ARRHYTMIAS

J. Pitha

OUTLINE

- CASE REPORT(S)
- DEFINITION
- PATHOPHYSIOLOGY
- (DIFF.) DIAGNOSIS – symptoms, evaluation
- THERAPY

DEFINITION

- Heart rate 50-100/min
- Heart rate regular

PATHOPHYSIOLOGY/CLASSIFICATION

- **Initiation x conduction of the el. impulse**
- **Supraventricular x Ventricular**
- **Cardiac x non-cardiac (electrolyte disturbances, acidosis/alkalosis, hyperthyreodism, anemia, …)**
- ***Structural heart disease x absence of structural heart disease (ventricular arr.)***

CAUSES

- ORGANIC HEART DISEASE
- DISRUPTION OF INTERNAL ENVIRONMENT
- IATROGENIC
- INBORNE/GENETIC
- UNKNOWN

EVALUATION/DIAGNOSIS

- HISTORY, incl. drugs
- ECG
- Laboratory findings
- Echocardiography
- Scintigraphy
- CT, MR, …

SYMPTOMS

- Asymptomatic
- Palpitation
- Signs of heart insufficiency: dyspnea, edema, …
- Angina pectoris
- Syncope
- Sudden death

Polyuria (atrial fibrillation)

ARRYTHMIAS

- BRADYcardia
- TACHYcardia

BRADYCARDIA

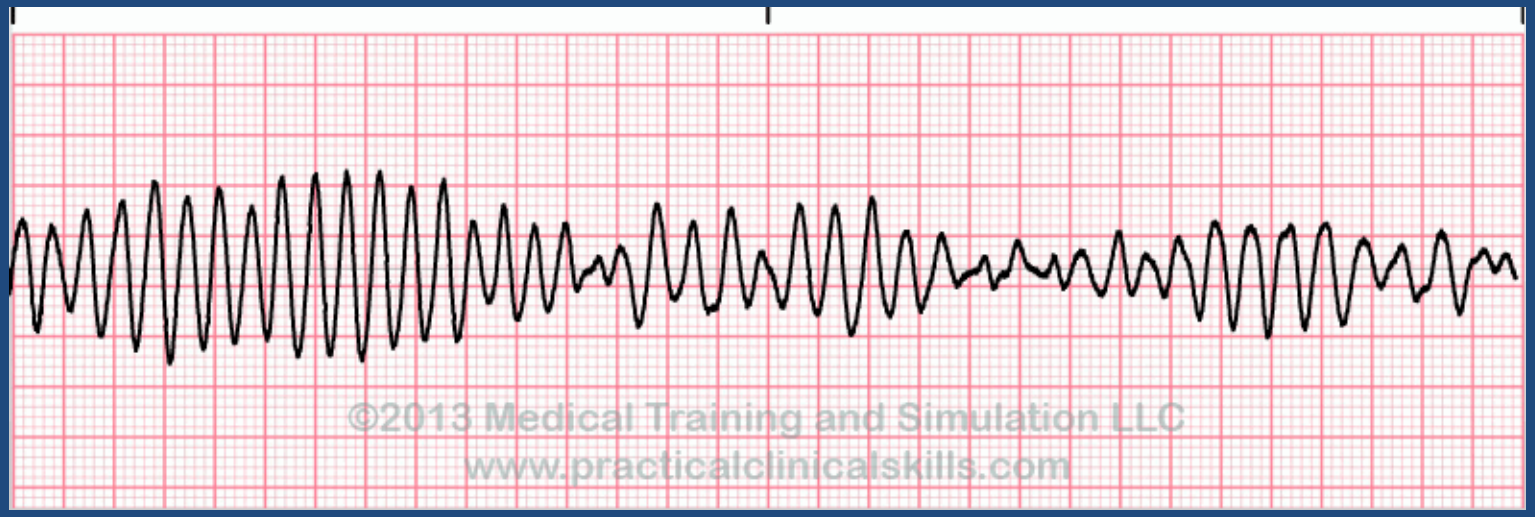
- SINUS BRADYCARDIA
- SA BLOCK(S)
- A-V BLOCKS – type I-III, II/1-2 gr.
- SSSyndrome ?

TACHYCARDIA – SUPRAVENTRICULAR

- SINUS TACHYCARDIA – INAPPROPRIATE ST
- SUPRAVENTRICULAR ES
- ATRIAL FIBRILLATION/FLUTTER
- AV-REENTRY …
- PREEXCITATION – WPW sy, LCL sy …

TACHYCARDIA - VENTRICULAR

- **VENTRICULAR ES**
- **VENTRICULAR TACHYCARDIA**
- **TORSADE DE POINTS**
- **VENTRICULAR *FLUTTER*/FIBRILLATION**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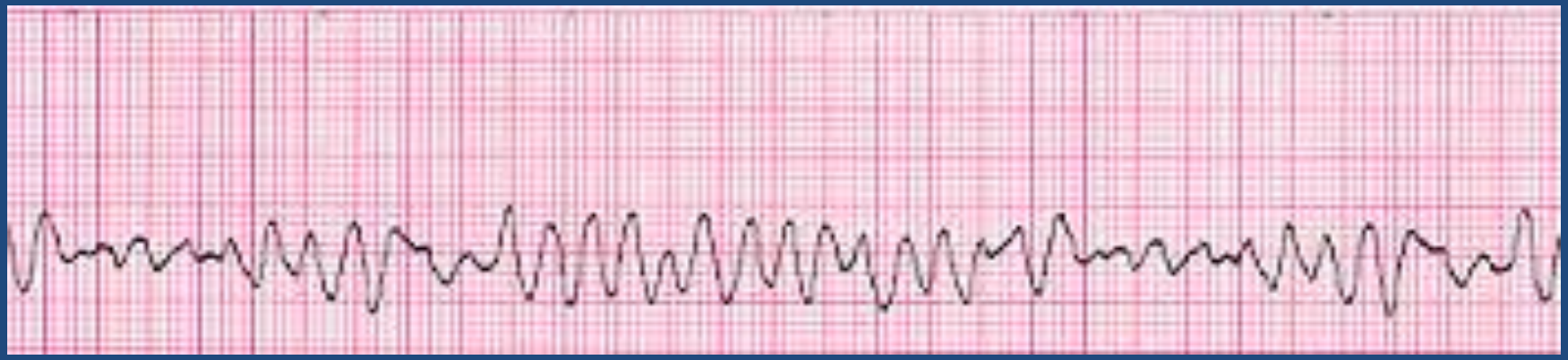
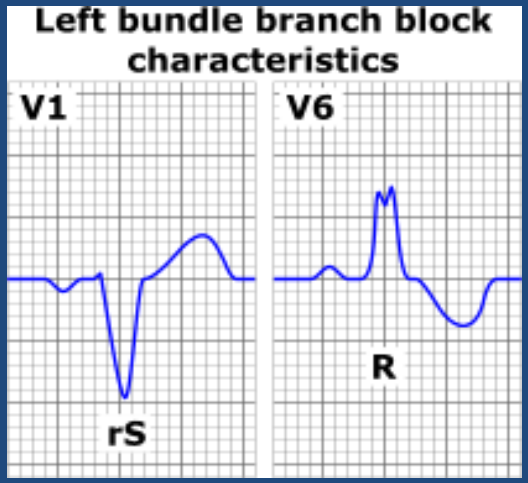
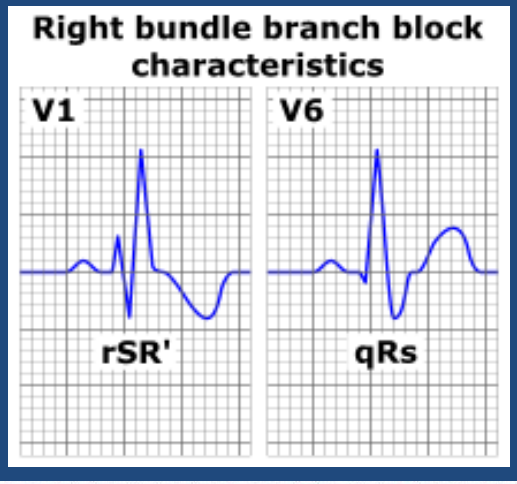
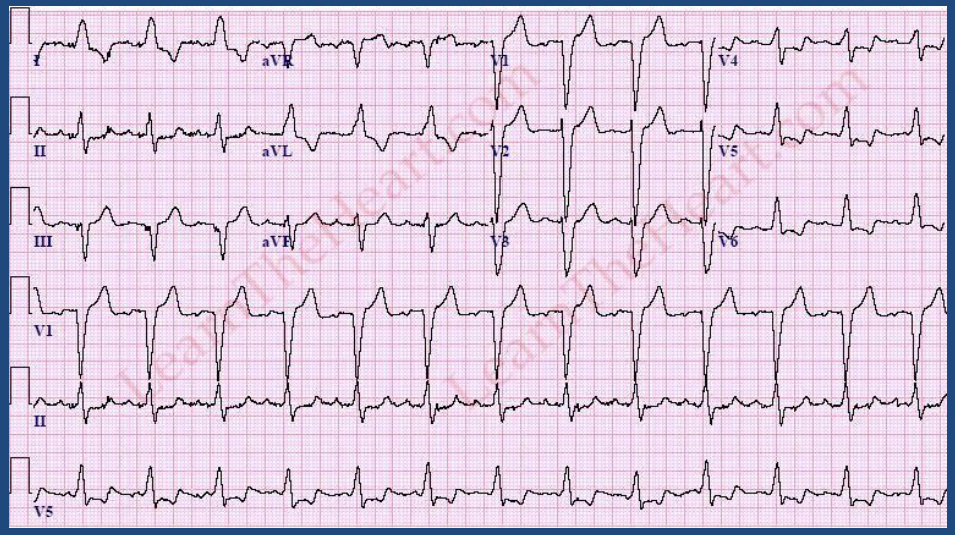
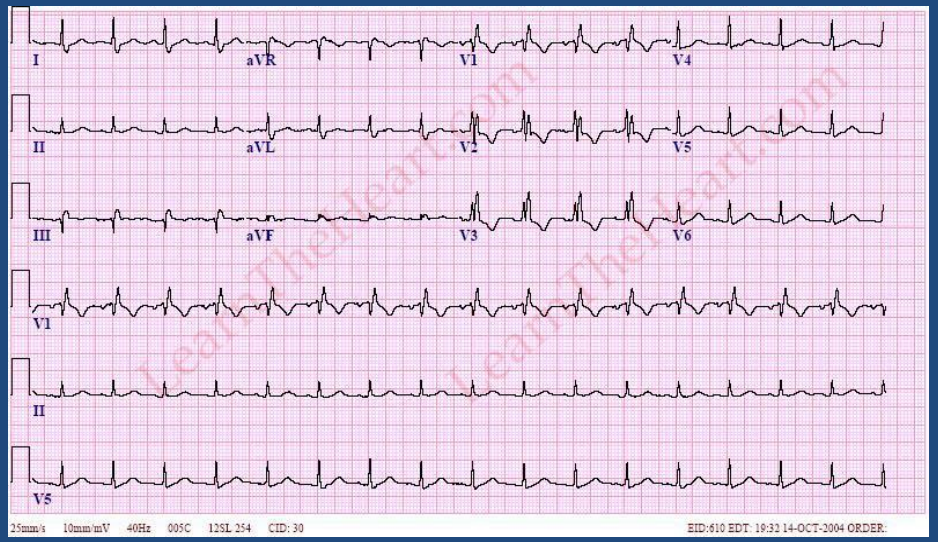


