PATOFYZIOLOGIE SRDEČNÍHO SELHÁNÍ

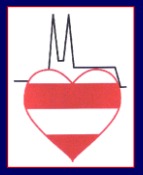
Špinar J, Špinarová L

Internní kardiologická klinika

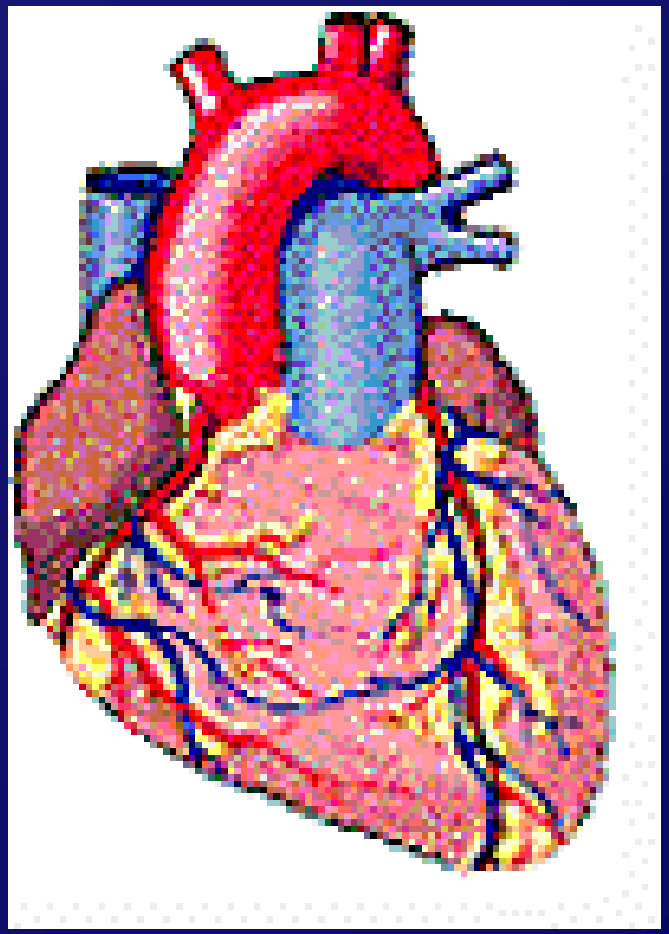
FN Brno a FN u sv. Anny v Brně

Masarykova univerzita

BRNO

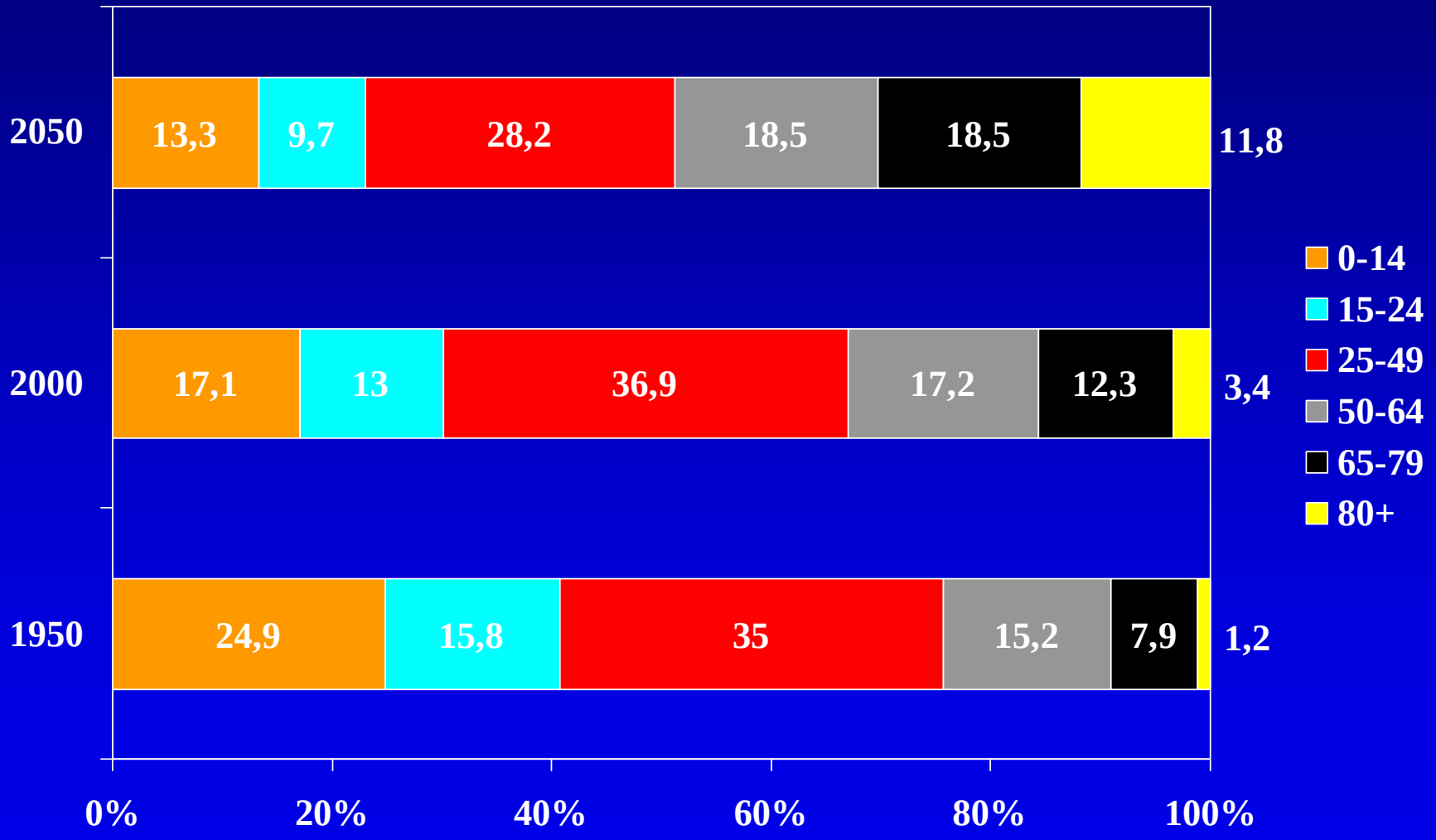


Češi se dožívají stále vyššího věku, aby žili déle v nemocnici



