

Common cardiac arrhythmia

From mechanisms to clinical presentations

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Rhythms included in this lecture

- Sinus rhythm, sinus tachycardia
- Atrial fibrillation
- Paroxysmal supraventricular tachycardia
- Premature ventricular complex
- Ventricular tachycardia
- Ventricular fibrillation

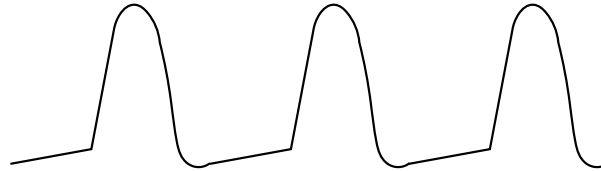
(esp. delayed afterdepolarisation)

Mechanisms of cardiac rhythm

arrhythmia with **increased** rate

Automaticity

- Normal/enhanced automaticity
- Abnormal automaticity

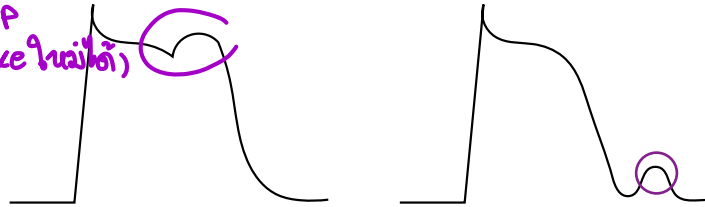


impulse initiation/formation

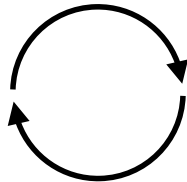
Triggered activity

- Early afterdepolarization
- Delay afterdepolarization

Rebound of AP
(ถ้าสูงพอ เกิด spike ในใจได้)



Reentry



impulse conduction

Automaticity of sinus node

~ pacemaker AP 104 (phase 4)

อัตราการเต้นหัวใจ

มีหน้าที่ปรับอัตราการเต้นหัวใจ

① HCN channel (I_f / funny current): manipulate by ANS (sym/parasymp)

② Ca^{2+} channel (T-type): influx $\uparrow$ = rate $\uparrow$

⊕
rate $\uparrow$

⊖

Autonomic influences changing automaticity rate

sym: rate $\uparrow$ para: rate $\downarrow$

can OA, Epi, NE

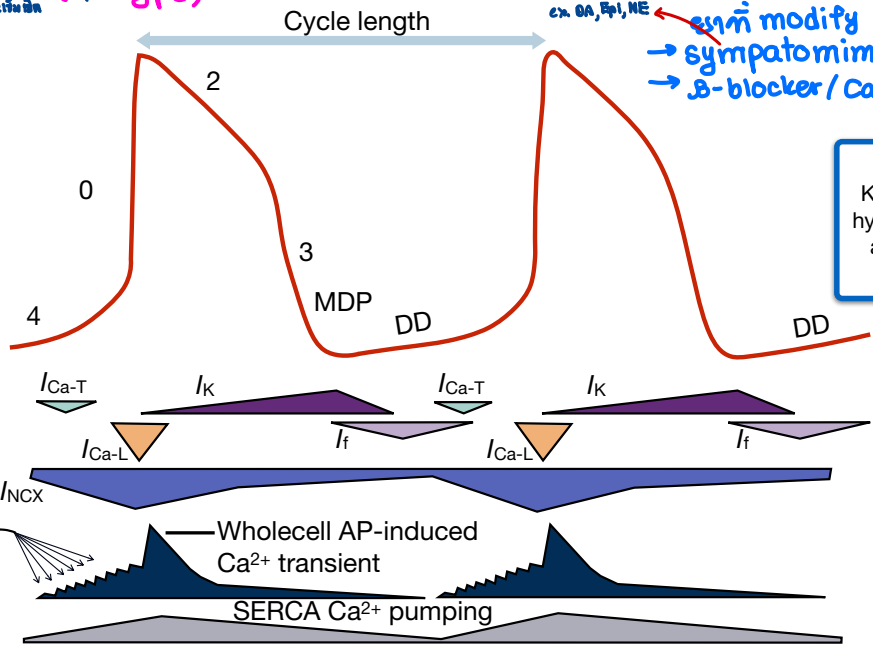
symp modify autonomic tone
→ sympathomimetic / lytic
→ β -blocker / Ca^{2+} -blocker → hr $\downarrow$

Change in resting membrane potential

Membrane potential

Membrane clock

Calcium clock



$I_{K_{ACh}}$ allow K efflux, induce hyperpolarization and decrease heart rate

β receptor activate cAMP, enhance HCN channel (I_f)

M2 receptor inhibit cAMP, inhibit HCN channel (I_f)

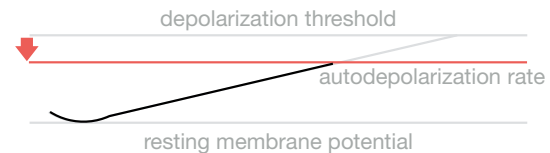
β receptor activate cAMP, enhance I_{CaL}

M2 receptor inhibit cAMP, inhibit I_{CaL}

depolarization rate (slope)

depolarization threshold

MDP = maximum diastolic potential; DD = diastolic depolarization; I_{Ca2T} = T-type voltage-dependent Ca²⁺ current; I_{Ca2L} = L-type voltage-dependent Ca²⁺ current; I_{NCX} = sodium-calcium exchange current; I_{K2} = delayed rectifier potassium current; I_f = funny current; SERCA = sarco-endoplasmic reticulum ATPase; LCRs = local Ca²⁺ releases



SA node

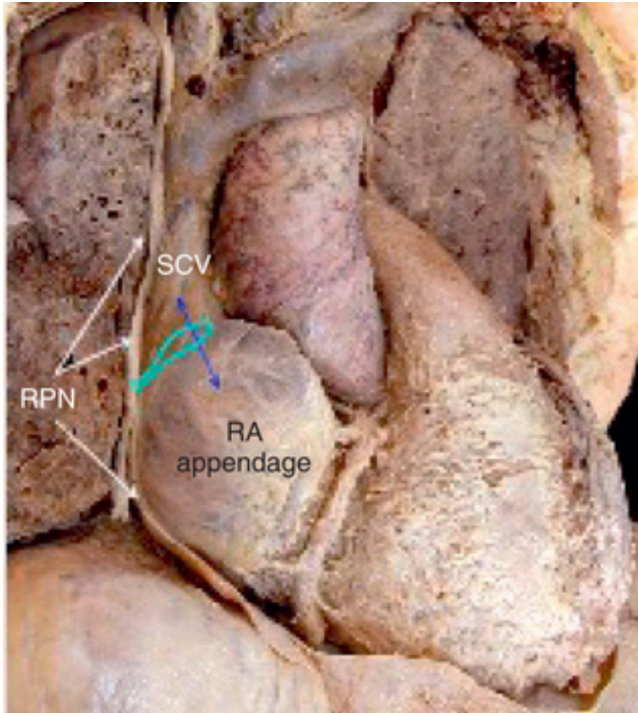
Blood supply

• SVC & RA junction

- Sinus nodal artery is a branch of the right coronary artery in 55% to 60% of patients and from the circumflex artery in 40% to 45%
- Distention of the artery slows the sinus rate, whereas collapse causes an increase in sinus rate *ischemia, metabolic disturbance → affect hr ↑/↓*

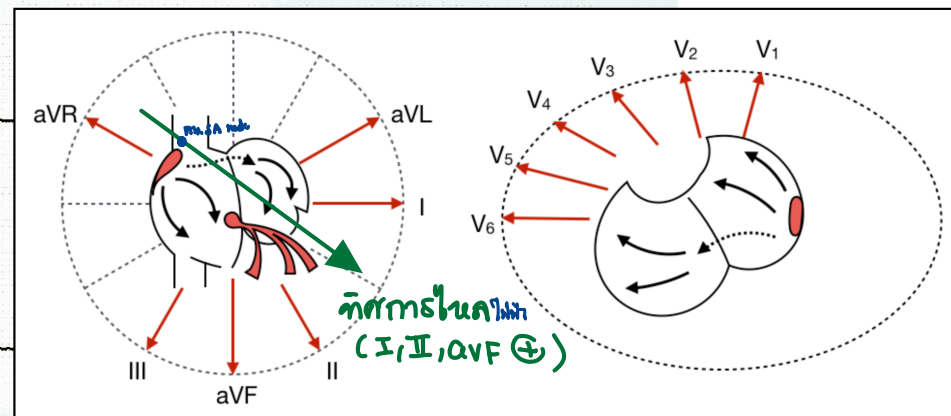
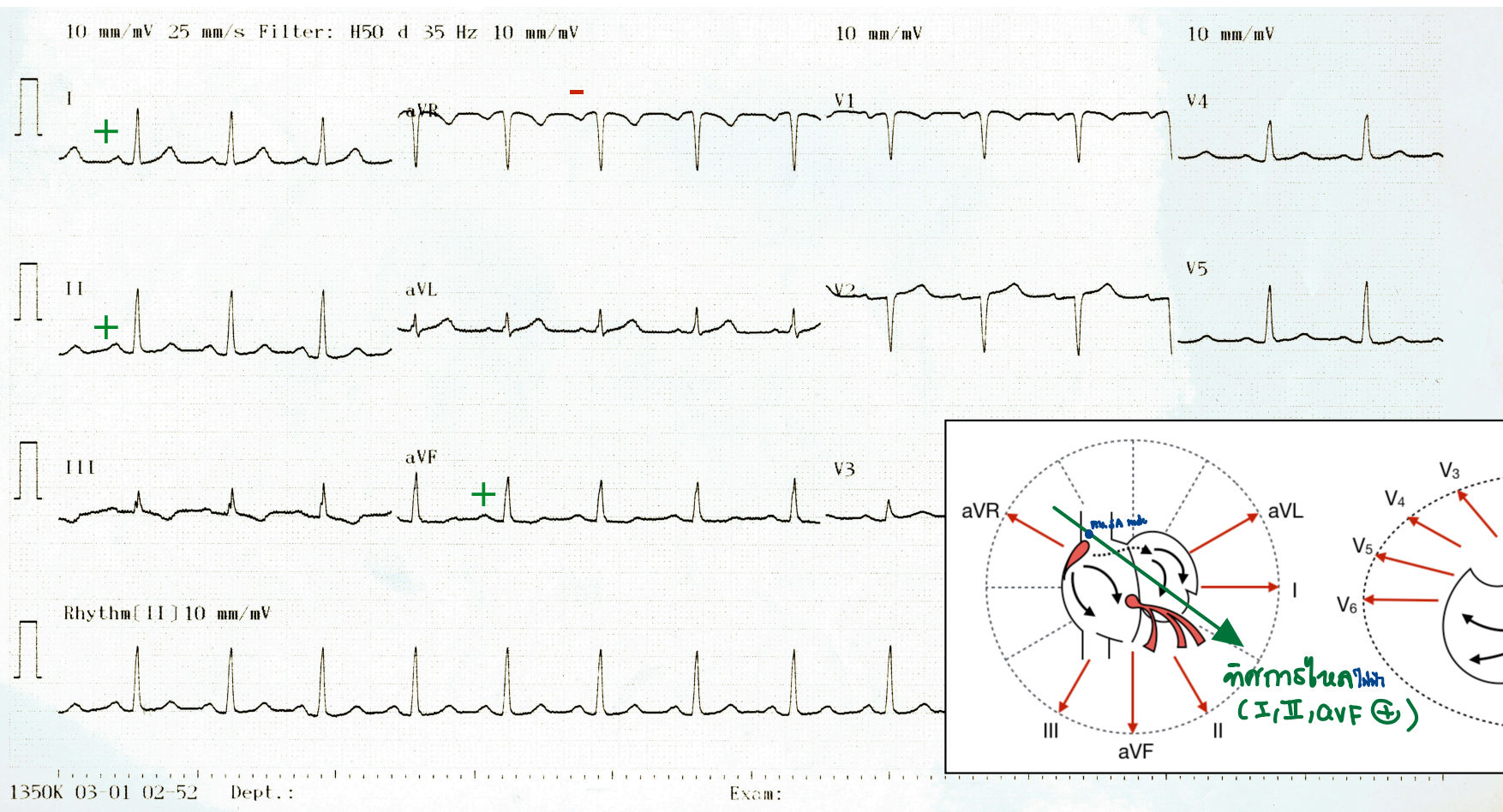
Innervation

- Densely innervated with postganglionic adrenergic and cholinergic nerve terminals (threefold greater density of beta-adrenergic and muscarinic cholinergic receptors than adjacent atrial tissue)