LEFT/RIGHT BUNDLE BRANCH BLOCK



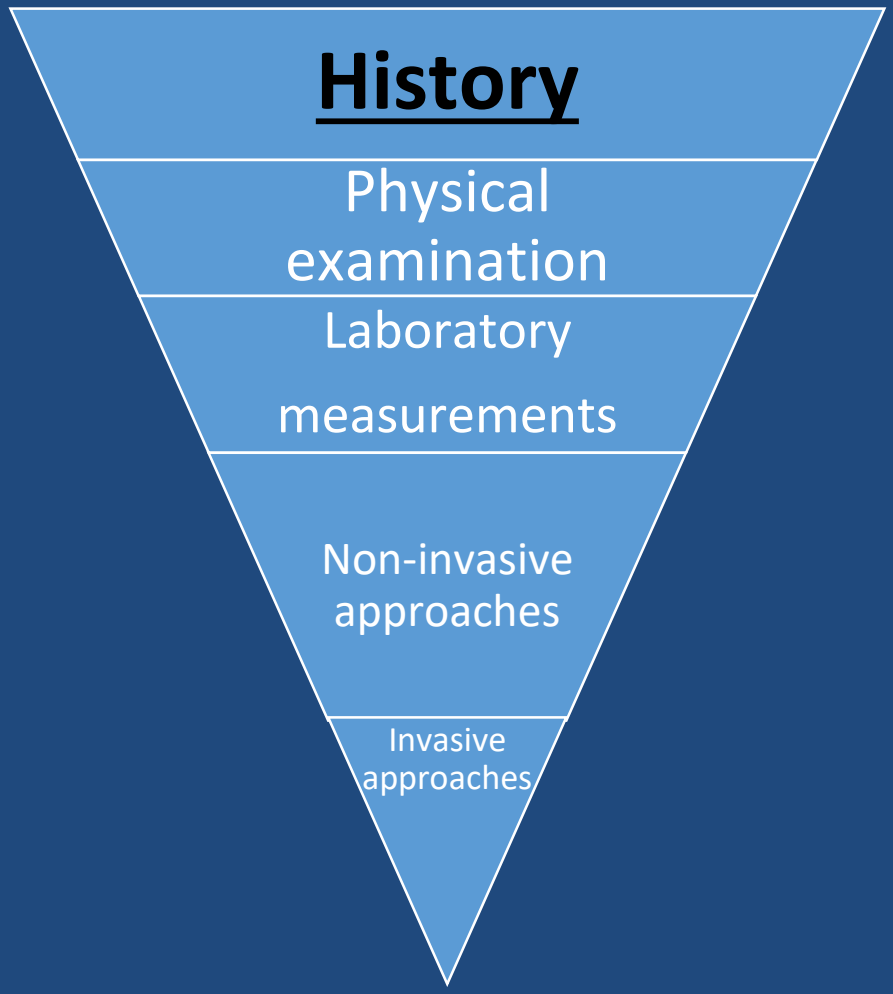




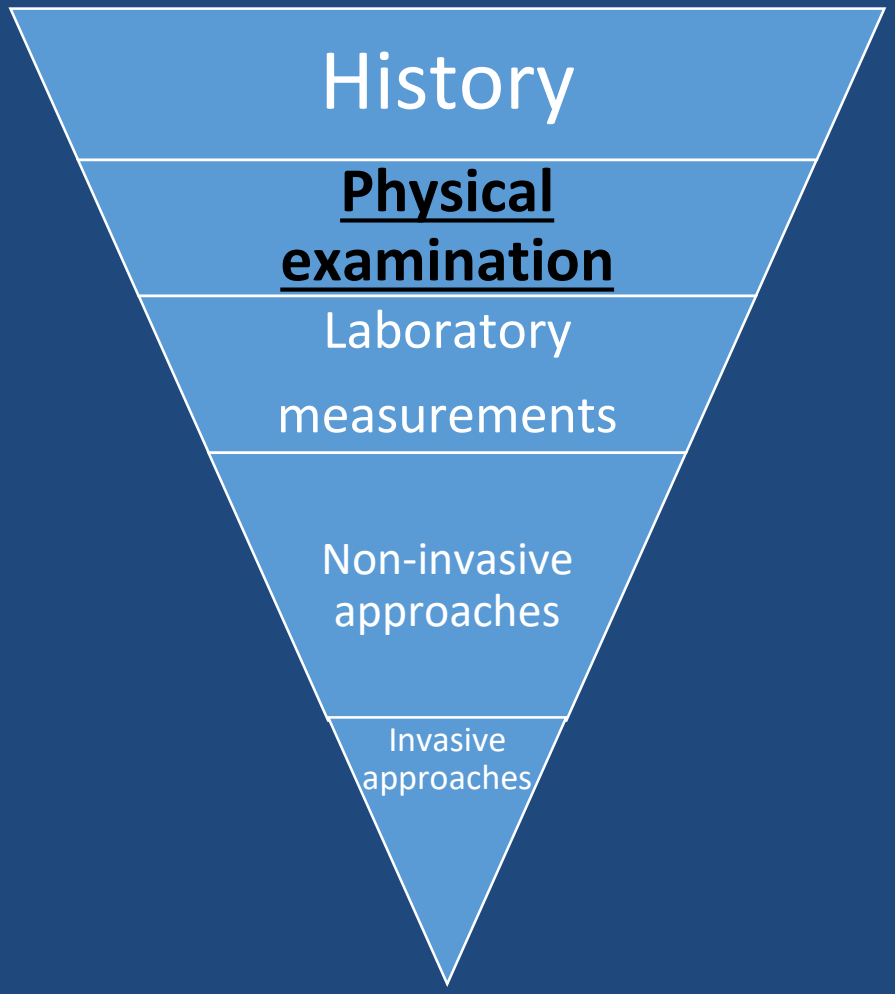


DIFF. DIAGNOSIS

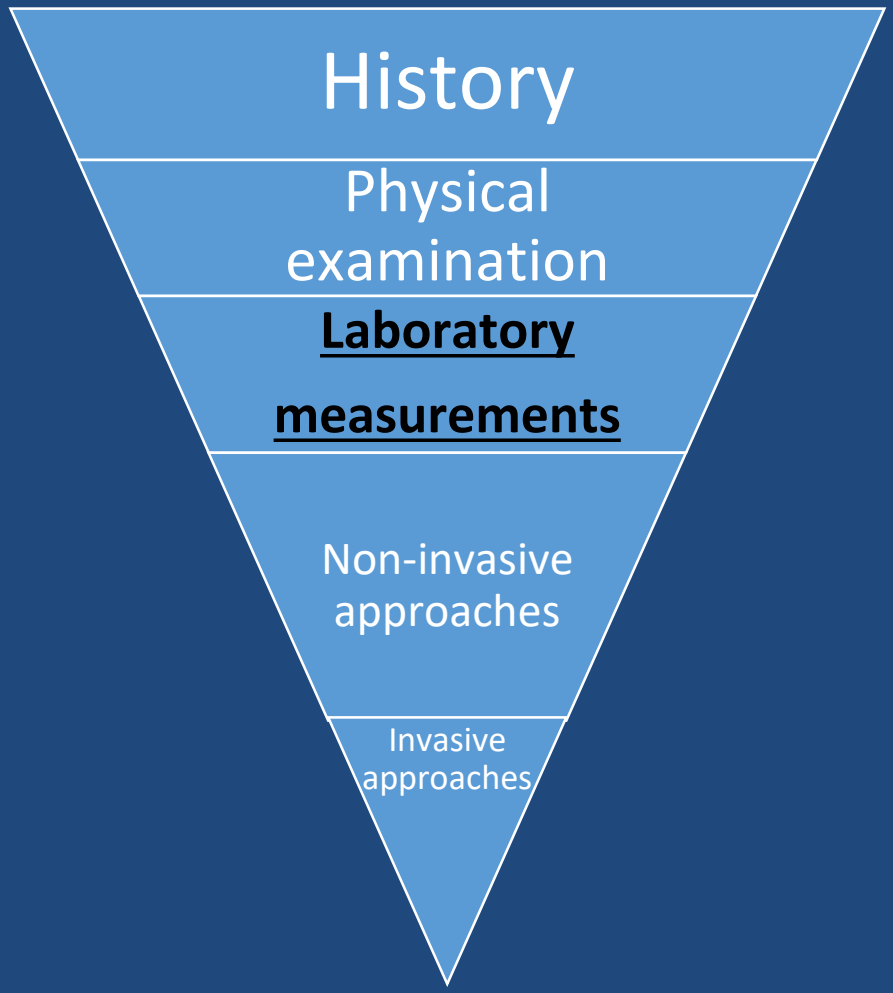
- Exclude structural heart disease/atrial flutter/fibrillation – critical for prognosis
- Exclude iatrogenic causes (drug induced bradycardia, hypo-, hyperkalemia, …).



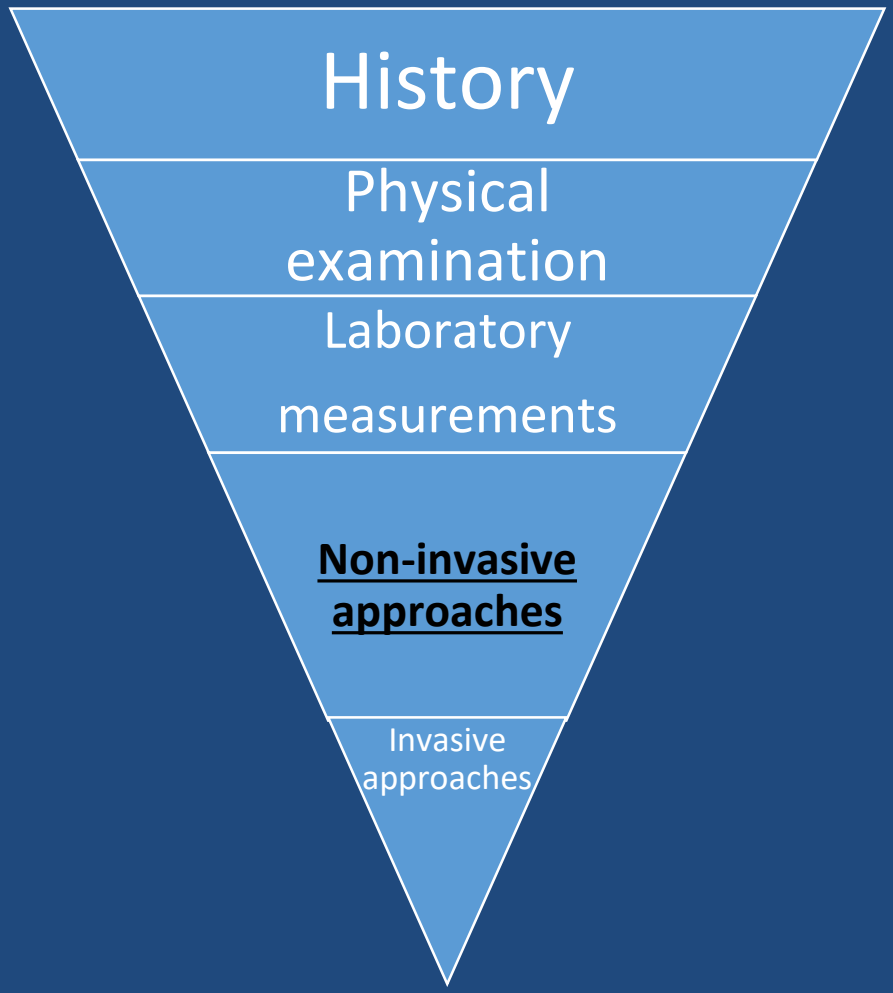
1. **What and where is the main problem (only 1)**
2. **Provocating/alleviating situations/maneuvres**
3. **Accompanying signs/risk factors, …**
4. **Intensity**
5. **Location**
6. **Time course/duration – new, long-lasting, worsening**



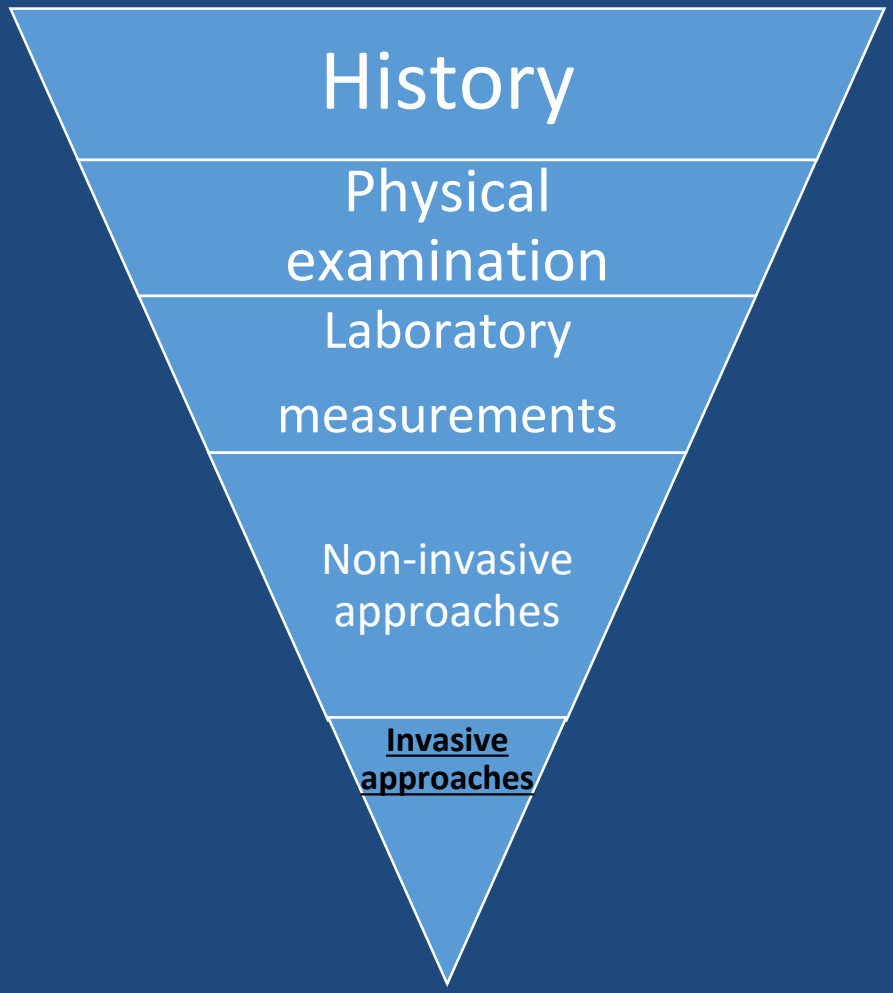
1. **General outlook – well, about to die, …**
2. **Hydratation, color, …**
3. **Vital signs BP (standing), HR, RR, Temperature, Saturation (02)**
4. **Location**
5. **Focus on suspicious area (auscultation, …)**



1. Glycemia
2. Blood gases (pH, pCO2, Po2, ...)
3. Cardiospecific markers
4. Blood count
5. Inflammatory markers: Sed. Rate, C-reactive protein, procalcitonine, interleukin-6, …
6. Minerals (Na, K, Cl, Ca, P, …)
7. Renal function – creatinine, urine analysis …
8. Status of coagulation INR/QUICK, aPTT, D-Dimers
9. Liver tests, bilirubin, amylases, albumin, …
10. Toxicology (unconsciousness of unknown origin …)
11. Bacteriology, parazitology
12. Other specific tests – hormonal status, imunology, ….