8.4.2016
Onkologie
Zámek LOKET

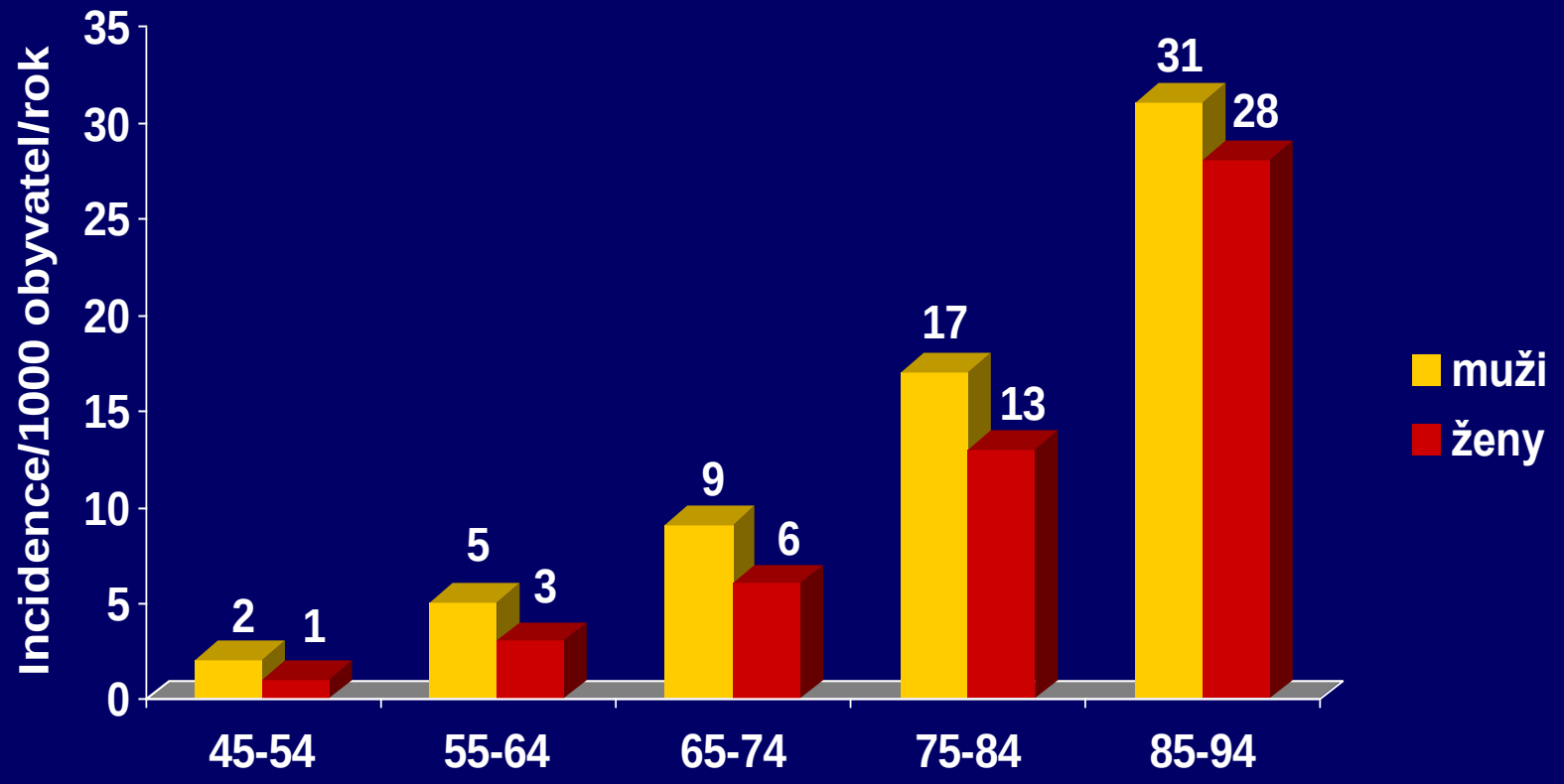
Stárnutí evropské populace (EU 25): distribuce podle věkových skupin



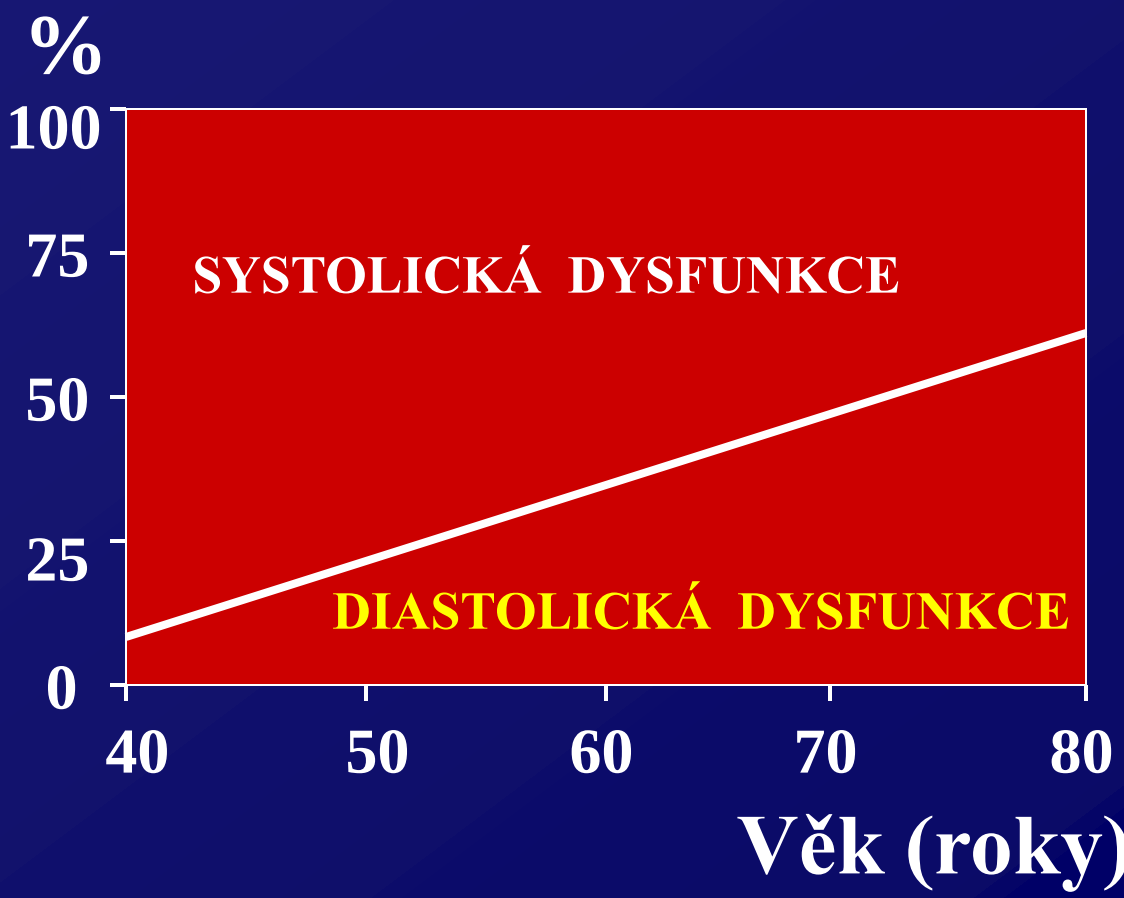
UN World Population Prospects (2002 Revision) nd Eurostat 2004. Demographic projection (baseline scenario)

