DISORDERS OF HEART RHYTHM; HYPERTENSION

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DEPARTMENT OF PATHOLOGICAL PHYSIOLOGY, 2025/2026

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DISORDERS OF HEART RHYTHM

ARRHYTHMIA - DYSRHYTHMIA

- Abnormal rhythm of heart (frequency, regularity)
- Aberant rhythm can be initiated anywhere from SA to myocytes

- Pathophysiology: Altered electrophysiological properties of cardiomyocytes
- abnormalities in structure or spatial distribution of gap junctions (ischemic heart disease, dilated cmp)
- Spontaneous aberant depolarisation of myocytes

- Causes: ischemic (most common!), metabolic, ion dysbalance, inflammation, bacterial toxins, depositions (amyloidosis), channelopathies, drugs
  - Ischemic injury- direct damage, dilation of chambers- altered conduction system firing

IMPAIRMENT OF CURRENT CONDUCTION

- SA blocks
- Preexcitatory syndromes
- AV block
- Tawar branch blocks
- Re-entry

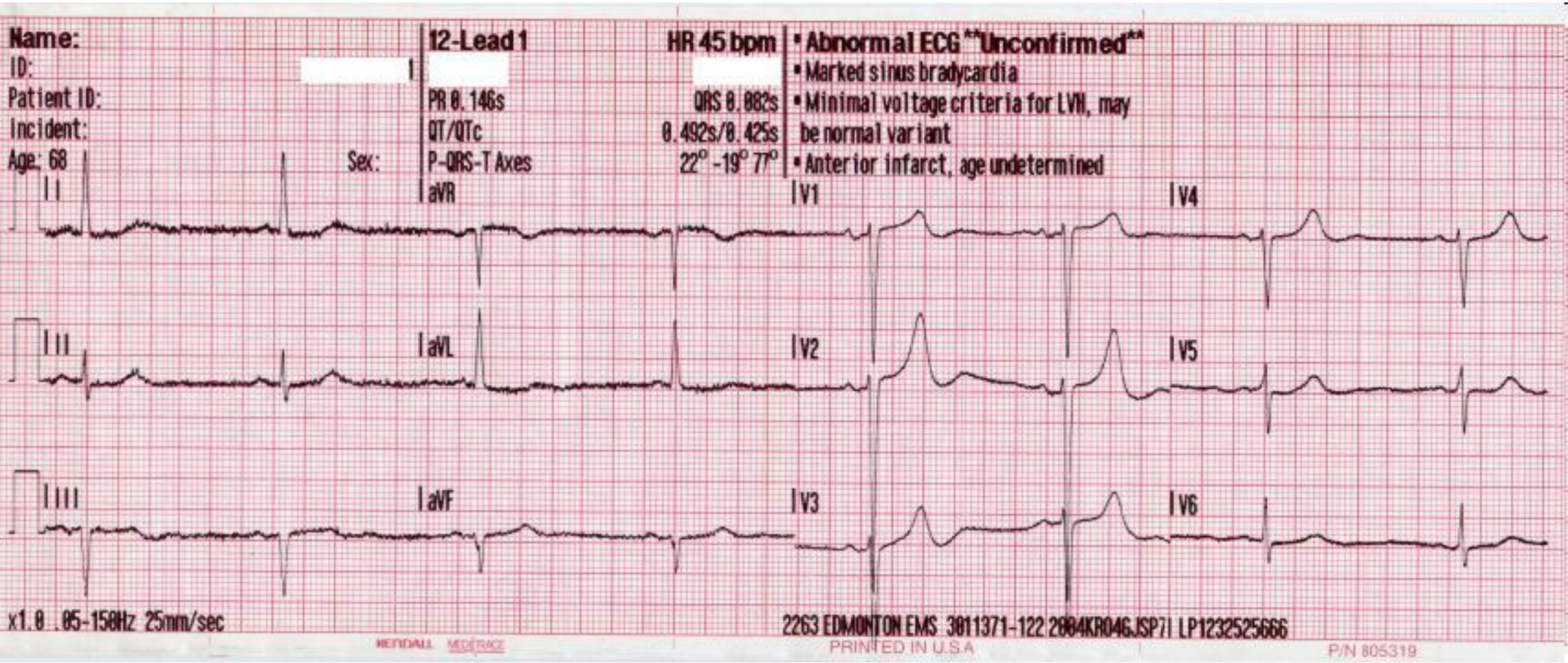
CLASSIFICATION

- Frequency
  - Regular, <60/min - Bradycardias
  - Irregular, <60/min - Bradyarrhythmias
  - Regular, >100/min - Tachycardias
  - Irregular, >100/min – Tachyarrhythmias
- Location
  - Supraventricular – SA node, atrial
  - Ventricular – AV node and ventricles

- *Pathophysiological classification is not possible currently due to lack of data*

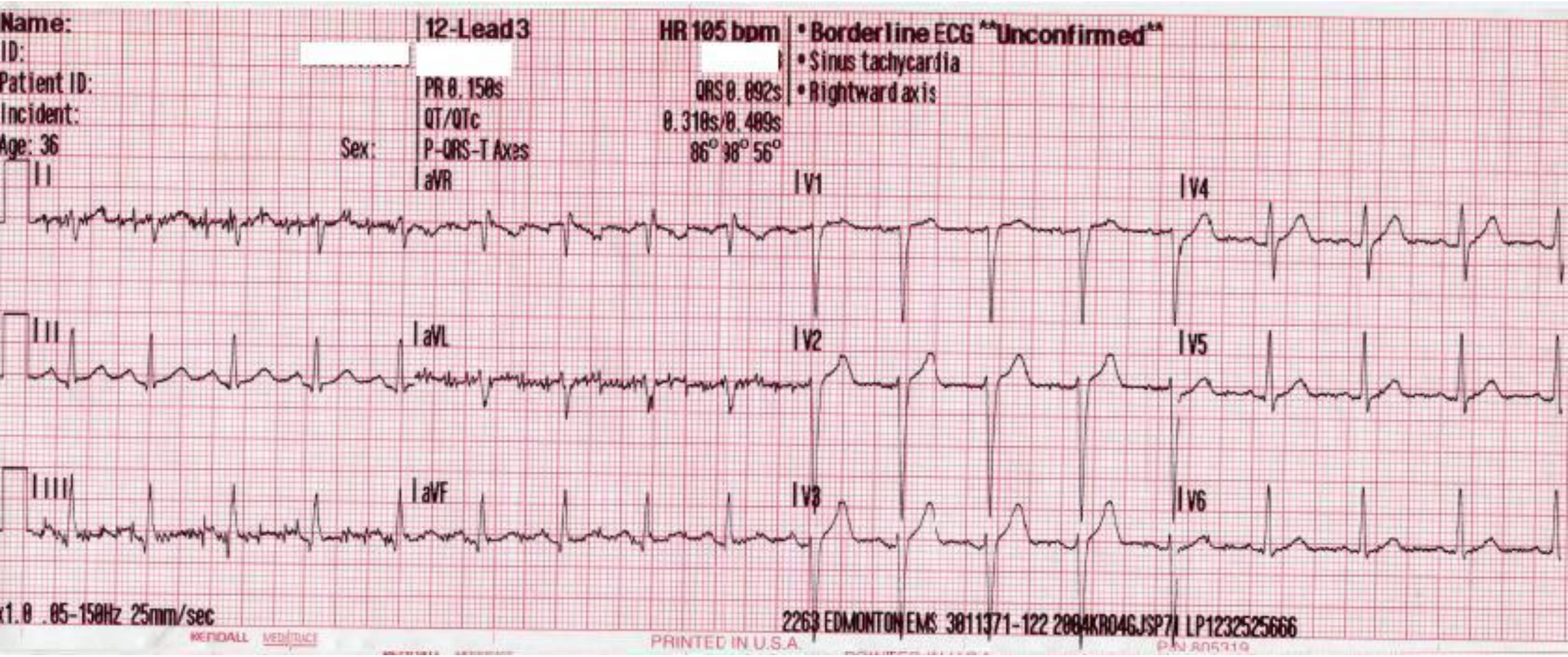
SINUS BRADYCARDIA

- Frequency: <50/min (cyclist F. Choppi only 40/min!)
- Regular: YES
- Causes:
  - Parasympathicus hyperactivity (vagal reflexes – blood pressure elevation, sinocardial reflex, oculocardial reflex, pain, fear)
  - May be accompanied with hypersalivation and nauzea
  - Hypothermia
  - Hypothyreosis
  - Trained persons (sport)
  - Part of physiological respiratory arrhythmia


Name:
ID:
Patient ID:
Incident:
Age: 68
Sex:
12-Lead 1
PR 0.146s
QT/QTc
P-QRS-T Axes
aVR
HR 45 bpm
QRS 0.082s
0.492s/0.425s
22° -19° 77°
• Abnormal ECG **Unconfirmed**
• Marked sinus bradycardia
• Minimal voltage criteria for LVH, may be normal variant
• Anterior infarct, age undetermined
I
II
III
aVL
aVF
V1
V2
V3
V4
V5
V6
x1.0 .05-150Hz 25mm/sec
2263 EDMONTON EMS 3011371-122 2004KR046JSP7I LP1232525666
KENDALL MEDITRACE
PRINTED IN U.S.A
P/N 805319

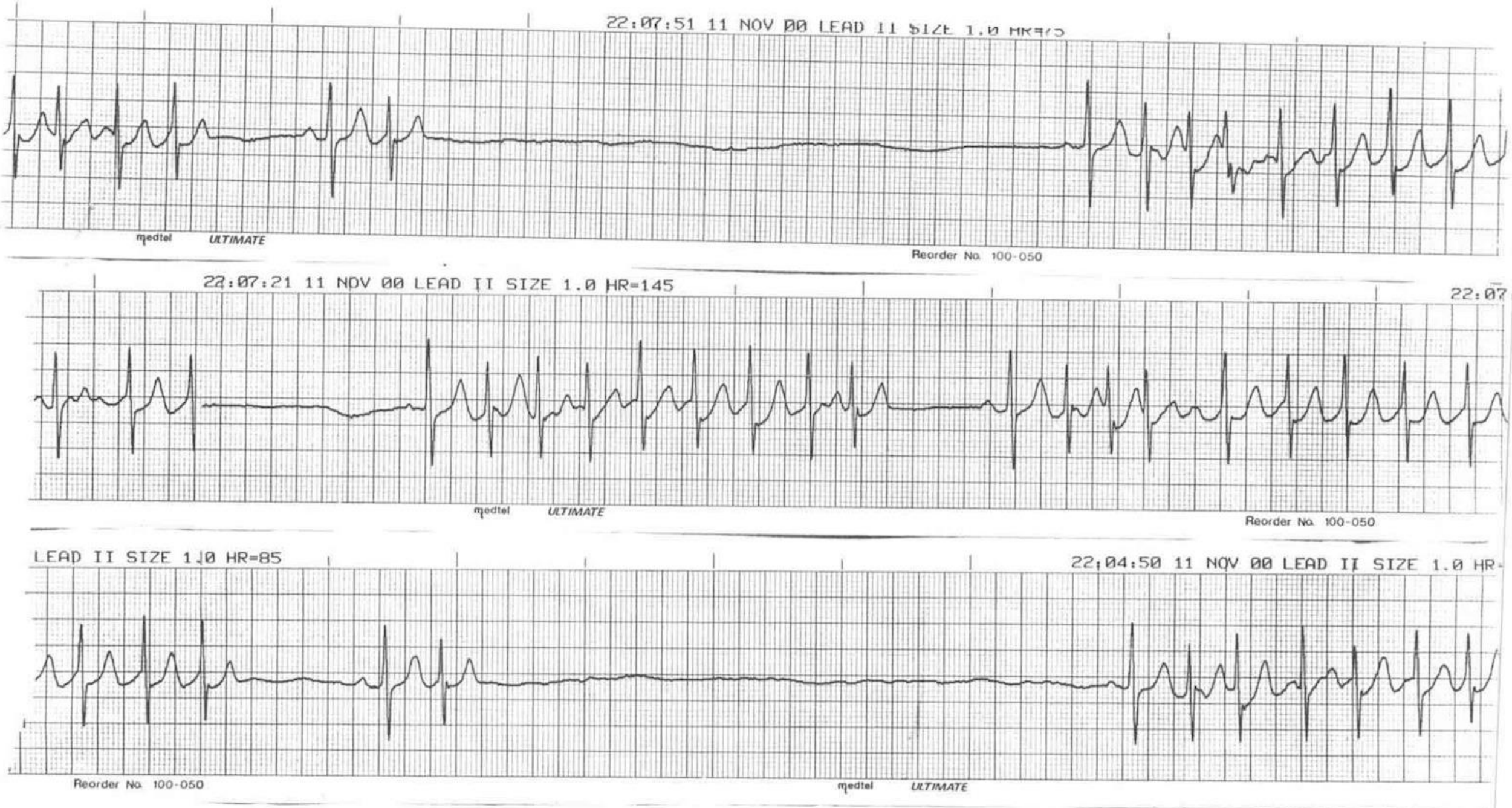
SINUS TACHYCARDIA

- Frequency: >100/min
- Regular: YES
- Causes:
  - Sympato-adrenal reaction – physical activity, emotions, blood pressure drop, hypoxia
  - Body temperature increase (each °C increases frequency for cca 10 %)
  - Hyperthyreosis


Name:
ID:
Patient ID:
Incident:
Age: 36
Sex:
12-Lead3
PR 0.150s
QT/QTc
P-QRS-T Axes
aVR
HR 105 bpm
QRS 0.092s
0.310s/0.409s
86° 38° 56°
• Borderline ECG **Unconfirmed**
• Sinus tachycardia
• Rightward axis
I
II
III
aVL
aVF
V1
V2
V3
V4
V5
V6
x1.0 .05-150Hz 25mm/sec
2263 EDMONTON EMS 3011371-122 2004KR046JSP7I LP1232525666
PRINTED IN U.S.A.

SINUS ARREST AND SICK SINUS SYNDROME (SSS)

- Frequency: <60/min (or >100/min)
- Regular: PARTIAL (escaped excitation/rhythm)
- Causes:
  - Ischemic damage
  - Acidosis, ion disbalance
  - Amyloidosis, etc.
- Manifestation:
  - Bradycardia with low increase during physical activity
  - Syncopes (heartbeat pause >3s)
  - Paroxysmal supraventricular tachycardia may occur

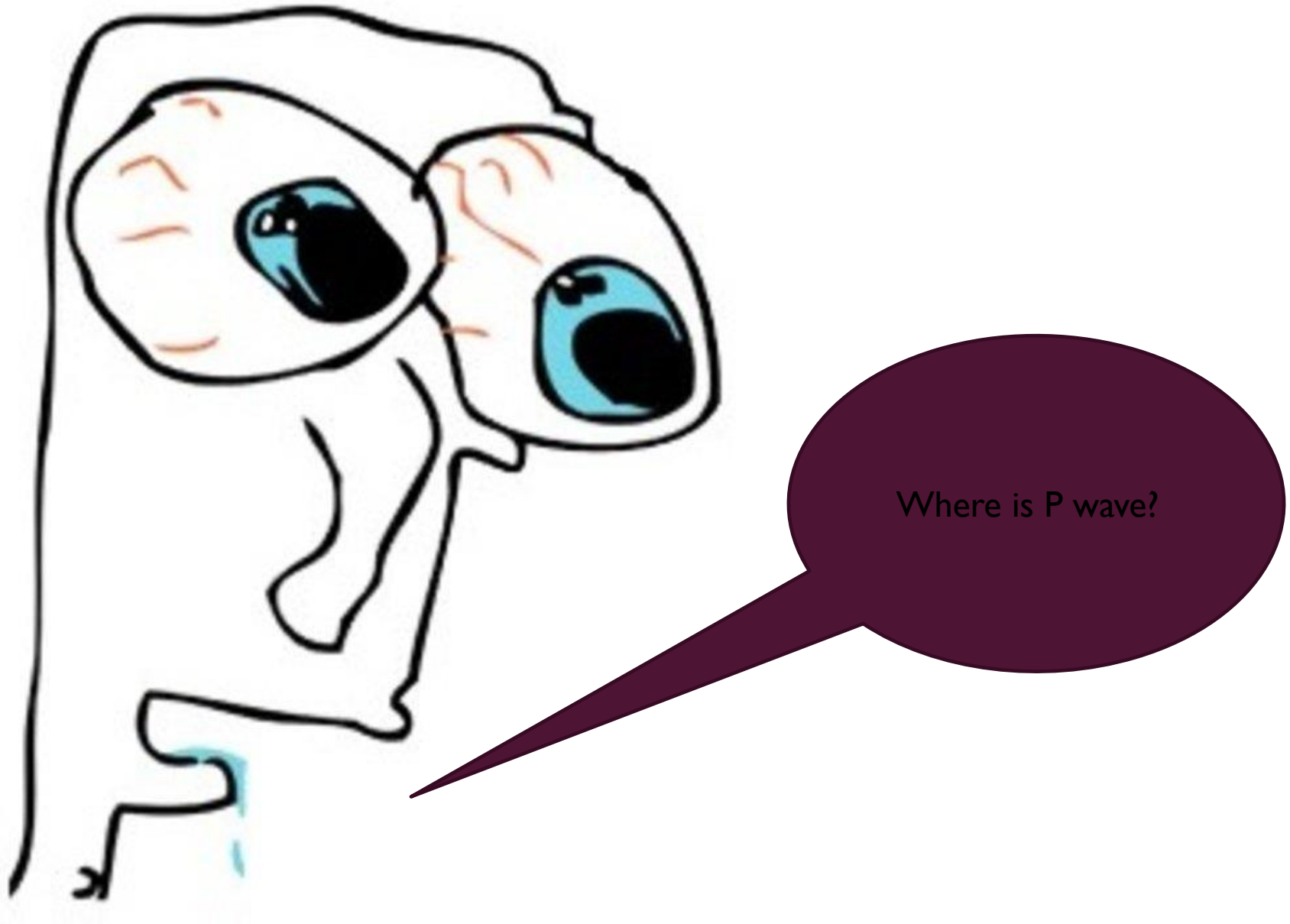

22:07:51 11 NOV 00 LEAD II SIZE 1.0 HR=75
medtel ULTIMATE
Reorder No. 100-050
22:07:21 11 NOV 00 LEAD II SIZE 1.0 HR=145
22:07
medtel ULTIMATE
Reorder No. 100-050
LEAD II SIZE 1.0 HR=85
22:04:50 11 NOV 00 LEAD II SIZE 1.0 HR=
Reorder No. 100-050
medtel ULTIMATE