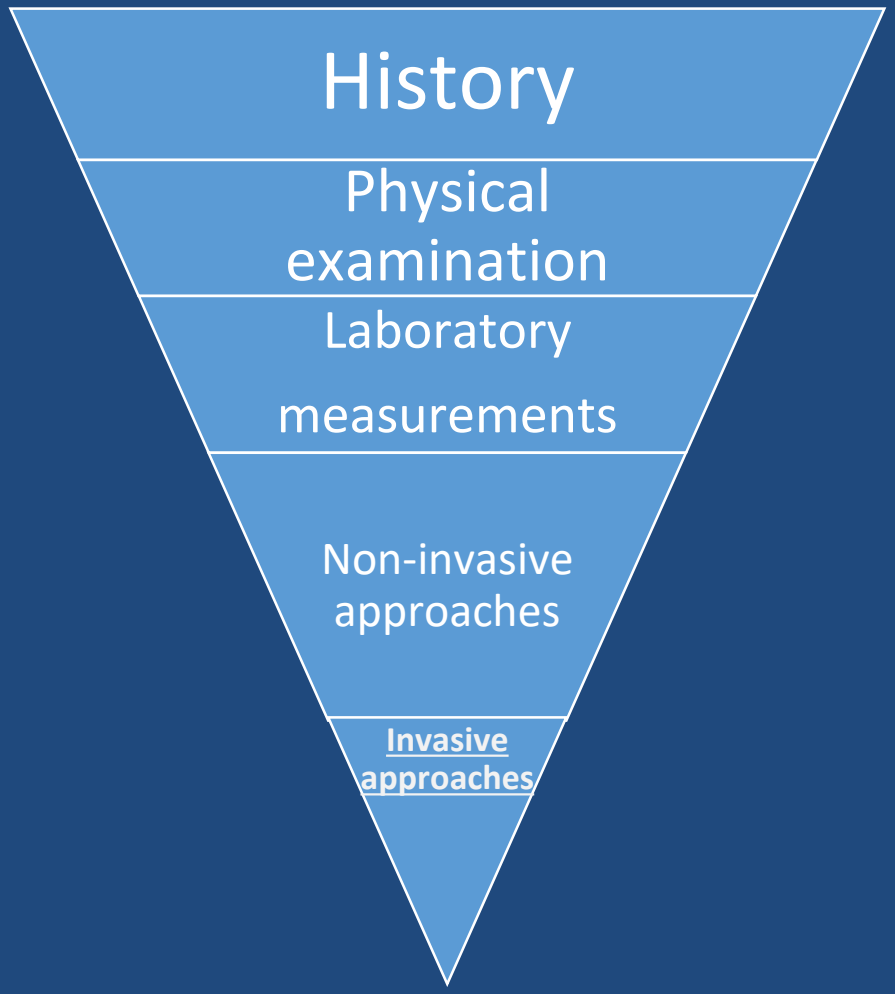


1. **ECG**
2. **Monitoring of ECG, Blood pressure**
3. **X-ray, ….**
4. **Ultrasound studies (echo in the case of heart)**
5. **Computer tomography (CT)**
6. **Magnetic resonance (MR)**
7. **Scintigraphy**
8. **Pozitron emission tomography (PET)**
9. **Functional tests– bicycle/treadmill ECG, tilt test, walking test**
10. **Combine 1-9, …**



1. Measurment of right heart pressures(CVP), intraarterial BP
2. Fibroscopy- gastro, broncho, …
3. **Angiography**
4. **Electrophys. studies**
5. Laparascopy
6. Sternal puncture
7. Biopsy
8. Lumbal puncture
9. Invasive imaging of body spaces
10....

FURTHER STEP(S)?



1. **Hospitalization –**

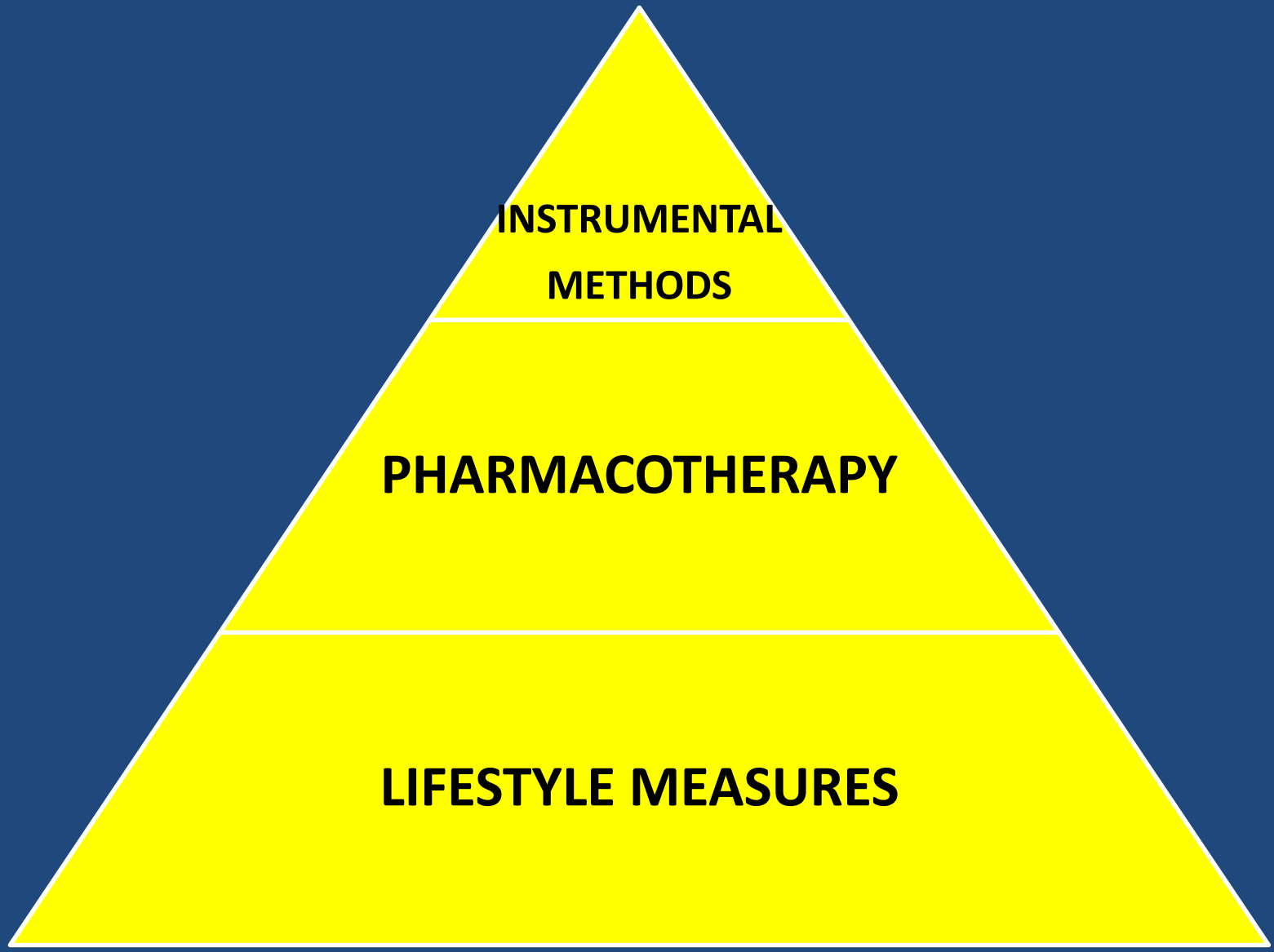
   monitoring of HR, …

2. **Implantation of PM**

3. **Defibrillation**

4. **EFStudies …**

5. **Correct pharmacotherapy**


INSTRUMENTAL METHODS
PHARMACOTHERAPY
LIFESTYLE MEASURES

MANAGEMENT

- CHECK THE PHARMACOTHERAPY (BRADYCARDIA, …)
- REVASCULARIZATION
- CORRECTION OF VALVE DISEASE
- MANAGEMENT OF HEART FAILURE INCL. TRANSPLANTATION
- MANAGEMENT OF INTERNAL ENVIRONMENT - - SUPPLEMENTATION/REMOVAL OF MINERALS
- SUPPLEMENTATION/BLOCK OF THYROID HORMONES
- CORRECTION OF ANEMIA …

LIFESTYLE MEASURES

- OMEGA3 FA
- SUPPLEMENTATION OF POTASSIUM AND MAGNESIUIM IN DIET
- DIET LOW IN ANIMAL FAT/SALT …

PHARMACOTHERAPY

DEFINITELY SAFE:

BETABLOCKERS,

IVABRADINE?

RELATIVELY SAFE

- AMIODARONE
- PROPAFENONE
- + ATROPIN, ISOPROTENEROL/ISOPRENALIN – BRADYCARDIA(S)
- **OTHERS** IN SPECIAL INDICATIONS … CAVEAT – PROARRHYTMOGENIC EFFECT (TRIMECAIN, …)

INSTRUMENTAL THERAPY

- **INCREASINGLY USED:**
- **CARDIOSTIMULATION – TEMPORARY, PERMANENT**
- **DEFIBRILLATION – ICD**
- **EL. CARDIOVERSION**
- **RADIOFREQUENCY ABLATION**
- **CRYOABLATION, ALCOHOL. ABLATION …**

ATRIAL FIBRILLATION – treatment

- Anticoagulation, rate control
- One trial of el. version recommended (according to the size of the left atrium)
- Control of rate x control of rhythm
- Exclude other causes: not well compensated hypertension, pulmonary emboli, hyperthyroidism, hypokalemia (plasma potassium optimally more than 4 mmol/l)
- Radiofrequency ablation ?

ATRIAL FLUTTER similar approach as in ATRIAL FIBRILLATION

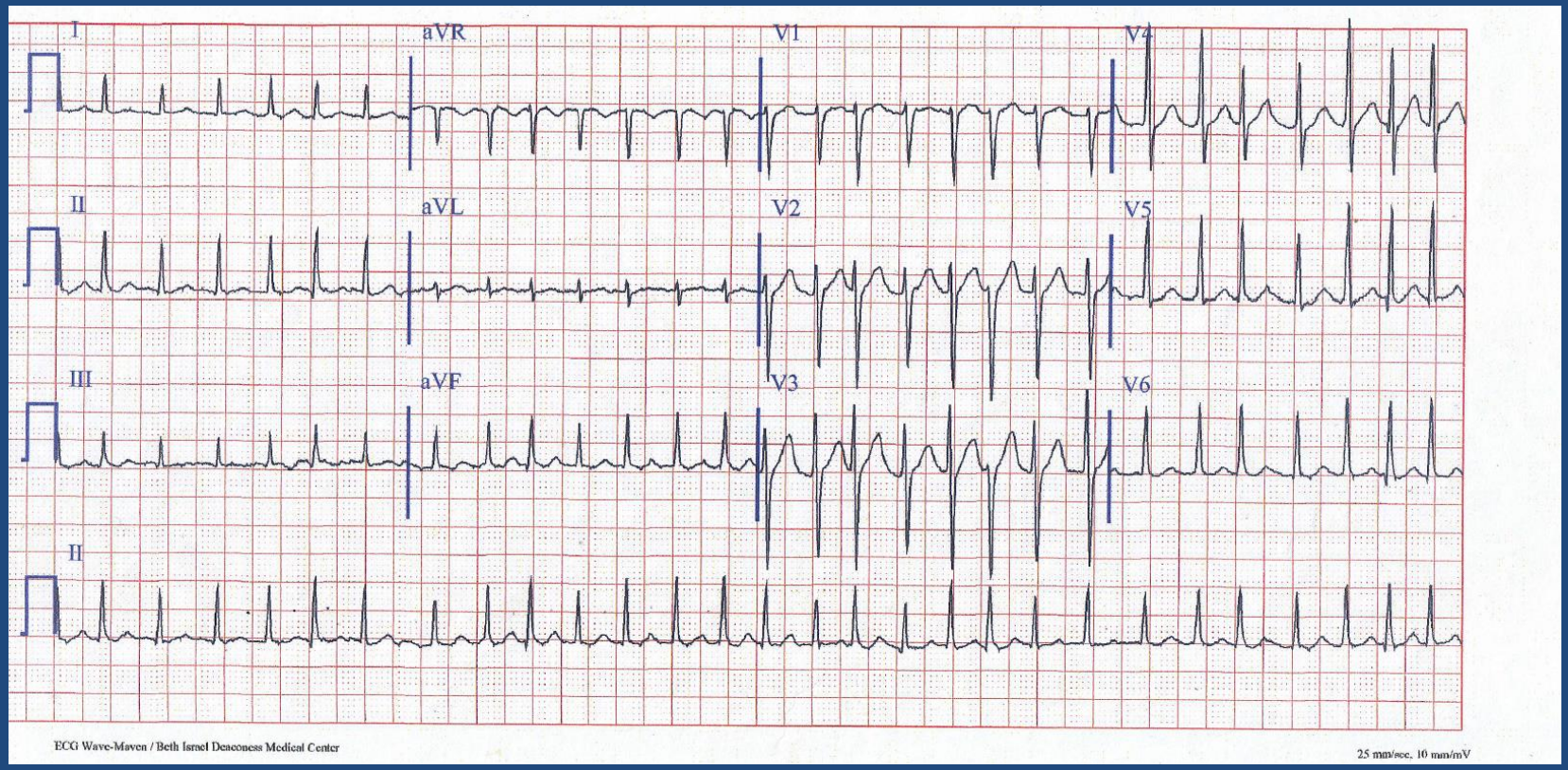
But:

MORE FREQUENTLY UNDERLYING ORGANIC HEART DISEASE

RFA MORE SUCCESSFUL

SPECIFIC MEASURES/MANAGEMENT

- ANTICOAGULATION – ATRIAL FIBRILLATION/FLUTTER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V1
V4
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aVL
V2
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V3
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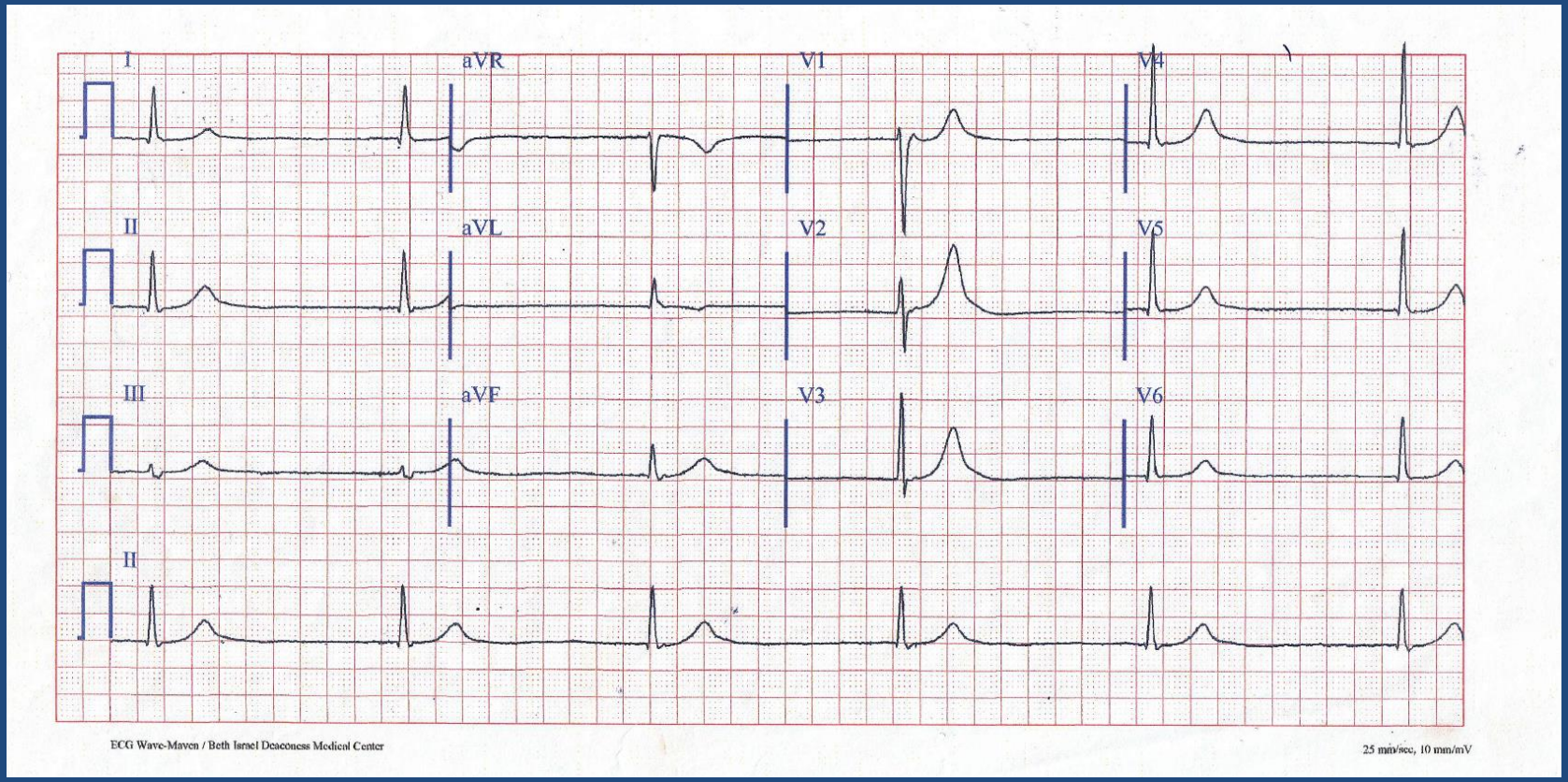
II
ECG Wave-Maven / Beth Israel Deaconess Medical Center
25 mm/sec, 10 mm/mV

ECG_1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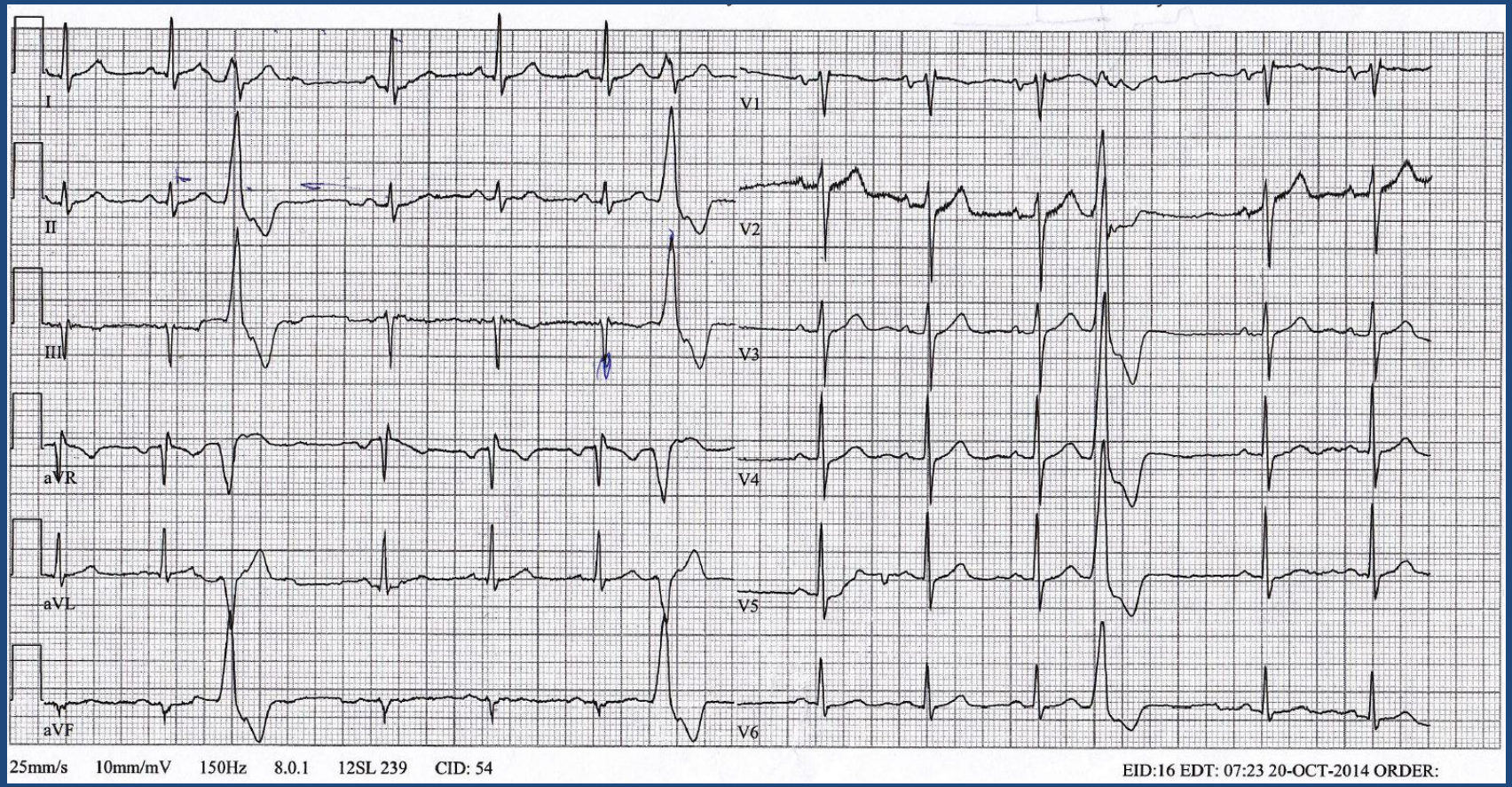
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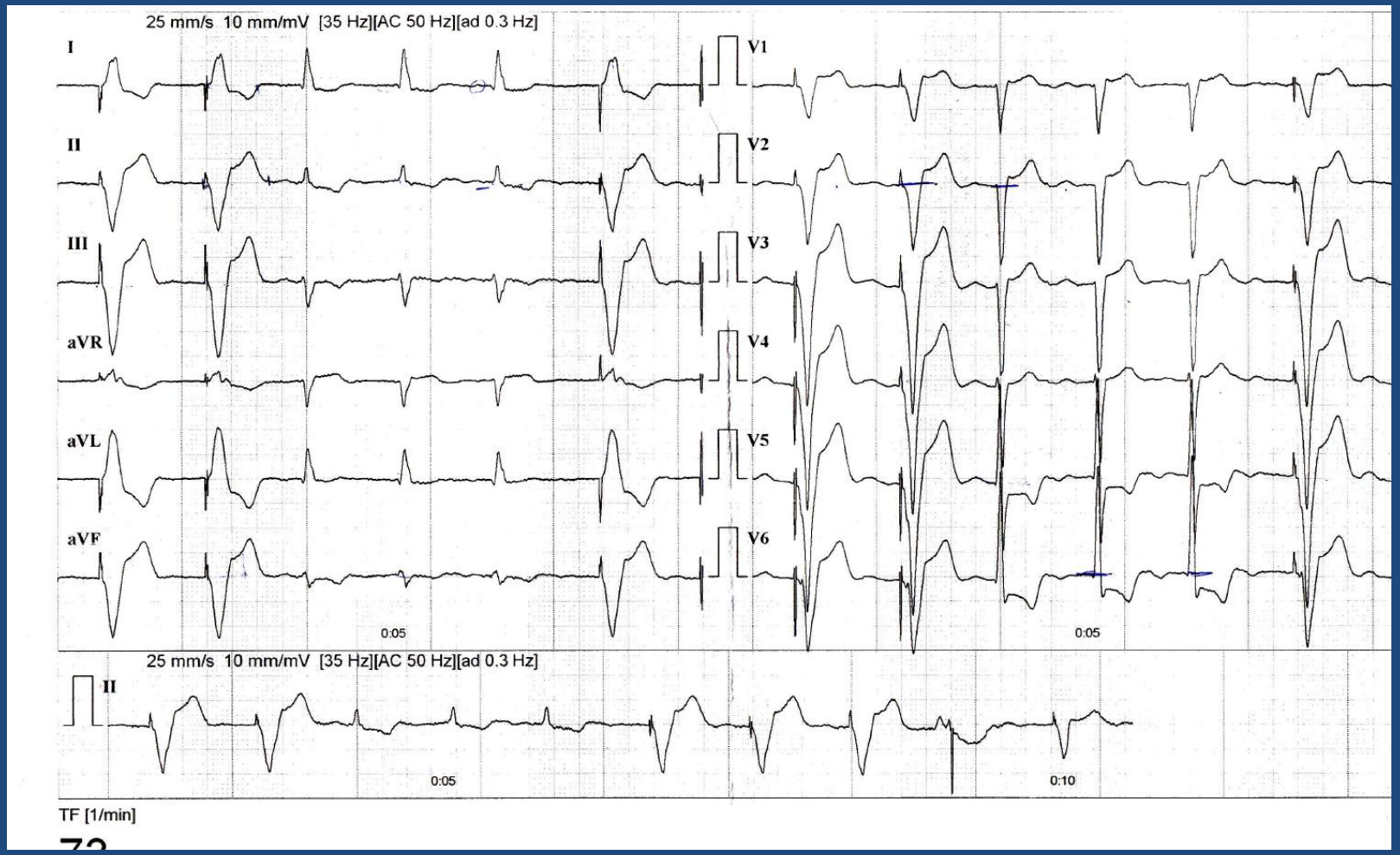
25 mm/s 10 mm/mV [35 Hz][AC 50 Hz][ad 0.3 Hz]
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V2
V3
V4
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0:05
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II
0:05
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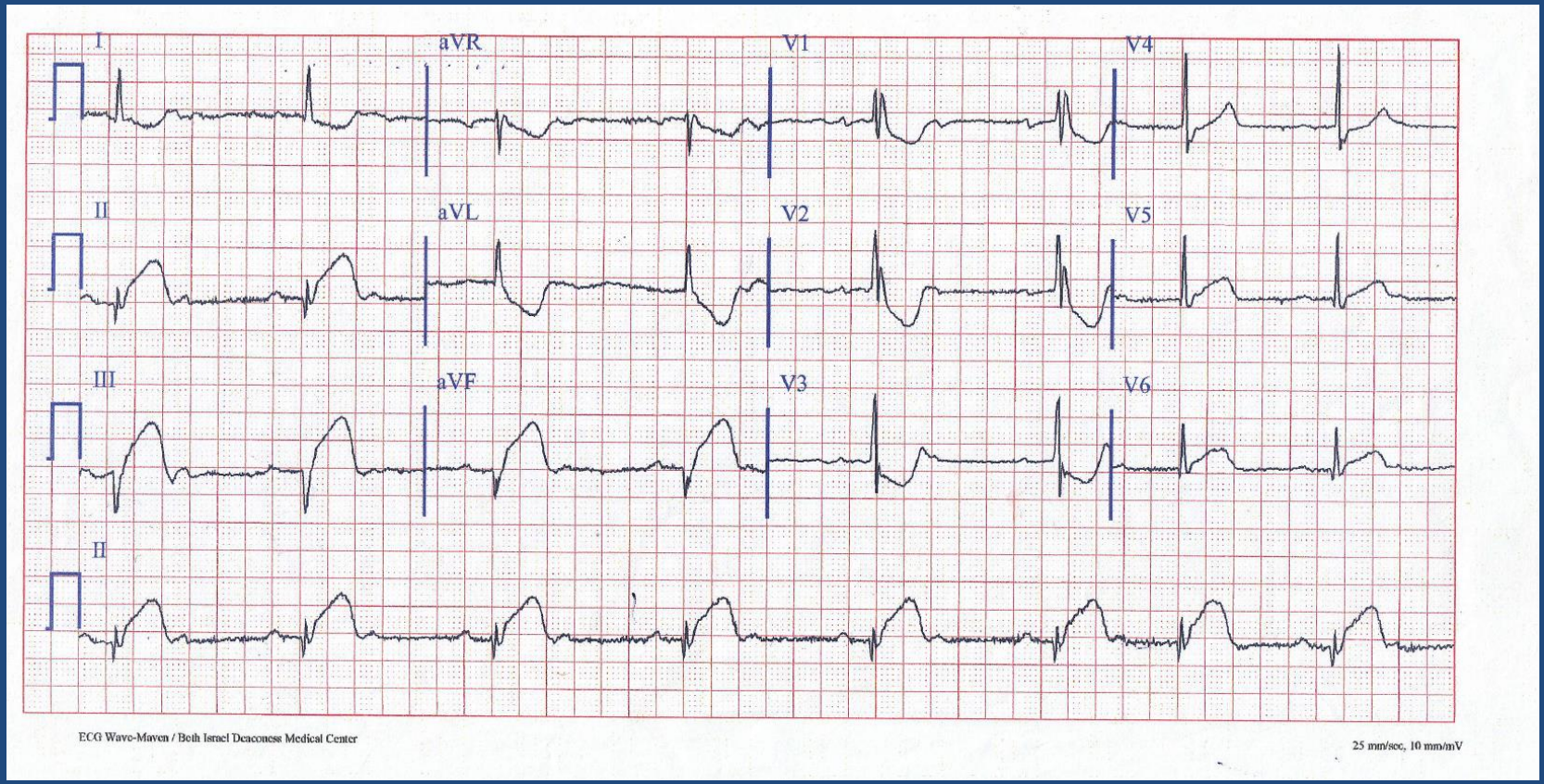
TF [1/min]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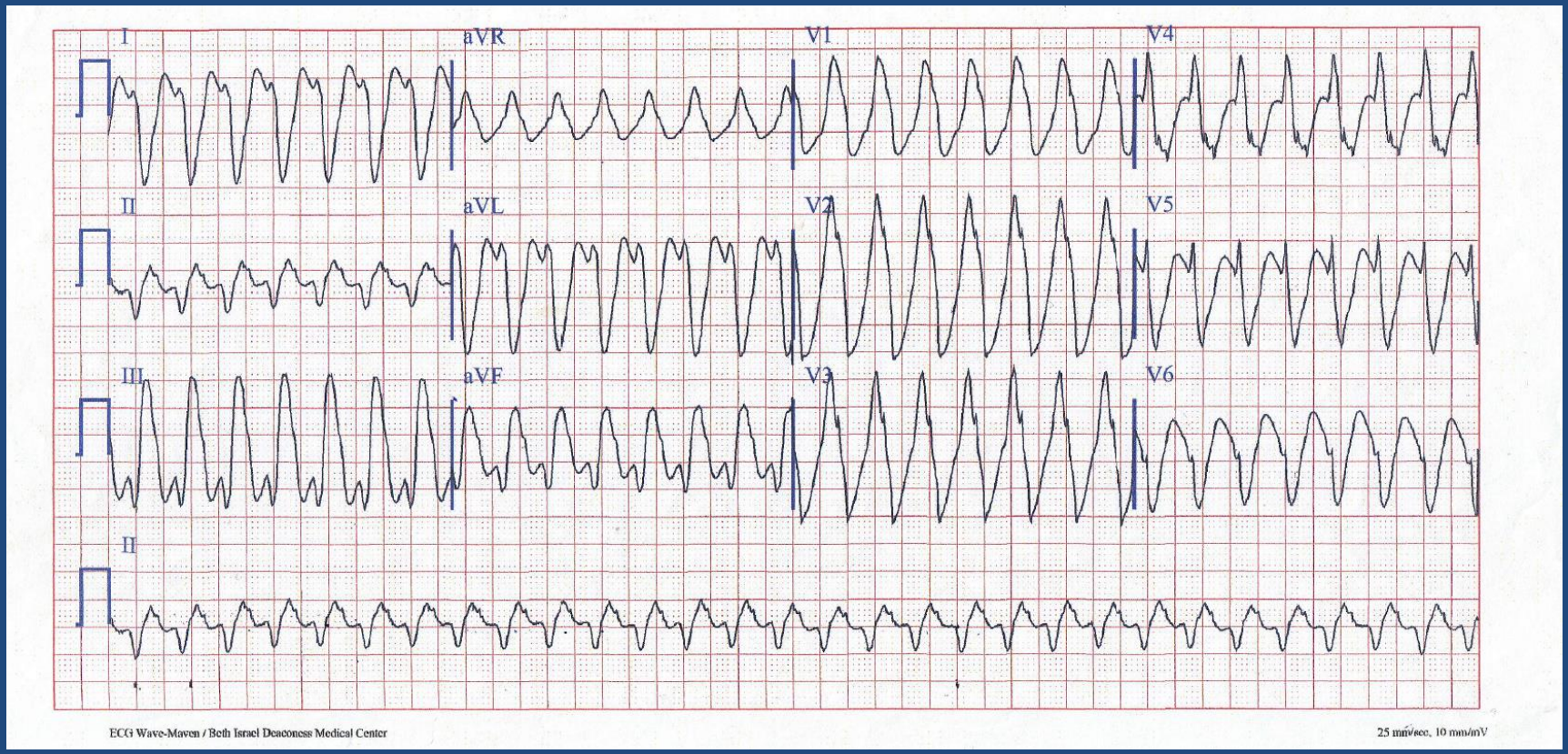
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ECG Wave-Maven / Beth Israel Deaconess Medical Center
25 mm/sec, 10 mm/mV