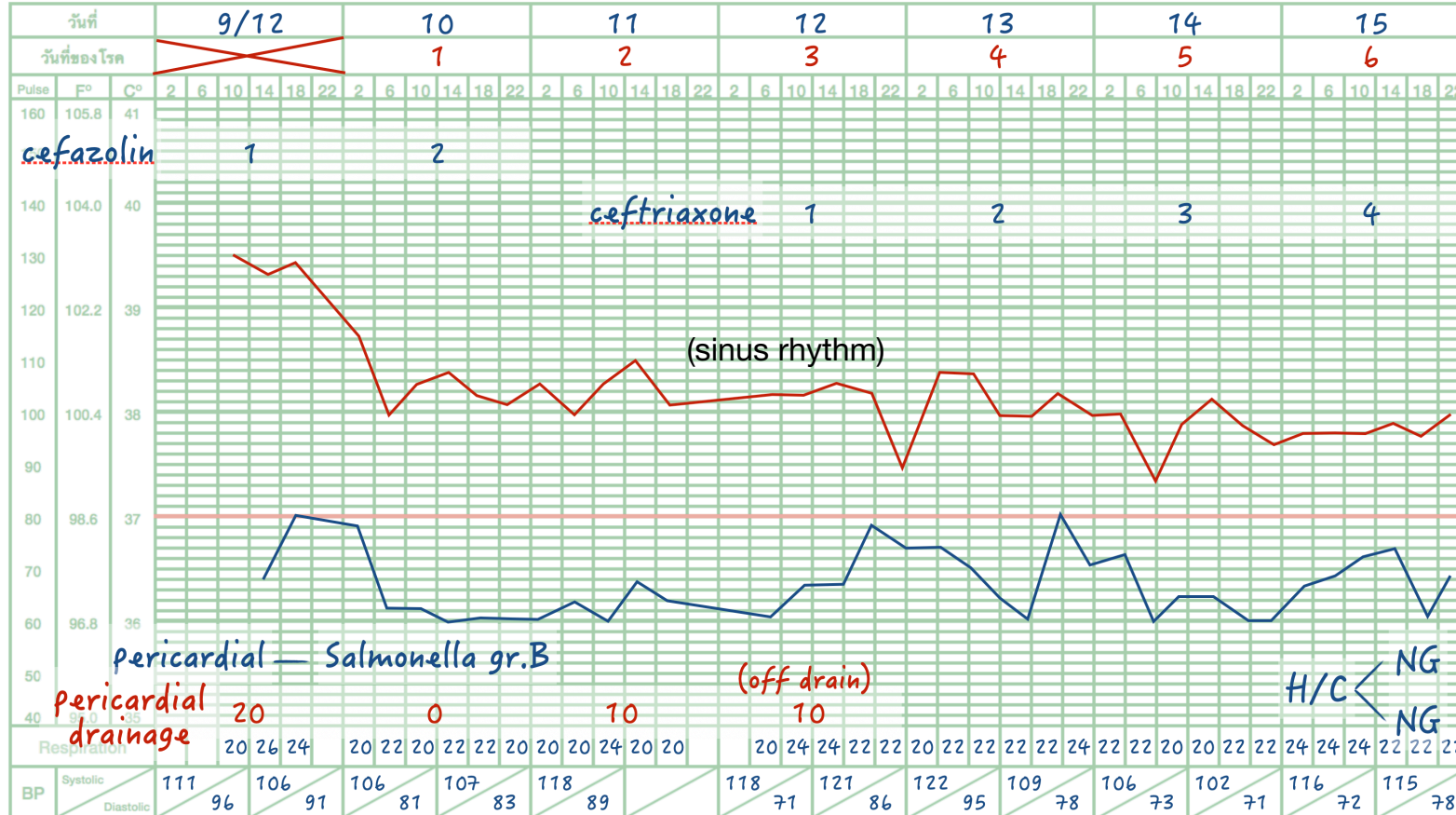
Sinus rhythm

P wave + in I, II, aVF P wave - in aVR



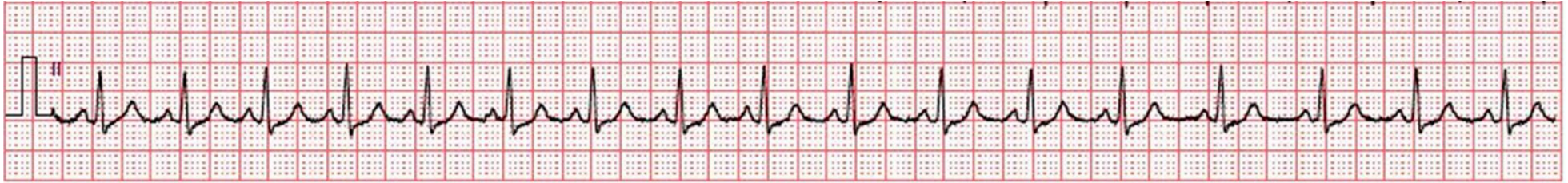
α sympathetic tone (main action: ex. to nerves)

Heart rate variation time to time



ex. คนไข้เดิน/วิ่ง

Sinus tachycardia, sinus rate 103/min



normal : 60-100 bpm

Sinus rhythm, sinus rate 68/min



ex. คนไข้หลับ

Sinus bradycardia, sinus rate 38/min



(in physiologic)

bp ↓



(Isi tachyaria su compensatiu)

genetic

Ca²⁺ channel stimulator

an automaticity without pacemaker cell

$\left. \begin{array}{l} \therefore \text{inhibition} \\ I_{Ca}, \text{HCN channel} \end{array} \right\}$

slow

AMP ↑

-: NO⁺ ATP maintains ion stability

Cause: Ischemia von ATP $\rightarrow$ NKA stillfalls $\therefore$ AMP $\uparrow$

No reperfusion: $\downarrow$ ATP $\rightarrow$ AMP maintain during change from ischemic $\rightarrow$ hr $\uparrow$

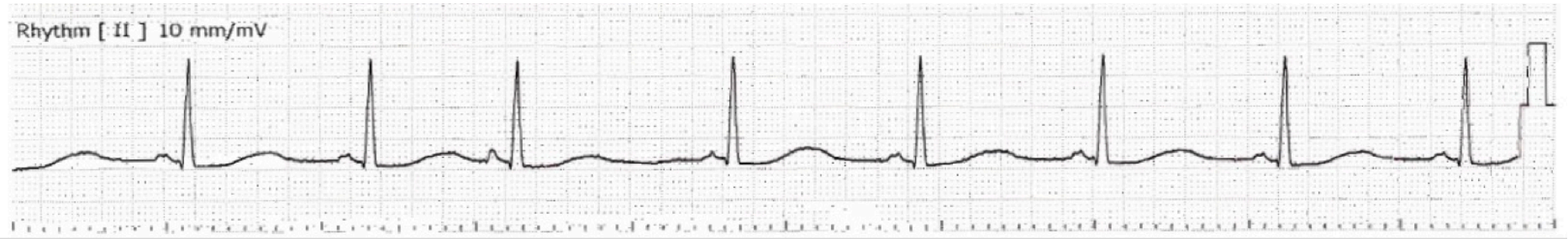
ex. $\text{[Na}^+]_i \uparrow \rightarrow \text{NCX} \uparrow \rightarrow \text{EC}^{2+}_i \uparrow \rightarrow$ delayed afterdepolarization
= triggered activity

rate of human reproduction

Premature atrial and ventricular complexes

foci ปลดปล่อยไฟฟ้าก่อนเวลาเป็นต้นเหตุ

Premature atrial complex



Premature ventricular complex

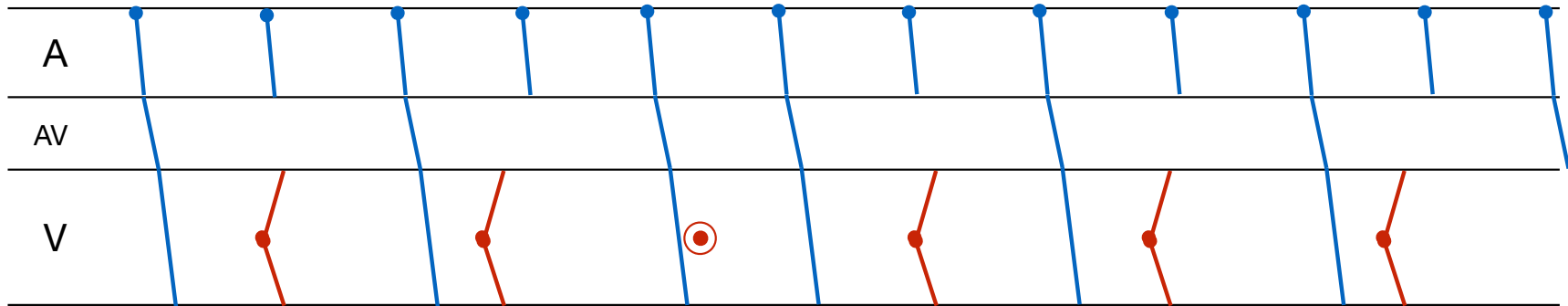


PVC from automaticity (ventricular parasystole)

อาจอยู่นิ่งในนาก็ได้ค่ะ ไม่ต้องตามนิจ (uncommon)



fixed PVC to PVC interval
foci ไม่เปลี่ยนตำแหน่ง rate เร่งขึ้นเอง



Why $Ca^{2+} \rightarrow DAD$?

$\rightarrow$ When AP⁺ in $\rightarrow Ca^{2+}$ activates DAD

Ca^{2+} influx $\downarrow$

- when hyperpolarization
- arrhythmogenic pathway $\rightarrow Ca^{2+}$ influx $\downarrow$

↑ Ca^{2+} influx in
ischemia: $ATP \downarrow \rightarrow [Na^+]_i \uparrow \xrightarrow{NCX} [Ca^{2+}]_i \uparrow \rightarrow DAD$
digitalis intoxication: $\nearrow$

arrhythmia $\rightarrow$ ADAs

Paroxysmal supraventricular tachycardia

Is atrium funny sustain

∴ มี reentry ที่ rate มี given arrhythmia ที่ rate นั้นพอ

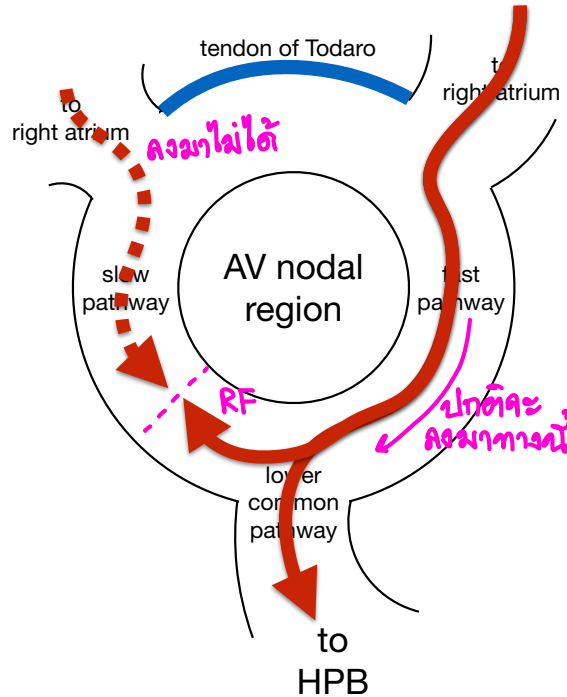
(reentry มี)



sustain in atrium only

most common SVT

Fast & slow pathway conduction



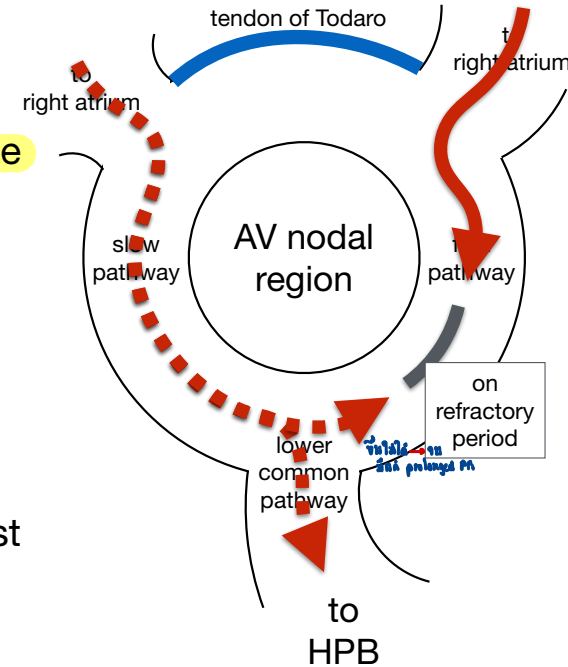
During sinus rhythm

Impulse entering AV node via **fast pathway** reach lower common pathway prior to impulse from slow pathway

Impulse from slow pathway collides with refractory period following impulse from fast pathway and stop

Only impulse from fast pathway past through HPB

unidirectional block
slow pathway
fast pathway
refractory period
fast pathway active



During PAC beat

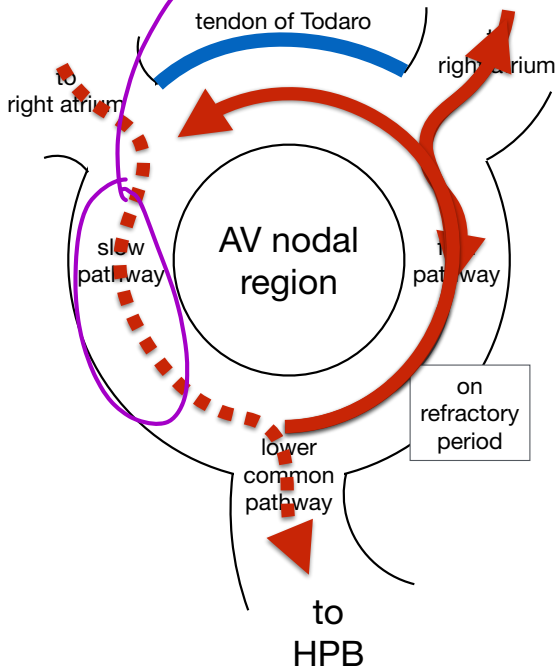
If PAC occurs at critical timing - **fast pathway** is still in **refractory period** while slow pathway is recovered (slow pathway naturally has shorter refractory period than fast pathway), slow pathway impulse instead reach lower common pathway.

