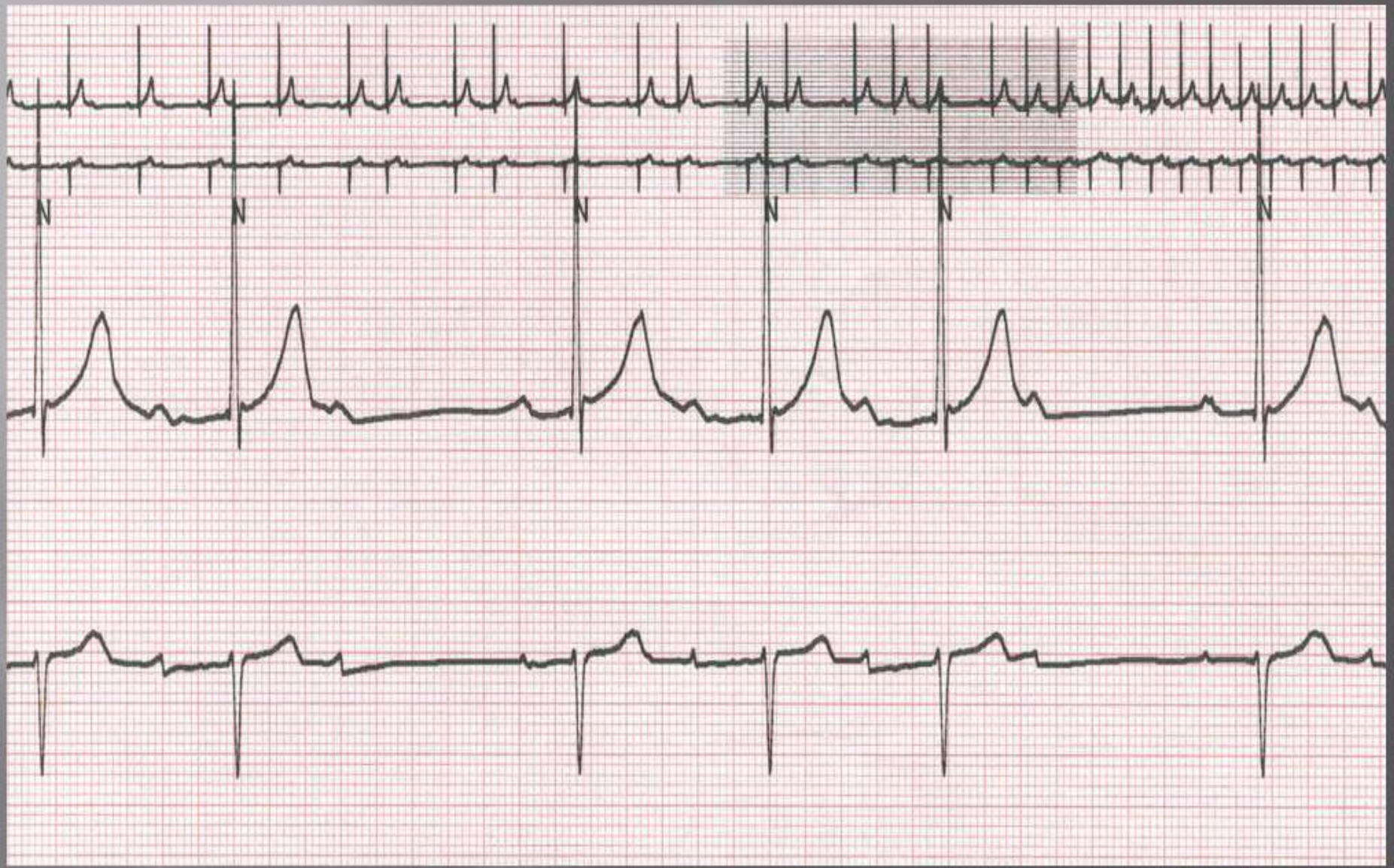
- PR interval=atrium+AV+HB
- INC PR=more common AVB
- Delay at the AV node results in prolonged PR interval
- PR interval>0.2 sec.
- Leave it alone

Second Degree AV Block Type 1 (Wenckebach)