THANK YOU FOR ATTENTION

ISCHEMIC HEART DISEASE CHRONIC

J. Pitha

OUTLINE

- INTRODUCTION
- PATHOPHYSIOLOGY
- CASES
- DEFINITIONS
- DIFF. DG. – SYMPTOMS, SIGNS, EVALUATION
- MANAGEMENT

Souhrn Doporučených postupů ESC pro diagnostiku a léčbu stabilní ischemické choroby srdeční – 2013.

Připraven Českou kardiologickou společností

(Summary of the 2013 ESC guidelines on the management of stable coronary artery disease. Prepared by the Czech Society of Cardiology)

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[b] *Kardiocentrum – Kardiologie, Nemocnice České Budějovice, a. s., Česká republika*
[c] *Kardioambulance, s. r. o., Praha, Česká republika*

DEFINITION/PATHOPHYSIOLOGY: ANGINA PECTORIS

Stable coronary artery disease is generally characterized by episodes of reversible myocardial demand/supply mismatch, related to ischaemia or hypoxia, which are usually inducible and reproducible by exercise, emotion or other stress, but may also be occurring spontaneously. Such episodes of ischaemia/hypoxia are commonly associated with transient chest discomfort (angina pectoris). SCAD also includes the stabilized, often asymptomatic, phases that follow an ACS. The various clinical presentations of SCAD are associated with different underlying mechanisms that mainly include: (i) plaque-related obstruction of epicardial arteries; (ii) focal or diffuse spasm of normal or plaque-diseased arteries; (iii) microvascular dysfunction and (iv) left ventricular dysfunction caused by prior acute myocardial necrosis and/or hibernation

DEFINITION/PATHOPHYSIOLOGY MISMATCH SUPPLY X DEMAND O2 IN MYOCARDIUM (hypoxia x ischemia)

LOWER SUPPLY

Obstructive coronary disease (atherosklerotic changes, vasculitis)

Spasms of coronary arteries

Aortic valve stenosis
Dissection of asc. aorta

Low (ox)Hb

INCREASED DEMAND

Myocardial hypertrophy

(hypertension, aortic valve stenosis)

Tachycardias (arrhythmias, febrile st., hyperthyrosis, …)

+ OVERLAP

DEFINITION(S)
CORONARY HEART DISEASE V.S. ISCHEMIC HEART DISEASE

ARTERIES	Blood flow	Clinical picture
Early plaque, non-significant stenosis	No limitation(s)	Asymptomatic *Emboli, rupture if vulnerable*
Stabile plaque stenosis more than 70 %	Limitation during exercise	Stabile angina pectoris
Unstable/vulnerable plaque	Generation of thrombi, spasms even at rest – flow limitation	Unstable angina pectoris
Rupture of unstable plaque, thrombus formation	Transitional thrombus its lysis, incomplete occlusion	NonSTEMI subendocardial ischemia
Complete thrombus on (ruptured) unstable plaque	Complete occlustion	STEMI transmural MI

MAIN FEATURES OF STABLE CORONARY ARTERY DISEASE (SCAD)/ ANGINA PECTORIS

Pathogenesis

Stable anatomical atherosclerotic and/or functional alterations of epicardial vessels and/or microcirculation

Natural history

Stable symptomatic or asymptomatic phases which may be interrupted by ACS

Mechanisms of myocardial ischaemia

- Fixed or dynamic stenoses of epicardial coronary arteries;
- Microvascular dysfunction;
- Focal or diffuse epicardial coronary spasm;
- The above mechanisms may overlap in the same patient and change over time.

Clinical presentations

Effort induced angina caused by:

- epicardial stenoses;
- microvascular dysfunction;
- vasoconstriction at the site of dynamic stenosis;
- combination of the above.

Rest angina caused by:

- Vasospasm (focal or diffuse);
- epicardial focal;
- epicardial diffuse;
- microvascular;
- combination of the above.

Asymptomatic:

- because of lack of ischaemia and/or of LV dysfunction;
- despite ischaemia and/or LV dysfunction.

Ischaemic cardiomyopathy

HISTORY – SIGNS AND SYMPTOMS – PROVOCING/ALLEVIATING FACTORS

- Asymptomatic
- Chronic IHD – stable angina pectoris (AP)
- Acut forms IHD – unstable AP (incl. new onset), acute myocardial infarction – STEMI, non-STEMI
- Heart failure
- Arrhytmias – atrial fibrillation
- Sudden death – malign arrhytmia(s)

DIAGNOSTIC APPROACH

ACUTE IHD

- HISTORY + PHYSICAL FINDINGS (BP in both upper extremities), ECG CHANGES (AT REST), LABORATORY MARKERS (TROPONINS)

CHRONIC form

- HISTORY (!) + PHYSICAL FINDINGS (?)
- ECG: TREADMILL/BICYCLE
- Echokardiography
- Scintigraphy
- CT, MR, … coronary angiography
- Laboratory tests (anemie, TSH, …)

CAUSES OF CHEST PAIN ACCORDING TO LOCATION AND IMPORTANCE/URGENCY – DIFFERENTIANL DIAGNOSIS

	Imminent threat to life	Less urgent
Cardiac	Acute forms of ischemic heart disease Dissection of (thoracic)aorta	Stabile angina pectoris Pericarditis Mitral valve prolapse Stenosis of aortic valve
Pulmonary	Hemodynamicaly signifficant pulmonary embolism Tension pneumothorax	Hemodynamicaly non-significant pulmonary embolism Non-tension pneumothorax Pleuritis
Other	Mediastinitis (rupture of esofagus, …) Rupture/penetration of gastric ulcer	Anemia, Gastroesofageal refflux, Costochondritis Vertebrogenic origin, …

EPIDEMIOLOGY: ANGINA PECTORIS

The prevalence of angina in population-based studies increases with age in both sexes, from 5 to 7% in women aged 45–64 years to 10–12% in women aged 65–84 and from 4 to 7% in men aged 45–64 years to 12–14% in men aged 65–84. Epidemiological data on microvascular angina and vasospastic angina are missing.

Estimates for annual mortality rates range from 1.2 to 2.4% per annum, with an annual incidence of cardiac death between 0.6 and 1.4% and of non-fatal myocardial infarction (MI) between 0.6% and 2.7%. The outcome is worse in patients with reduced left ventricular ejection fraction, heart failure, a greater number of diseased vessels, more proximal locations of coronary stenoses, greater severity of lesions, more extensive ischaemia, more impaired functional capacity, older age and more severe angina.

CHARACTERISTICS OF ANGINA PECTORIS

Typical angina	Meets all three of the following characteristics: • substernal chest discomfort of characteristic quality and duration; • provoked by exertion or emotional stress; • relieved by rest and/or nitrates within minutes.
Atypical angina (probable)	Meets two of these characteristics
Non-anginal chest pain	Lacks or meets only one or none of the characteristics

CANADIAN CLASSIFICATION OF ANGINA PECTORIS

Class I	Ordinary activity does not cause angina such as walking and climbing stairs. Angina with strenuous or rapid or prolonged exertion at work or recreation
Class II	Slight limitation of ordinary activity. Angina on walking or climbing stairs rapidly, walking or stair climbing after meals, or in cold, wind or under emotional stress, or only during the first few hours after awakening. Walking more than two blocks on the level and climbing more than flight of ordinary stairs at a normal pace and in normal conditions
Class III	Marked limitation of ordinary physical activity. Angina on walking one to two blocks[a] on the level or one flight of stairs in normal conditions and at a normal pace
Class IV	Inability to carry on any physical activity without discomfort – angina syndrome may be present at rest

[a] Equivalent to 100–200 m.