Typical AVNRT circuit

กรณี lesion ที่ฉันทักใน slow pathway 1 ชั่วโมง

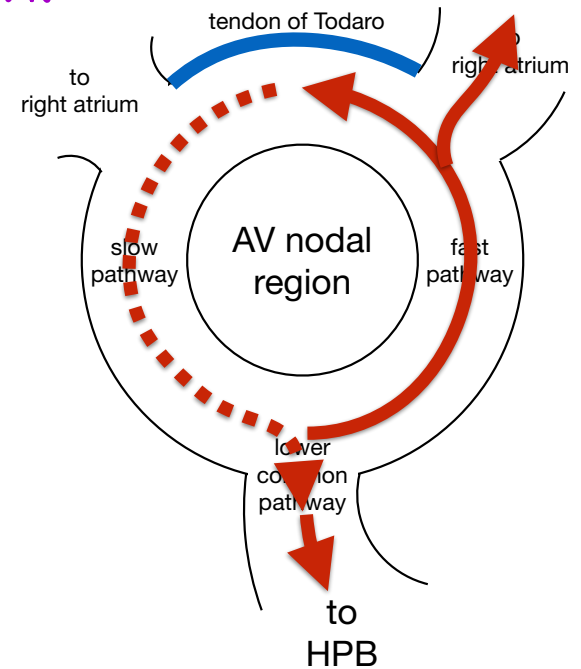
จนพ้น RF ของ fast pathway $\Rightarrow$ reentry บน slow pathway เดิม

จนกลับมา slow pathway เดิม



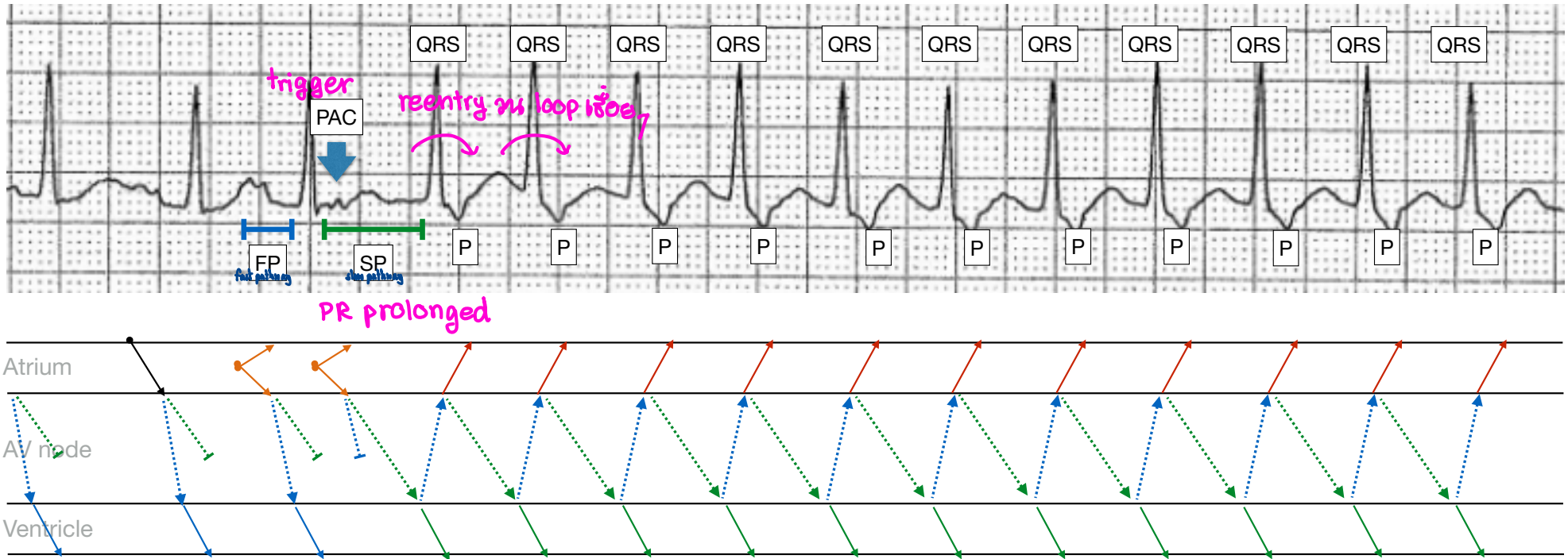
Initiate

If slow pathway impulse following the critical PAC manage to reach the recovered fast pathway, impulse can endlessly go retrogradely via fast pathway and antegradely via slow pathway.



Sustain

Typical AVNRT initiation in ECG



มี bypass tract ¹⁴

1st atrium $\Rightarrow$ ventricle 1st atrial AV node

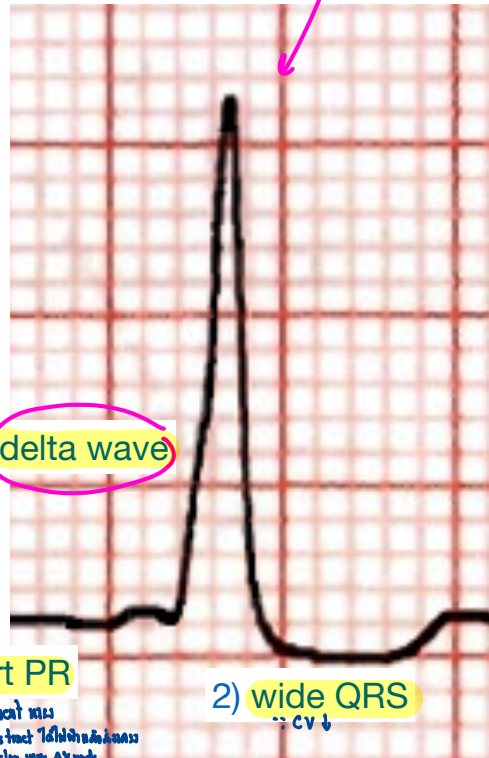
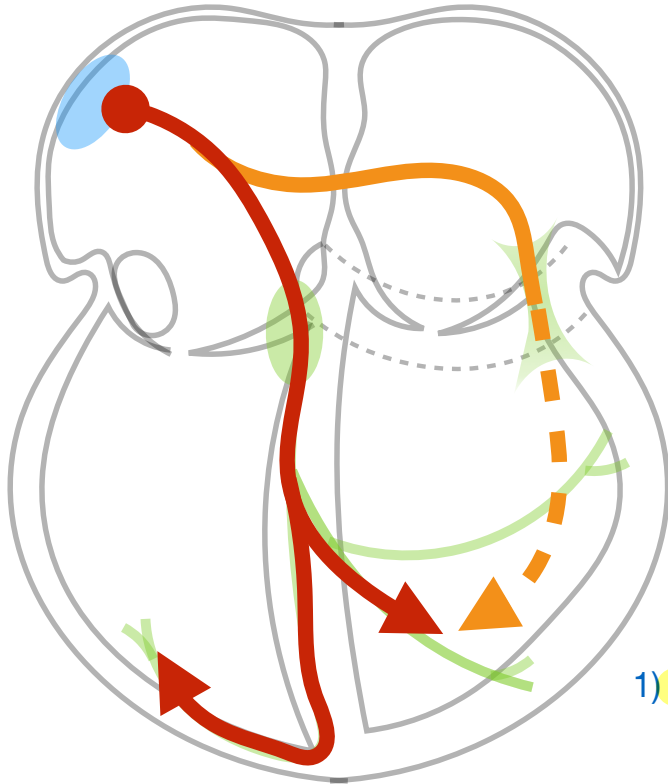
AV
node

“ပြော့ bypass tract ခုထုတ်ပေါ့” (congenital)

Wolff-Parkinson-White (WPW) syndrome

bypass tract ခေါ်သော ventricle ကို

“Preexcitation”



ပြော့ bypass tract

1) short PR

PR segment သေး
∴ bypass tract ကိုယ်စားပြုသော
တစ်ခုခုက AV node

2) wide QRS

∴ CV ↓

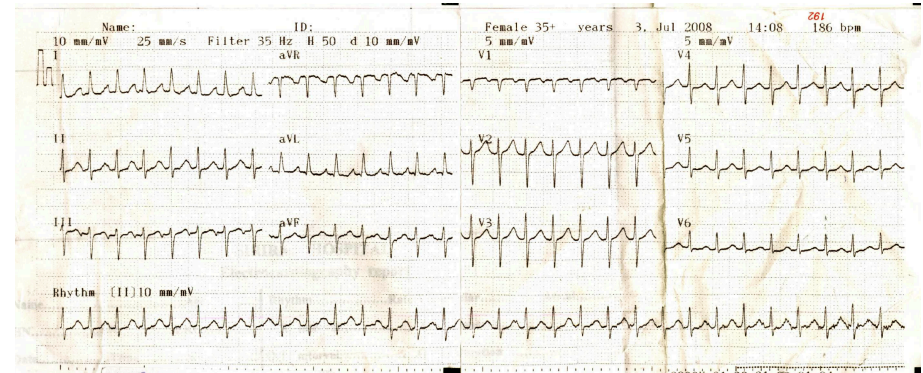
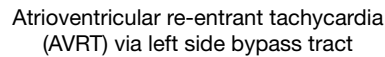
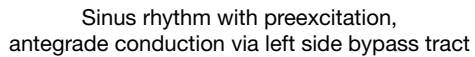
pre-excitation !

Atrioventricular bypass tracts
(bundle of Kent) are typically
fast fiber

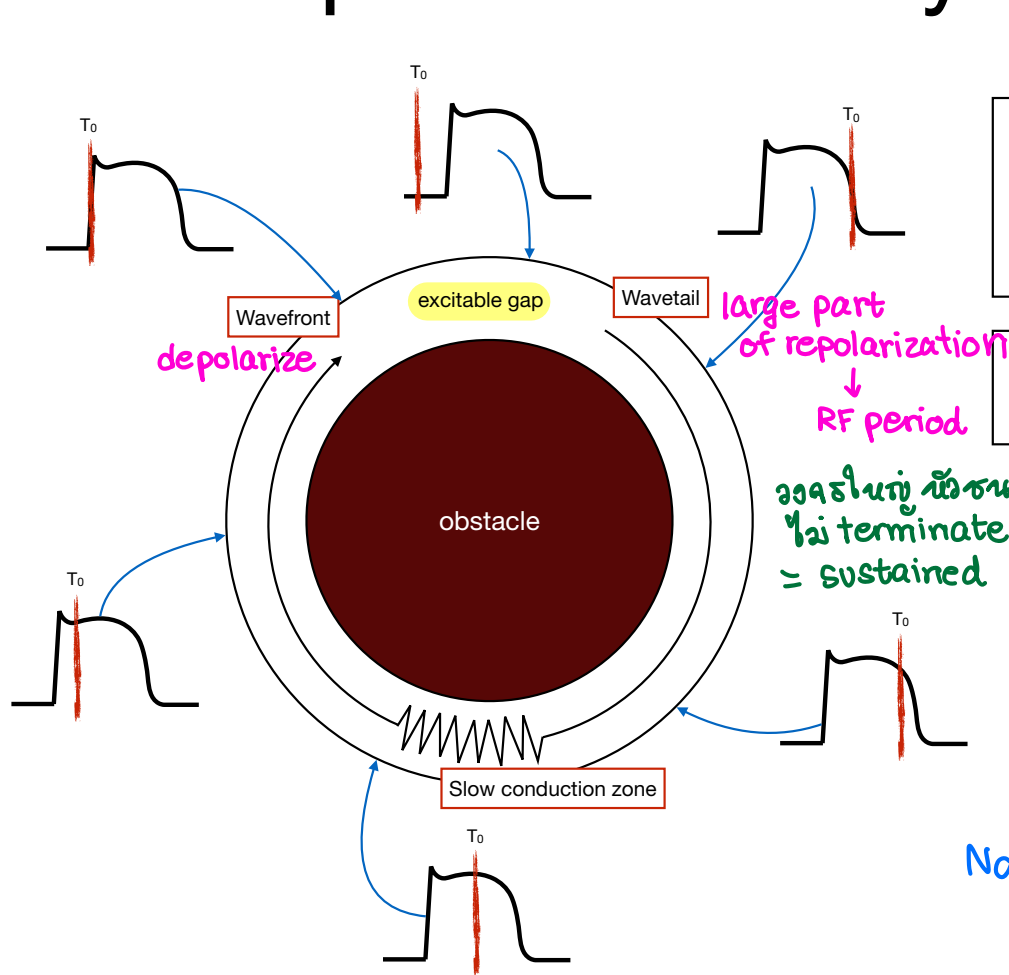
- Minimal conduction delay
- No decremental property
- Short refractory period
- Insensitive to CCB or beta blocker

WPH : 90% หารยา byram triol
AVRT : 90% หารยา AV node ablation byram triol

พรีลิมินารี เมดิเลชัน
 ∴ normal (WPH) → wide QRS
 หรือ AVRT → QRS แคบผิดปกติ



Principles of reentry (anatomical reentry)



AP duration prolonged / รีบยาว RF ↑
 ถ้าทำให้ AP duration prolonged / รีบยาว RF → terminate arrhythmia

$$T_{\text{Tachycardia cycle}} = T_{\text{Max refractory period}} + T_{\text{Excitable gap}}$$

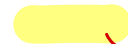
$$\frac{\text{Circuit distance}}{\text{Mean conduction velocity}} = T_{\text{Max refractory period}} + T_{\text{Excitable gap}}$$

$$\text{Tachycardia rate (bpm)} = \frac{60000}{T_{\text{Tachycardia cycle (milliseconds)}}$$