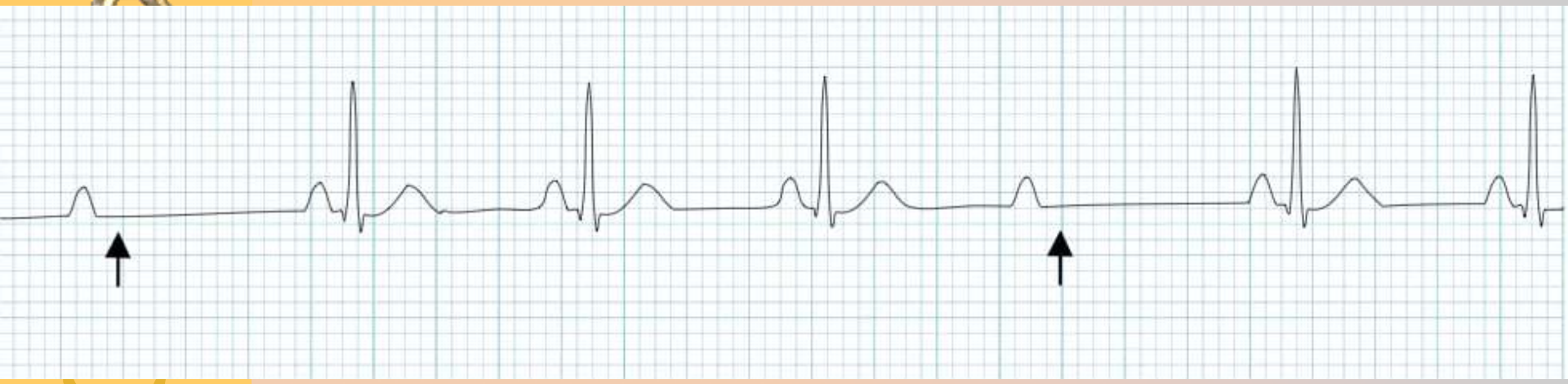


- Increasing delay at AV node until a p wave is not conducted.
- Often comes post inferior MI with AV node ischemia
- Gradual prolongation of the PR interval before a skipped QRS. QRS are normal! In most cases
- Generally benign and does not advance to more advanced AV block
- NL QRS=block in AV OR PROX HIS
- Can occur in normal children and well-trained athletes
- No pacing as long as no bradycardia.

Wenckebach Block



Second Degree AV Block Type 2



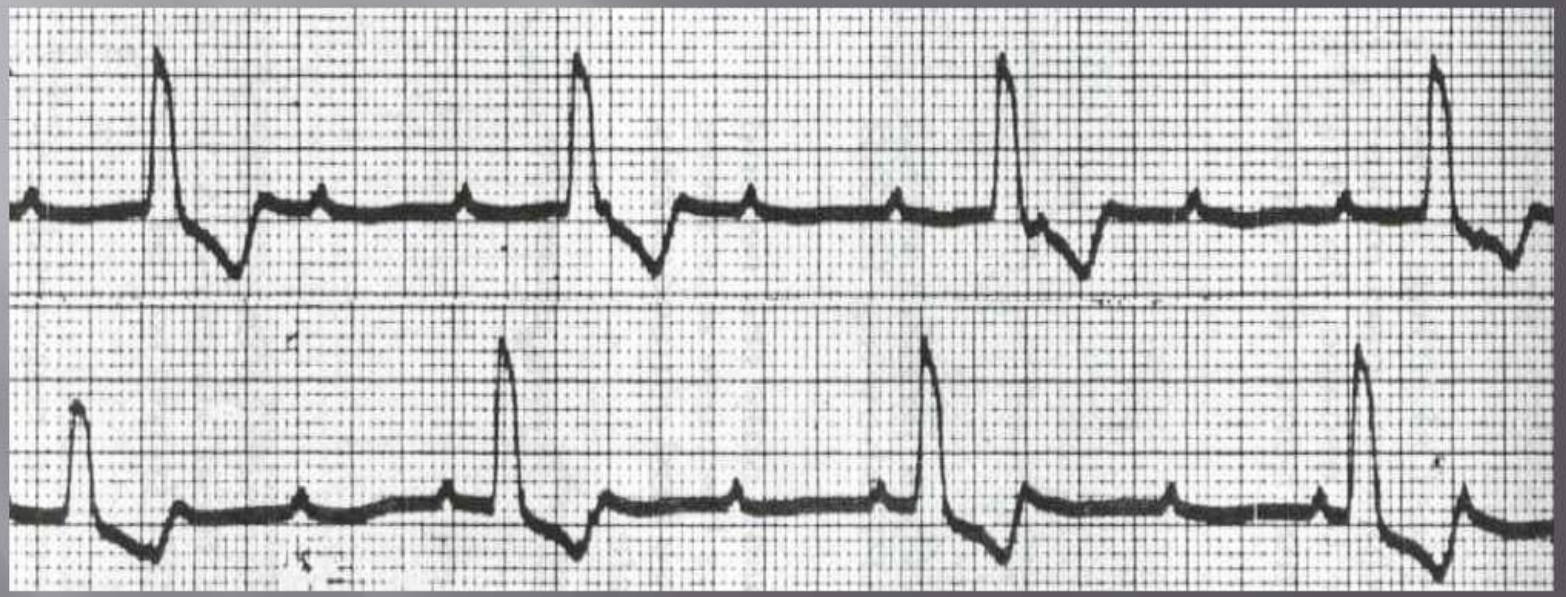
- The RR interval surrounding the dropped beat(s) is an exact multiple of the preceding RR interval
- usually due to failure of conduction at the level of the HP (i.e. below the AV node)
- In around 75% of cases, the conduction block is located *distal to the Bundle of His*, producing *broad QRS complexes*.
- In the remaining 25% of cases, the conduction block is located within the His Bundle itself, producing narrow QRS complexes.
- **Sudden loss of a QRS wave because p wave was not transmitted beyond AV node. May be precursor to CHB and needs pacing.**