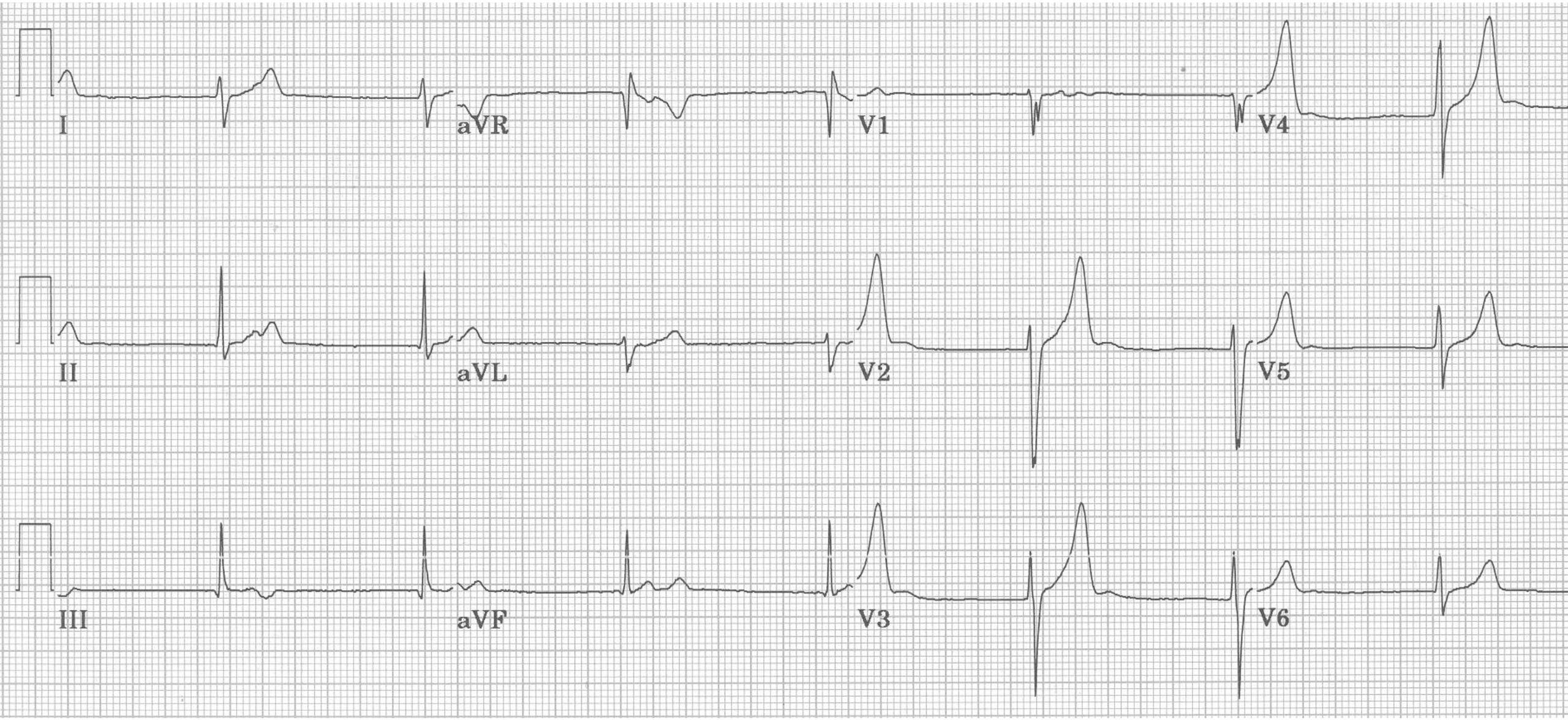
HETEROTOPOUS EXCITATIONS

- The location of origin is outside the heart conduction system
- Classification
  - Passive (escaped rhythms and excitations)
  - Active (extrasystoles or tachyarrhythmias)

PASSIVE HETEROTOPOUS EXCITATIONS

- AV nodus can depolarize in case of SA disorder
- Excitation is delayed, has tendency to progress to bradycardia
- Frequency: <45-50/min
- Regular: YES
- Cause:
  - Damage of SA node or total isolation of atrias


Where is P wave?


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

ACTIVE HETEROTOPOUS EXCITATIONS

- Other locus is more active than SA node
- Classification:
  - Single – extrasystole
  - Plural – tachyarrhythmias

EXTRASYSTOLES

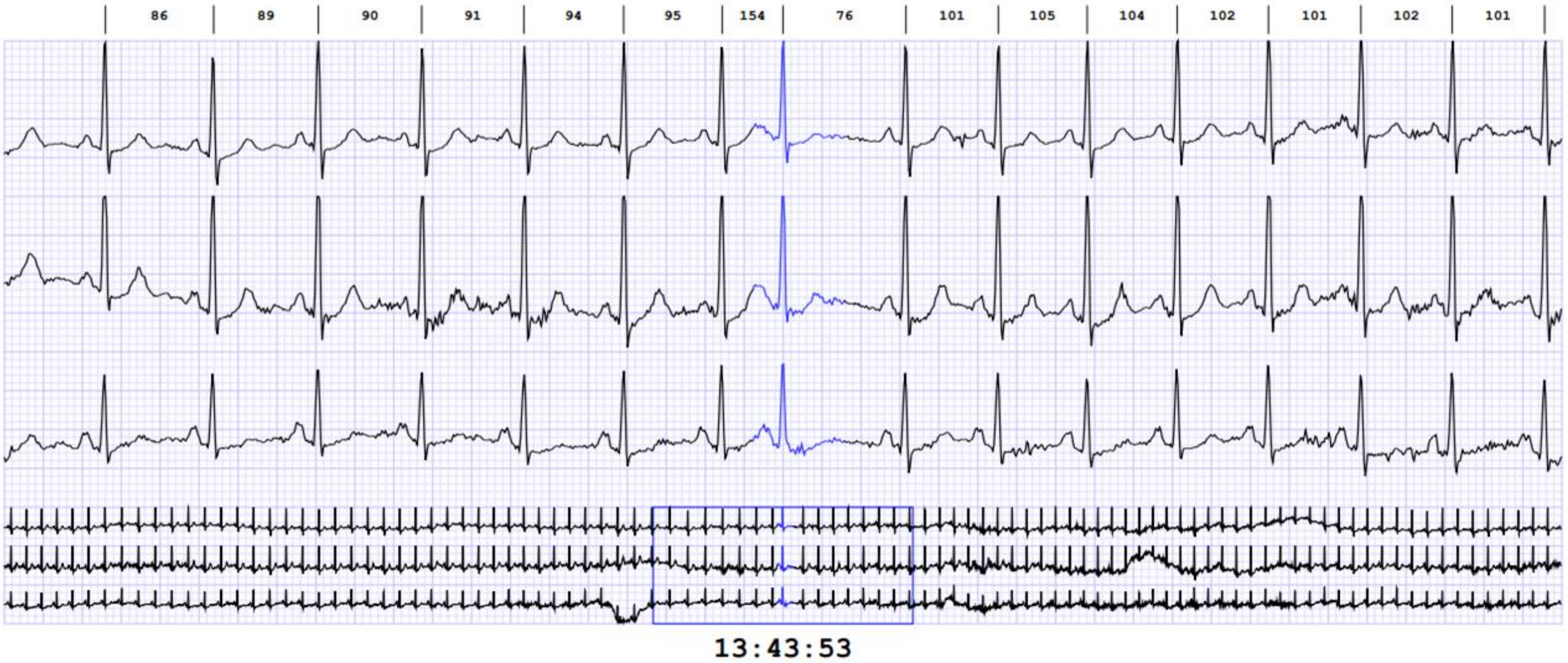
- Frequency: Varies
- Regular: mostly YES
- Cause:
  - Ischemia/myocardial hypoxia
  - Ion disbalance/acidosis
  - Coffein, drugs may enhance
- Pathophysiology
  - Ectopy or reentry
  - SA node potential is ignored – ventricles are depolarized – SA node may or may not be reset

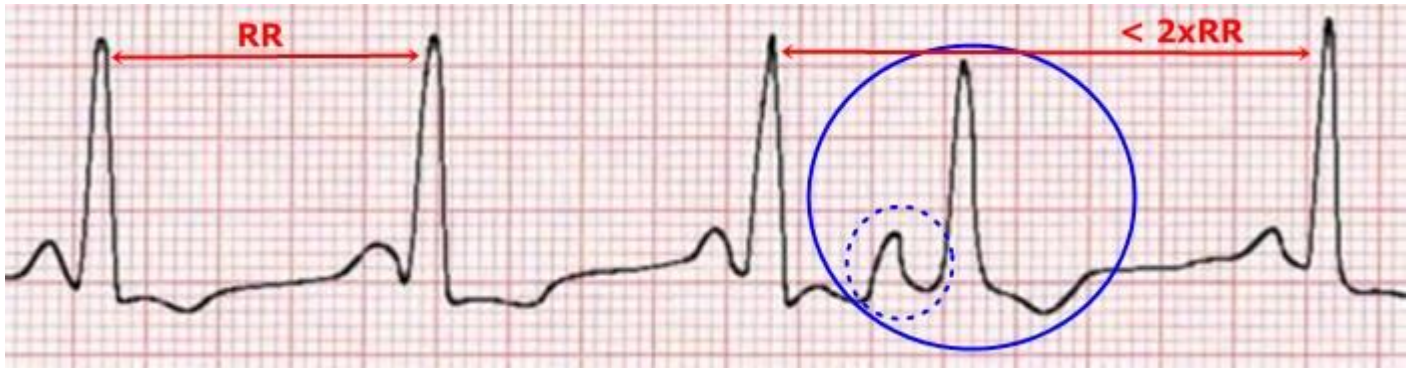
EXTRASYSTOLES

- Maximum 5/min is considered to be normal (even ventricular!)
- Classification in order of origin:
  - Supraventricular
  - Junctional
  - Ventricular

SUPRAVENTRICULAR EXTRASYSTOLES

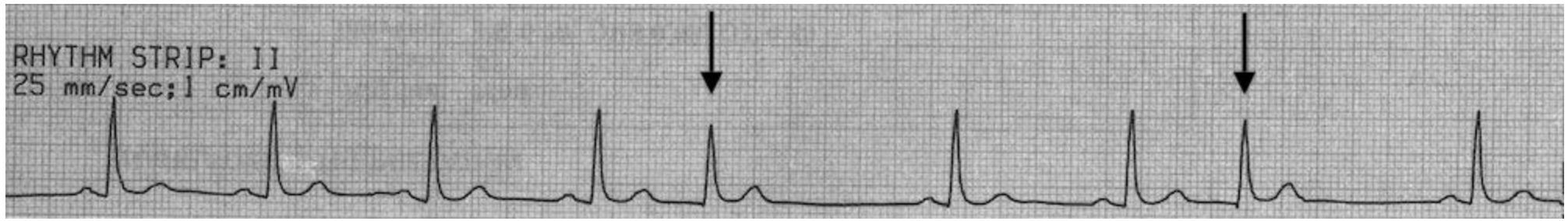
- Frequency: varies
- Regular: partly YES
- Manifestation:
  - Abnormal P wave – depolarization pathway changed (biphasic, inverted, etc.)
  - PQ interval changed (the smaller the closer to AV node)
  - QRS complex not changed
  - Compensation pause is incomplete (R-R‘ distance is changed, depolarization is resetting SA node)
  - R-R‘ coupling – remains same (reentry), varies (ectopy)
  - No hemodynamics change


86
89
90
91
94
95
154
76
101
105
104
102
101
102
101
13:43:53


RR
< 2xRR

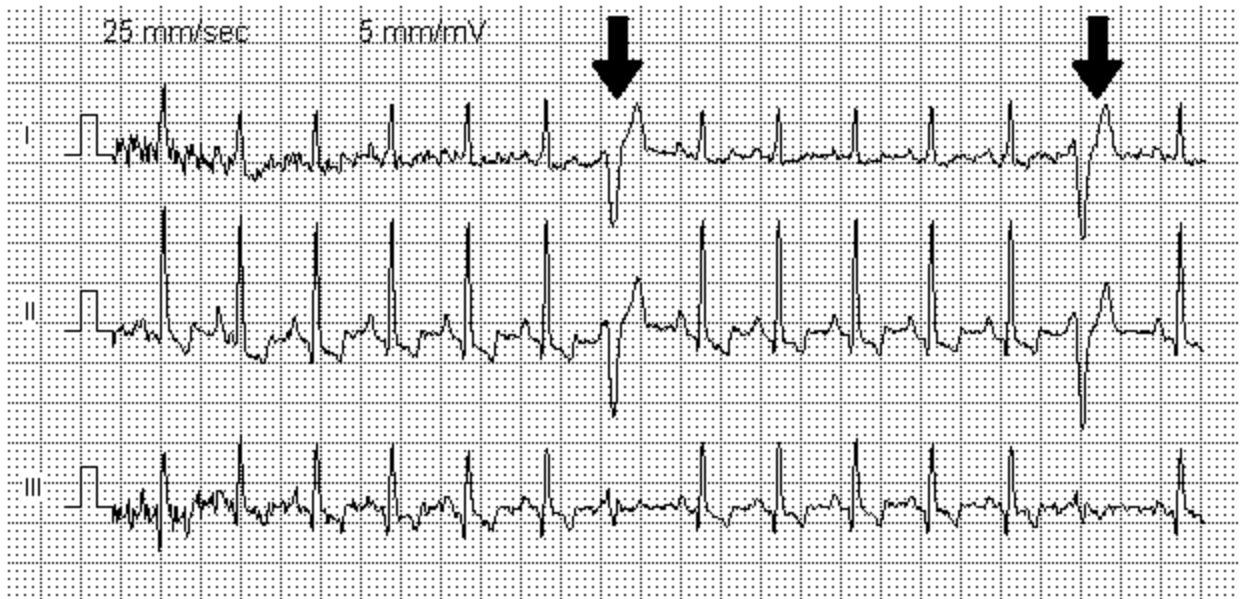
JUNCTIONAL EXTRASYSTOLES

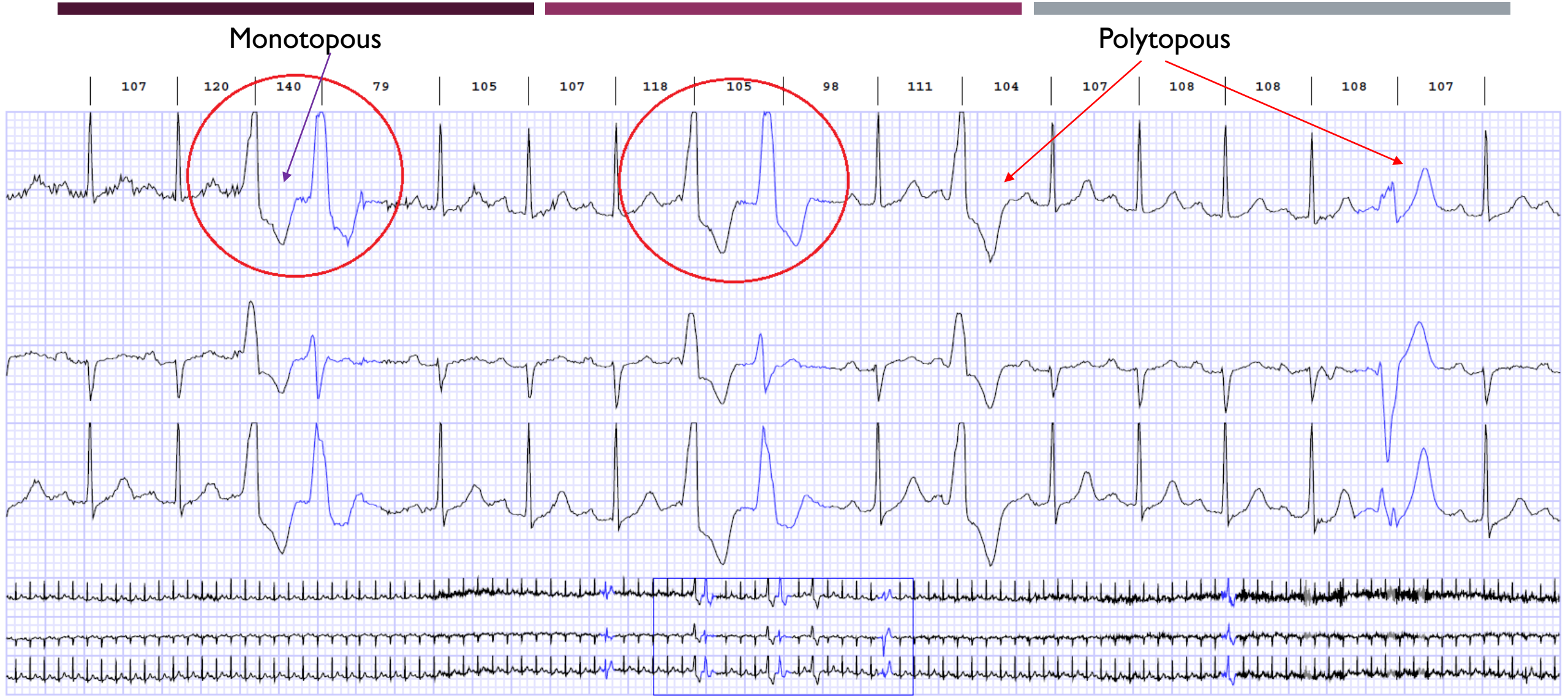
- Frequency: varies
- Regular: partly YES
- Manifestation:
  - No P wave (hidden behind ventricular depolarization)
  - No hemodynamics change
  - QRS mostly normal
  - SA node may or may not be reset


RHYTHM STRIP: II
25 mm/sec;1 cm/mV

VENTRICULAR EXTRASYSTOLES

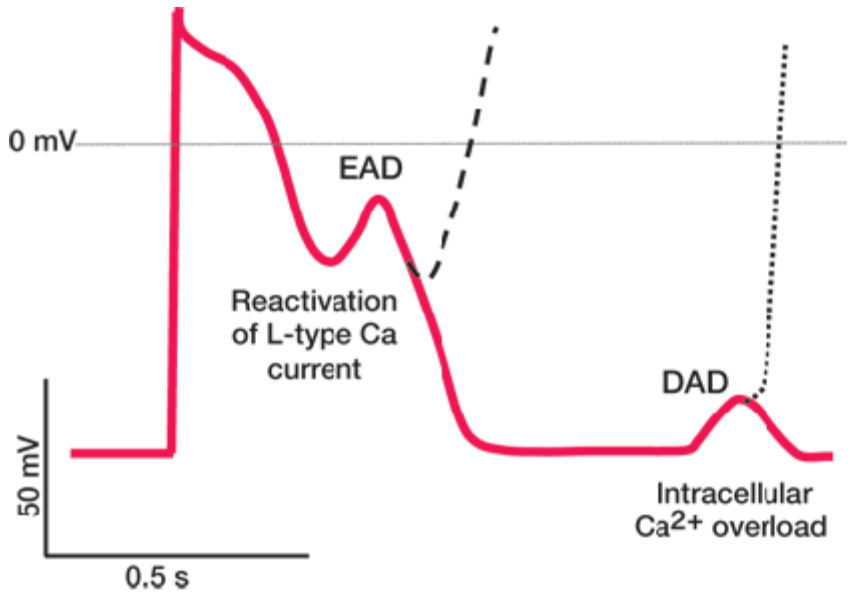
- Frequency: varies, may progress to tachyarrhythmia
- Regular: mostly YES
- Manifestation:
  - QRS bizarre and deformed (locus is distal from Hiss branch, pathway aberrant)
  - SA node is not affected (AV node will stop retrogradeous potential)
  - Totally compensated (SA impulse may catch ventricles depolarised)
  - R-R‘ distance remains still same
  - May be sole or coupled (bigeminal, trigeminal, etc.)
  - May be monotopous or polytopous (QRS shape is changing, more dangerous!)
  - May progress to ventricular tachycardia! (QRS shaped as initial extrasystole)
  - Reentry or early/delayed depolarisation


25 mm/sec
5 mm/mV
I
II
III

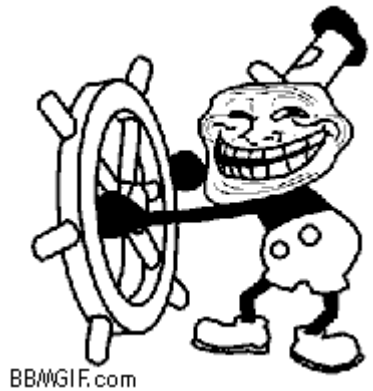

Monotopous
Polytopous
107
120
140
79
105
107
118
105
98
111
104
107
108
108
108
107