- Sustainability of reentry $\propto T_{\text{Excitable gap}}$
- ↑ Sustainability $\leftarrow$ ↓ refractory period, ↑ circuit distance, ↓ conduction velocity $\rightarrow$ $\text{Na}^+\text{Ch. blocker} : \text{CV} \downarrow$ (ลดไฟฟ้า) ทำให้ arrhythmia terminate ยาก !!
- ↓ Sustainability $\leftarrow$ (↑ refractory period, ↓ circuit distance, ↑ conduction velocity) ex. amiodarone block นาน AP ยาวขึ้น อาจทำให้ CV ↓ และ ไล่กดที่ถาวร normal AP ยาวเกินกว่า → terminate ยาก
- ↓ Tachycardia rate $\leftarrow$ ↓ conduction velocity ยกเว้น High dose + VT storm

target neuron

AVNAT



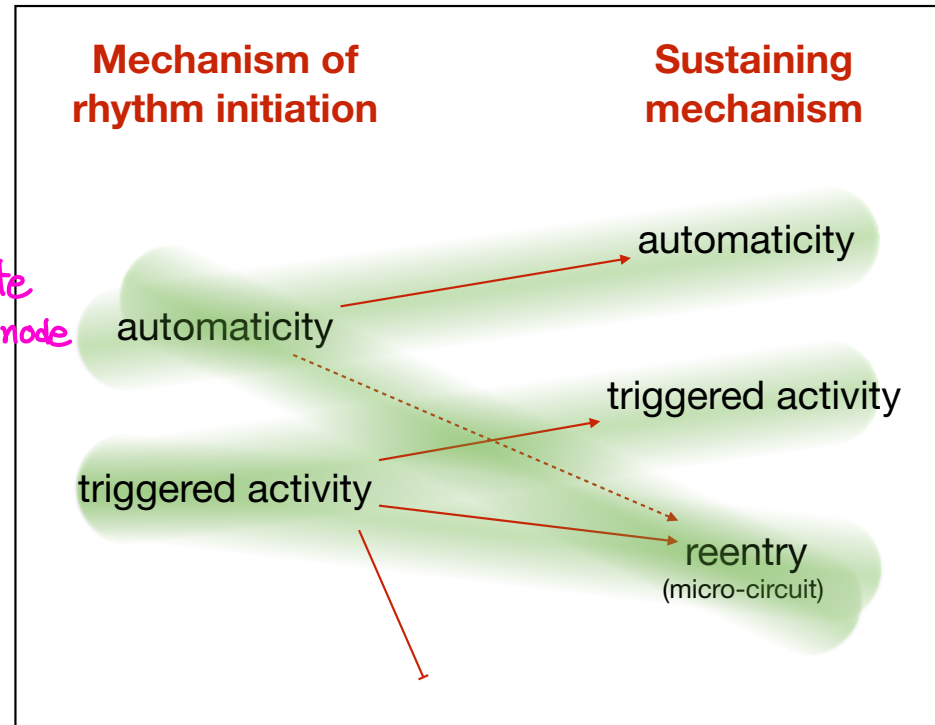
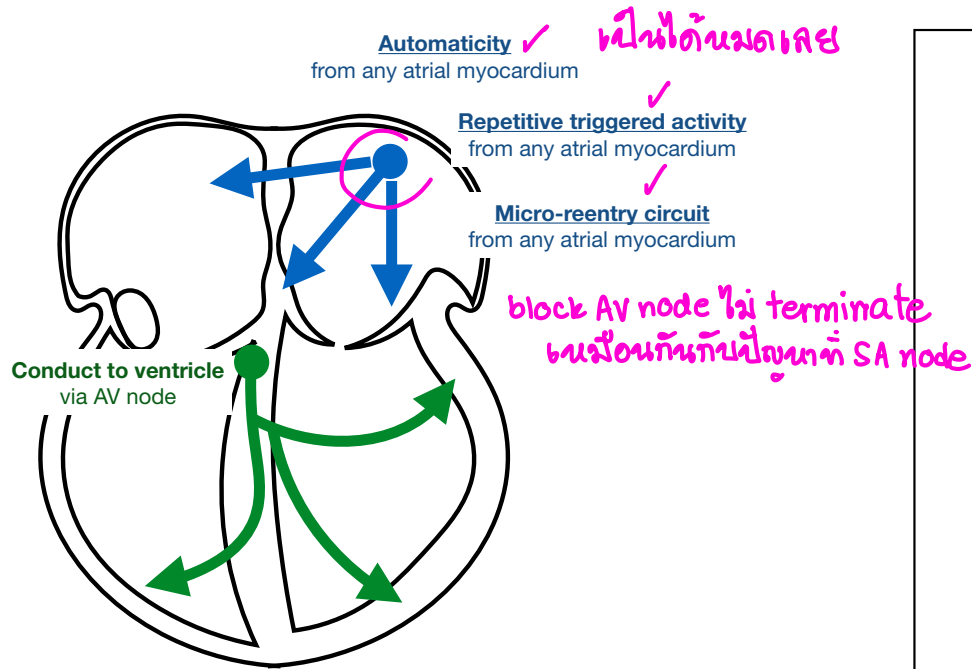
is hyperpolarization
AV node conduct is reduced
∴ terminate MU



→ fast fiber is Na⁺ non phase 0

Focal atrial tachycardia *

24 hr active only $\therefore$ block AV node ง่าย

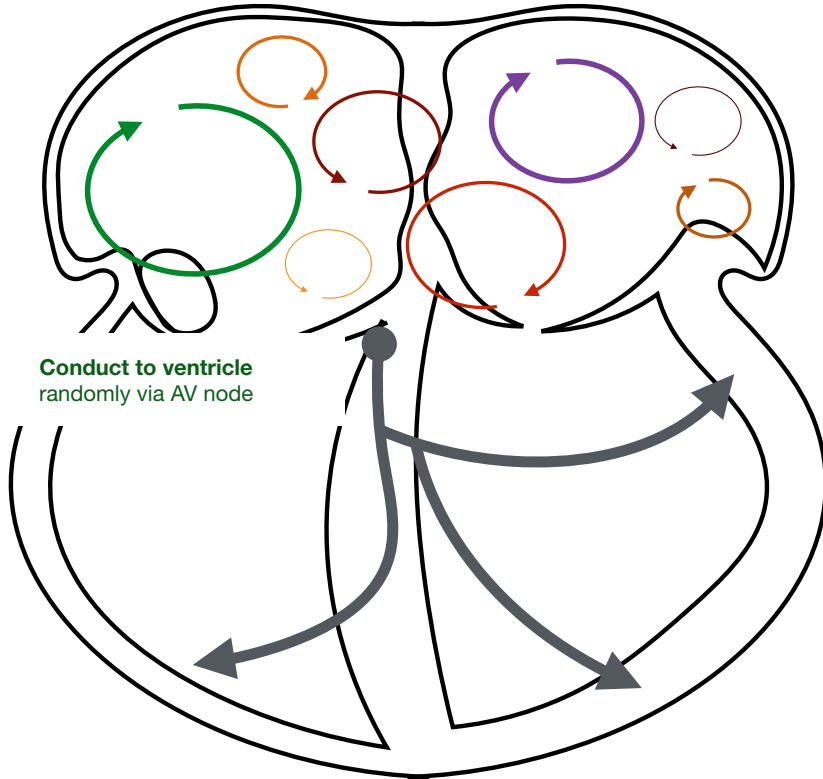


P wave
P wave in lead II
∴ indicates SA node

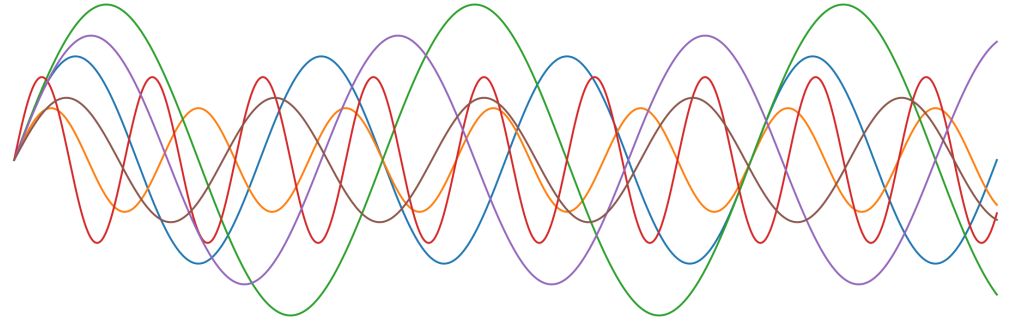
s P wave: 1 QRS complex
= if AV block, no rhythm to terminate

Atrial fibrillation

Multiple wavelet reentry (multiple functional reentry)



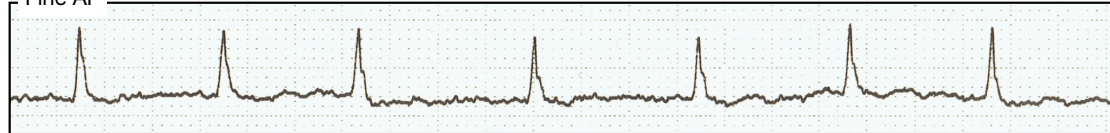
Superimposed of each individual wavelets



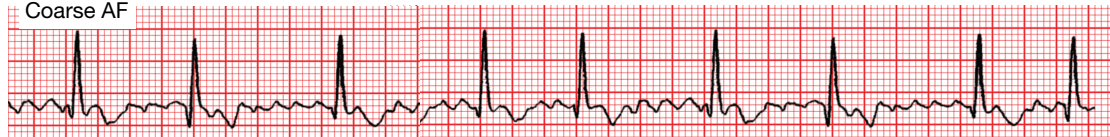
Summation of wavelets visible from ECG



Fine AF

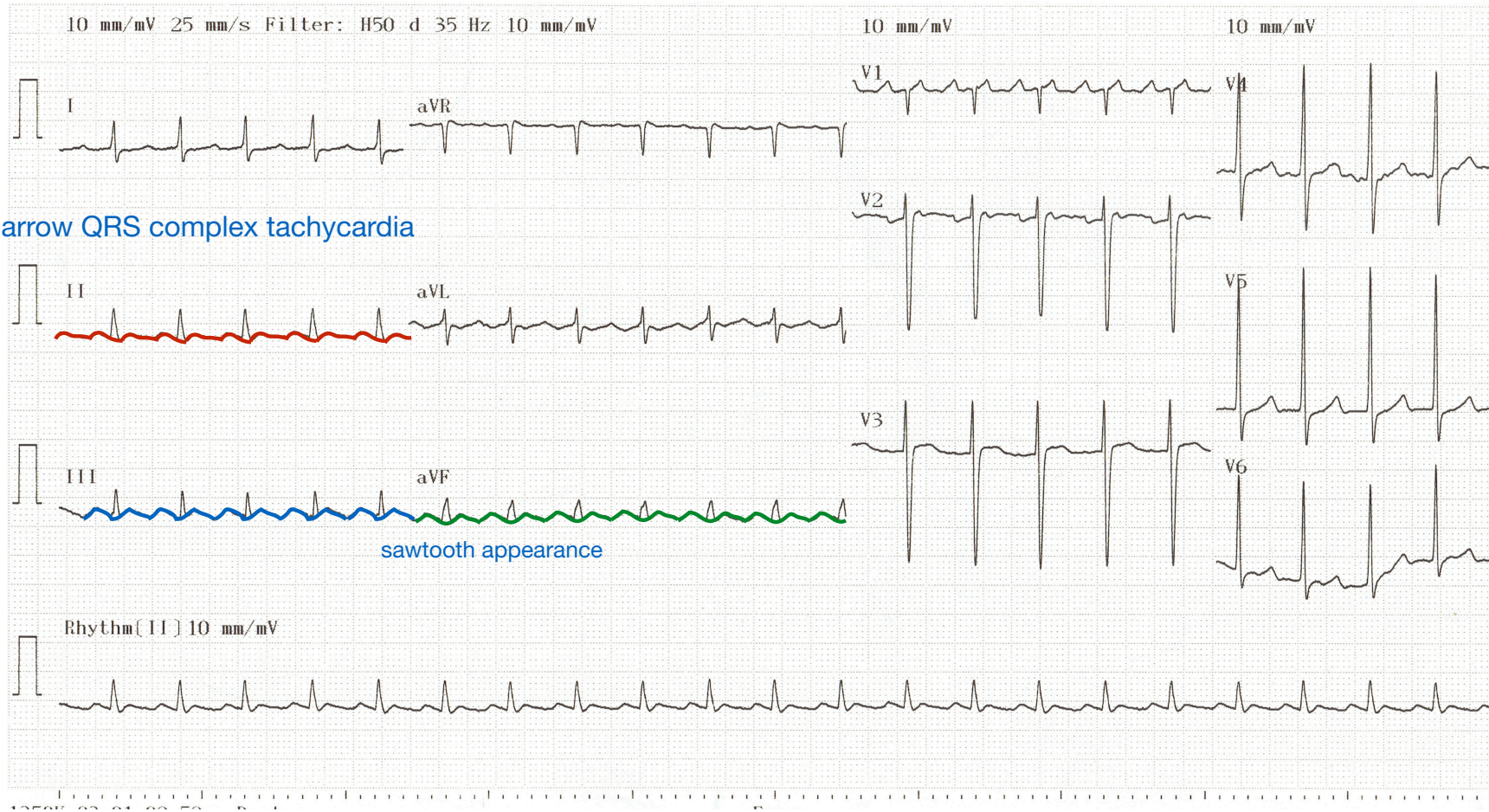


Coarse AF



Atrial flutter (2:1 AV conduction)