Complete heart block

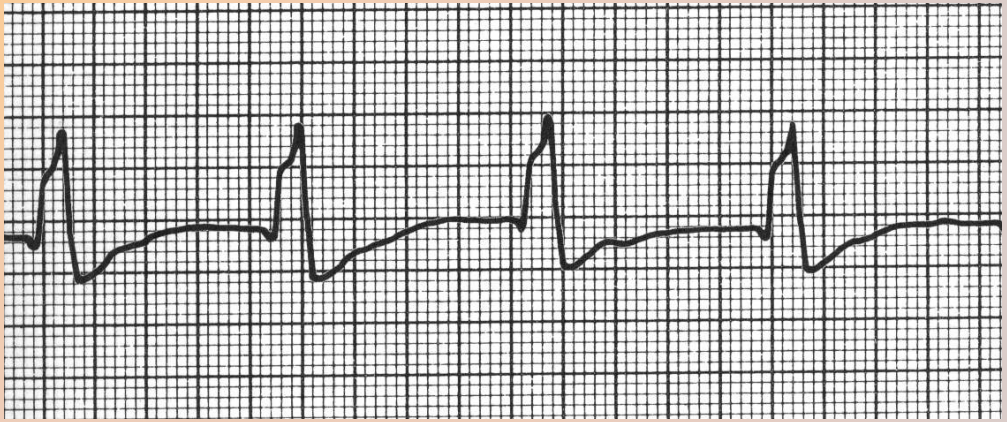
- atria and ventricles beat independent & atria beat faster than ventricles.
- no atrial activity conducts to ventricles
- AV dissocoation
- Ventriculat focus is usually located just below the site of block
- Higher sites are more stable with a more faster escape rate
- Must treat with pacemaker.
- Atropin:worse block under AV