TRIGGERED ACTIVITY

- Cyclic action with repeated spontaneous myocardial depolarization
- Electric conduction system is not affecting these
- Predispose to tachycardia, tachyarrhythmia
- Classification according to cause:
  - Early after depolarzation
  - Delayed after depolarization



Source: Fauci AS, Kasper DL, Braunwald E, Hauser SL, Longo DL, Jameson JL, Loscalzo J
Harrison's Principles of Internal Medicine, 17th Edition: http://www.accessmedicine.com


BBWGIF.com

EARLY AFTER DEPOLARIZATION

- Negative potentials are not achieved – Na^{+} and Ca^{2+} canals reopened too early – repetetive depolarization independent on conduction system
- Frequency: >100/min
- Regular: YES
- Manifestation:
  - Sinusoid changes in amplitude of R wave
  - Torsade de pointes – sudden invertion of R wave, then revertion, repeated (may be fatal, in case of diagnosing defibrilation indicated)

EARLY AFTER DEPOLARIZATION

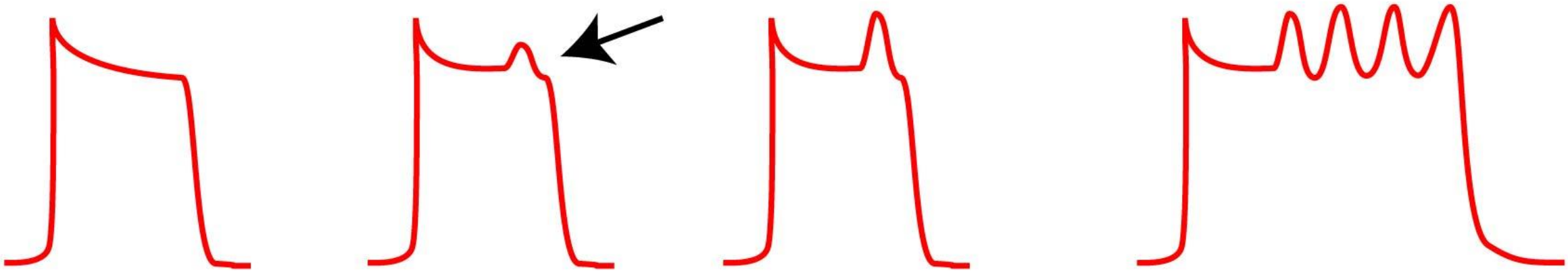
- Cause:
  - Hypoxia
  - Hypokalemia
  - Hypothermia
- Consequences
  - Hemodynamic changes may result in lowered cardia output (diastole affected, contraction not properly effective)

normal
action potential

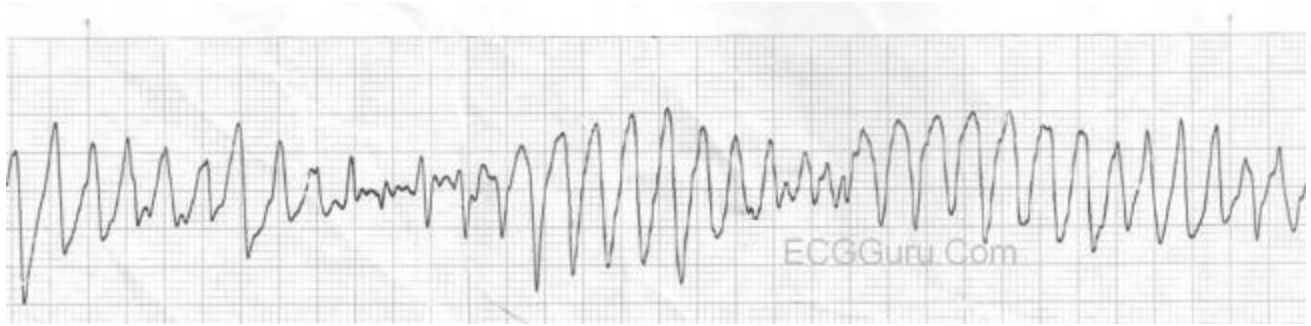
Early
After Depolarization

Single
Triggered AP

Multiple
Triggered AP's

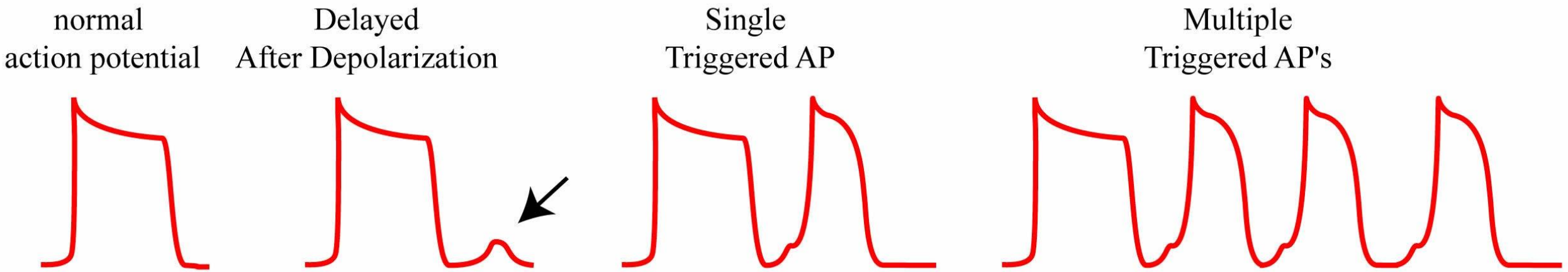


Torsade de pointes

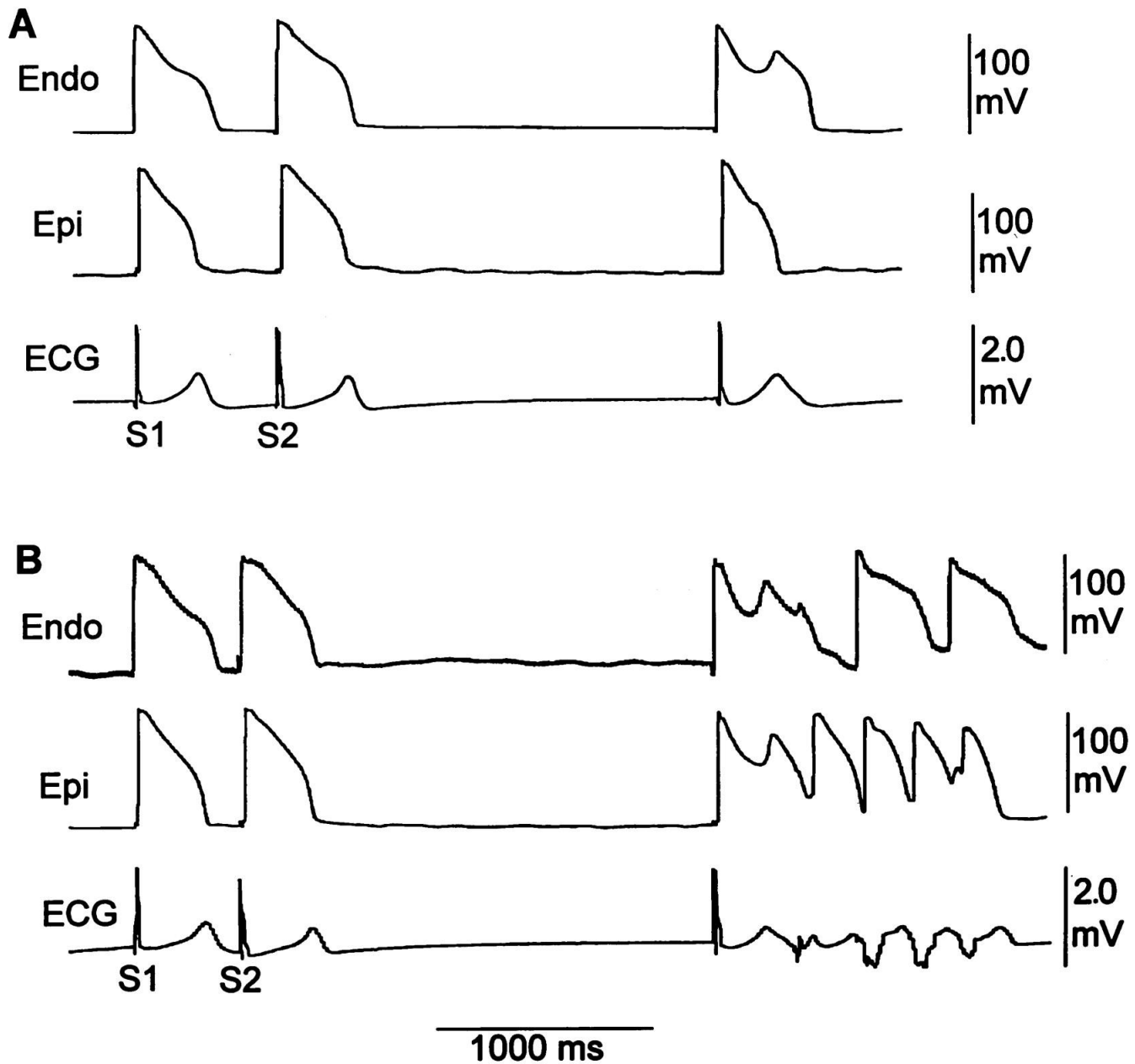


DELAYED AFTER REPOLARIZATION

- Spontaneous fluctuation between total repolarization/hyperpolarization and depolarization
- Cause:
  - Hyperkalcemia
  - Digitalis overdose
- Pathophysiology
  - Na^+ channels reopened
- Frequency: >100/min or normal (may lead to tachyarrhythmia)
- Regular: mostly NO


normal
action potential
Delayed
After Depolarization
Single
Triggered AP
Multiple
Triggered AP's

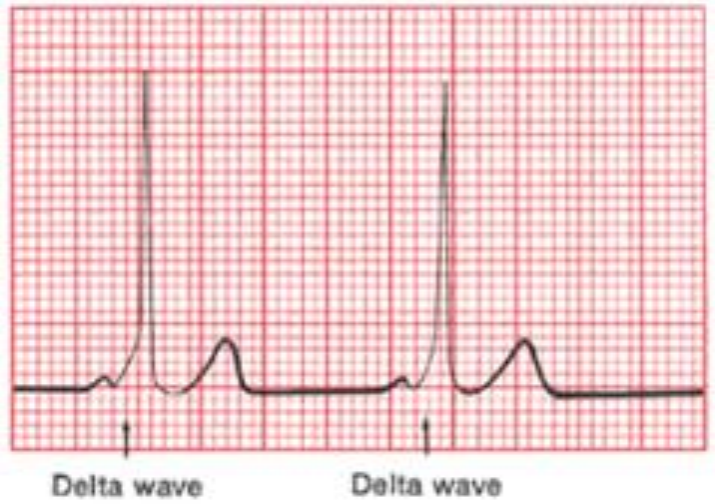
Isolated Rabbit Left Ventricle
dl-sotalol 100 μM



PREEXCITATORY SYNDROMES

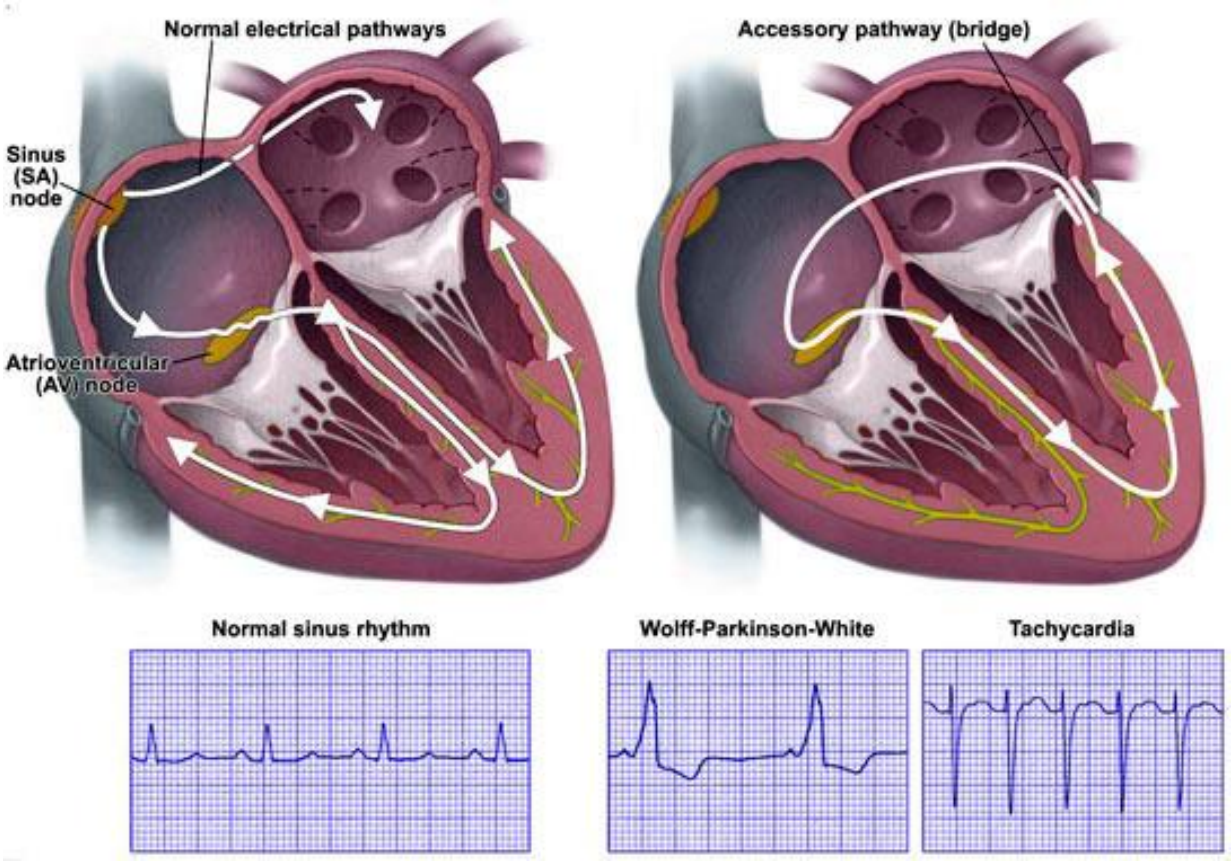
- Cause: aberrant abnormal connection between atrial and ventricular myocardium, paralel to AV node; through anulus fibrosus
  - Less delayed conduction than AV block – aberant pathway
- Most common anatomic variant : Kent´s fascicle

- Early depolarisation of ventricles – ECG changes:
  - short PQ interval
  - δ- wave : part of QRS complex, together > 0,1 s



- Congenital preexcitatory: Wolf-Parkinson-White syndrome (WPW-syndrome): paroxysmal achycardia

- Syndrome of short PQ = Lown-Ganong-Levin´s syndrome (LGL-syndrome) : absent δ-wave, short PQ interval



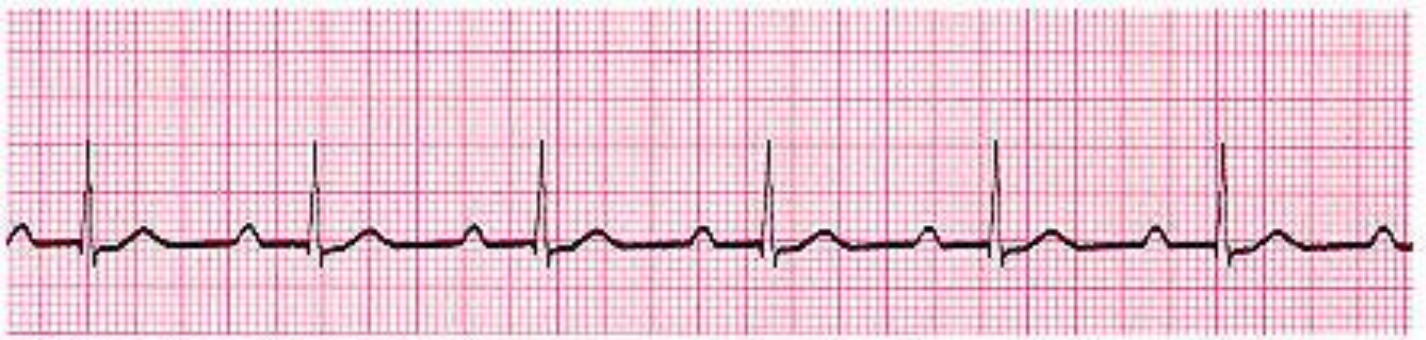
SA BLOCKS

- I. degree: delayed depolarisation wave from SA node
  - No hemodynamic consequences, no changes on a classic ECG graph
- II. degree: some impulses are not transmitted to contractile myocardium of atria nor AV node
  - Absence of the relevant P wave and QRS, hemodynamic consequence: absence of pulse wave
- III. Degree: no impulse exits SA node
  - Substitutional rhythm takes place
  - Same presentation as SA arrest (can be distinguished by a special ECG monitoring)

AV- BLOCKS- DEGREE I

- AV- block Degree I.: transmission of current from atria to ventricles is constantly prolonged over 0,2 s (PQ is same)
- No hemodynamic effect, silent 1st heart sound
- Cause- damage of AV node: metabolic, drugs (digitalis, beta-blockers, Ca2+ channel blockers), increased tone of n. X.

- ECG: prolonged, regular PQ

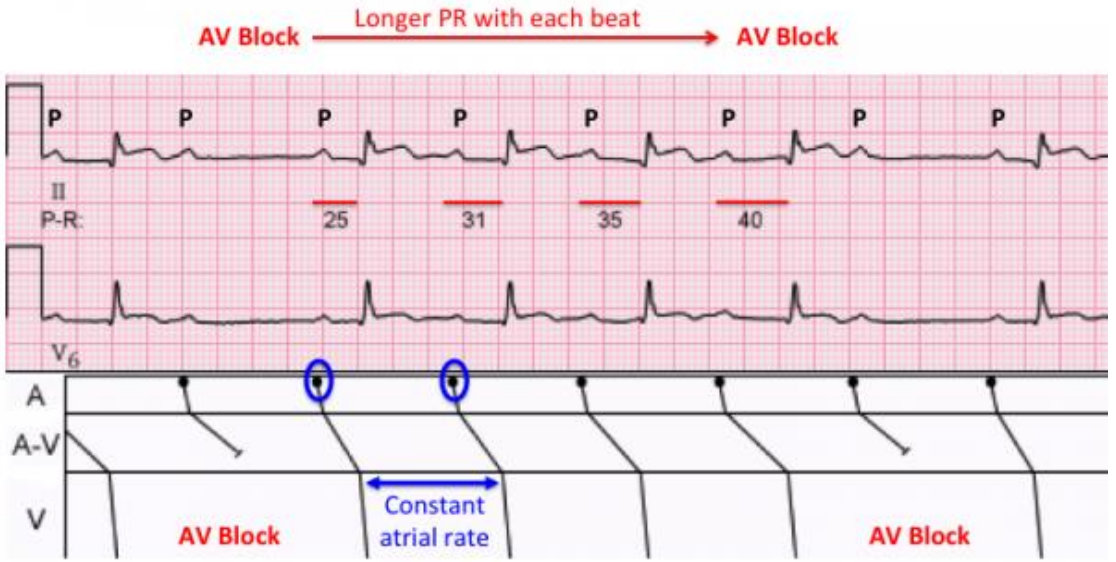

1st Degree Heart Block

AV BLOCKS – DEGREE II

Type I = Mobitz I = Wenkenbach type

- Wenkenbach periods = proggressive prolongation of PQ interval, until QRS complex skips with only P wave present, and new cycle begins
- Periods gradually prolong from less than 0,2 s to more than 0,2 s

- n: n-1
- E.g. 5:4, 4:3, 3:2, ...