Regular narrow QRS complex tachycardia

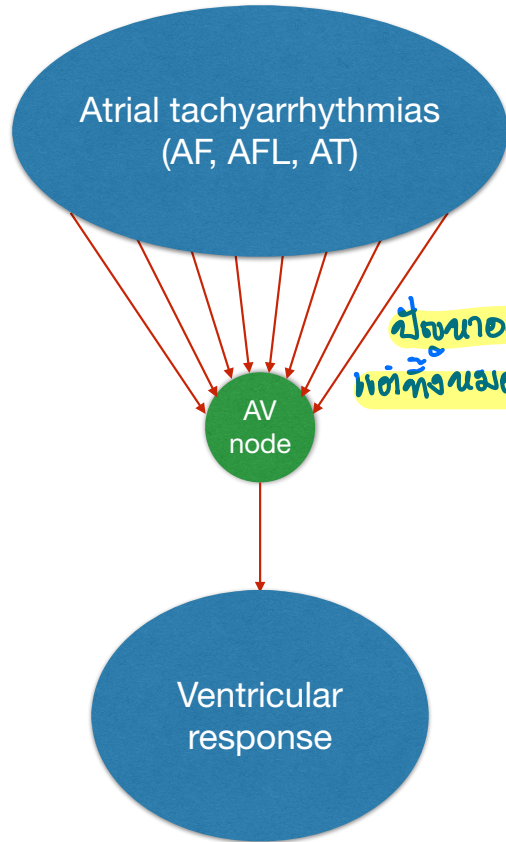


most common: regurg of tricuspid valve

circuit is not active
1st AV node

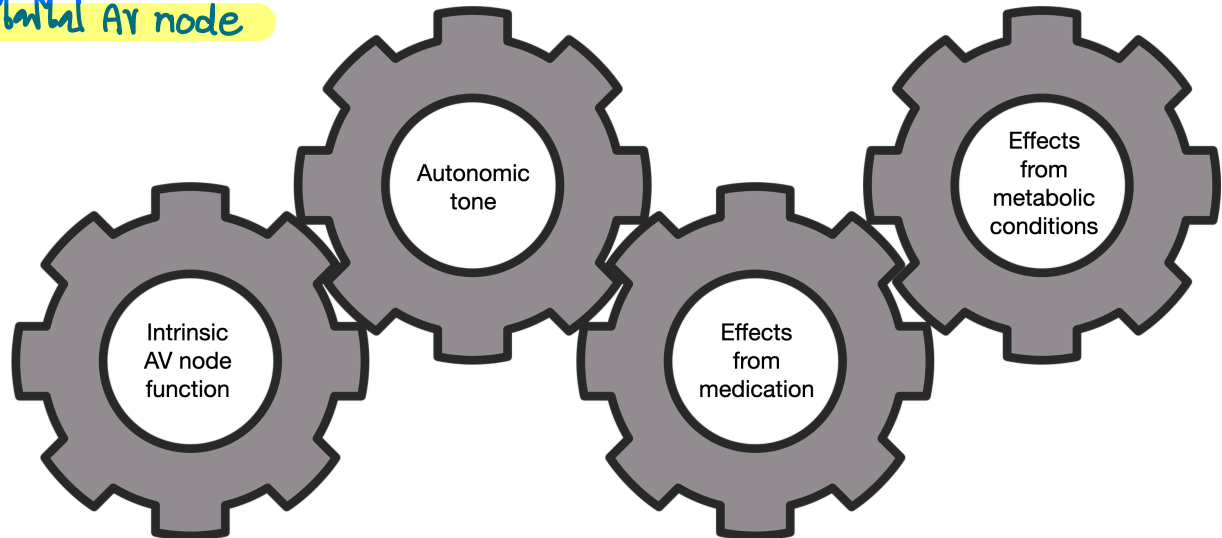
∴ AV node in refractory period is not active

Ventricular rate during atrial tachyarrhythmias



ปัจจัยที่มีผลต่อ
การทำงานของ AV node

- Ventricular response (ventricular rate) during atrial tachyarrhythmias (atrial fibrillation, atrial flutter, or atrial tachycardia) is determined by AV node refractoriness
- Factors determine AV node refractoriness are including 1) intrinsic node function, 2) autonomic tone, 3) medication effects, and 4) metabolic effects



Atrial fibrillation with various ventricular rate

q34 AV node refractory period $\rightarrow$ q34 ventricular response q34

AF, ventricular rate 48 /min

Rhythm (II) 10 mm/mV



AF, ventricular rate 114 /min

Rhythm (II) 10 mm/mV



AF, ventricular rate 150 /min



Atrial flutter with various AV conduction ratio



AF : Sustained arrhythmia ใจห้องบนเต้นเร็ว adenosine (ลองกดหัวใจ 6-8s แล้วจะหาย)

- β -blocker
 - Ca^{2+} -blocker
 - amiodarone
- ใจห้องบนเต้นเร็ว
modify AV node

4:1



4:1 - 5:1

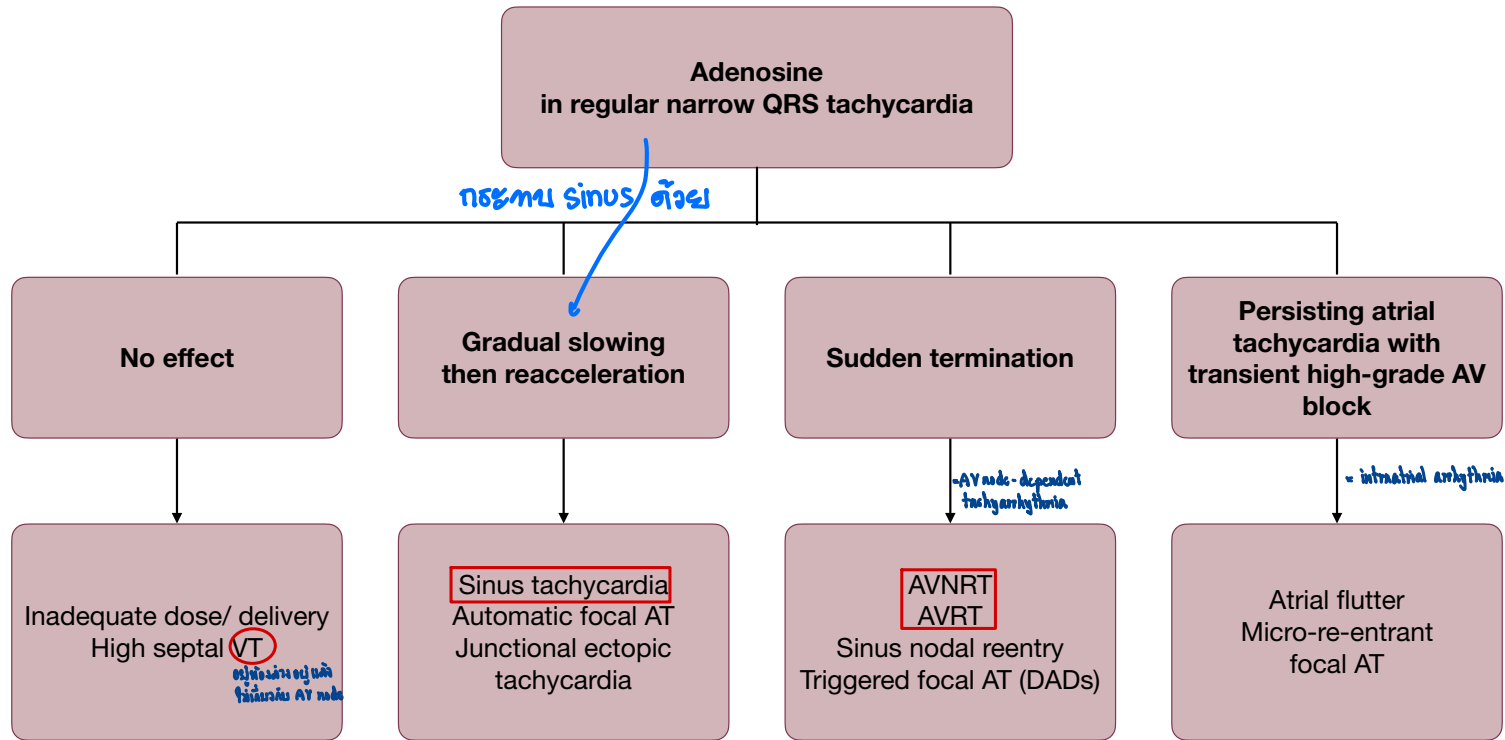


Responses of narrow-complex tachycardias to

adenosine

ใช้ 1st line for SVT (AVNRT/AVRT) 6 ไม่นาน !

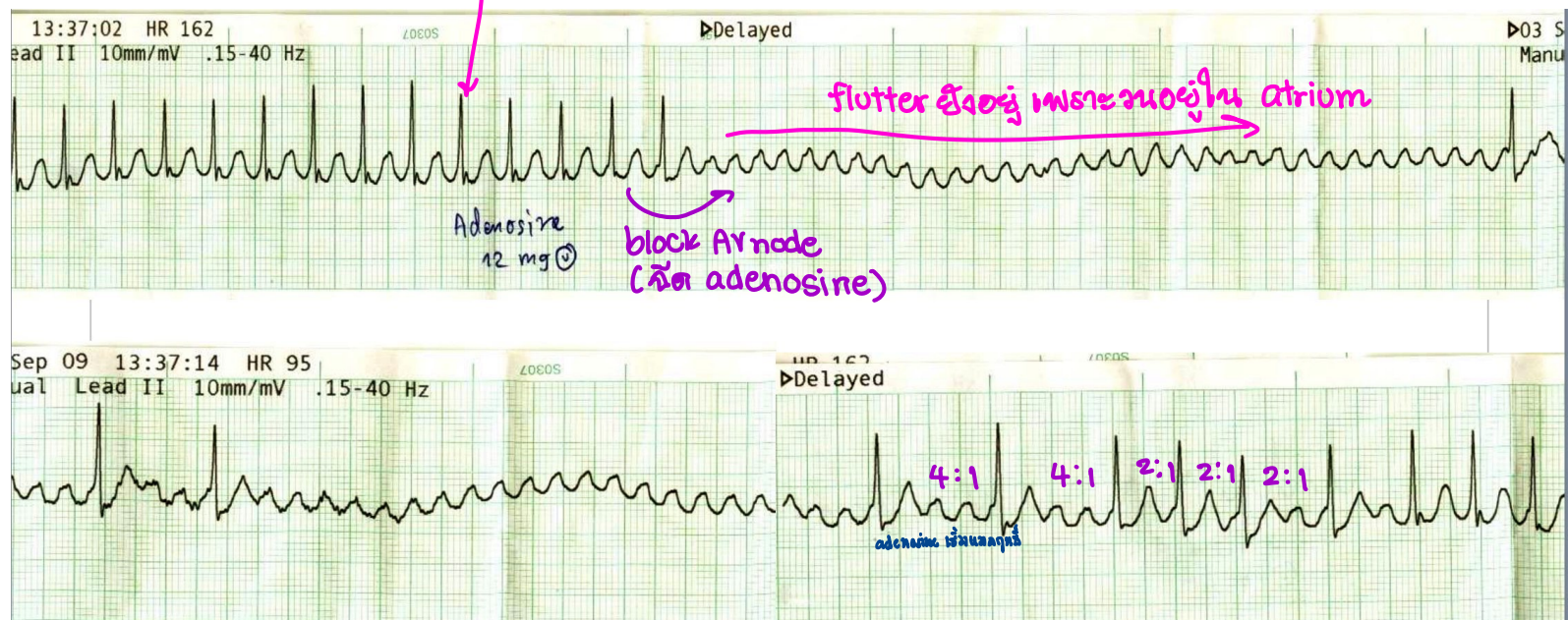
เพราะ AV node-dependent 9:10 นาที
คือ ทำให้ AV node hypopolarization



หาคำวินิจฉัย
จาก SVT เป็น Sinus tac.