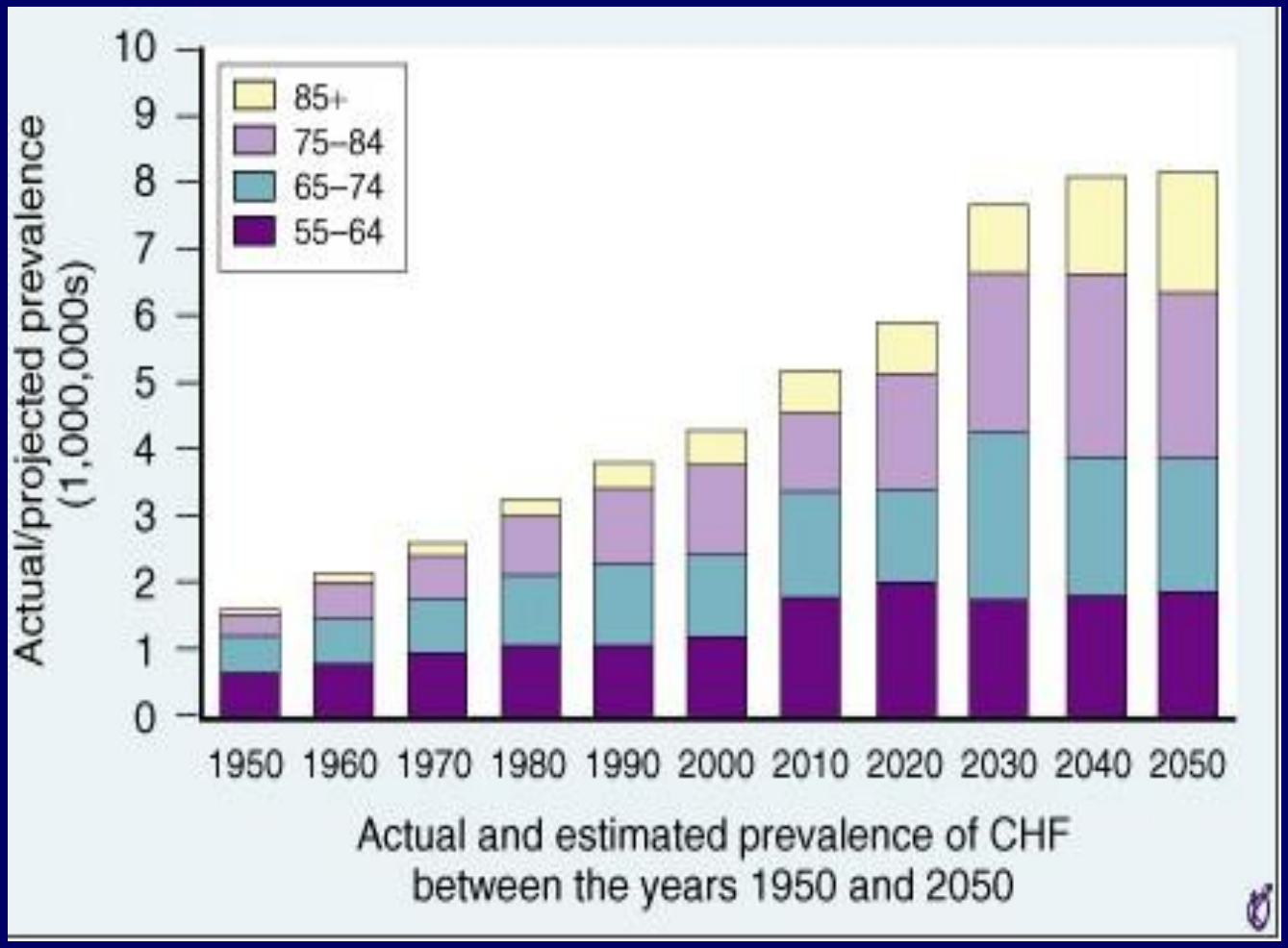
Epidemiologie

Incidence srdečního selhání ve věkových kategoriích - údaje z Framinghamské studie



VÝSKYT SYSTOLICKÉ A DIASTOLICKÉ DYSFUNKCE LK JAKO PŘÍČINY SRDEČNÍHO SELHÁNÍ





Motto: Kvalita života není o nic méně významný cíl léčby než statisticky vyčíslitelná mortalita!