AV BLOCKS – DEGREE II

Hemodynamic effect: small significance, irregularity of pulse

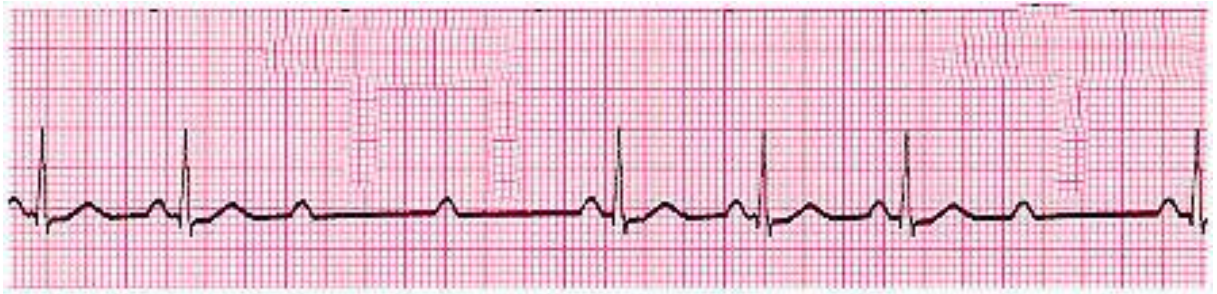
- Heart sounds: similar to grade I.

- Rarely in young people, children, trained sportsmen with increased tone of n. X.

AV BLOCKS - DEGREE II

- Type II. = Mobitz 2
- Constant length of PQ interval with normal period (<0,2s), but occassional absence of transmission of impulse from atria to ventricle
- (same PQ, with occasional absent QRS)

- More QRS can be missed, or AV block degree III. can occur !
  - Sudden decrease in cardiac output, cardiac syncope, Adams-Stokes syndrome



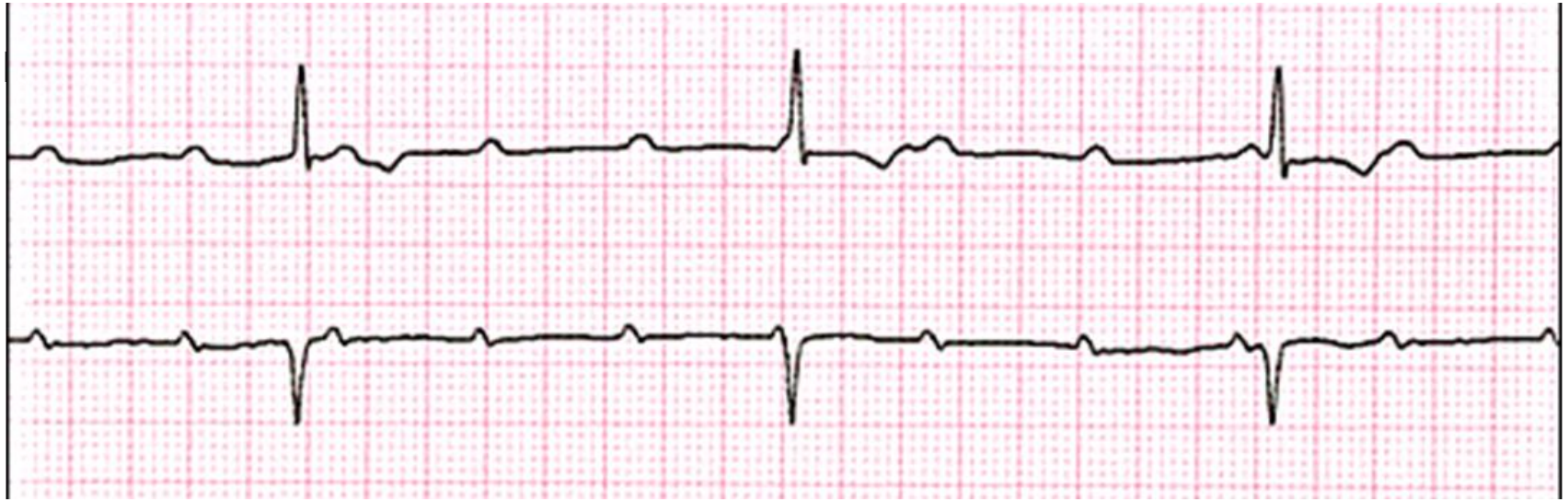
Mobitz Type II

AV BLOCKS – DEGREE III.

- No impulses are transmissed from atria to ventricle
- Activation of substitutional rhythm – (AV-nodal rhythm, or more distal;, f= 35-50/min, stable and slow)
- Atria and ventricles work separately (no coordination) = **disociation** of atria and ventricles
- AV is less affected by sympato-adrenal activity – stable frequency also during physical activity

AV BLOCKS – DEGREE III.

- ECG: same



RE-ENTRY PHENOMENON

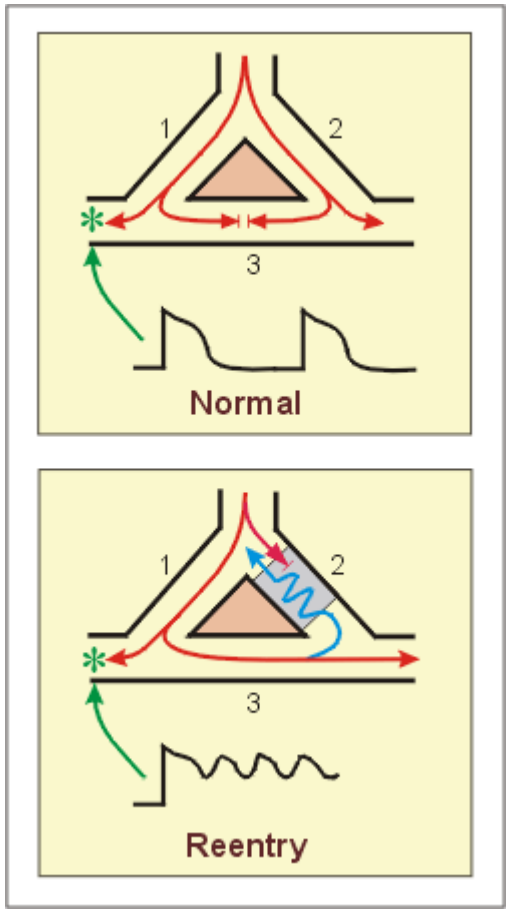
- Re-entry: block of depolarisation to certain locus, and its depolarisation of retrocedent pathway

- Constant depolarisation and repolarisation of a certain circuit of myocardium
- Aberant spreading, pathologic excitatory pathway- faster transmission of impulse that takes place instead of SA
- Depolarisation – contraction
- Causes increased heart rate- tachyarrhythmias

RE-ENTRY PHENOMENON

- micro- re-entry, macro-re-entry
- Random-reentry, sequenced (regular) re-entry

- Functional or morphologic change of myocardium that changes excitatory properties of myocardium (scar, congenital heart disease)

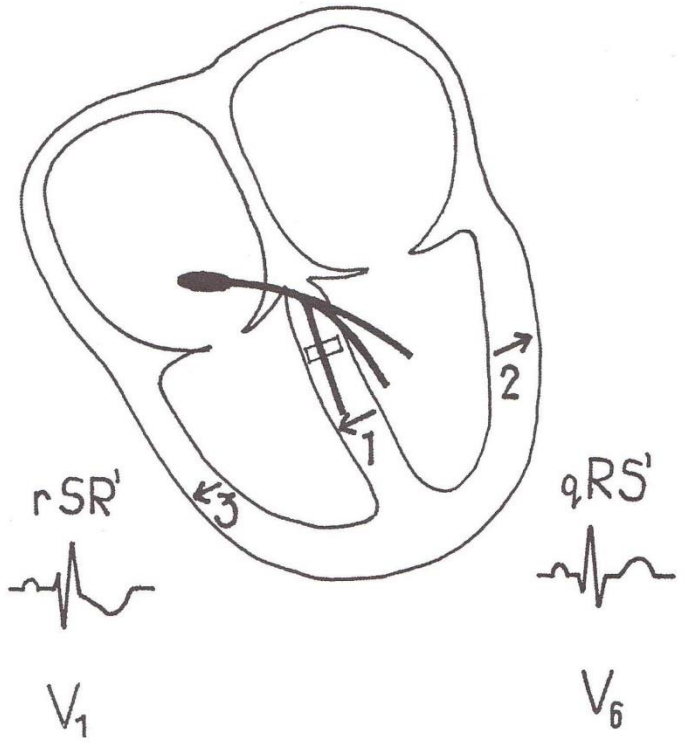


BLOCKS OF TAWAR´S BRANCHES

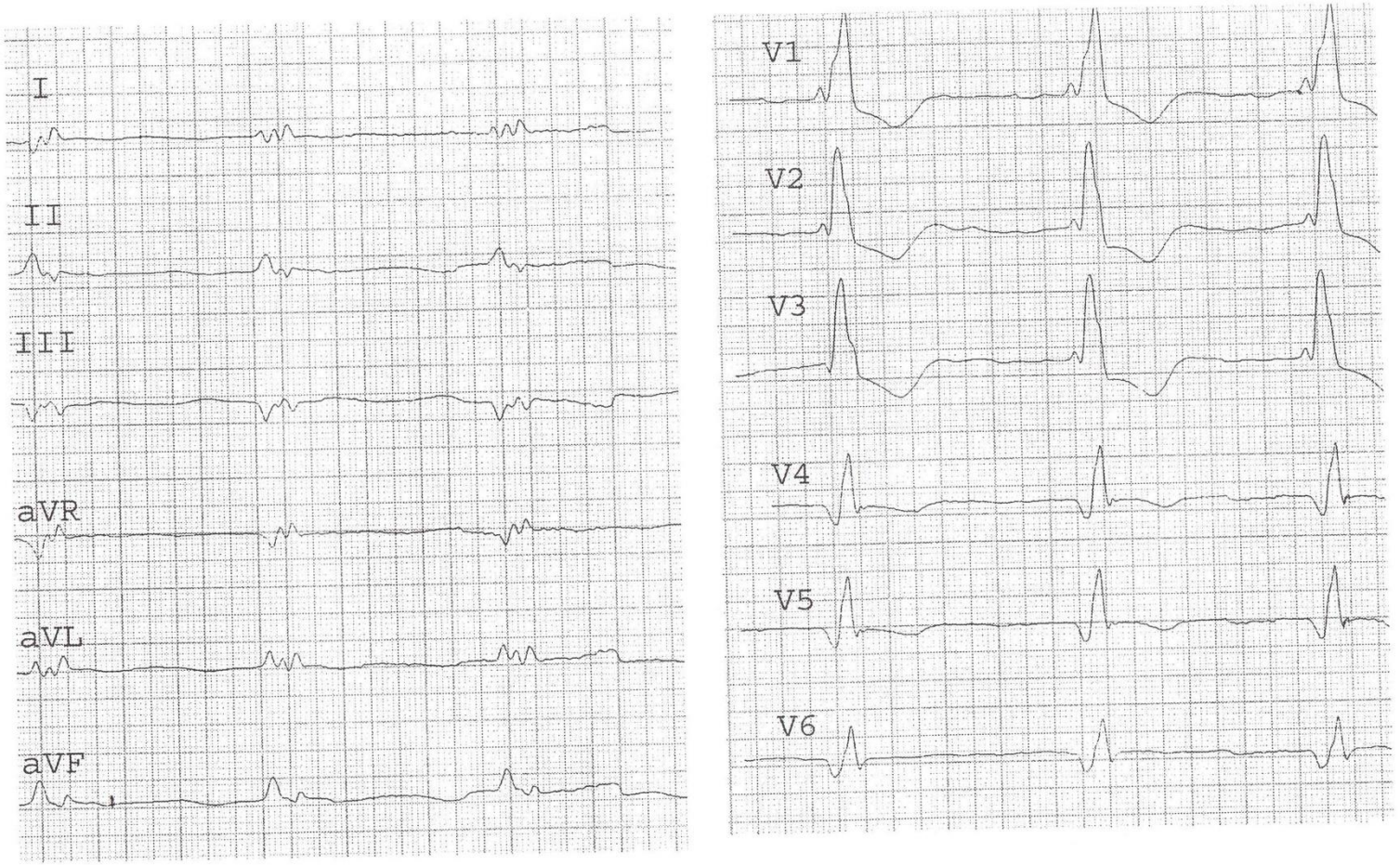
- Delayed depolarisation of affected ventricle
- ECG: QRS is broader, „cleft“ QRS: shape of W or M on V1 or V; longer than 0,1s
- Hemodynamic change: not significant

- Right Tawar´s branch block
- Left Tawar´s branch block
  - Left anterior fascicular block
  - Left posterior fascicular block
- Combined

RIGHT TAWAR´S BRANCH BLOCK

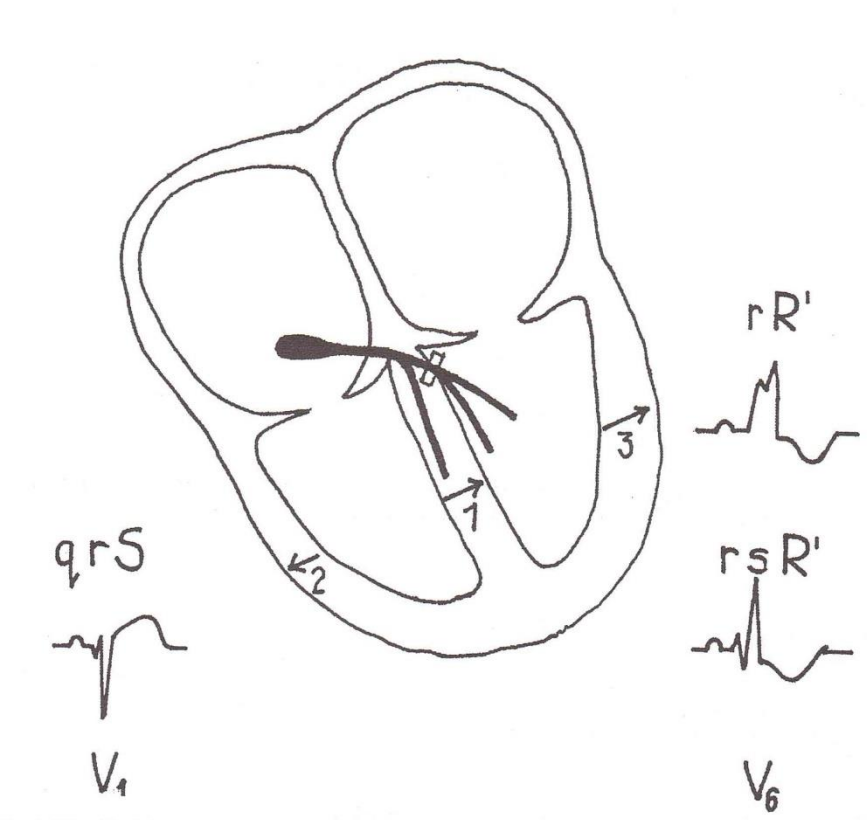


Right Tawar´s branch block. Adapted from: Mitro, P. Základy elektrokardiografie. 2003.

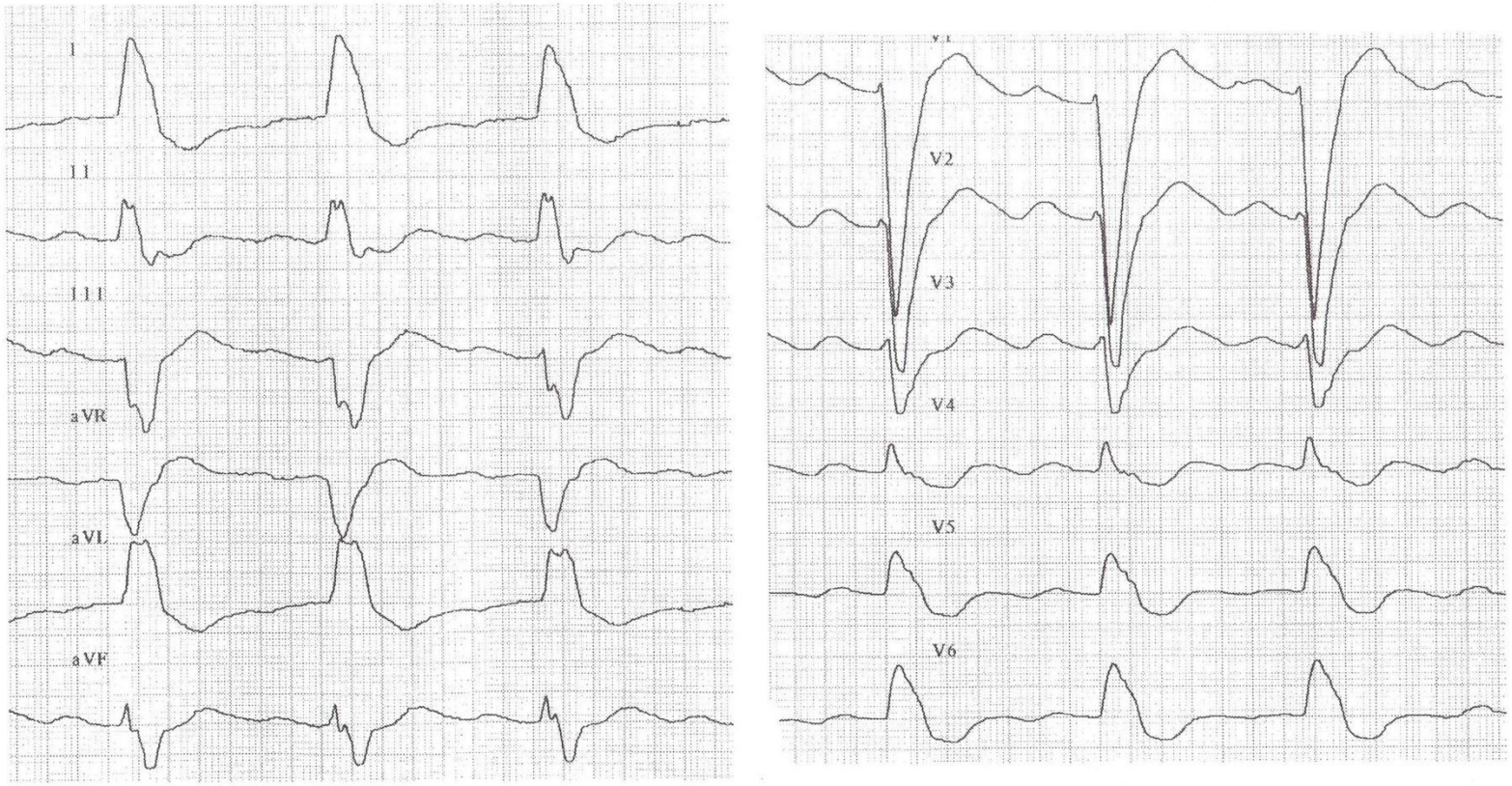


Right Tawar´s branch block. Adapted from: Mitro, P. Zákaldy elektrokardiografie. 2003.