Atrial flutter : effect of adenosine rapid injection

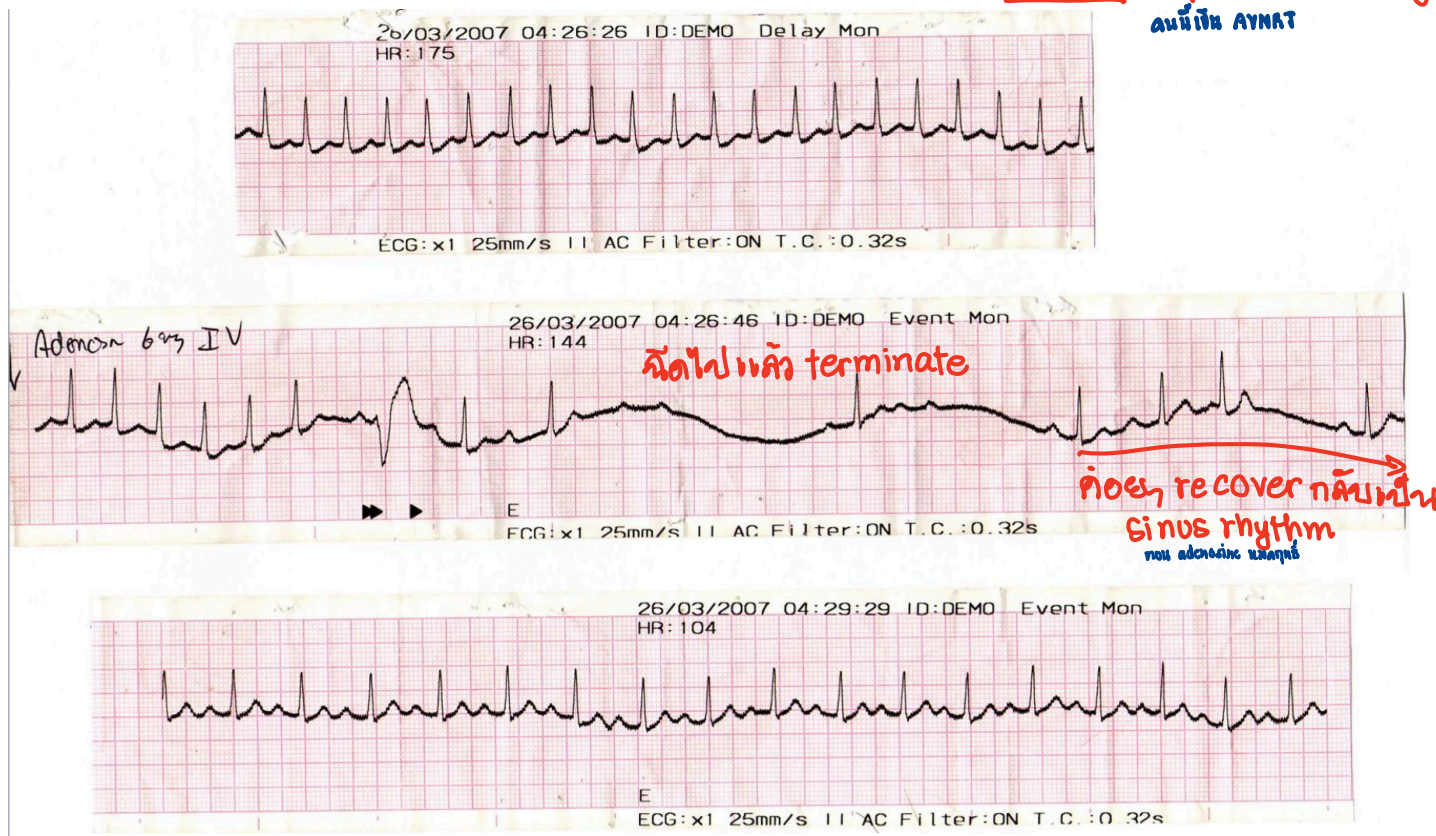
តំណាង adenosine អាច terminate



Adenosine rapid injection in patients with PSVT

Av node dependent entry ↙

อันนี้ AVNAT



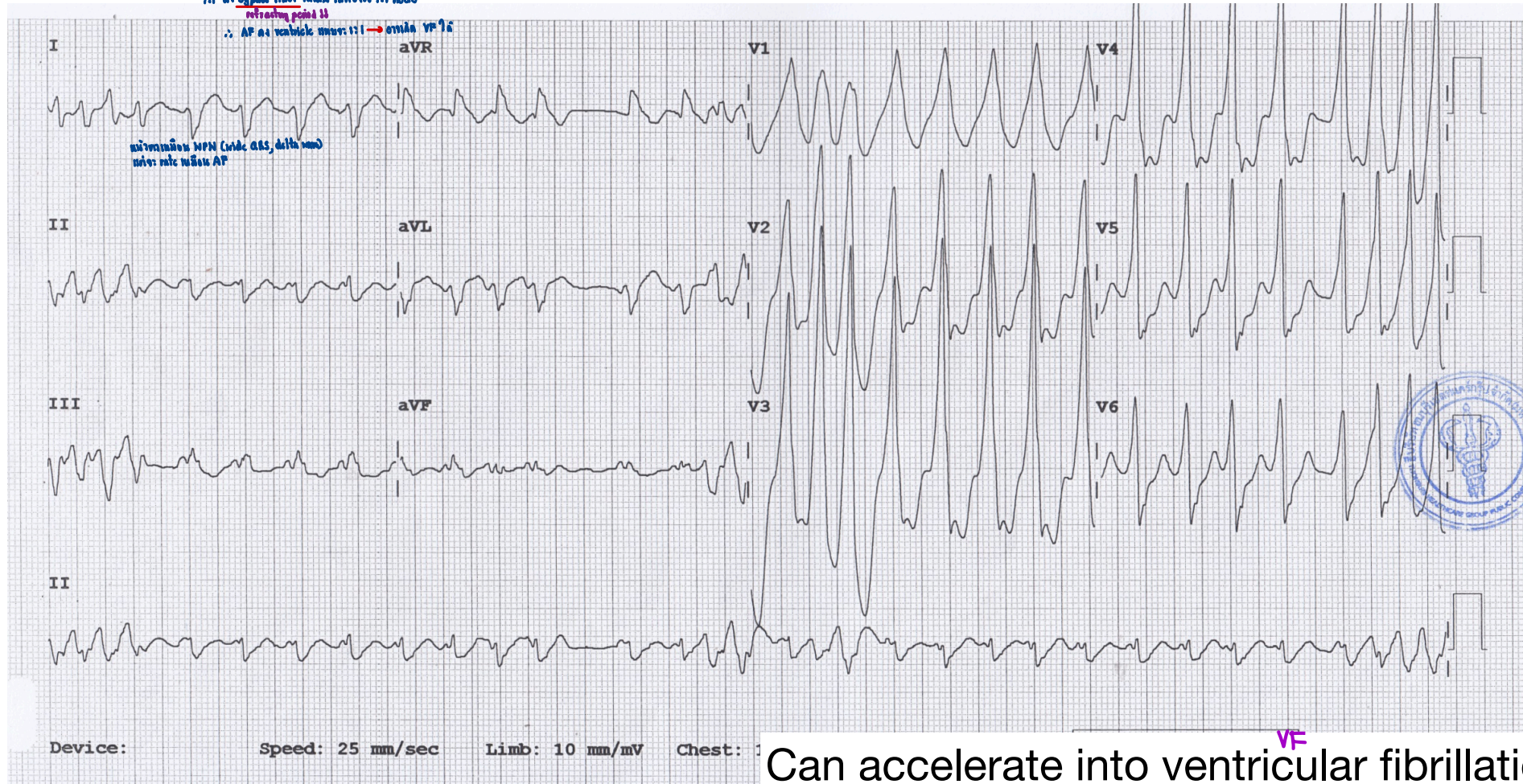
ប្រើ β -blocker បន្ថែម ពេលមានរោគសញ្ញា fast fiber

↑ **amiodarone** block fast block slow **AV node** → **decel VF**

AF with preexcitation - rapid conduction via bypass tract

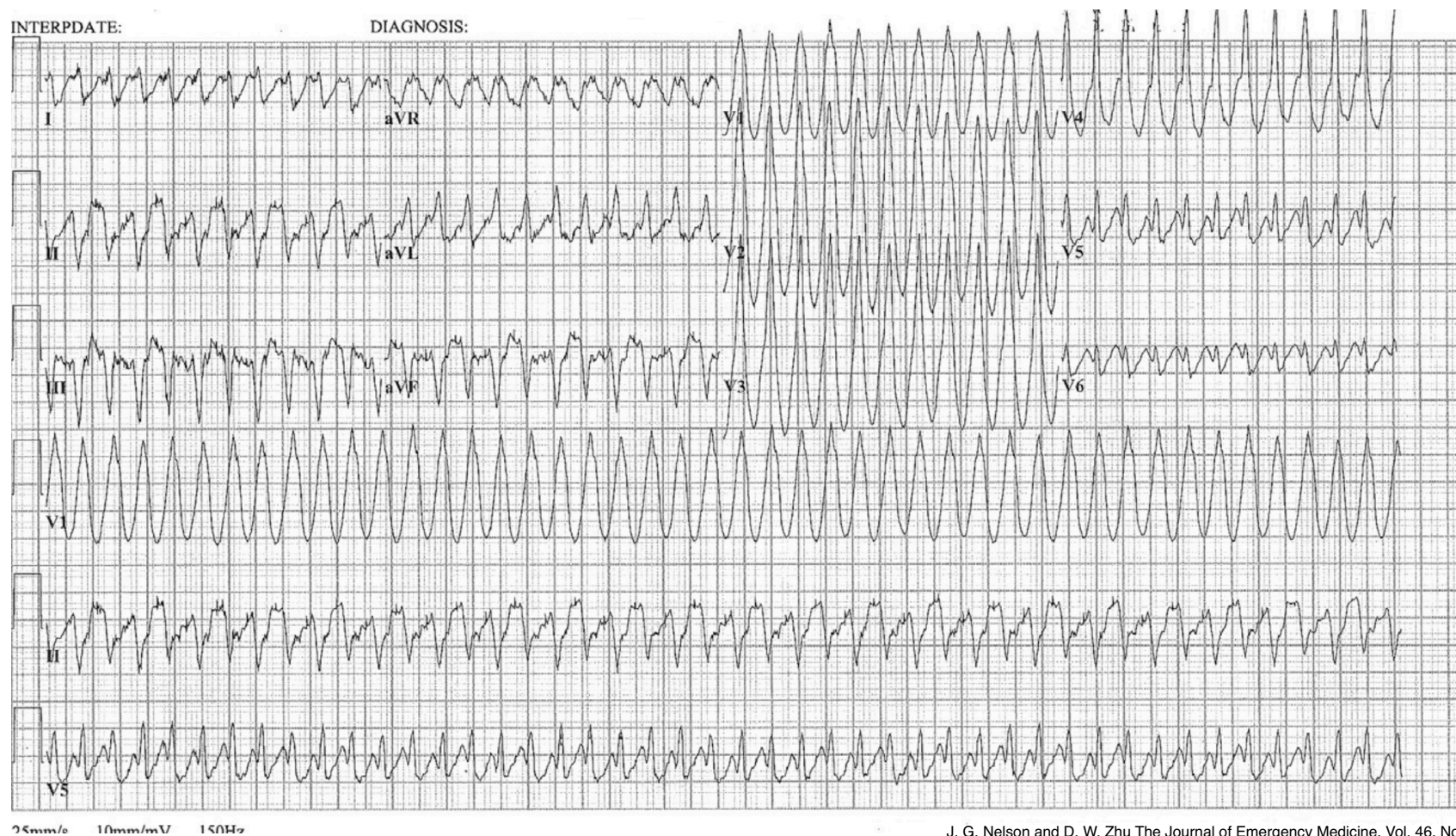
AF as bypass tract allows initiation of AF node
refractory period !!

$\therefore$ AF in ventricle causes: 1:1 $\rightarrow$ atrial VF



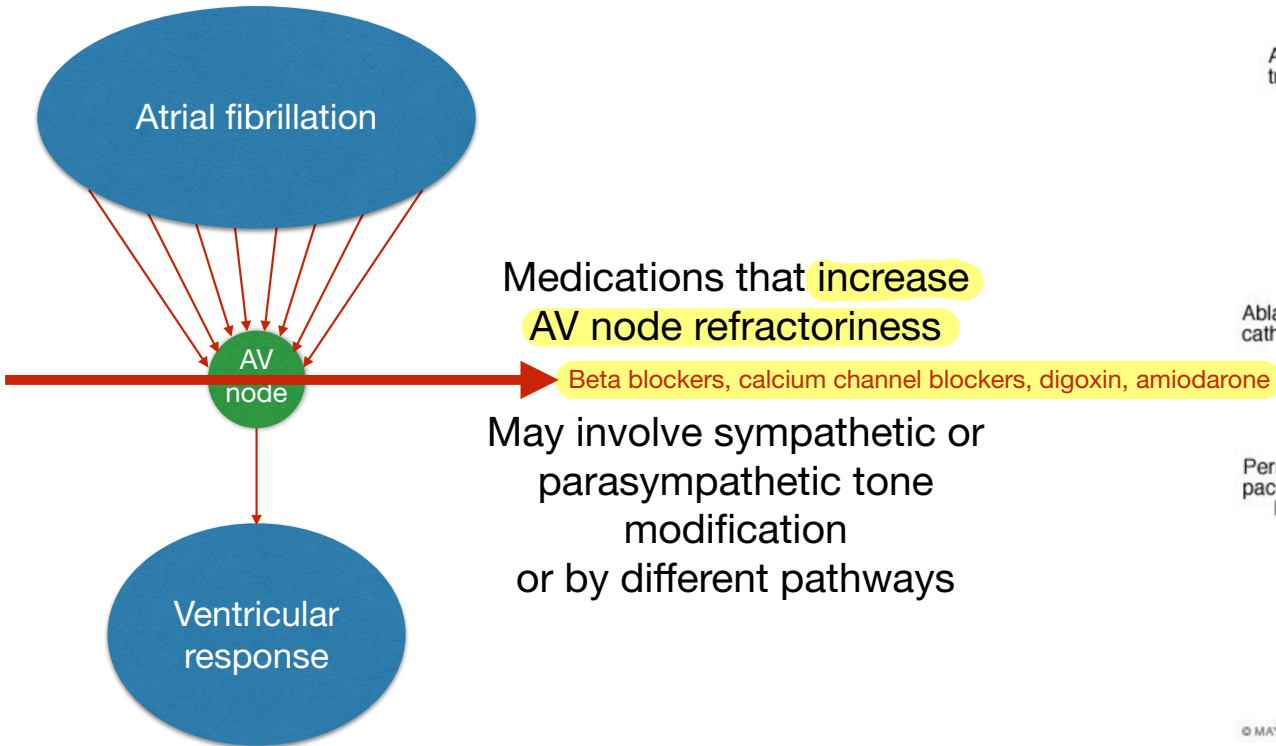
Can accelerate into ventricular fibrillation VF