Surveys of the Study Group on Diagnosis of the WG on Heart Failure

IMPROVEMENT of HF

IMprovement PROgram in eValuation and managEMENT

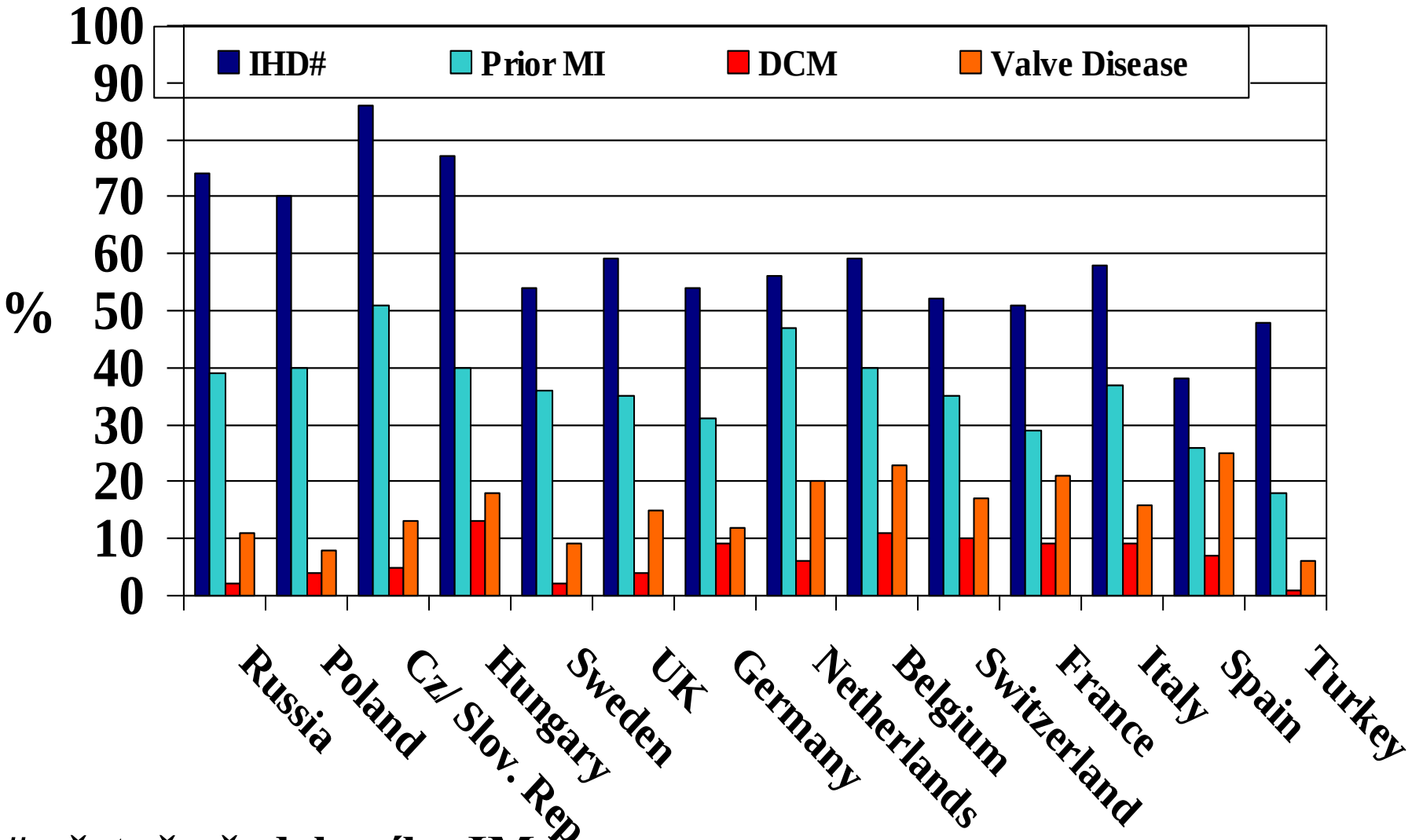
Heart Failure

Primary Care (1999-2000)

- 14 ESC Countries
- 1,363 PC Physicians

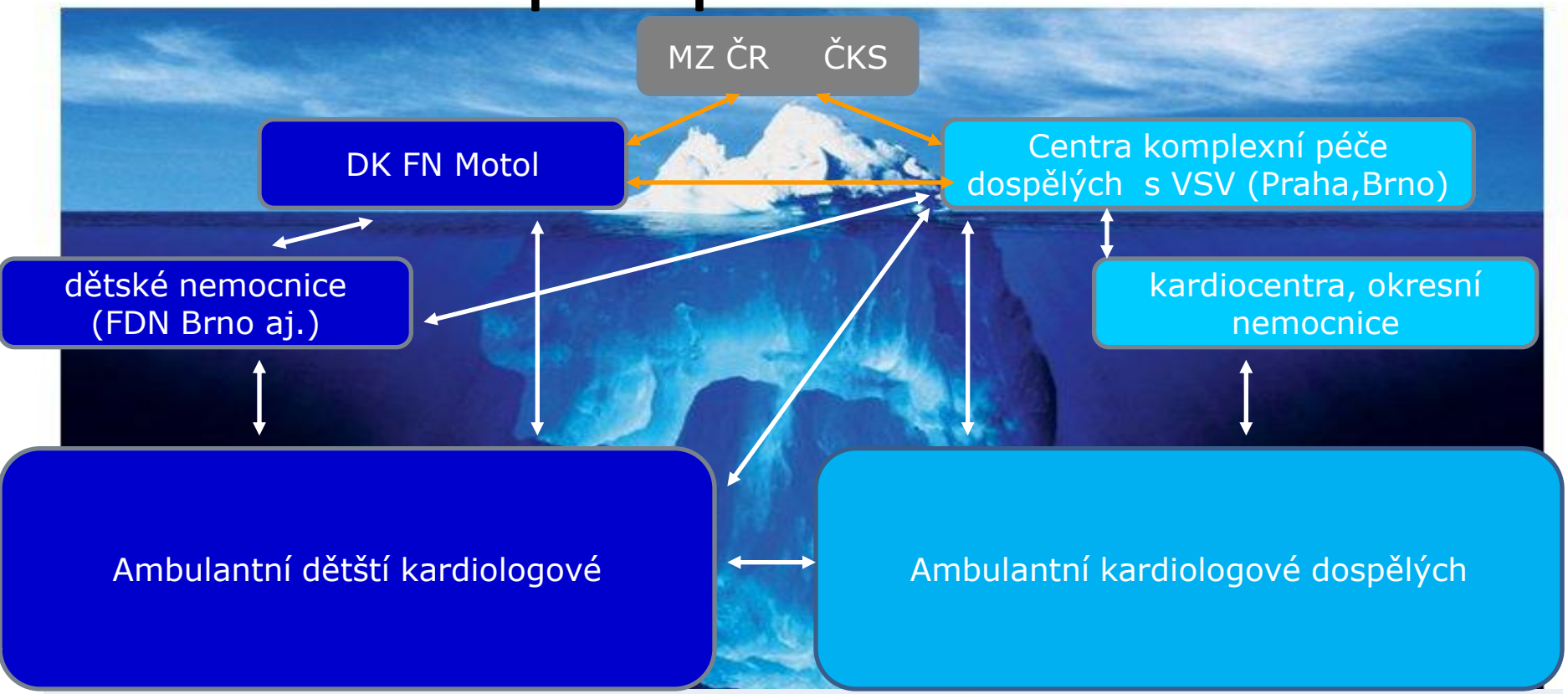
- 11,062 patients
- Mean age 70 years
- 45% Women
- In Hospital in last yr 41%