TESTS FOR CORONARY HEART DISEASE

	Sensitivity	Specificity
Exercise ECG[a]	45–50	85–90
Exercise stress echocardiography	80–85	80–88
Exercise stress SPECT	73–92	63–87
Dobutamine stress echocardiography	79–83	82–86
Dobutamine stress MRI[b]	79–88	81–91
Vasodilator stress echocardiography	72–79	92–95
Vasodilator stress SPECT	90–91	75–84
Vasodilator stress MRI[b]	67–94	61–85
Coronary CTA[c]	95–99	64–83
Vasodilator stress PET	81–97	74–91

AGGRAVATING FACTORS

- Anemia
- Hyperthyrosis
- Not well controlled blood pressure

MANAGEMENTS OF VASCULAR DISEASE

Strategy

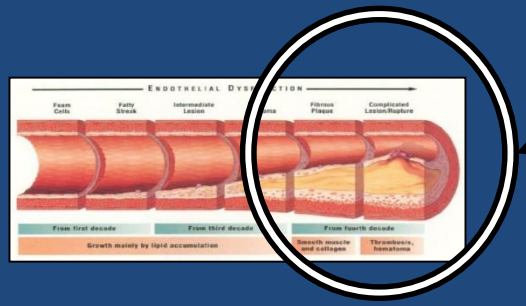
- **To offer better and longer life**

Tactics

- **Find (out) and remove as much of CV risk as possible**

PREVENTION:

DEVELOPMENT OF ATHEROSCLEROSIS



(NEW) BIOMARKERS

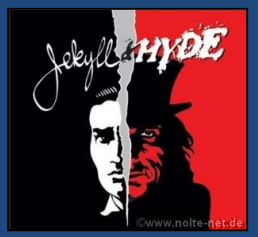
Cardiovascular events

Non-fatal 33 %

Fatal - 66 %

Chambless L et al,Population versus clinical view of case fatality from acute coronary heart disease: results from the WHO MONICA Project 1985-1990, Multinational MONItoring of Trends and Determinants in CArdiovascular Disease, Circulation, 1997;3849-59,

ATHEROSCLEROSIS



LDL particles

(LDL cholesterol more than 2.0 mmol/L)

Dysfunction of the arteries Endothelial dysfunction

HDL particles/reverse cholesterol transport ?

(HDL cholesterol in men more than 1.1, in women more than 1.3 mmol/L)

Remnant lipoproteins

(Triglycerides more than 2.0 mmol/L)

Morphollogical changes - plaques

Destabilisation of plaques

Endothelial progenitor cells/stem cells

Inflammatory markers

(hsCRP, IL-6, ICAM,

V-CAM, TNF alfa, …)

(Endothelial) microparticles

Clinical events caused by atherosclerosis.

Stabilisation of atherosclerotic plaques

MAIN RISK FACTORS FOR CVD

NON-MANAGEABLE

1) AGE (45y m, 55y w)

2) MALE GENDER

3) GENETICS

(Fam. history – 60 y m, 65 y w)

MANAGEABLE

1) SMOKING

2) DYSLIPIDEMIA

3) HYPERTENSION (140/90 mm Hg and above)

4) DIABETES M.

(fasting/nonfasting glycemia more than 7/11 mmol/l)

5) RENAL DISEASE

ORBITA STUDY

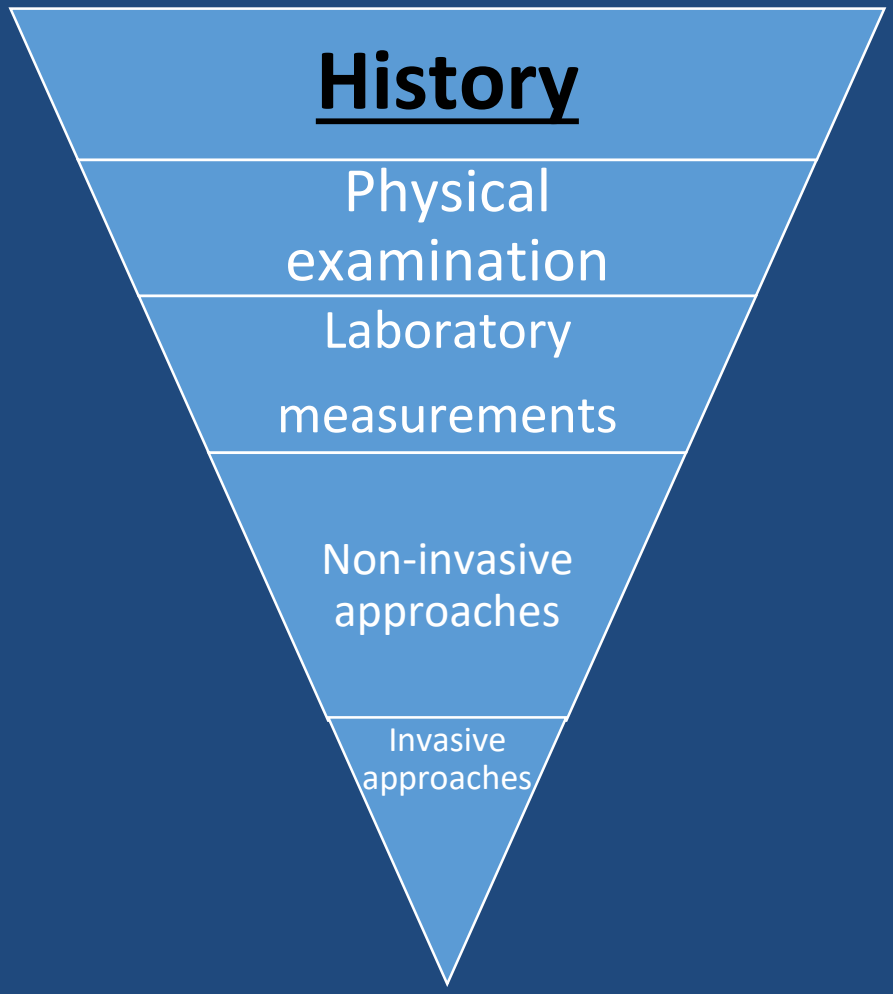
- Patients with stable angina and evidence of severe single-vessel stenosis were randomized in a 1:1 fashion to either PCI or a placebo sham procedure. After enrollment, patients received 6 weeks of medication optimization. Coronary angiography was done via a radial or femoral arterial approach with auditory isolation achieved by placing over-the-ear headphones playing music on the patient throughout the procedure. In all patients, a research invasive physiological assessment of fractional flow reserve (FFR) and instantaneous wave-free ratio (iFR) was done. The operator was blinded to the physiology values and therefore did not use them to guide treatment. Randomization occurred after this physiological assessment.
- For patients allocated to PCI, the clinical operator used drug-eluting stents (DES) to treat all lesions that were deemed to be angiographically significant, with a mandate to achieve angiographic complete revascularization. After PCI, iFR and FFR were measured again. In the placebo group, patients were kept sedated for at least 15 minutes on the catheter laboratory table and the coronary catheters were withdrawn with no intervention having been done.

ORBITA STUDY

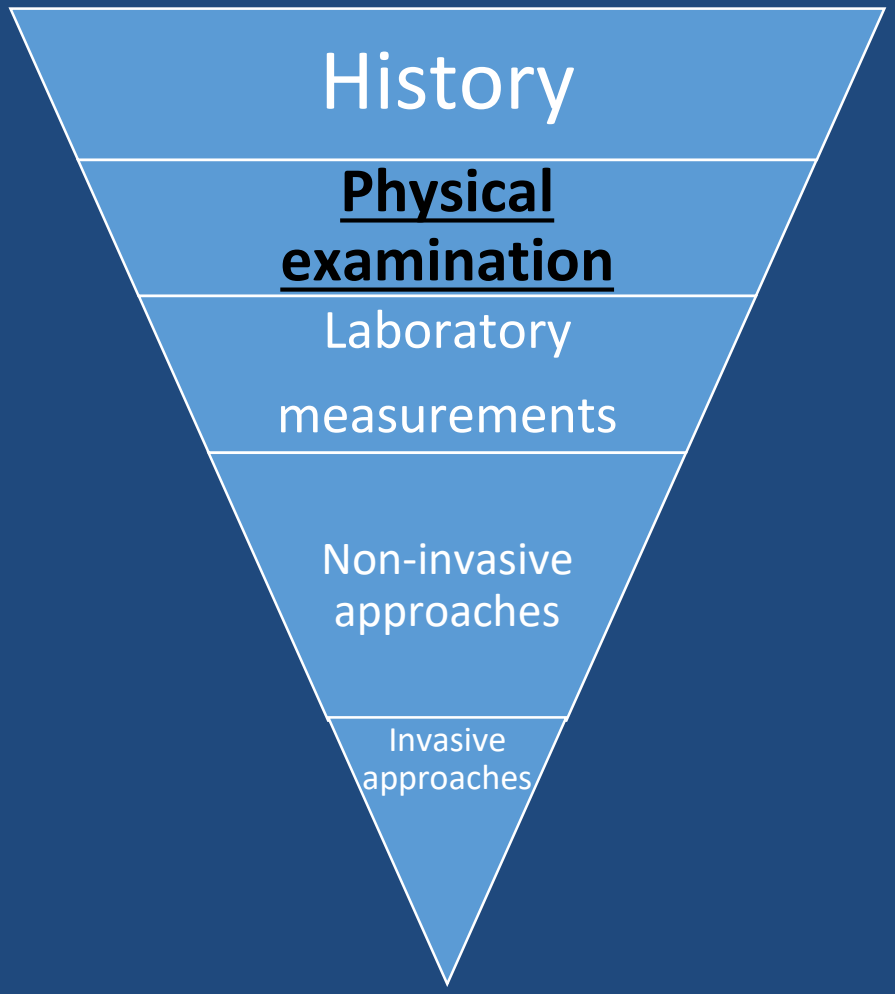
- Total number of enrollees: 200
- Duration of follow-up: 6 weeks
- Mean patient age: 66 years
- Percentage female: 27%
- Inclusion criteria:
- Age 18-85 years
- Stable angina/angina equivalent
- At least one angiographically significant lesion (≥70%) in a single vessel that was clinically appropriate for PCI
- Principal Findings:
- The primary outcome, change in exercise time from baseline for PCI vs. sham, was 28.4 vs. 11.8 seconds, $p = 0.2$.
- Secondary outcomes for PCI vs. sham:
- Change in Seattle Angina Questionnaire (SAQ)-physical limitation from baseline: 7.4 vs. 5.0, $p = 0.42$
- Change in SAQ-angina frequency from baseline: 14.0 vs. 9.6, $p = 0.26$
- Change in Duke treadmill score from baseline: 1.22 vs. 0.1, $p = 0.10$
- Complete freedom from angina: 49.5% vs. 31.5%, $p < 0.05$

ORBITA STUDY

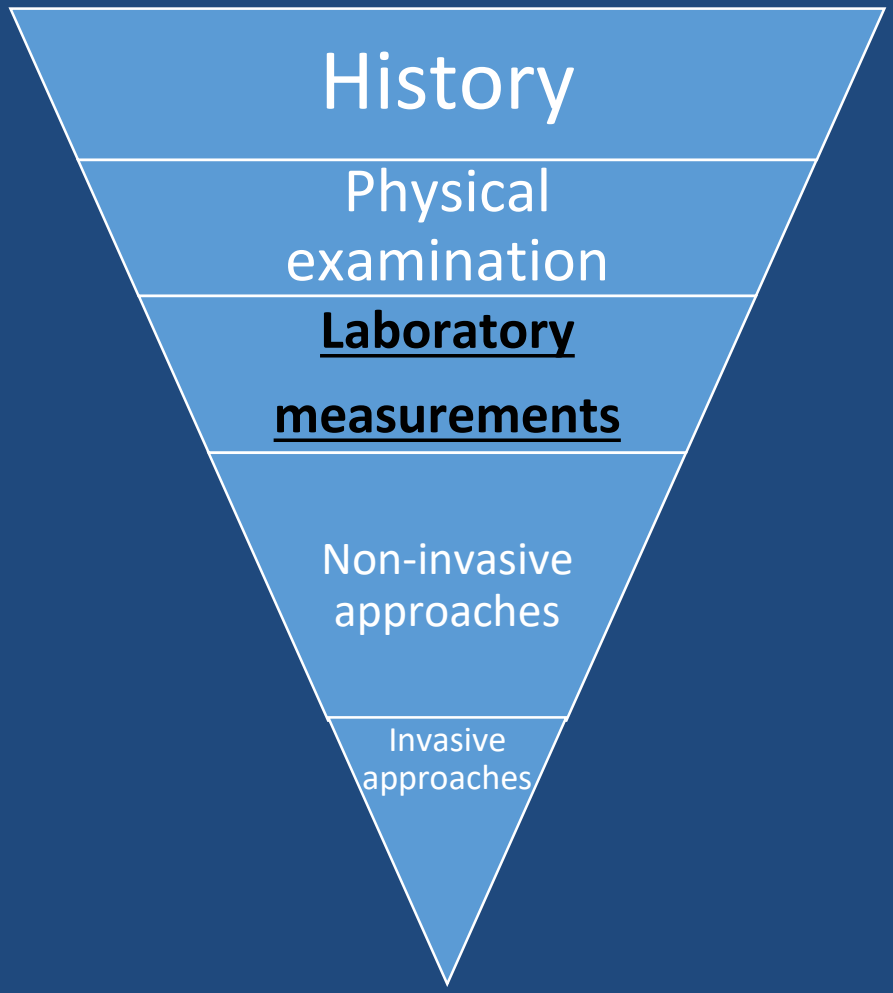
- The results of this trial indicate that among patients with stable angina, PCI does not result in greater improvements in exercise times or anginal frequency compared with a sham procedure. This was despite the presence of anatomically and functionally significant stenoses. PCI did however resolve ischemia more effectively, as ascertained by follow-up stress echocardiography.