Third Degree AV Block

Complete AV block
Isorhythmic AV Dissociation

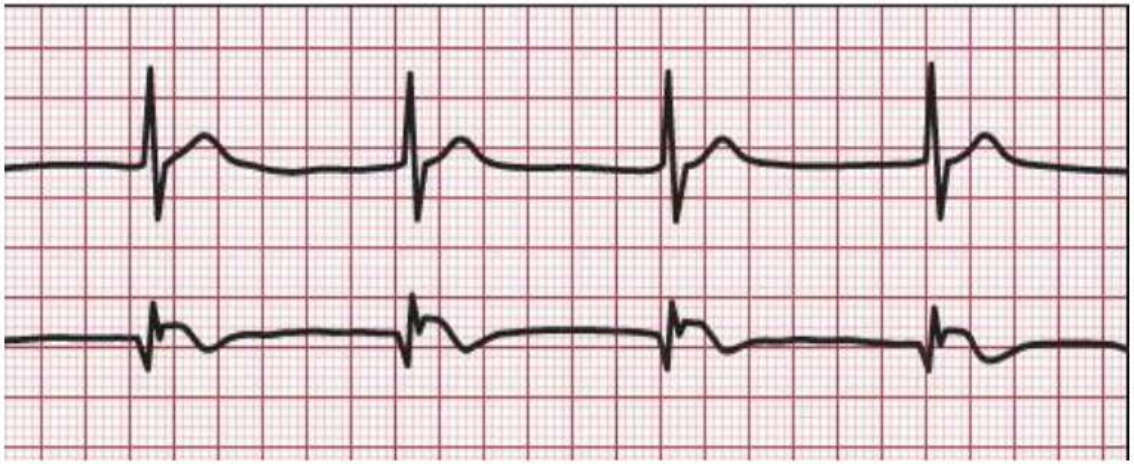


Idioventricular Rhythm



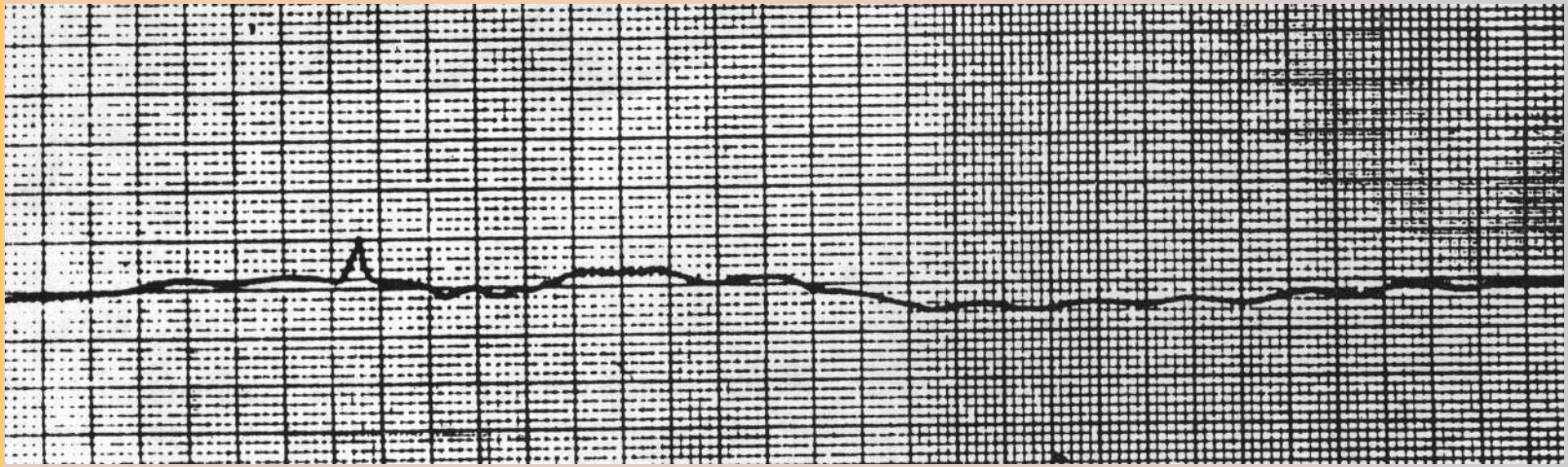
- Ventricles depolarizing on their own because of no conduction from above
  - Rate will be between 20-40
- A rate of 60-120 (all PVCs) is sometimes called “Slow Vtach”

Junctional Rhythm



Heart Rate	Rhythm	P Wave	PR interval (s)	QRS (s)
40-60 bpm	regular	Variable (none, antegrade, or retrograde)	None, short or retrograde (<0.12)	Normal (< 0.12)

Agonal Rhythm Leading to Ventricular Standstill (Asystole)



agonal heart rhythm

- It is a variant of asystole. Agonal heart rhythm is usually ventricular in origin. Occasional P waves and QRS complexes can be seen . The complexes tend to be wide and bizarre in morphological appearance.an agonal rhythm is regarded as asystole and should be treated equivalently, with CPR and administration of adrenaline. As in asystole, the prognosis is very poor. Sometimes this appears after asystole or after a failed resuscitation attempt.

Who gets a pacemaker?

- ♥ Syncope, presyncope or exercise intolerance that can be attributed to bradycardia
- ♥ Symptomatic 2nd or 3rd degree AV block
- ♥ Congenital 3rd degree AV block with wide QRS
- ♥ Advanced AV block after cardiac surgery
- ♥ Recurrent type 2 2nd degree AV block after MI
- ♥ 3rd degree AV block with wide QRS or BBB.

THANK YOU