Etiologie srdečního selhání



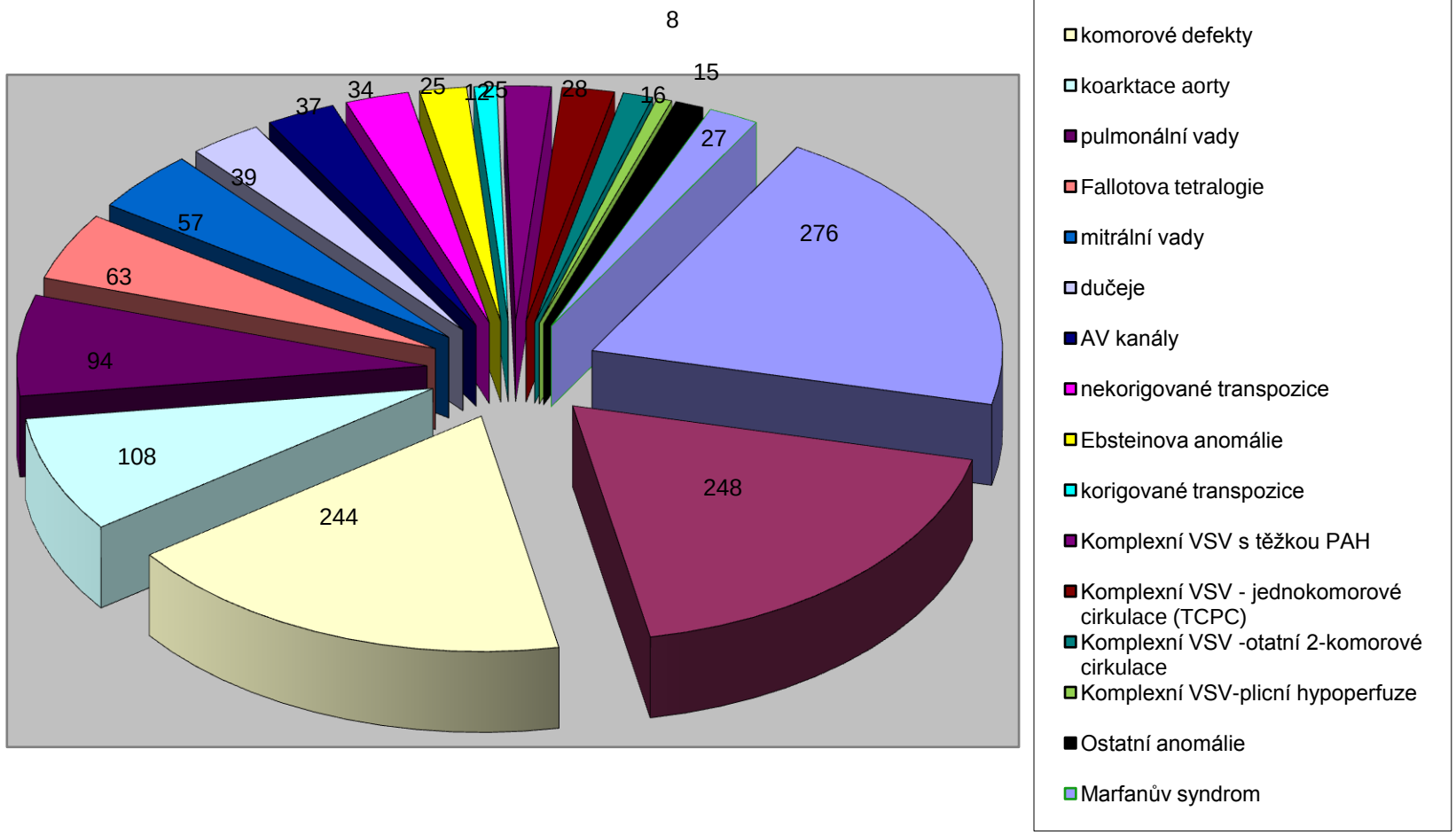
včetně předchozího IM

Koncepce péče o VSV v ČR



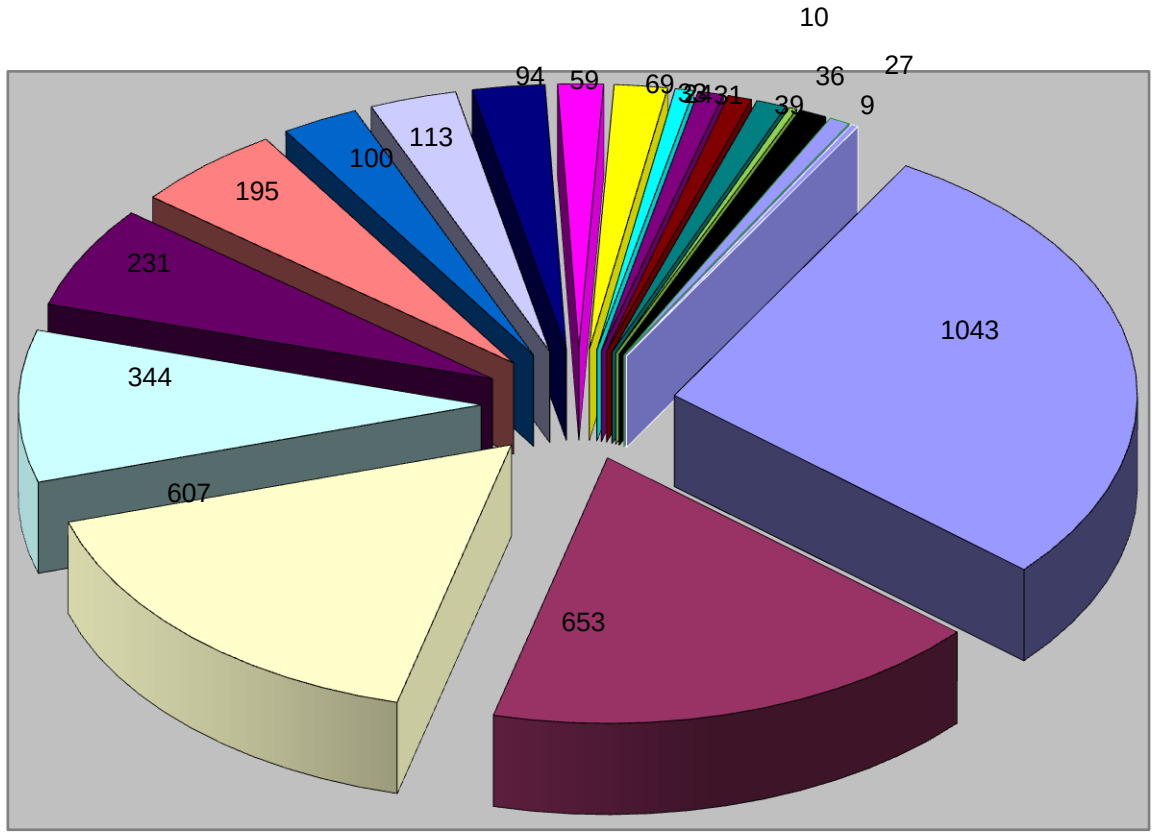
Ambulance VSV v dospělosti FN Brno Bohunice dle zákl. dg. n=1356

(k 21.9.2015)