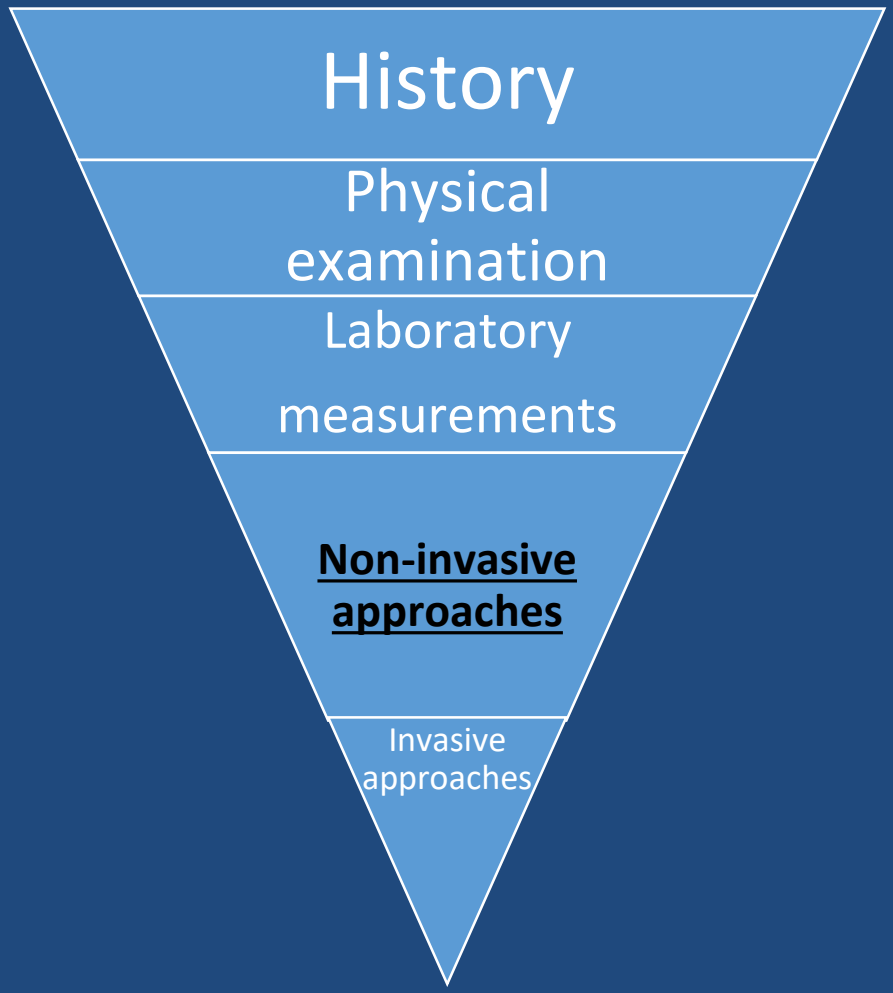
1. **What and where is the main problém**
2. **Provocating/alleviating situations/maneuvres**
3. **Accompanying signs/risk factors, …**
4. **Intensity**
5. **Location**
6. **Time course/duration – new, long-lasting, worsening**



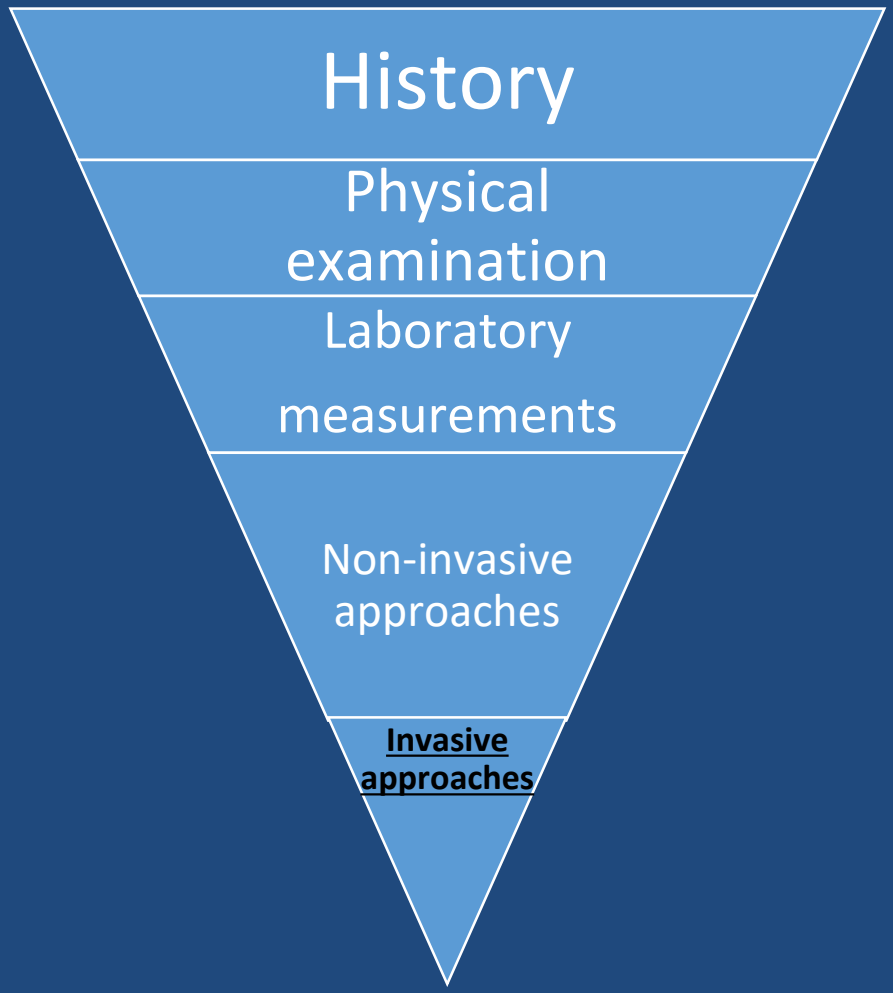
1. General outlook – well, about to die, …
2. Hydratation, color, …
3. Vital signs BP (standing), HR, RR, Temperature, Saturation (02)
4. Location
5. Focus on suspicious area (auscultation, …)



1. Glycemia
2. Blood gases (pH, pCO2, Po2, ...)
3. Cardiospecific markers
4. Blood count
5. Inflammatory markers: Sed. Rate, C-reactive protein, procalcitonine, interleukin-6, …
6. Minerals (Na, K, Cl, Ca, P, …)
7. Renal function – creatinine, urine analysis …
8. Status of coagulation INR/QUICK, aPTT, D-Dimers
9. Liver tests, bilirubin, amylases, albumin, …
10. Toxicology (unconsciousness of unknown origin …)
11. Bacteriology, parazitology
12. Other specific tests – hormonal status (TSH), imunology, ….

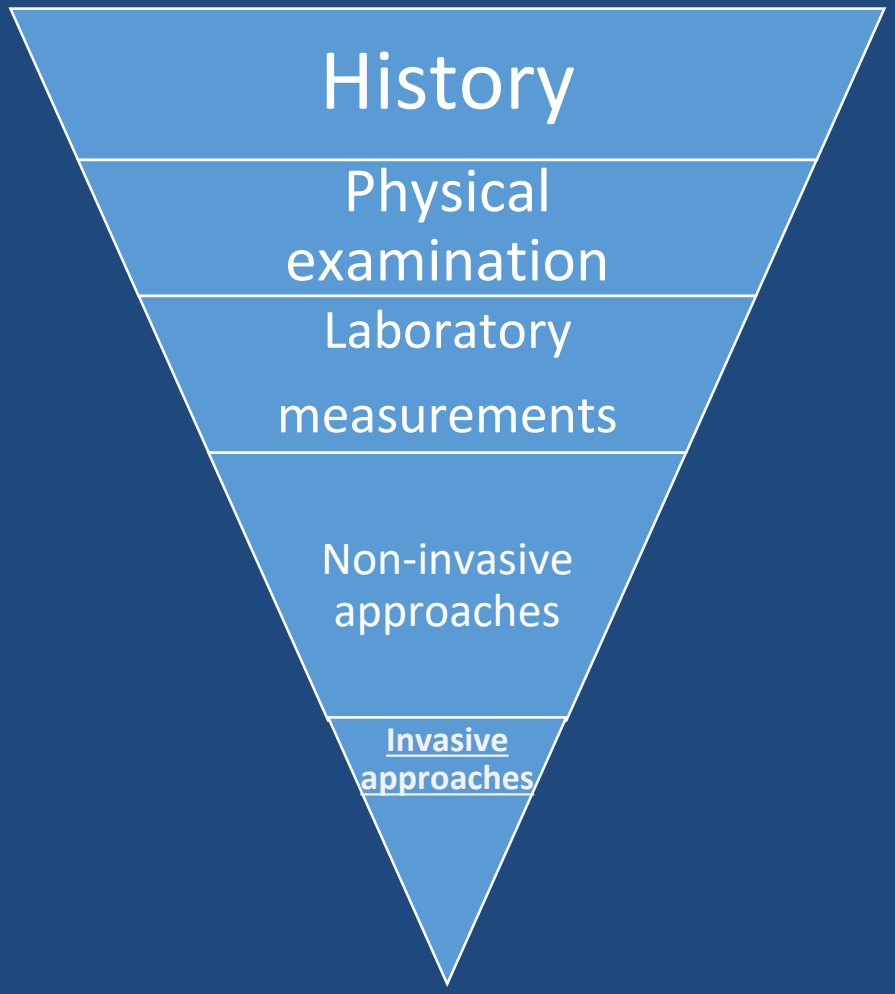


1. ECG
2. Monitoring of ECG, Blood pressure
3. X-ray, ….
4. Ultrasound studies (echo in the case of heart)
5. Computer tomography (CT)
6. Magnetic resonance (MR)
7. Scintigraphy
8. Pozitron emission tomography (PET)
9. Functional tests– bicycle/treadmill ECG, tilt test, walking test
10. Combine 1-9, …



1. Measurment of right heart pressures(CVP), intraarterial BP
2. Fibroscopy- gastro, broncho, …
3. Angiography
4. Electrophys. studies
5. Laparascopy
6. Sternal puncture
7. Biopsy
8. Lumbal puncture
9. Invasive imaging of body spaces
10....

FURTHER STEP(S)?



1. **Correct pharmacotherapy**
2. **Remove aggravating factors**
3. **Revascularize**

CASE REPORT

- 63 y. old woman chest pain/dyspnea during walking (uphill), cease at rest. Duration 1-2 years, progressive nature. Non-smoker, 5 years treated for high blood pressure, knows about high cholesterol levels for several years, untreated. At examination without any problems.

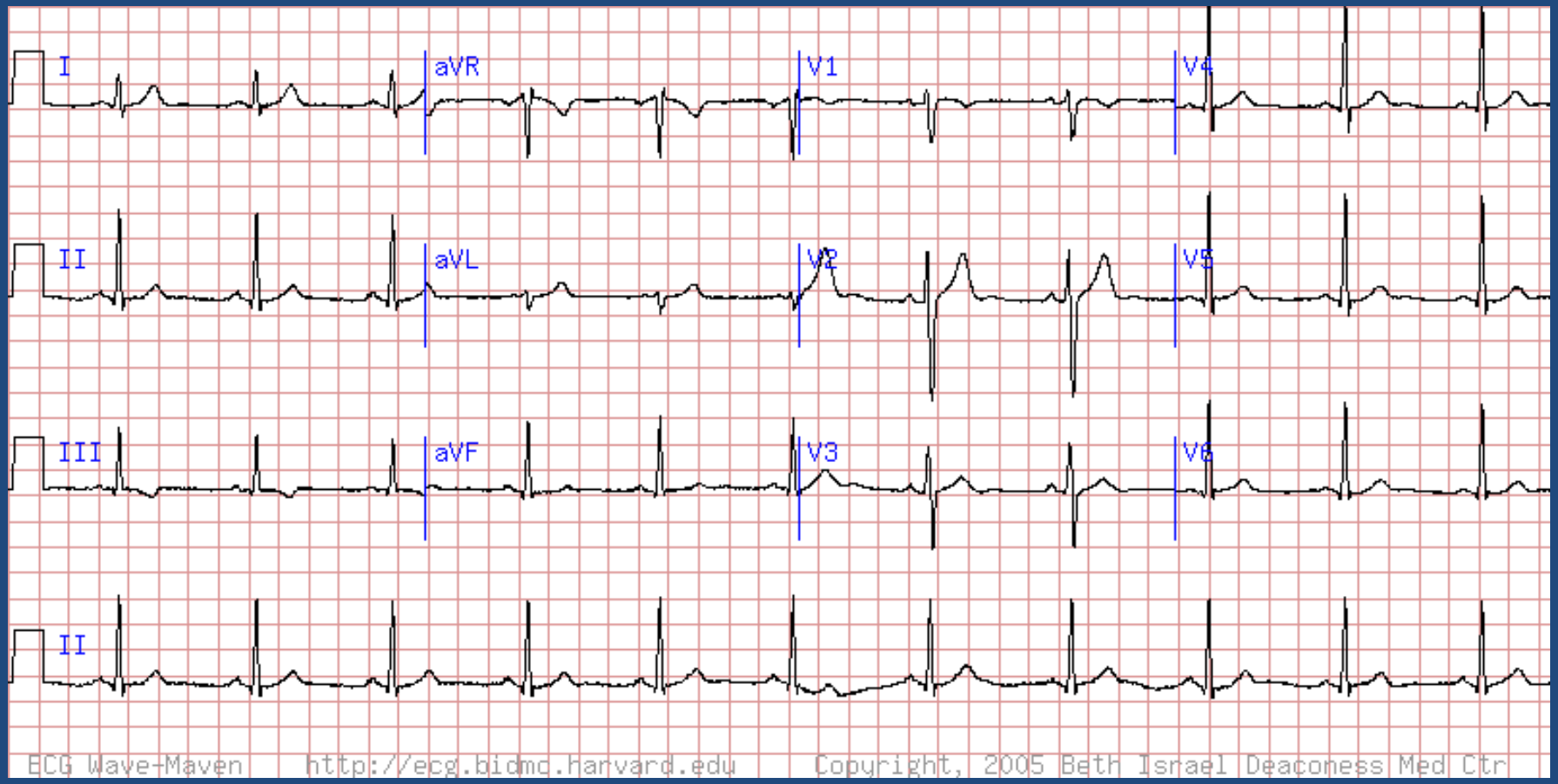
CASE REPORT (63 yo. Woman): other diseases