LEFT TAWAR´S BRANCH BLOCK

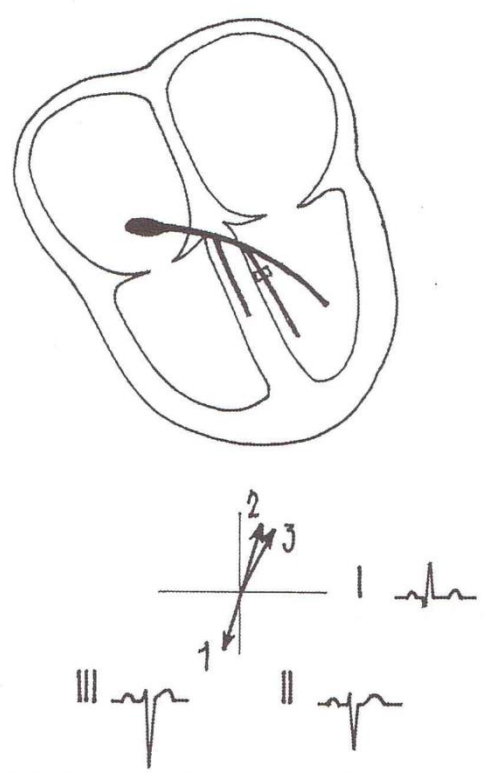


Left Tawar´s branch block. Adapted from: Mitro, P. Základy elektrokardiografie. 2003.

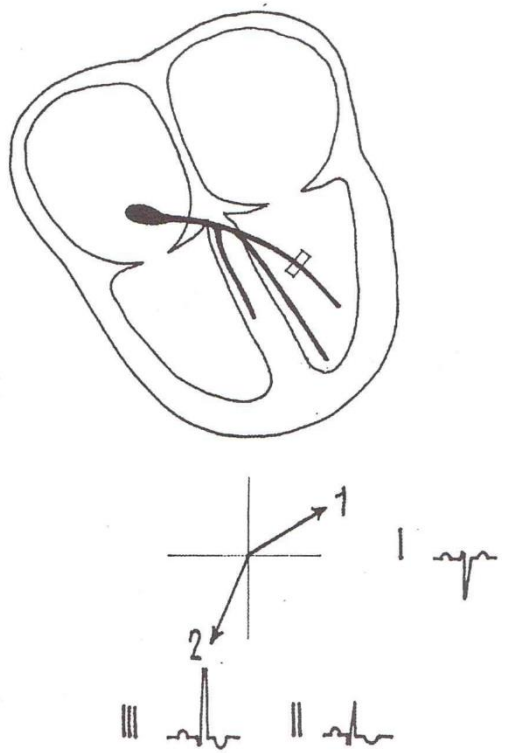


Left Tawar´s branch block. Adapted from: Mitro, P. Základy elektrokardiografie. 2003.

LEFT ANTERIOR AND LEFT POSTERIOR FASCICULAR BLOCK



A. Left anterior fascicular block.



B. Left posterior fascicular block

Adapted from: Mitro, P. Základy elektrokardiografie. 2003.

TACHYCARDIA

- Frequency: 100-180/250/min
- Regular: YES
- Cause:
  - Reentry/early/delayed after depolarization
- Classification:
  - Supraventricular
  - Ventricular

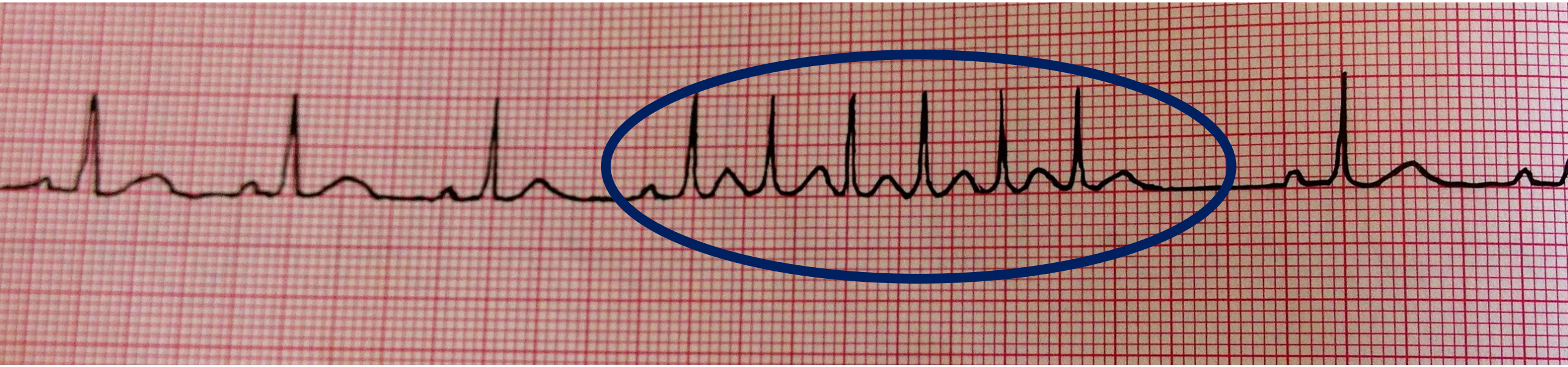
SUPRAVENTRICULAR TACHYCARDIA (SVT)

- Source:
  - Atrial myocardium, AV node
- Manifestation:
  - High frequency, QRS normal, P wave may be present
- Classification:
  - Atrial SVT
  - Atrioventricular junctional SVT
  - Atrioventricular reentry SVT

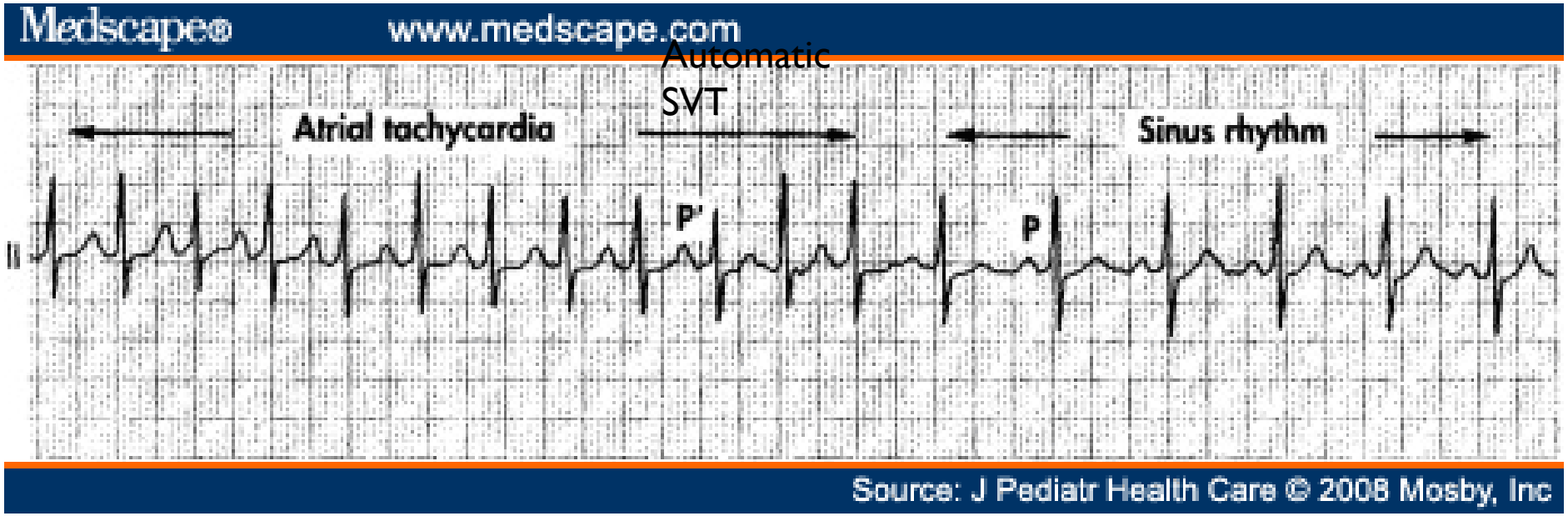
ATRIAL SVT

- Frequency: 100-200/min
- Regular: YES, may be paroxysmal
- Cause:
  - Reentry – paroxysmal SVT
  - Depolarization disorders – non-paroxyzmal SVT
  - Multiple locuses – multifocal – P waves various
- Manifestation
  - Paroxysmal – sudden beginning/end
  - Non-paroxysmal – continuously rises and ceases

Paroxysmal SVT

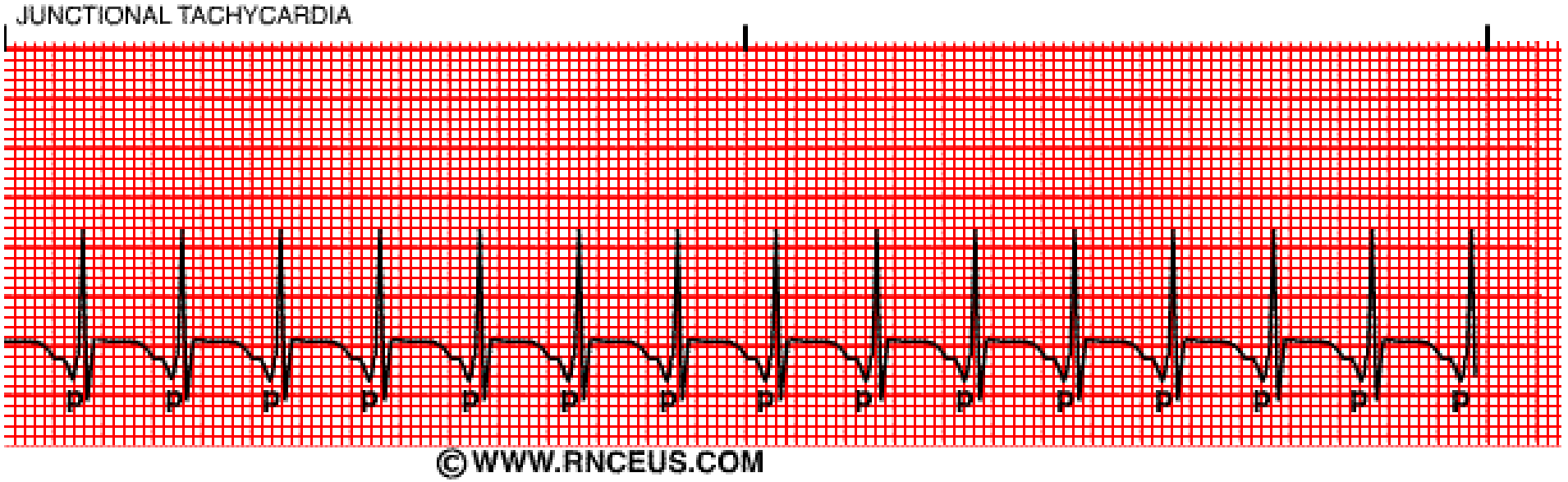


Automatic
SVT



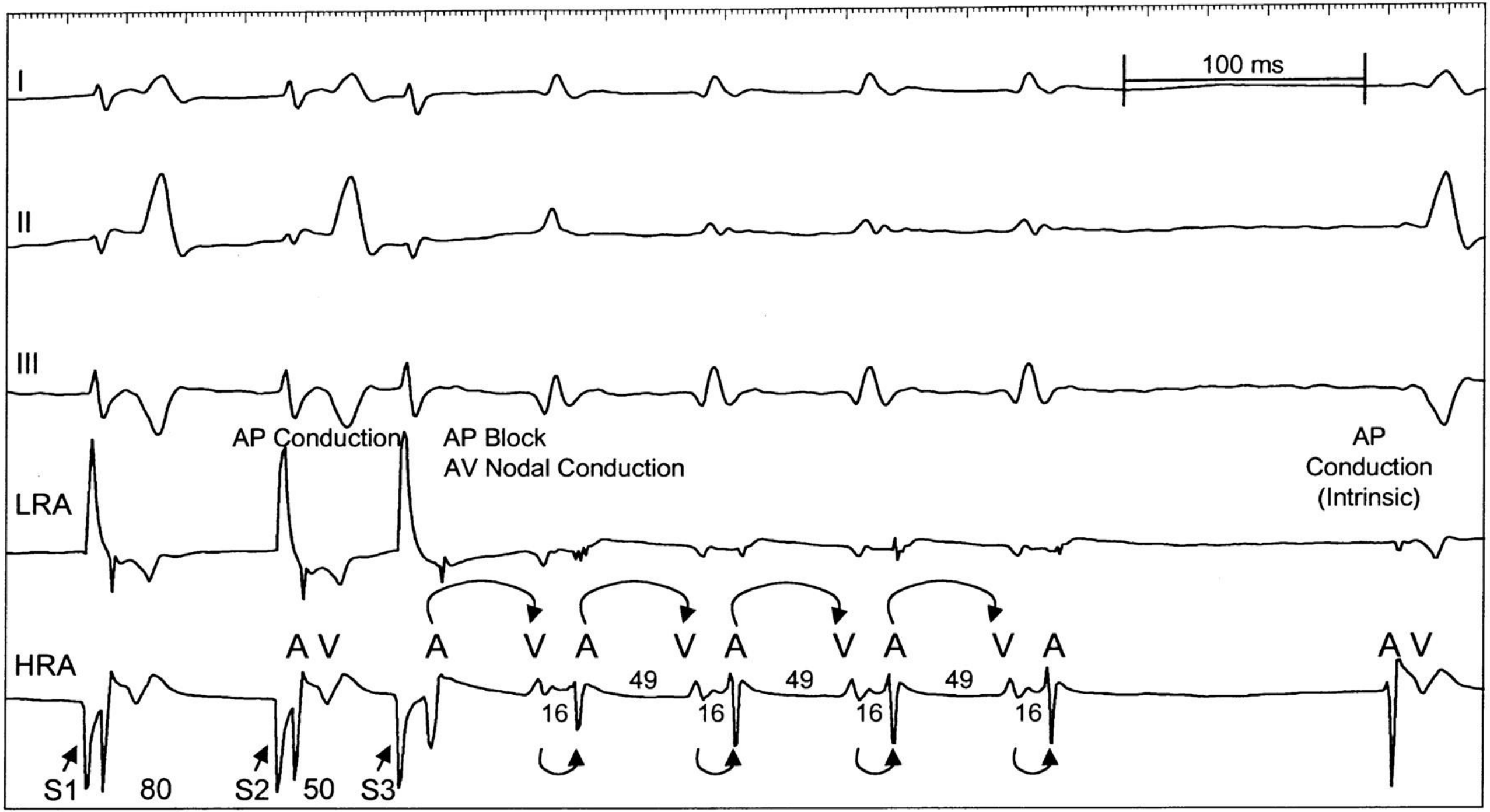
ATRIOVENTRICULAR JUNCTIONAL SVT

- Frequency: 140-180/min
- Regular: YES, paroxysmal form
- Cause:
  - AV and junction reentry
- Manifestation:
  - No P wave
  - QRS varies
  - Can be ceased by increased vagal activity (oculocardial reflex)


JUNCTIONAL TACHYCARDIA
P
©WWW.RNCEUS.COM

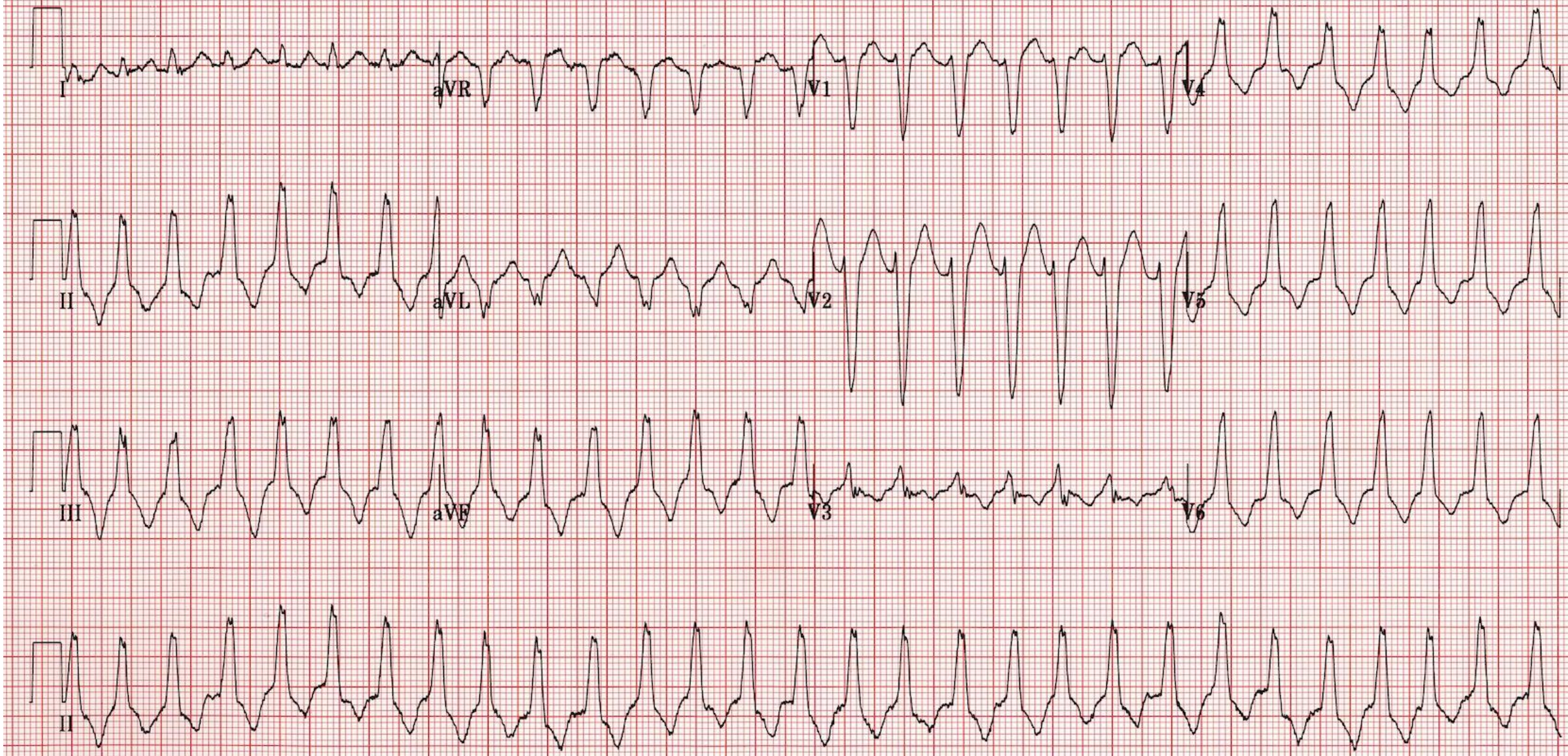
ATRIOVENTRICULAR REENTRY SVT

- Frequency: 100-250/min
- Regular: YES, paroxysmal form
- Cause:
  - Additional conduction pathway among atrias and ventricles
    - Ortodromous – antegradous pathway in AV node
    - Only retrogradeous pathway is ceased under sinus rhythm (silence pathways, hidden pathways)


I
100 ms
II
III
AP Conduction
AP Block
AV Nodal Conduction
AP
Conduction
(Intrinsic)
LRA
HRA
A V
A V
A V
A V
A V
A V
49
49
49
16
16
16
16
S1
80
S2
50
S3

VENTRICULAR TACHYCARDIA

- Frequency: >100/min (at least 3 extrasystoles)
- Regular: YES, paroxysmal
- Cause:
  - Acute or chronic ischemic heart disease
  - Hypertrophic or dilatation cardiomyopathy
  - Elonged QT-interval syndrome
  - Medication, Idiopatic
- Manifestation
  - Mono/polytopous (more dangerous)
  - QRS bizarre, >0,12 s
  - Hemodynamic changes – lowered cardiac output may be present
  - Palpitations, syncope, ventricula fibrilation, heart arrest