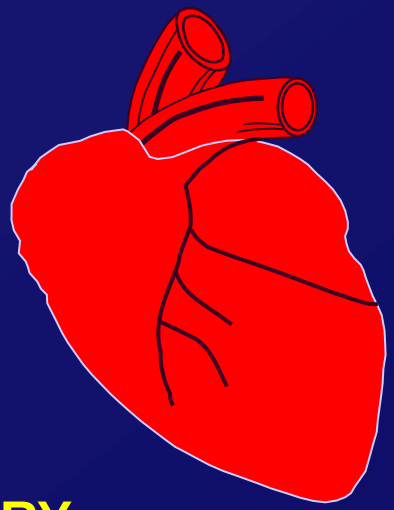
Centrum komplexní péče o VSV v dospělosti Brno dle zákl. dg. n=3717

(k 21.9.2015)



- síňové defekty
- aortální vady
- komorové defekty
- koarktace aorty
- pulmonální vady
- Fallotova tetralogie
- mitrální vady
- dučeje
- AV kanály
- nekorigované transpozice
- Ebsteinova anomálie
- korigované transpozice
- Komplexní VSV s těžkou PAH
- Komplexní VSV - jednokomorové cirkulace (TCPC)
- Komplexní VSV -otatní 2-komorové cirkulace
- Komplexní VSV-plicní hypoperfuze
- Ostatní anomálie
- Marfanův syndrom
- OTS pro VSV

KV kontinuum



Thrombóza

Náhlá smrt

Infarkt myokardu

Re-infarkt

Ztráta srd. svalu

Remodelace

Srdeční selhání

OTS

Ischemie

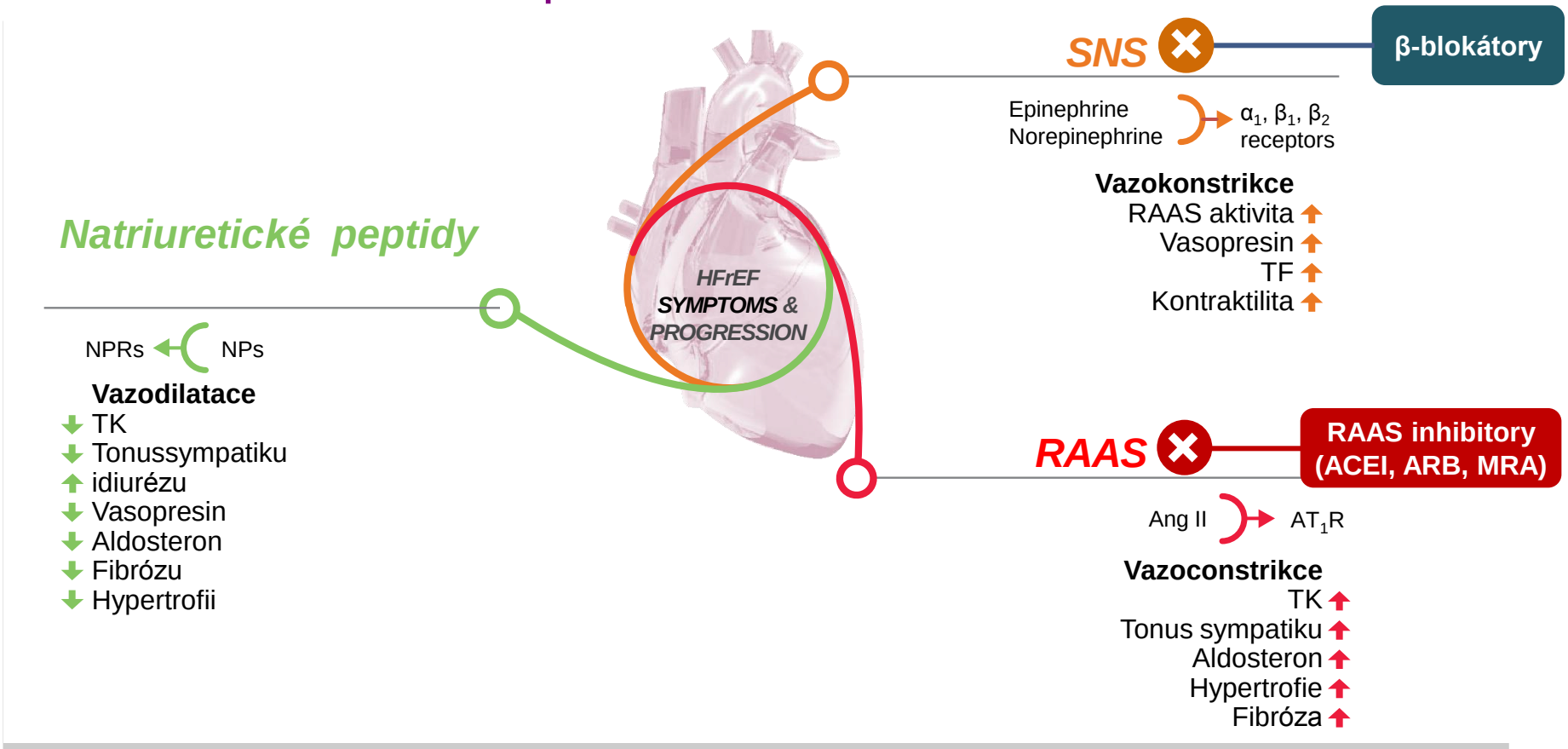
Atherom

RIZIKOVÉ FAKTORY

Hypertenze
Hypertrofie LK
Diabetes mellitus

Dzau and Braunwald, 1991

Nadměrná RAAS a SNS je škodlivá u CHSS, její ovlivnění je základem farmakoterapie



- Základním význam RAAS je podpořen příznivým účinkem ACEIs, ARBs a MRA[1]
- Prospěch β-blokátorů ukazuje, že SNS hraje také klíčovou roli

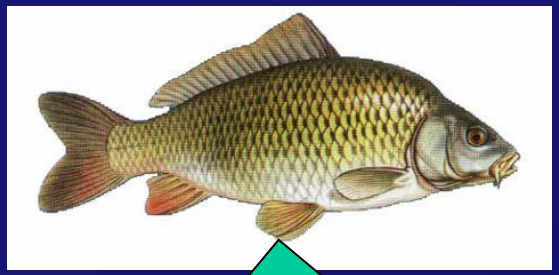
1. McMurray et al. Eur Heart J 2012;33:1787–847
Figure references: Levin et al. N Engl J Med 1998;339:321–8; Nathisuwan & Talbert. Pharmacotherapy 2002;22:27–42; Kemp & Conte. Cardiovascular Pathology 2012;365–371; Schrier & Abraham. N Engl J Med 2009;341:577–85;

Renin angiotenzin aldosteronový systém

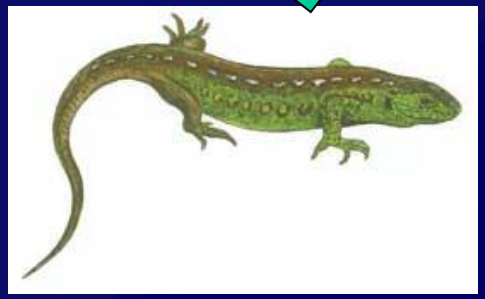
Co o něm víme ?