- During excretory urography (15 years ago) severe and typical anaphylaxis.
- Potential pulmonary asthma (betablockers well tolerated ?) Not ideal control of hypertension well responded to improvement of therapy.
- Allergies: contrast agent, dust, pollens
- Medication: Lokren 20 mg (betaxolol) 1-0-0, Stacyl (ASA) 100 mg, Lorista H 50/12,5 mg (ARB + HCTH) 1-0-0, Tenaxum 0-0-0-1 (imidazo rc. Agonist), Sortis 40 mg (atorvastatin) 0-0-0-1, Elicea 10 mg 1-0-0, Calcichew, Vitamin D, Panzytrát,Tensiomin (captopril) as neeed, Ventolin inh. d.p.

CASE REPORT (63 y.o. Woman): physical findings

Height: 172 cm, Weight: 79.2 kg, waist circumference 79 cm. BP-128/88, 134/80 mm Hg, bpm-66/min, regular. Heart: regualar 2 sounds, short systolic murmur above apex, no spreading. LE – without edema, otherwise normal.

Resting ECG


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
ECG Wave-Maven
http://ecg.bidmc.harvard.edu
Copyright, 2005 Beth Israel Deaconess Med Ctr

NEXT STEP(S) ?

3 STEPS:

1. Probality of IHD
2. Non-invasive tests
3. Risk/prognosis assessment revaskularize ?

Step I is the determination of the pre-test probability. In patients with intermediate probability step 2 consists of non-invasive testing to establish the diagnosis of SCAD including non-obstructive atherosclerosis. The latter may also be useful in patients with a PTP for SCAD < 15% but intermediate probability of atherosclerosis e.g. measured by SCORE system. Step 3 consists of stratifying for risk of subsequent events usually on the basis of available non-invasive tests in patients at intermediate PTP. Usually, optimal medical therapy will be instituted between steps 2 and 3. In patients with severe symptoms who have a high-intermediate or high pre-test probability of disease, early invasive coronary angiography (ICA) with appropriate invasive confirmation of the significance of a stenosis (usually by FFR) and subsequent revascularization may be appropriate bypassing non-invasive testing in steps 2 and 3.

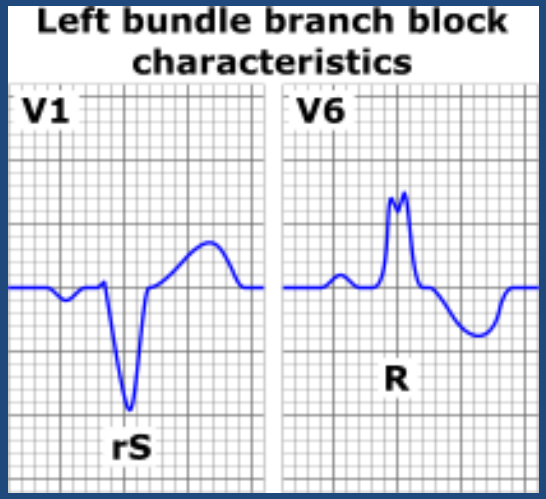
63 y. o. woman : ECHOKARDIOGRAPHY

- Mitrální chlopeň: bpn, bez deg. změn, dnes insuficience do 2/3 LS, E/A 99/81 cm/s. Aortální chlopeň: bpn, 3 cípy dobrá hybnost, bez deg. změn, stopová insfuicience, max. gradient 8 mm Hg.Trikuspidální chlopeň: bez deg. změn, insuficience do 2/3 PS, nmax, gradient PK/PS 36 mm Hg, stopová Pulmonální chlopeň: stopová insuficience KOŘEN AORTY: - 27 (20-37mm) PRAVÁ KOMORA:27 LEVÁ SÍŇ: - 33 (19-40mm) M-mode - (9-26mm) PRAVÁ SÍŇ(dlouhá osa) - (<50mm) 4 dutin.apik. - 29 (<31mm) LEVÁ KOMORA: end diastol.: 46 (35-57mm) systol.: (23-36mm) zadní stěna: 10 (6-11mm) septum: 9 (6-11mm) EJEKČNÍ FRAKCE LK:
- Odhad: 55-60 - % Poruchy kinetiky: bpn Perikard: bpn
- **CONCLUSION: GOOD SYSTOLIC FUNCTION OF LV, MODERATE DIASTOLIC DYSFUNCTION, NO DEFFECT OF LOCAL CONTRACTILITY, NORMAL DIMENSIONS OF CARDIAC COMPARTMENTS, NO SERIOUS DEGENERATIVE CHANGES OF THE VALVE APPARATUS, BORDERLINE MITRAL AND TRICUSPID INSUFFICIENCY, OTHERWISE WITHOUT SIGNS OF A HEMODYNAMICALLY SIGNIFICANT VALVE DEFECT, SIGNS OF BORDERLINE PULMONARY HYPERTENSION. POSSIBLE MODIFICATION BY HIGHER BLOOD PRESSURE.**
- **BP 180/100, 175/100 mm HG**

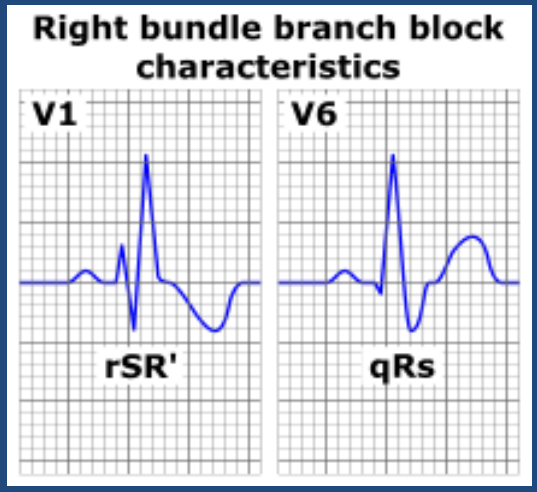
63 y. o. woman : BICYCLE ERGOMETRY

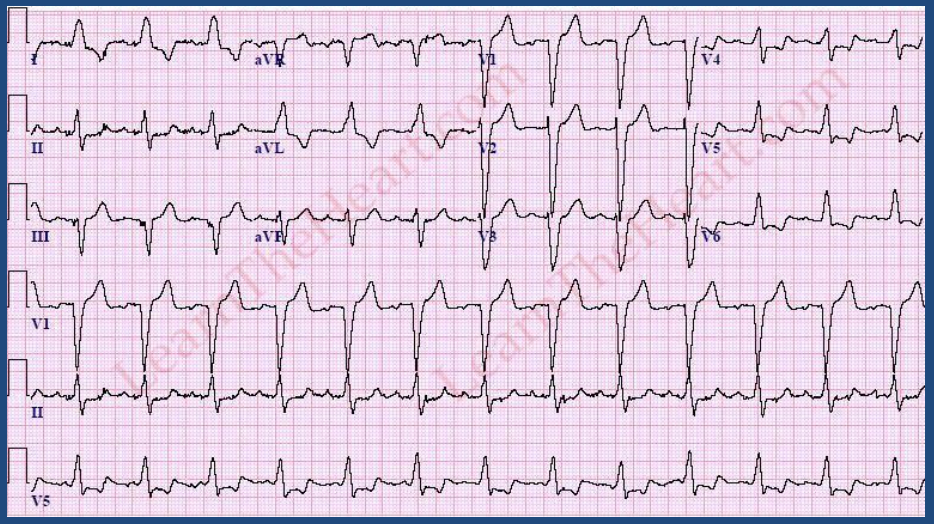
CONCLUSION:

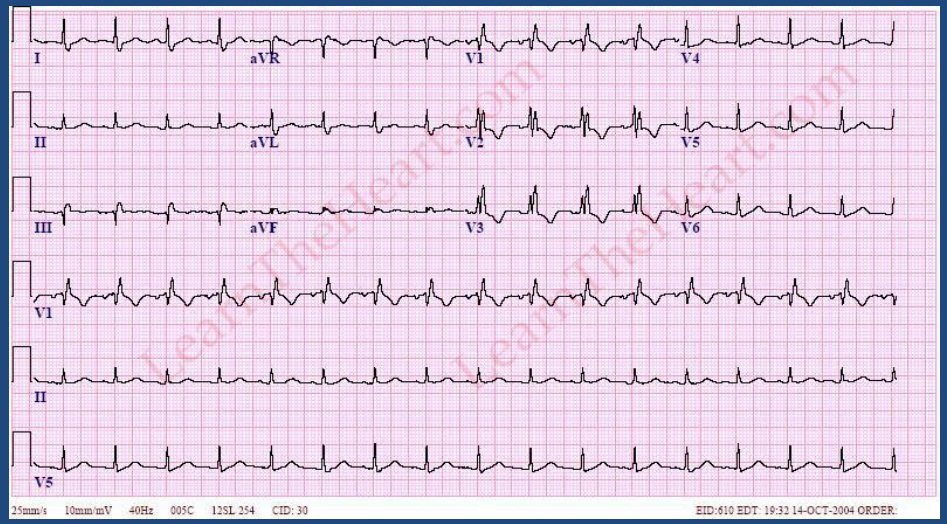
TEST TERMINATED FOR POSITIVE FINDINGS (ST DEPRESSION) AT 75 W FOR 1 MIN., MAXIMUM HEART RATE APPROX. 79 % MAC. DURING THE TEST DYSPNEA.


Left bundle branch block characteristics
V1
V6
rS
R

???


Right bundle branch block characteristics
V1
V6
rSR'
qRs


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


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 40Hz 005C 12SL 254 CID: 30
EID:610 EDT: 19:32 14-OCT-2004 ORDER:

63 y. o. woman : LAB. FINDINGS.

- **BIO 23.01.2018-06:35:** SPEC.HMOTN: 1,011 kg/l pH: 5,5 LEUKOCYTY: 1 NITRITY: - BILKOVINA: - GLUKOSA: Normal KETOLATKY: - UROBILINOG: Normal BILIRUBIN: - KYS.ASK.: - BARVA: světle žlutá ZÁKAL: průhledná KREV: - Erytrocyty: 4 částic/ul Leukocyty: 13 částic/ul Hyal.valce: 0 částic/ul Dlazdic.ep: 12 částic/ul
- **BIO 23.01.2018-06:35:** Na: 137 mmol/l K: 3,8 mmol/l OSMpoč: 285 mmol/kg P-GLUK: 5,6 mmol/l ALT: 0,42 ukat/l GGT: 0,41 ukat/l UREA: 5,2 mmol/l KREA: 68 umol/l eGFR(Schw): zrušeno ml/s/1,73 m2 do 1 roku orientační výsledek eGFR(CKD): 1,37 ml/s/1,73 m2 **TRIGL: 1,20 mmol/l CHOL: 4,1 mmol/l HDL-CHOL: 0,86 mmol/l non-HDL CH: 3,24 mmol/l LDL-CHOL: 2,61 mmol/l**
- **BIO 23.01.2018-06:35:** GHBC A1c: 34,00 mmol/mol eAG: 5,8 mmol/l
- **KOAG 23.01.2018-06:35:** APTT: 31.50 s APTTN: 28.00 s APTT-R: 1.13 -- PT: 11.20 s PTN: 11.40 s PT-INR: 0.98 -- PT-R: 0.98 **KO 23.01.2018-06:35:** WBC: 4.7 x10^9/l RBC: 4.22 x10^12/l HGB: 127 g/l HCT: 0.362 l/l MCV: 85.8 fl MCH: 30.1 pg MCHC: 350.8 g/l RDW: 13.3 % PLT: 230 x10^9/l MPV: 10.4 fl PCT: 0.240 % PDW-SD: 11.5 fl NRBC-A: 0.0 % NRBC#-A: 0.000 x10^9/l P-LCR: 27.4 %

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63 y. o. woman : SUMMARY

-
- **CONCLUSION:**

- **RECOMMENDATION**:

SUMMARY – STABLE IHD

DIAGNOSIS:

- HISTORY – IMPORTANT
- NON-INVASIVE TESTS (TREADMILL ECG)

MANAGEMENT:

- IMPROVE QUALITY OF LIFE: revaskularization, nitrates, beta blockers, ca antagonists
- IMPROVE SURVIVAL: statins/RF management