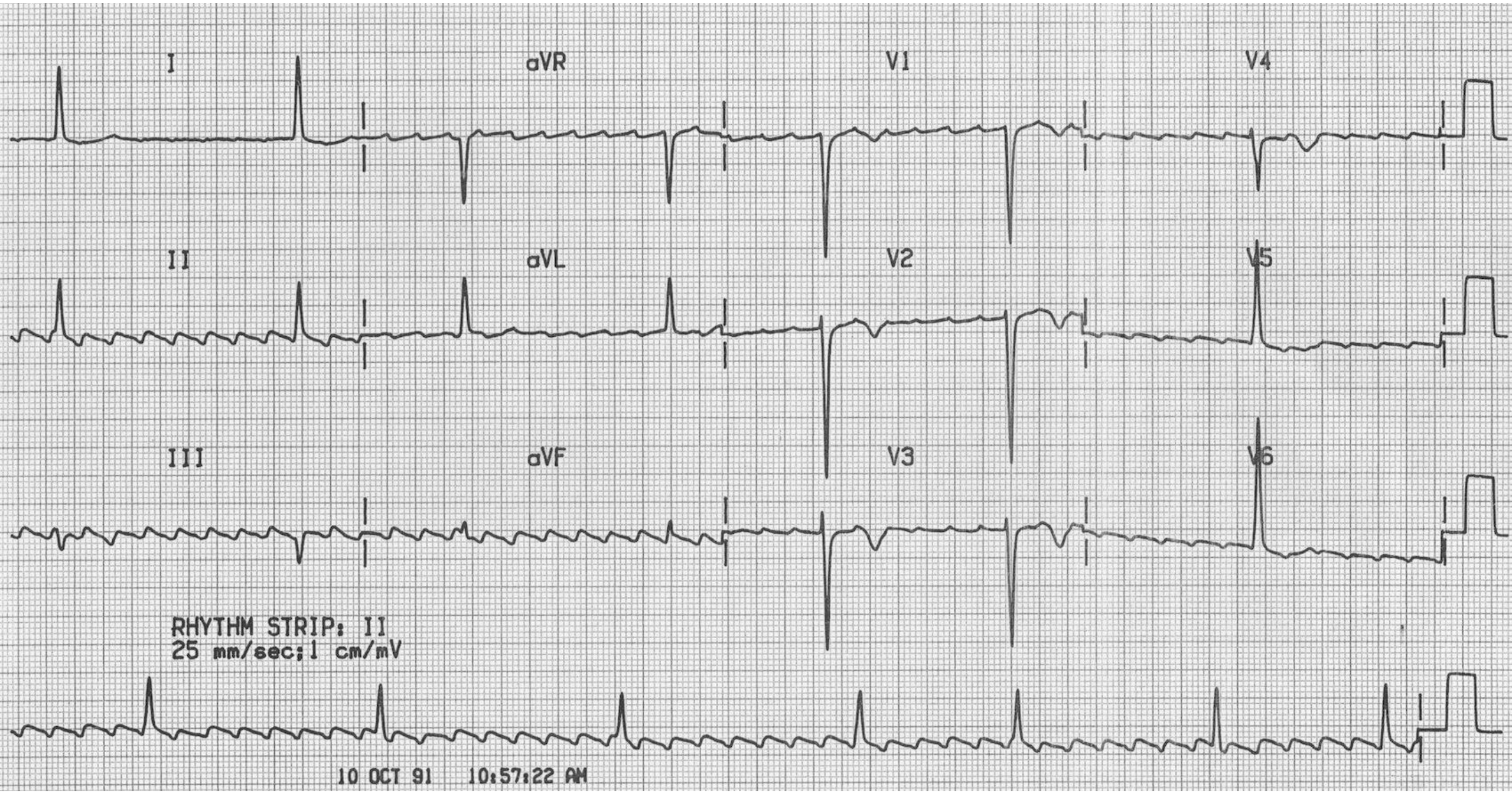
150 Hz 25.0 mm/s 10.0 mm/mV 4 by 2.5s + 1 rhythm ld MAC35 009B.1 12SL™v239

HEART FLUTTER

- Frequency: 250-350/min
- Regular: YES!!! (depolarization-repolarization)
- Cause:
  - Organised reentry phenomenon or early after depolarization
- Manifestation:
  - „sawtooth“ shape waves on ECG
  - Hemodynamic defect varies

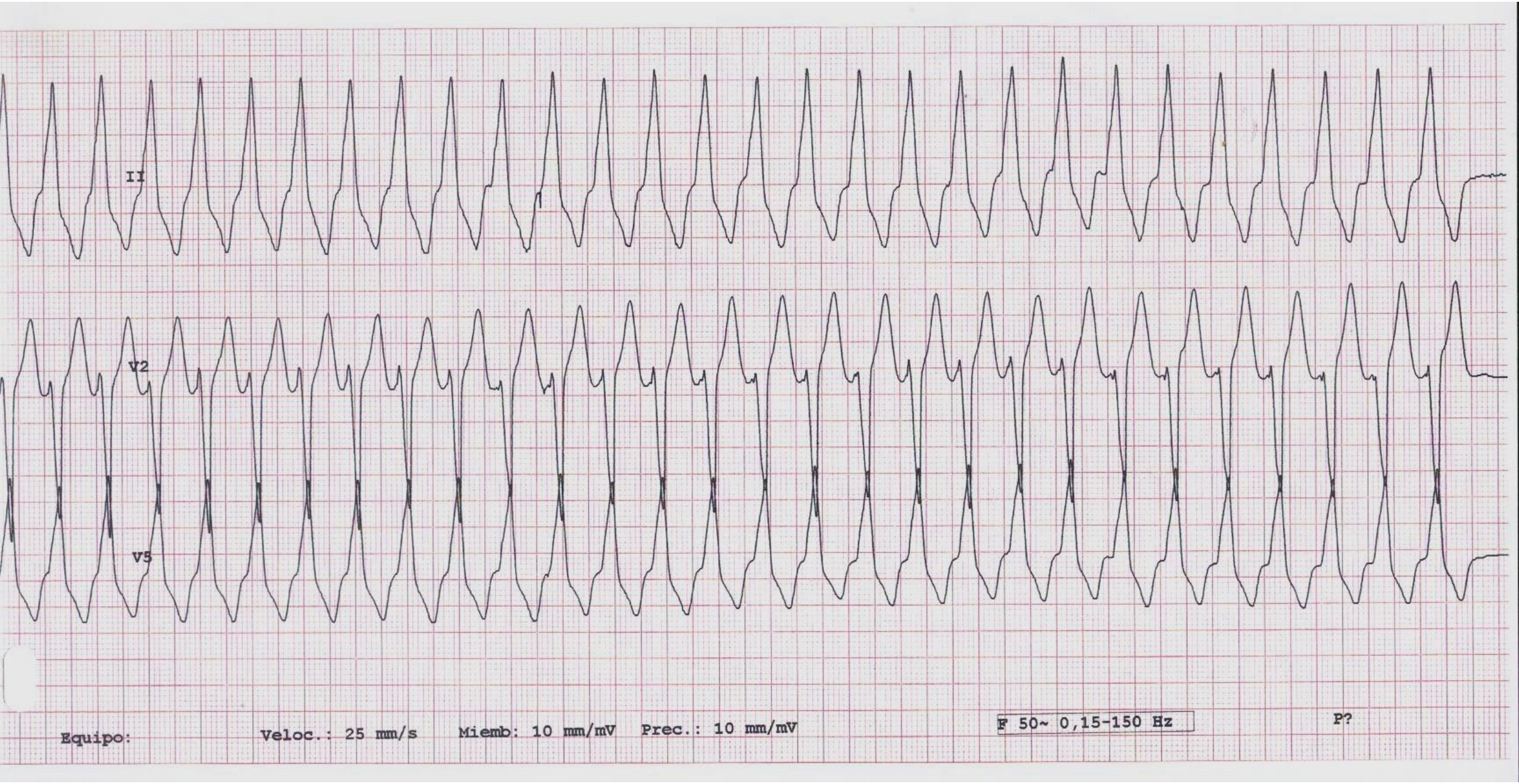
ATRIAL FLUTTER (OSCILLATION)

- Frequency of atria: 250-350/min (may be even 440/min)
- Regular: YES!!!
- Cause: as heart flutter
- Pathophysiology:
  - AV node transfers only every 2nd or 3rd wave (2:1, 3:1, 4:1, etc.)
  - „Improvement“ of transfer will worsen patient‘s condition! (diastole shortened)
  - Frequency of ventricles may be normal (can be survived for a long time)


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec; 1 cm/mV
10 OCT 91 10:57:22 AM

VENTRICULAR FLUTTER

- Frequency: 250-350/min
- Regular: YES!!!
- Cause:
  - Elongated QT-interval syndrome, Torsade de pointes
  - Early after depolarization
- Manifestation:
  - Flutter waves on ECG
  - Hemodynamics highly impaired (diastole almost does not exist)

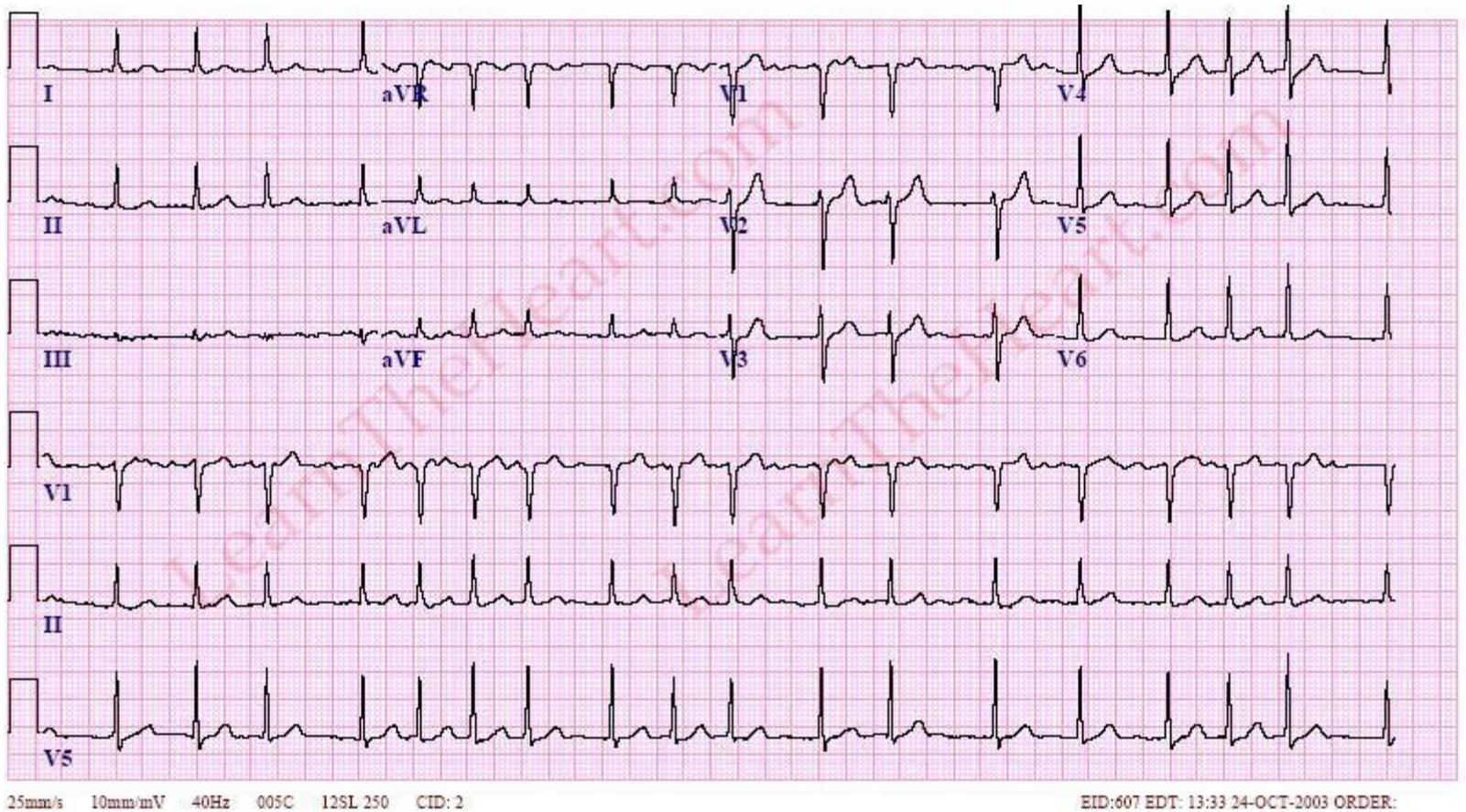

II
V2
V5
Equipo:
Veloc.: 25 mm/s
Miemb: 10 mm/mV
Prec.: 10 mm/mV
F 50~ 0,15-150 Hz
P?

HEART FIBRILLATION

- Frequency: 350-600/min
- Regular: HELL NO!
- Cause:
  - Electric current injury
  - K^+, Ca^{2+}, H^+ concentration disbalance
  - Hypertrophic or dilatation cardiomyopathy
  - Ischemic heart disease, hypoxia
- Manifestation
  - Fibrillation waves on ECG (irregular pulse – „minicontractions“)
  - Hemodynamics heavily impaired (chaotic current conduction – no pressure)
  - Oxygen demands of myocardium decreased

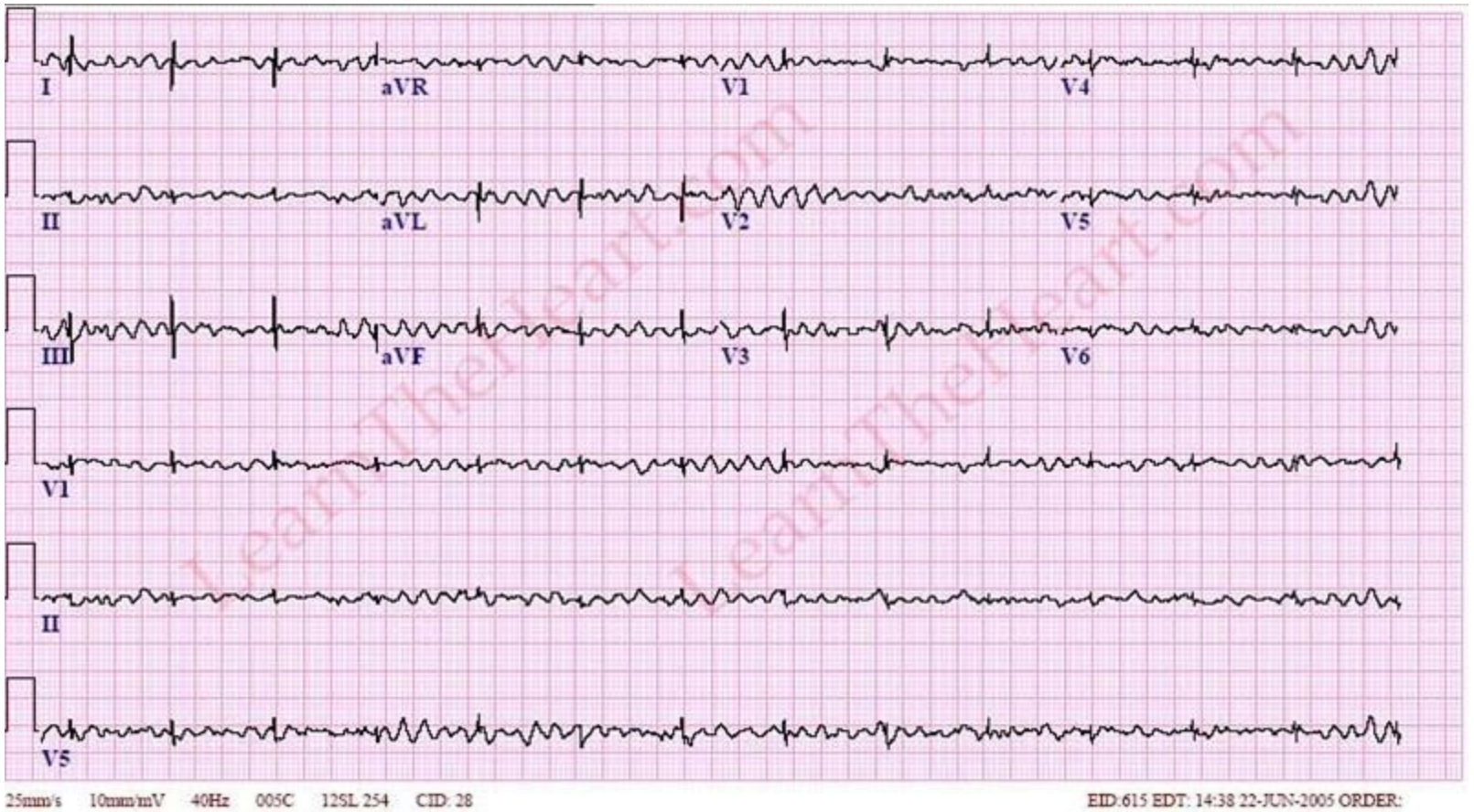
ATRIAL FIBRILLATION (TWINKLING)

- Frequency of atria: 350-600/min
- Regular: NO!
- Cause: same as heart fibrillation
- Manifestation:
  - Fibrillation waves instead of P waves, AV node activated irregularly
  - Hemodynamics decrease for about 15 %
  - Pulsus irregularis et inequalis (beat volumen varies)
  - Thrombi formation (fibrinolytics before cardioversion)
  - Ventricular frequency may be normal! (can be survived for a long time)


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 40Hz 005C 12SL 250 CID: 2
EID:607 EDT: 13:33 24-OCT-2003 ORDER:

VENTRICULAR FIBRILLATION

- Frequency: >300/min
- Regular: ABSOLUTELY NO!!!
- Cause: same as heart fibrillation
- Manifestation:
  - ECG completely chaotic
  - Cardiac output = 0!!! (hemodynamic defect is absolute)
  - Life threatening condition! -> 10 seconds to consciousness loss, 4-5 mins irreversible brain damage may occur
  - DEFIBRILLATE!!! (15 mins after start of ventricular fibrillation heart activity ceases)


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 40Hz 005C 12SL 254 CID: 28
EID:615 EDT: 14:38 22-JUN-2005 ORDER:

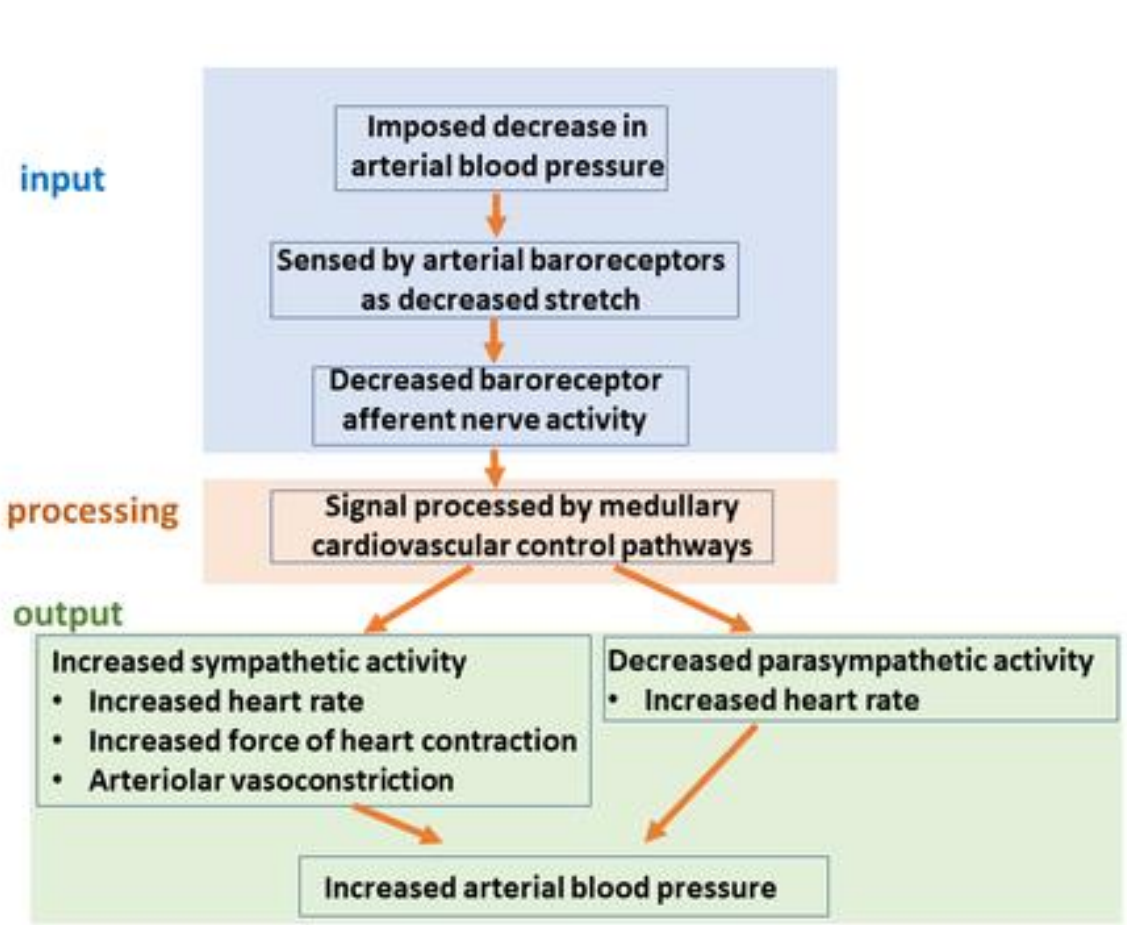
HYPERTENSION

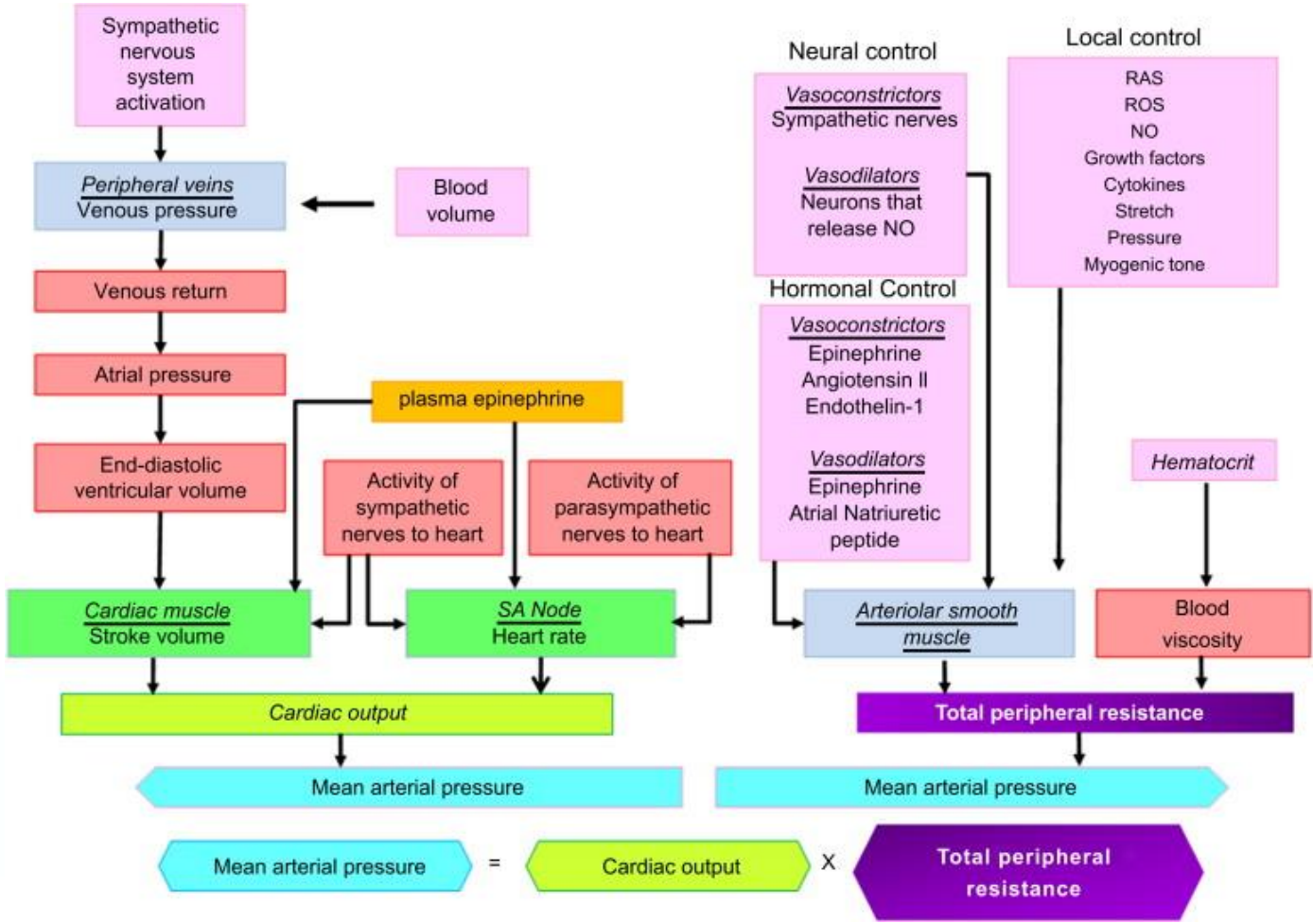
SYSTEMIC VS. PULMONARY

PHYSIOLOGICAL MECHANISMS OF BLOOD PRESSURE REGULATION

- Increase of systolic and/or diastolic blood pressure in systemic/lung circulation may occur due to (P = Q x R)
  - Increased blood flow (Q)
  - Increase resistance of blood vessels (R)
  - Both mechanisms engaged (QxR)
- Vasomotoric centre in oblongate medulla
  - High-pressure baroreceptors -> carotid sinuses, aortic arch
  - Low-pressure baroreceptors -> atria, ventricles, pulmonary vessels

  - Baroreceptors react to short-term changes only -> reset within days -> possible adaptation (hypertension prone)





https://www.physio-pedia.com/images/thumb/a/af/Baroreceptor_reflex_block_diagram.png/450px-Baroreceptor_reflex_block_diagram.png
https://ars.els-cdn.com/content/image/3-s2.0-B9780124052062000144-f14-01-9780124052062.jpg

FACTORS INFLUENCING BLOOD PRESSURE (BP)

Systolic blood pressure (sBP)

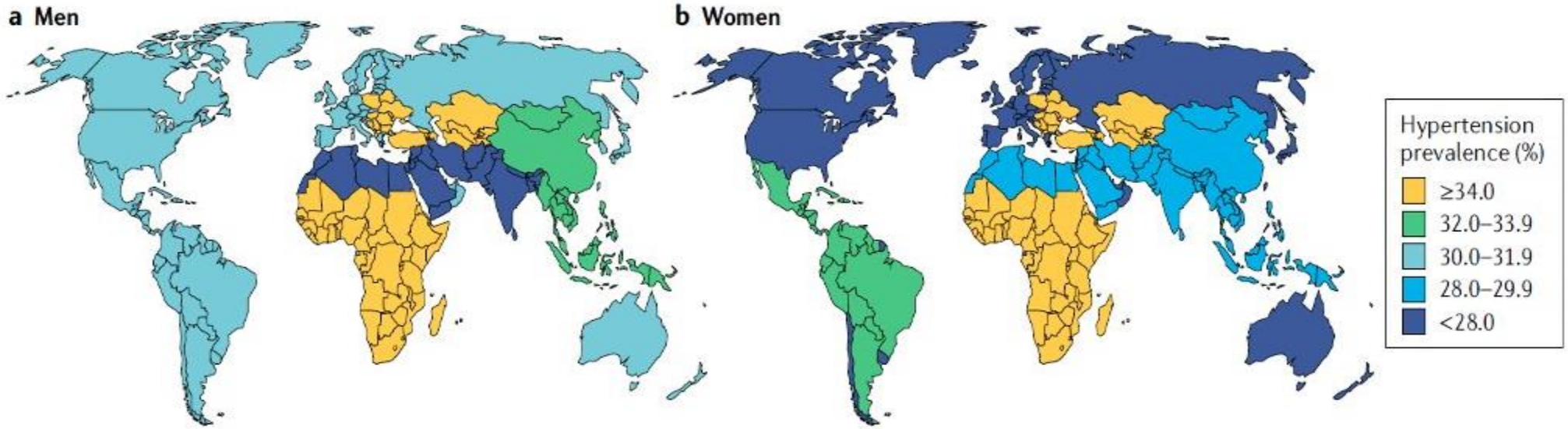
- Venous return and ventricles preload
- Myocardial contractility
- Pulse volume – amount of blood expulsed into vessels during one systolic contraction
- Expulsion velocity of blood
- Compliance of large arteries
- Arterial resistance – de facto diastolic blood pressure
- Peripheral resistance working against blood flowing from large arteries

Diastolic blood pressure (dBP)

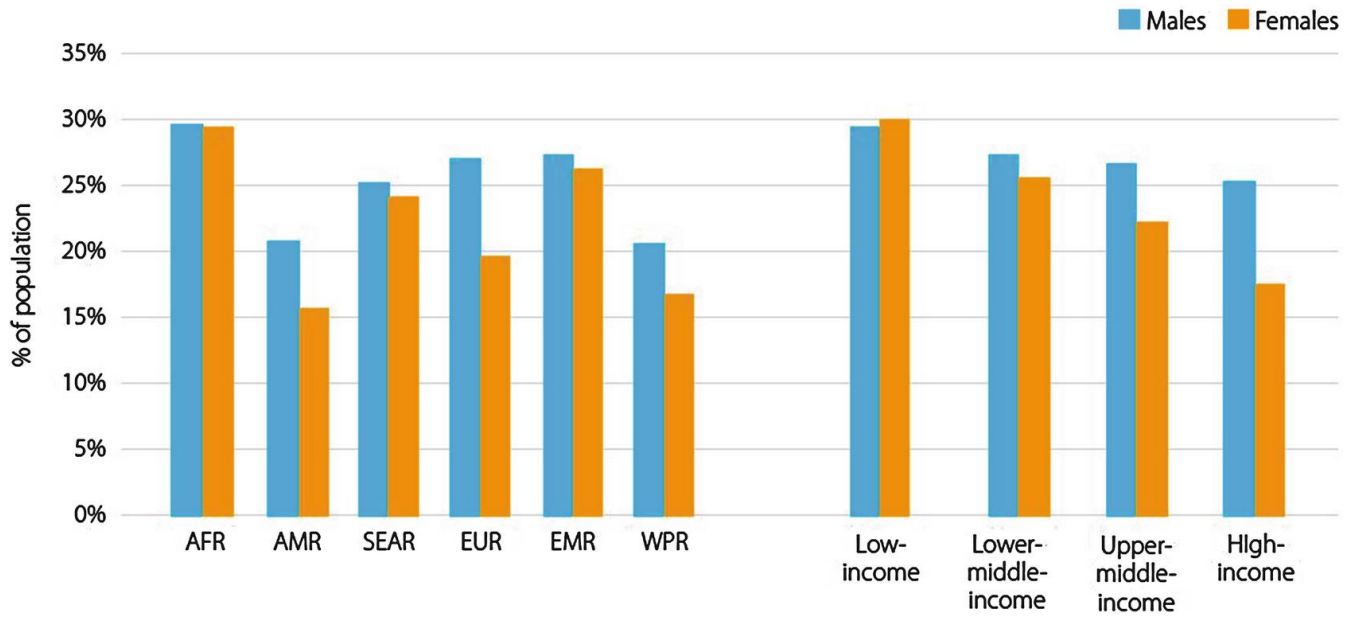
- Systolic BP
- Peripheral resistance working against blood flowing from large arteries
- Time period between two systoles

HYPERTENSION – DEFINITION AND CHARACTERISTICS

- Definition
  - ESH 2024 – sBP >140 mmHg and/or dBP >90 mmHg (isolated systolic hypertension when sBP elevated only)
    - „Pre-hypertension“ cancelled -> optimal BP (<120/80 mmHg) and „normal“ BP (120-130/80-90 mmHg)
  - AHA 2020 – sBP >130 mmHg and/or dBP >80 mmHg
- Classification
  - Systemic arterial vs pulmonary hypertension
  - Primary (essential) vs. secondary (known cause)
- Epidemiology
  - Prevalence – 1.3 billion worldwide (2023)
    - 46 % patients unaware, 42 % patients diagnosed and treated
    - Only 21 % patiens with controlled hypertension



Mills et al. Nat Rev Nephrol. 2020 Apr;16(4):223-237



https://pbs.twimg.com/media/FTCjdeUaMAAMOUX.jpg
https://media.springernature.com/full/springer-static/image/chp%3A10.1007%2F978-3-319-31107-4_31/MediaObjects/145541_4_En_31_Fig1_HTML.png?as=webp

PHYSIOLOGY SUMMARY AND TRANSITION TO PATHOLOGY

- Conditions leading to arterial hypertension (HT)
  - „Hypercirculation“ associated conditions
  - Conditions connected with increased peripheral resistance
- Amplitude between systolic and diastolic BP may change leading to increase to one or both BP
- BP arises due to dysregulation of functional relationship between amount of circulating blood and resistance to blood flow
  - Cardiac output vs. peripheral resistance

Guideline Similarities	2017 ACC/AHA	2023 ESH
Accurate Blood Pressure Measurement	Office-based BP measurements and use of validated, cuffed devices and home/ambulatory BP monitoring are recommended prior to diagnosing hypertension.	
Cardiovascular Risk Calculator for Treatment Thresholds	Pooled Cohort Equation and SCORE2/SCORE2-OP provide estimates for 10-year risk of fatal and non-fatal cardiovascular events and should be used to guide treatment decisions.	
Initial Pharmacotherapy Recommendations	Initial therapeutic choices include ACE inhibitors, angiotensin-receptor blockers, thiazide or thiazide-like diuretics, and calcium channel blockers. Single pill combination therapy is a first-line strategy for many patients.	

Guideline Differences	2017 ACC/AHA	2023 ESH
Hypertension Definition	≥ 130/80	≥ 140/90
Normal BP Ranges (mmHg)	Normal: < 120/80 Elevated: 120-129/<80	Optimal: < 120/80 Normal: 120-129/80-84 High-Normal: 130-139/85-89
Hypertensive BP Ranges (mmHg)	Hypertension Stage 1: 130-139/80-89 Hypertension Stage 2: ≥ 140/90	Hypertension Grade 1: 140-159/90-99 Hypertension Grade 2: 160-179/100-109 Hypertension Grade 3: ≥ 180/110
BP Targets for Treatment		
18 – 64 years (mmHg)	< 130/80	< 130/80
65-79 years (mmHg)	< 130/80	< 140/80*
≥ 80 years (mmHg)	< 130/80	140-150/<80
Pharmacotherapy	Initial therapy with beta-blockers reserved for specific conditions including ischemic heart disease or heart failure	Beta blockers included as first-line therapy for hypertension.

** Target < 130/80 if tolerated*

ACC/AHA 2020

Blood Pressure Category	Systolic mm Hg (upper number)		Diastolic mm Hg (lower number)
NORMAL	LESS THAN 120	and	LESS THAN 80
ELEVATED	120 - 129	and	LESS THAN 80
HIGH BLOOD PRESSURE (Hypertension) Stage 1	130 - 139	or	80 - 89
HIGH BLOOD PRESSURE (Hypertension) Stage 2	140 OR HIGHER	or	90 OR HIGHER
HYPERTENSIVE CRISIS (consult your doctor immediately)	HIGHER THAN 180	and/or	HIGHER THAN 120

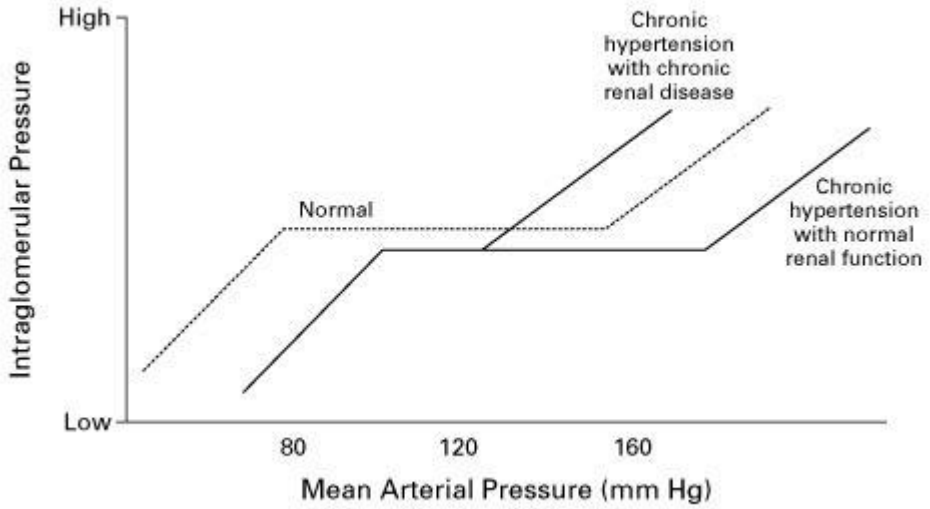
https://www.acc.org/-/media/Non-Clinical/Images/Latest-in-Cardiology/Articles/2024/02/PREV-EA-Vemu-Table1.png
https://blog.ohiohealth.com/wp-content/uploads/2022/02/Blood-Pressure-Guidelines-Update_Infographic-2022.jpg

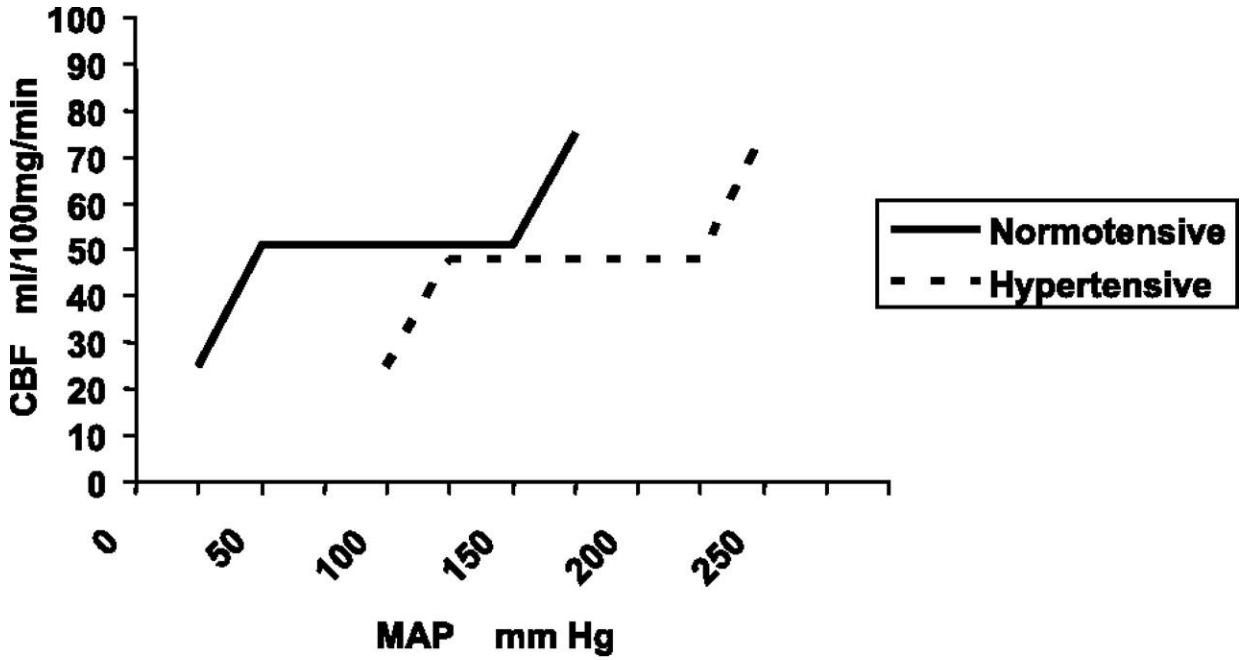
SYSTEMIC ARTERIAL HT

- Definition – applies from ESH 2024 or ACC/AHA 2020
  - Europe – sBP >140 mHg and/or dBP >90 mmHg
- Only arterial circulation affected
- Organs affected
  - Heart
  - Kidney
  - Brain
  - Arteries
- Classification
  - 95 % - essential (primary)
  - 5 % - known conditions (secondary)