Atrial flutter with 1:1 conduction via bypass tract

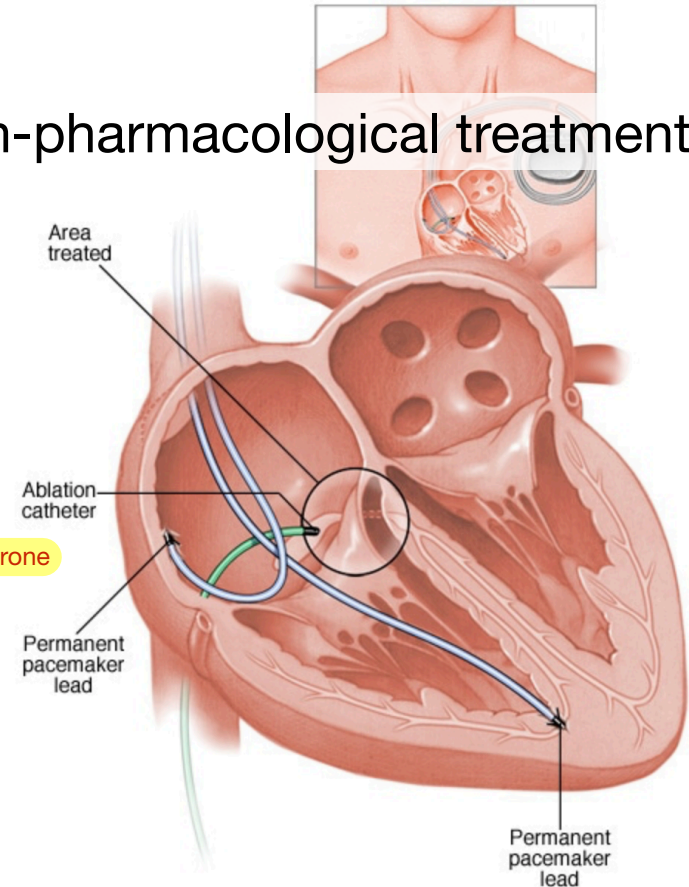


Ventricular rate control during AF/AFL/AT

Pharmacological treatment

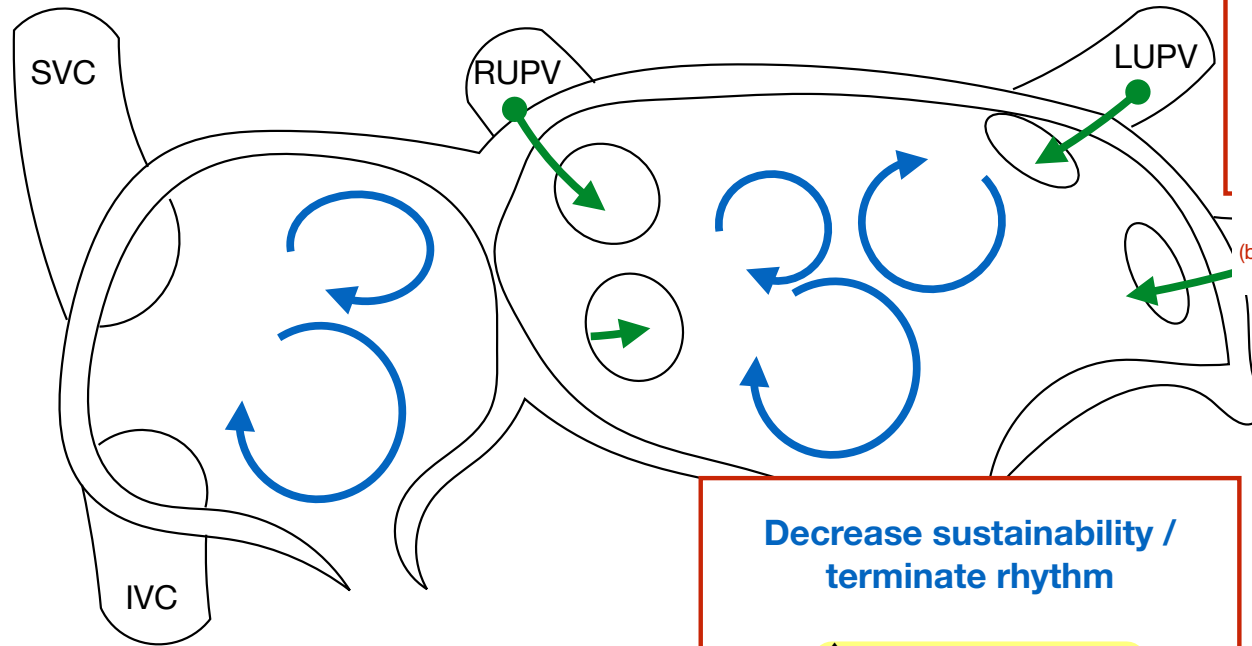


Non-pharmacological treatment



Pharmacological targets for rhythm control in AF

With HF → use β -blocker
 with LV dysfunction → use Na^+ blocker
 ↪ contraction



Inhibit triggers

Sympathetic blockers
 Reduce intracellular calcium

Medication to ^{prevent recurrent} suppress triggers
 (beta blockers, calcium channel blockers, class Ic, amiodarone)
 to prevent initiation

บนมือนาฬิกา ⌚ (แก้ DADs)

Decrease sustainability / terminate rhythm

↑ refractory period

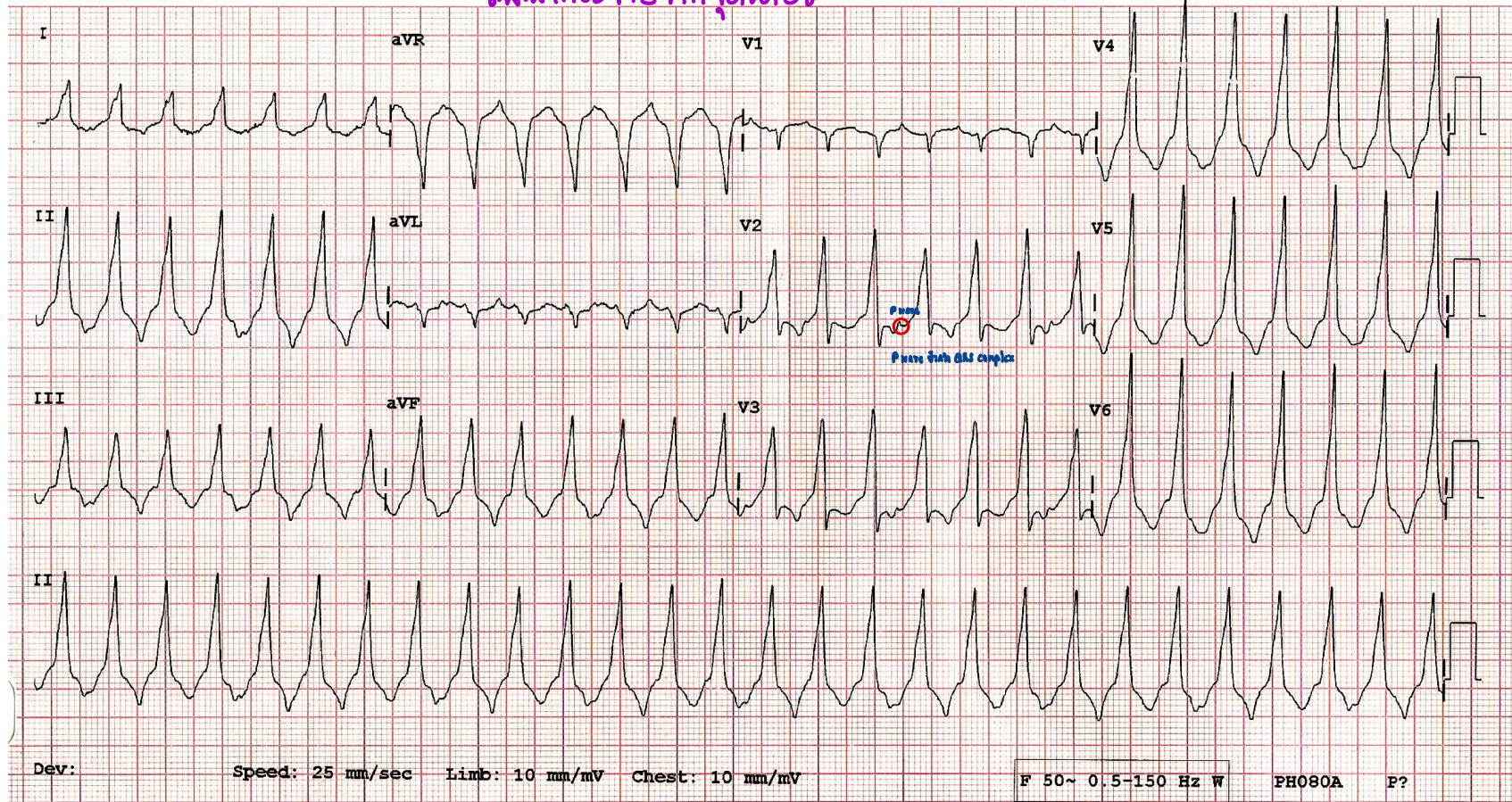
Medication to interfere sustainability

(amiodarone, other class III, high-dose class Ic) Na^+ blocker
 prolonged AP duration

MI → scar เกิดที่ V
 ถ้า CV ↓ จะยิ่งทำให้อัตรา reentry ↑
 ∴ Na^+ blocker ห้ามใช้ใน
 Structural disease
 ex. cardiomyopathy, CAD, ischemic

Monomorphic ventricular tachycardia

ไฟฟ้ากระตุกจากจุดเดียว



Monomorphic ventricular tachycardia

- Monomorphic ventricular tachycardia → electrocardiographic phenotype
- Mechanisms
 - Abnormal automaticity (so-called accelerated idioventricular rhythm - AIVR) ✓
rate = 100-120
 - (Repetitive) triggered activity ✓
0A0 47
 - Reentry ✓
highway scar

cell depolarizes excess Ca^{2+} $\rightarrow$ depolarizes $\dot{I}$ sodium Ca^{2+} channel leaky Ca^{2+}

triggered
= PVC

untriggered
= repetitive triggers

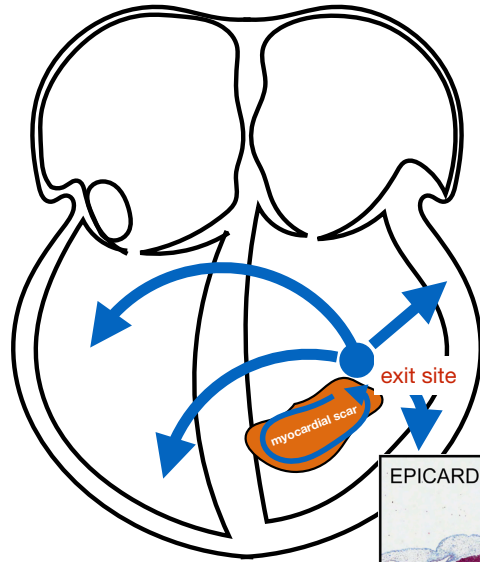
right ventricular outflow tract

ex.3 MF, M1 → อาจเกิด structural disease

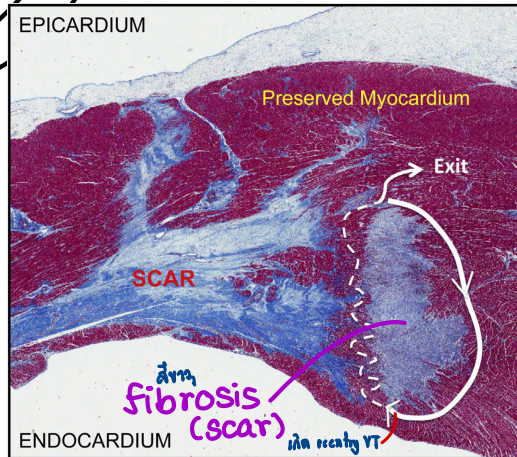
↓ Ca^{2+}

Scar-related ventricular tachycardia

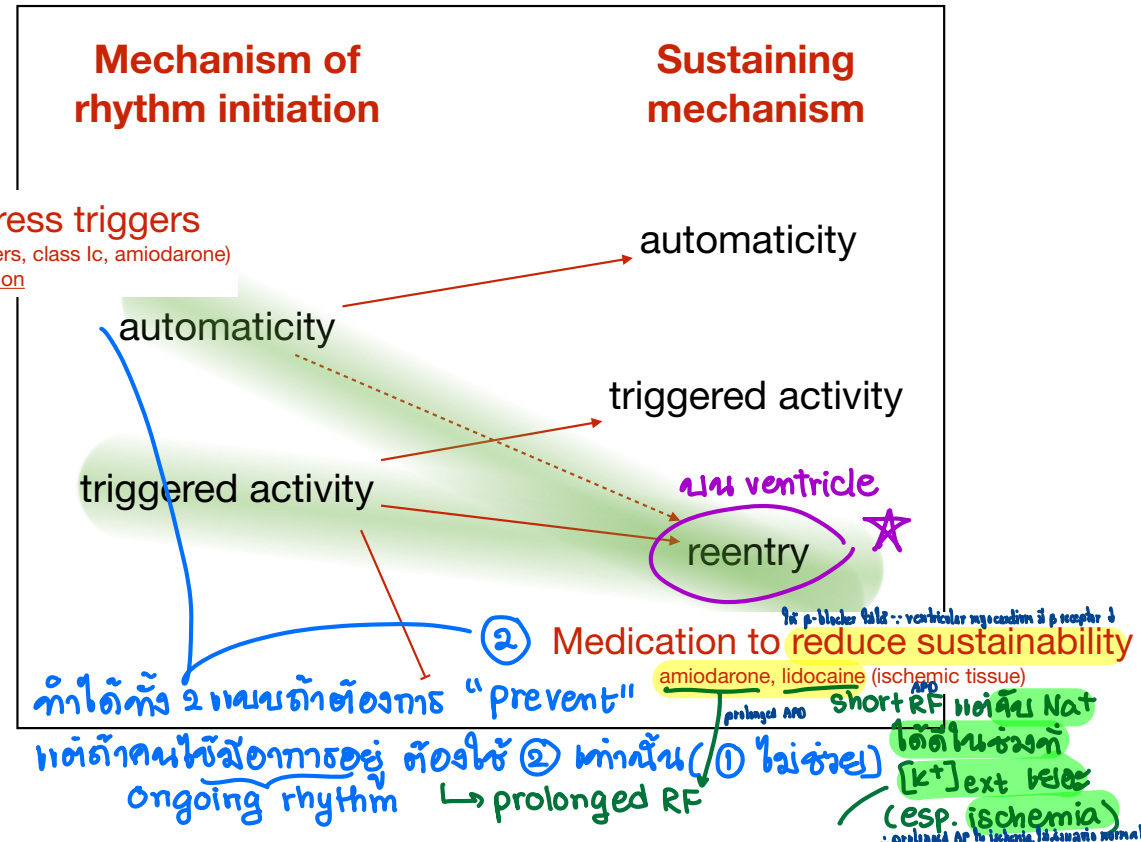
VT_m structural disease



① Medication to suppress triggers
(beta blockers, calcium channel blockers, class Ic, amiodarone)
to prevent initiation