Proč inhibujeme systém RAA?

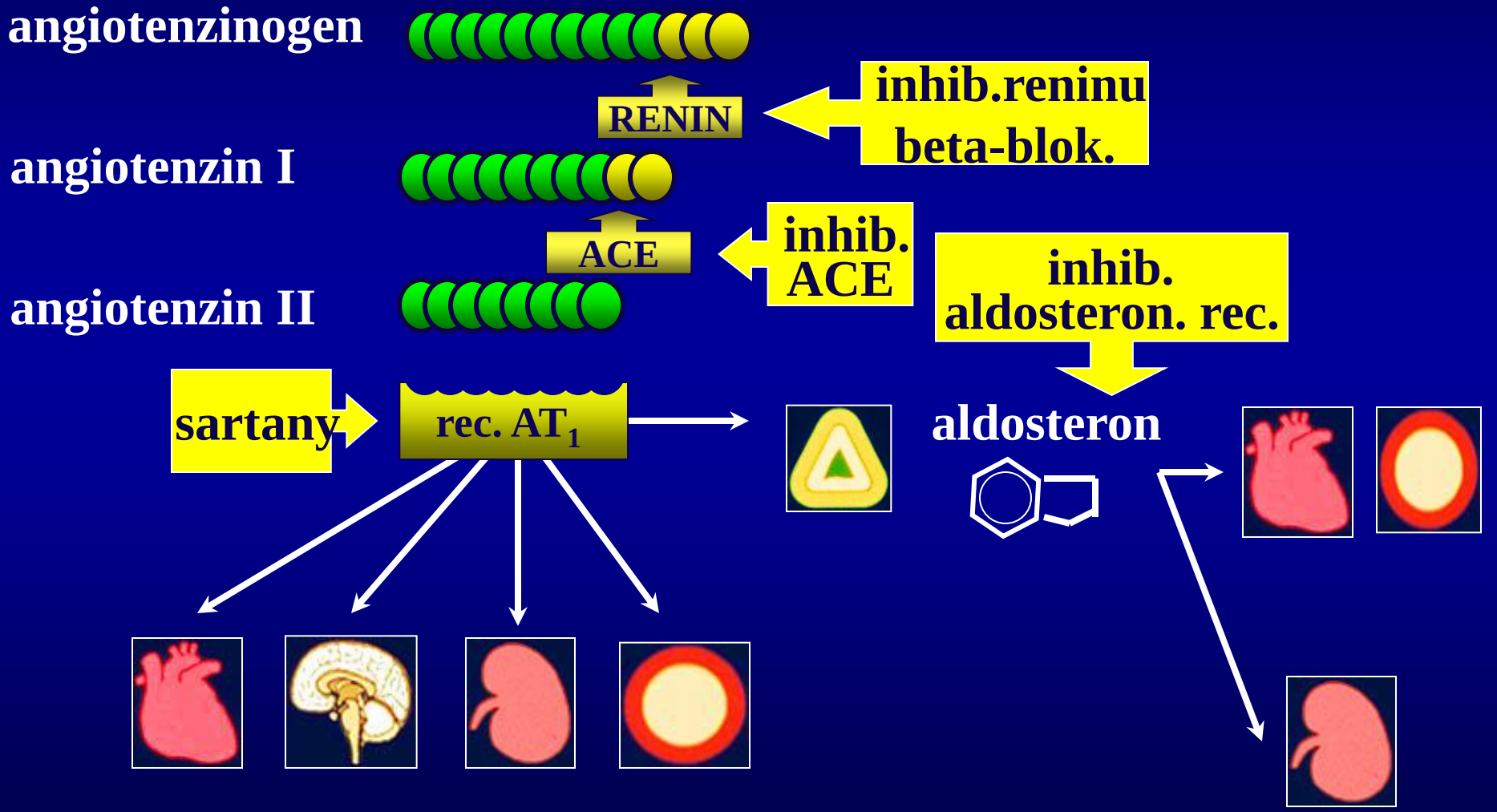
Jsou rozdíly v klinickém účinku blokády?