HYPOTHESES OF ESSENTIAL BP DEVELOPMENT

1. „Hypertension by volume increase "
   - Primary change -> circulating volume elevates
   - Result – hypercirculatory state -> peripheral resistance arises
2. Circulation compensating primary kidney malfunction
   - Lowered excretion of sodium and water -> increase in BP -> pressure diuresis -> homeostasis achieved (damages kidney over a time!)
3. *Increased sympathetic activity*
   - *Vasoconstriction -> hypertension*
   - *Lack of proof*
     - *Sympathetic hyperactivity conditions as heart failure, liver cirrhosis are rarely accompanied by HT*





CBF=cerebral blood flow; MAP=mean arterial pressure

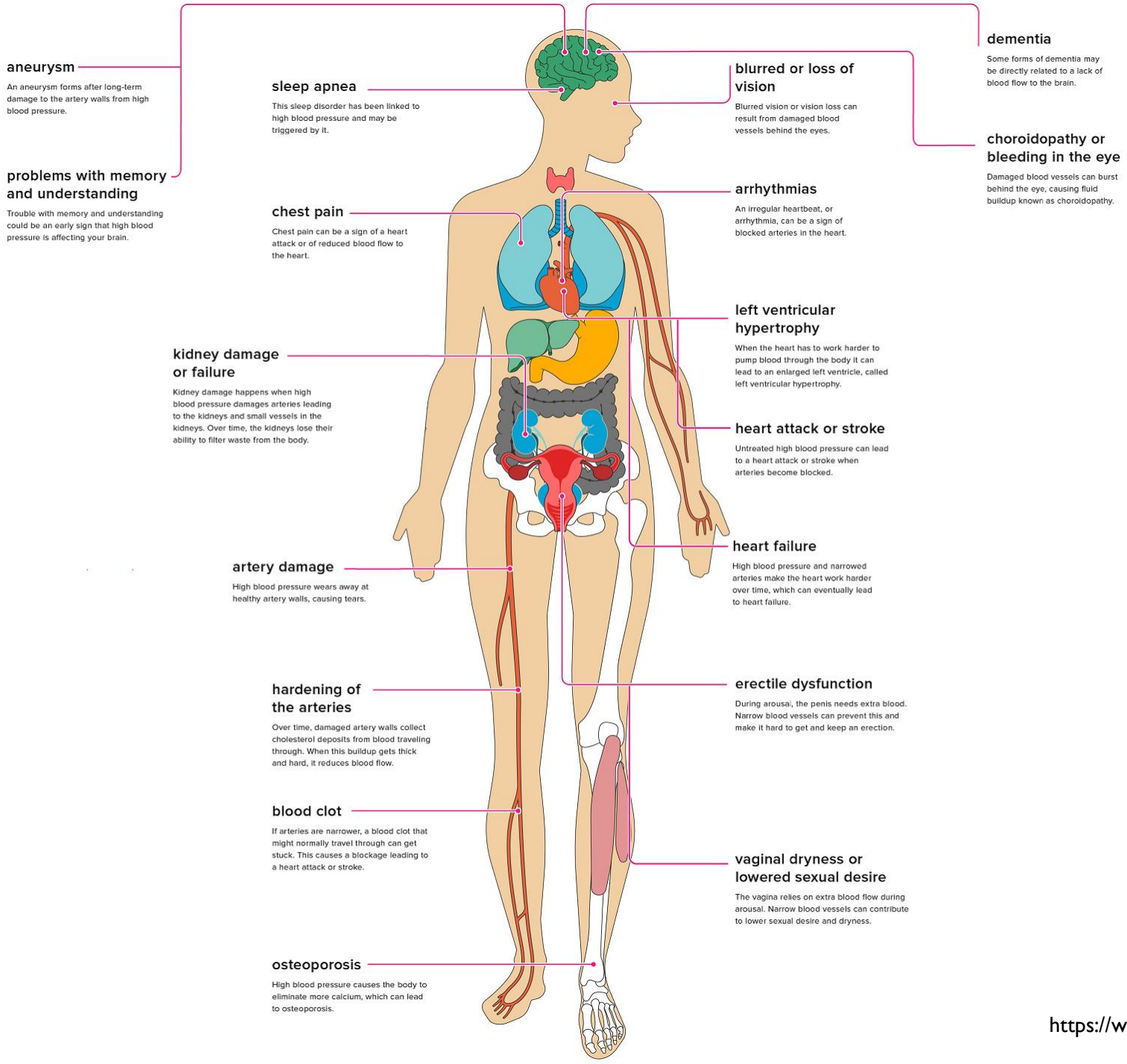
http://www.nejm.org/na101/home/literatum/publisher/mms/journals/content/nejm/2002/nejm_2002.347.issue-16/nejmra020676/production/images/img_medium/nejmra020676_f1.jpeg
http://hyper.ahajournals.org/content/hypertensionaha/49/5/977/F1.large.jpg

ESSENTIAL HT

- Dyscrepancy between circulating volume and peripheral vessels resistance
- Dysregulation between perfusion of organs and peripheral resistance
  - Kidney (RAAS) and brain mostly
    - Angiotensin II increases peripheral resistance
    - Aldosterone -> Na^+ retention -> hypervolemia
- During HT there is no hyperperfusion of organs
  - After crossing certain limit (higher in HT than normal) -> reaction of peripheral circulation in adjusting of blood flow
- „Stabilisation of HT“ -> ↓BP -> ↓organ perfusion (kidney, brain) -> tendency to return back hypertension conditions
- Arterial HT as a health burden and a risk factor for cardiovascular diseases

ESSENTIAL HT

- Increase in cardiac output + hypercirculation conditions
- Chronic increase of tissue perfusion -> vasoconstriction of peripheral vessels -> increase in peripheral resistance
- Consequences
  - Endothelial damage (strengthened pulsations) -> atherosclerosis acceleration
  - Afterload increase -> concentric hypertrophy of left heart ventricle
  - Increase in heart work -> increased oxygen consumption by myocard
  - Risk of circulation disruption and arterial bleeding – epistaxis, stroke, „ruptured vessels on sclera“, retinal bleeding etc.
  - Hyperfiltration in kidney -> glomeruli fibrotisation -> chronic kidney failure (hypertension nephropathy)



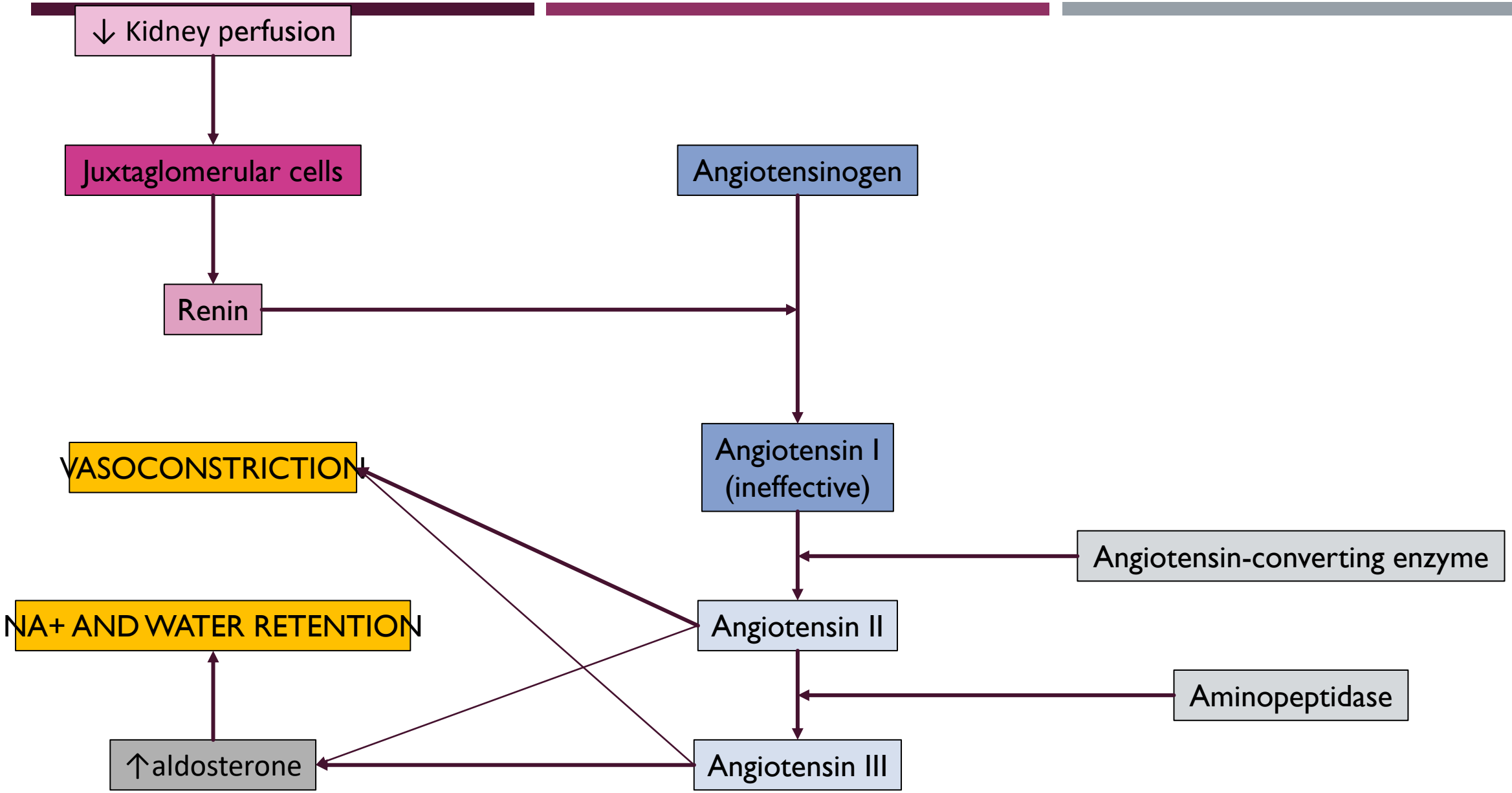
https://www.healthline.com/hlcmsresource/images/Effects_Hypertension/Hypertension_Effects_Pinterest_crop.jpg

SECONDARY SYSTEMIC ARTERIAL HT

Pathomechanism	Examples
Prevailing peripheral resistance increase	Pheochromocytoma Unilateral a. renalis stenosis
Prevailing increase of blood volume and hypercirculation	Primary hyperaldosteronism (Conn sy.) Surgical removal of kidney (renoprive HT) Cushing sy. (hypercorticism) Acromegaly Polycytemia vera Estrogen administration
HT caused by both peripheral resistance and circulating volume increase	End-stage renal insufficiency – last stage Bilateral ischemia of kidney Pregnancy induced hypertension (?)

SECONDARY HT CAUSED BY PERIPHERAL RESISTANCE INCREASE

- Pheochromocytoma
  - Origin – suprarenal medulla/sympathetic ganglions
  - Catecholamines produced – norepinephrine mainly -> alfa1-adrenergic receptors
  - Paroxysmal/persistent HT with BP fluctuations -> may reach 300/150 mmHg during paroxysm
- Unilateral a. renalis stenosis (renovascular HT)
  - Perfusion decrease behind the stenosis -> juxtaglomerular cells irritated -> renin production -> RAAS activation
  - Hypervolemia -> preload increase -> increased cardiac output


↓ Kidney perfusion
Juxtaglomerular cells
Renin
Angiotensinogen
Angiotensin I
(ineffective)
Angiotensin-converting enzyme
Angiotensin II
Aminopeptidase
Angiotensin III
VASOCONSTRICTION
NA+ AND WATER RETENTION
↑aldosterone

SECONDARY HT CAUSED BY INCREASE OF BLOOD VOLUME AND HYPERCIRCULATION

- Conn sy. (primary hyperaldosteronism)
  - Increased natrium retention -> increased blood volume
- Renoprive HT
  - Accumulation of bodily fluids -> blood volume increase
- Cushing sy.
  - Increased cardiac output suspected

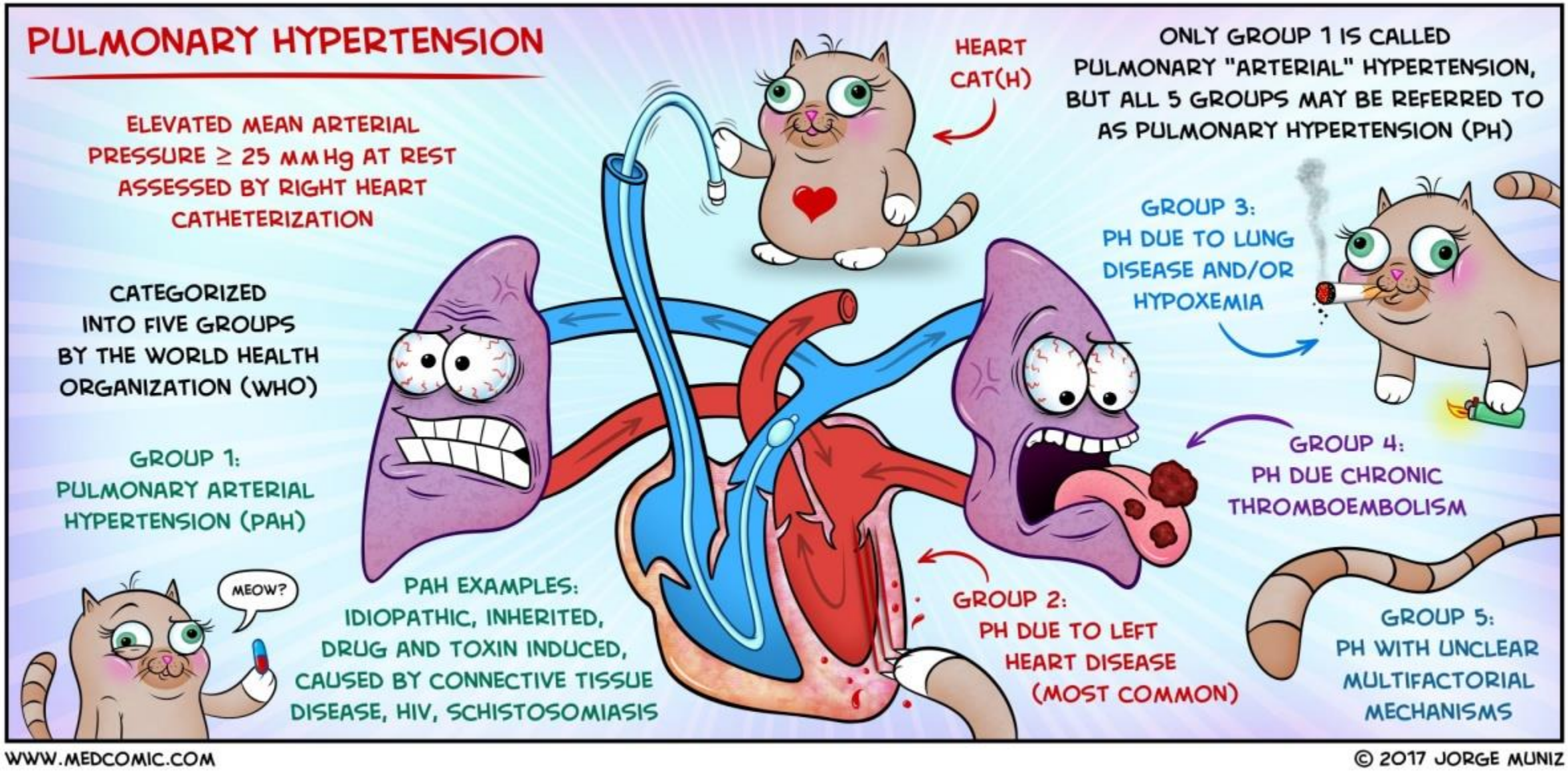
SECONDARY HT CAUSED BY BOTH PERIPHERAL RESISTANCE AND CIRCULATING VOLUME INCREASE

- Bilateral ischemia of kidney and ESRI
  - Fluid retention -> vasoconsctriction as a reactive change
  - Dehydratation/dialysis -> renin production intensified -> worsening of HT
  - Volume HT changes to resistance HT

- Pregnancy induced hypertension (5-14 % pregnant!)
  - Maldevelopment of placenta
    - Lack of aa. Spirales proper transformation and/or malinvasion to decidual layer
    - Placental circulation remains as high-resistance circulation (normal is a decrease in resistance)
    - Proteinuria, edemas my occur as well (preeclampsia – edemas + Ht+ proteinuria = EPH gestosis)
    - Maternal metabolism affected (adipokines, cytokines, immune system involved)
    - Increased risk of complications without a treatment

PULMONARY ARTERIAL HT

- Physiological BP in pulmonary artery – 8-20 mmHg
- Pulmonary HT
  - BP >25 mmHg resting; >30 mmHg during physical activity
- Often has an acute manifestation
  - Systemic is more chronic
- Significant influence of pulmonary veins
  - In systemic HT venous part is almost only slightly affecting BP
- Classification (see next slide)



https://kevinmd-kevinmdcom.netdna-ssl.com/blog/wp-content/uploads/Pulmonary_Hypertension.jpg

PULMONARY HT FORMS

1. Hyperkinetic pulmonary HT
   - L->R shunt -> increased amount of blood flowing
   - Pulmonary arterioles remodelation – thicker muscular layer
   - Correction of cardial mistake has to happen sooner then fixation of HT occurs
2. Postcapillary pulmonary HT
   - Primary – increase of pressure in left atrium (left-sided heart failure – backward, mitral stenosis and insufficiency, cardiomyopathies)
   - Chronic – cor pulmonale chronicum (right ventricle hypertrophy)

PULMONARY HT FORMS

1. Reactive pulmonary HT
   - Lung circulation hypoxia -> arterioles vasocontriction
   - Hypoventilation states – chronic bronchitis, emphysema
   - Mountain sickness
2. Obliteration and obstruction pulmonary HT
   - Idiopatic
   - Dysregulation of pulmonary arterioles composition - BMP (bone morphogenic proteins) cytokines mutation – familiar/sporadic
     - Progressive, irreversible, at physical activity, later resting dyspnoe, right-sided heart failure
   - Pulmonary parenchyme reduction – surgery including
   - Repeated thrombembolism to lungs
     - Right ventricle myokardium may arise BP in a. pulmonalis to 40-100 Torr at once, without preceding hypertrophy, risk of blood pooling in right ventricle and heart failure

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https://patfyz.medic.upjs.sk/ehome.htm