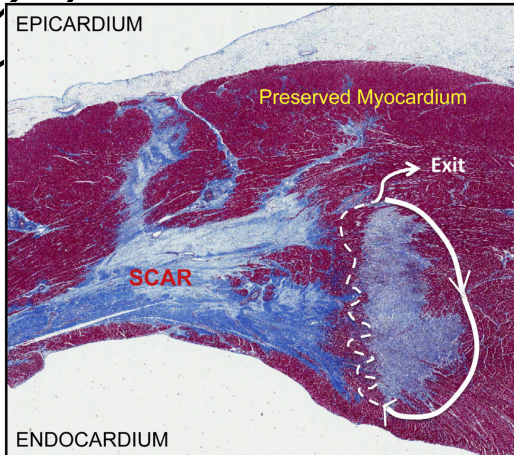
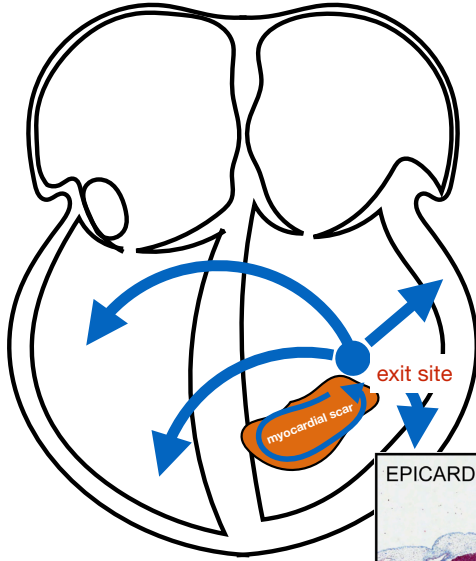
Olujimi A. Ajiola, et al. Indian Heart J. 2014 Jan; 66(Suppl 1): S24-S34.



prolonged RF เพราะคลื่นที่ abnormal 😊

Scar-related ventricular tachycardia

Pharmacological management



Control triggers

Aim → control DAD of at risk foci

- Class Ib : Lidocaine ^{Na⁺ channel blockers}
- Class Ic : flecainide, propafenone → poor clinical outcome in RCT ^{ไม่ช่วย stable HF}
- Class II : beta blockers → precaution in active HF
- Class III : amiodarone
- Class IV : contraindicated in LV dysfunction ^{suppress myocardial contraction}

Rhythm conversion ^{terminate reentry}

Aim → ↑ ventricular tissue refractory period

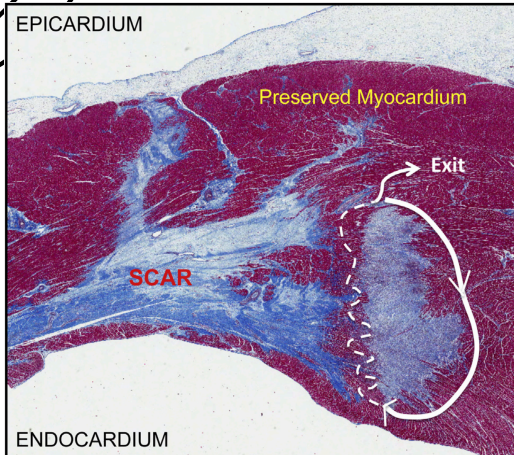
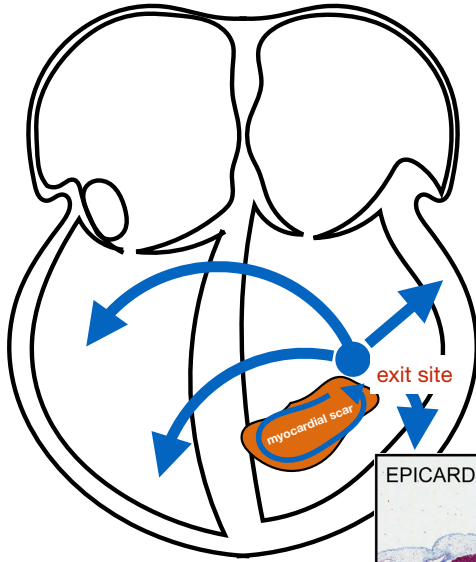
- Class Ia : procainamide ^{ไม่ช่วย}
- Class Ib : Lidocaine ^(esp. ischemic tissue)
- Class III : amiodarone

Longterm substrate modification

Effective heart failure/cardiomyopathy management

Scar-related ventricular tachycardia

Non-pharmacological management



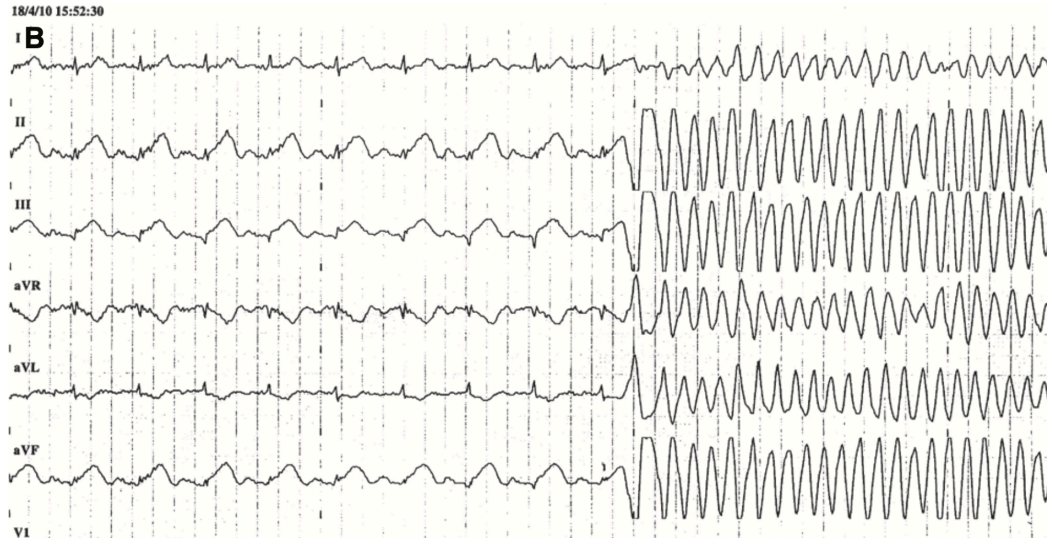
depolarize excitable gap $\rightarrow$ reentry $\rightarrow$ fibrillation
Electrical cardioversion ✓
for acute rhythm termination reentry only ✗

Ablation to modify scars
(moderate effectiveness)

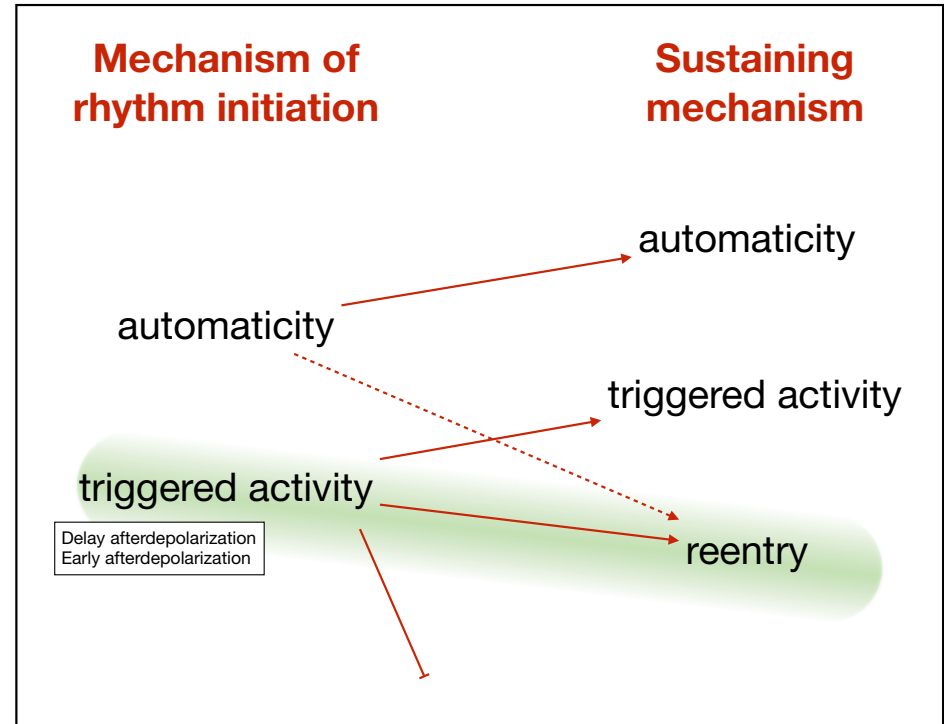
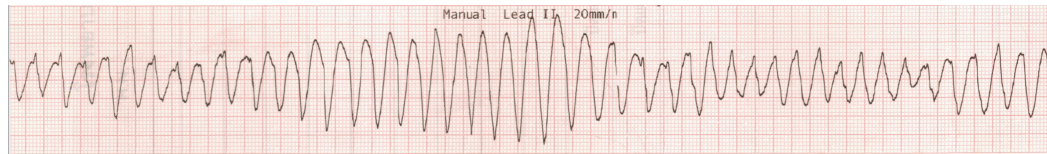
Defibrillator implantation

Polymorphic ventricular tachyarrhythmia

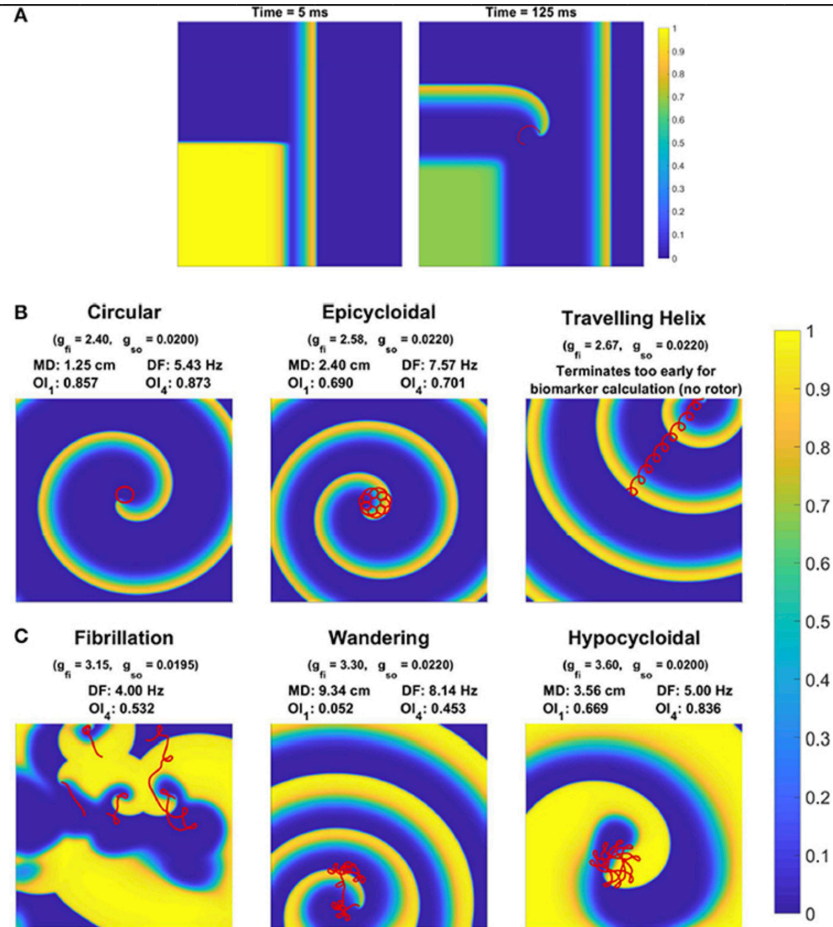
= Ventricular fibrillation



S Viskin, et al. Circulation. 2021;144:823–839.

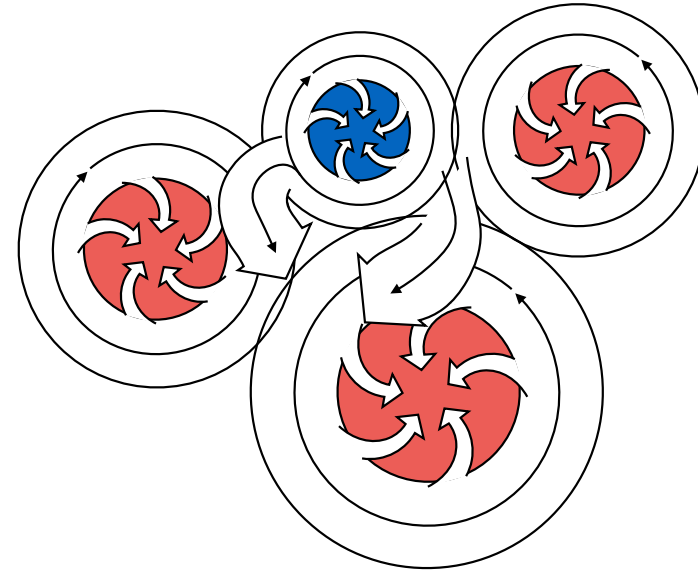


mechanism for sustain polymorphic VT/ventricular fibrillation



Functional reentry

-ไม่พบบน myocardium ที่ heterogeneous → repolarization ไม่สม่ำเสมอ
ทั้งหมด



Multiple wavelets → ventricular fibrillation

Ventricular fibrillation

Electrical therapy 100



Polymorphic VT → Ventricular fibrillation (VF)

disorganized
ventricular contraction

Loss all
cardiac output

cardiac
arrest

Thank you for your attention