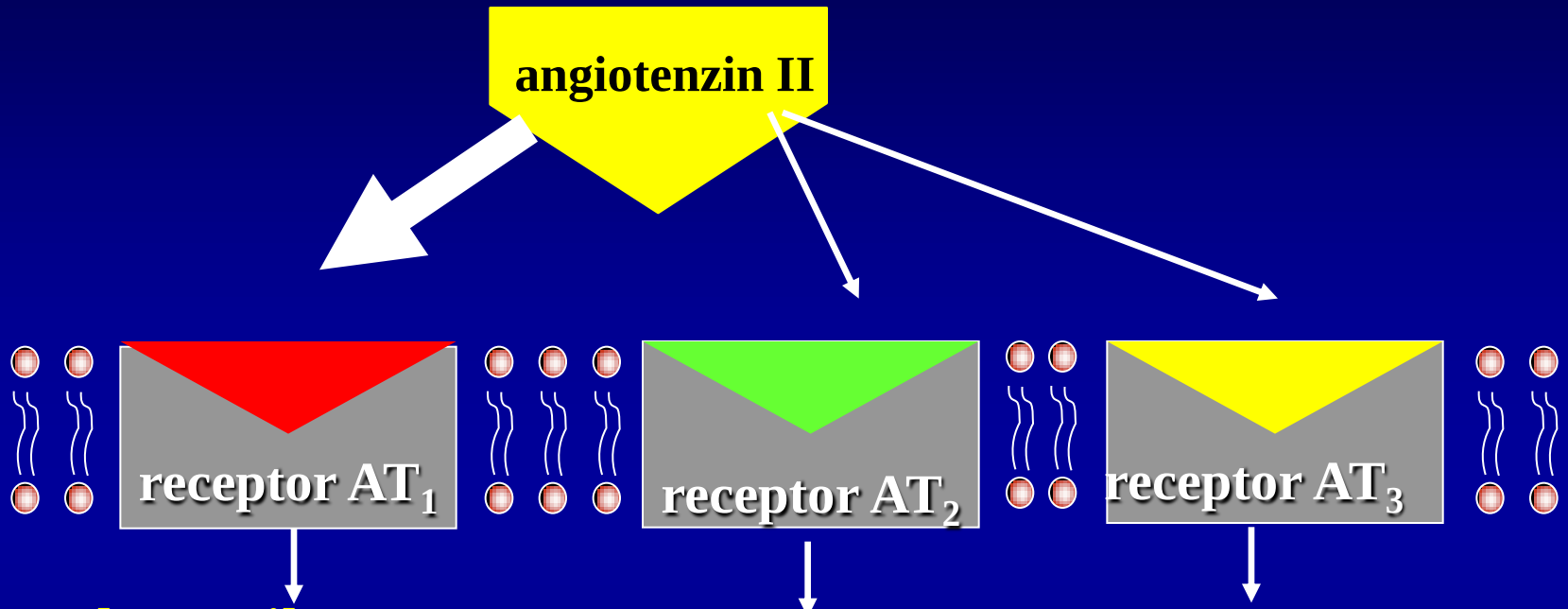

Gina Mikel
www.scientificillustrator.com



Osa renin-angiotenzin-aldosteron



RECEPTORY AT1, AT2 a AT3 pro AII

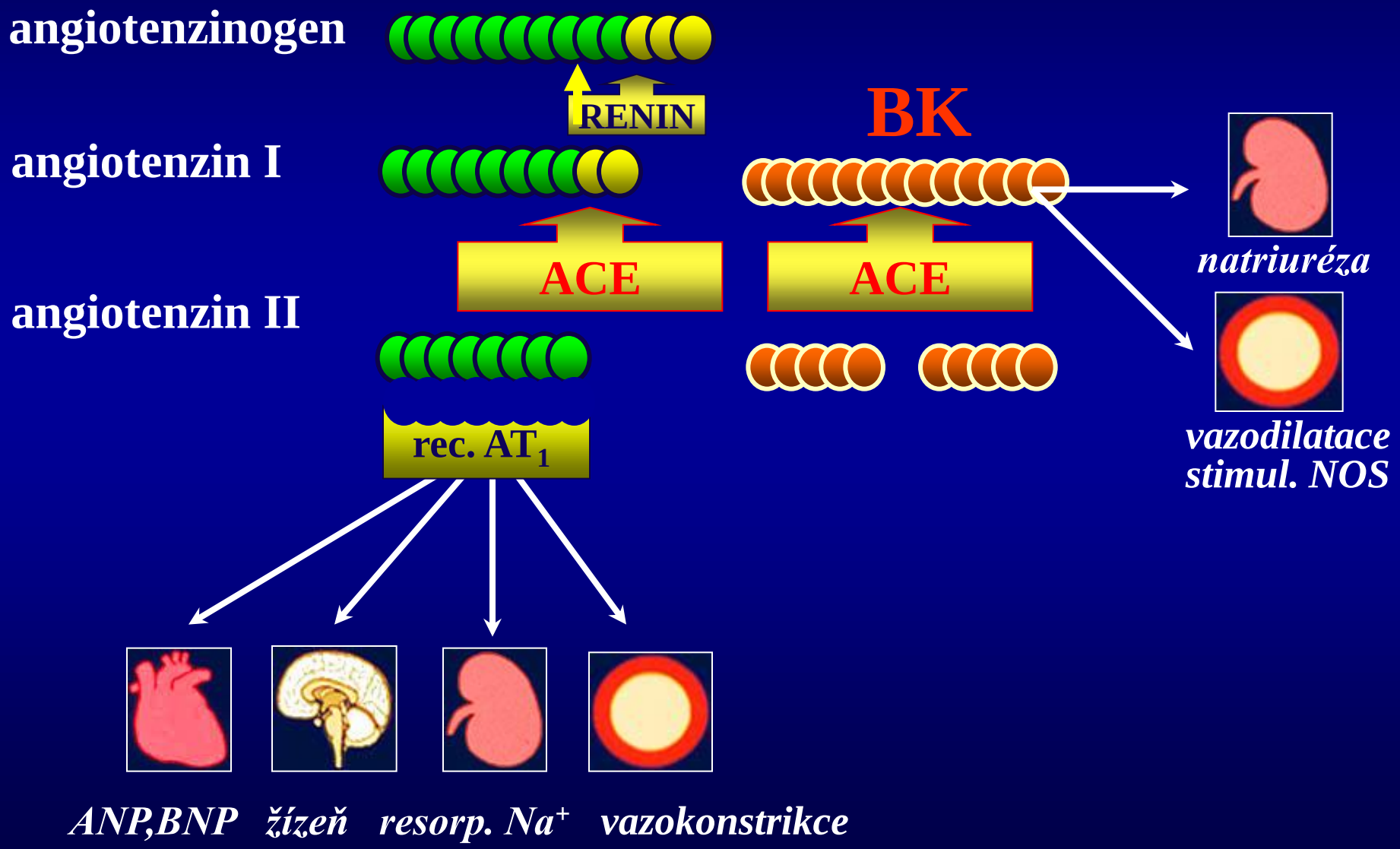


vazokonstrikce
aktivace sympatiku
retence sodíku a vody
uvolnění vazopresinu
hypertrofie a proliferace myocytů
stimulace fibrinolýzy

mitogenní efekt
vazodilatace
antiproliferace

aktivace trombocytů

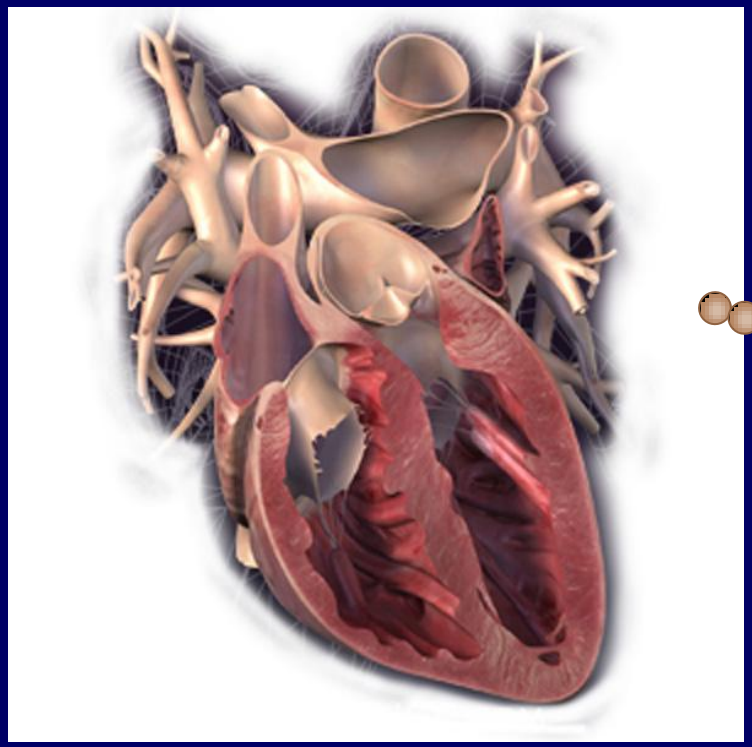
Osa RAA a kininy

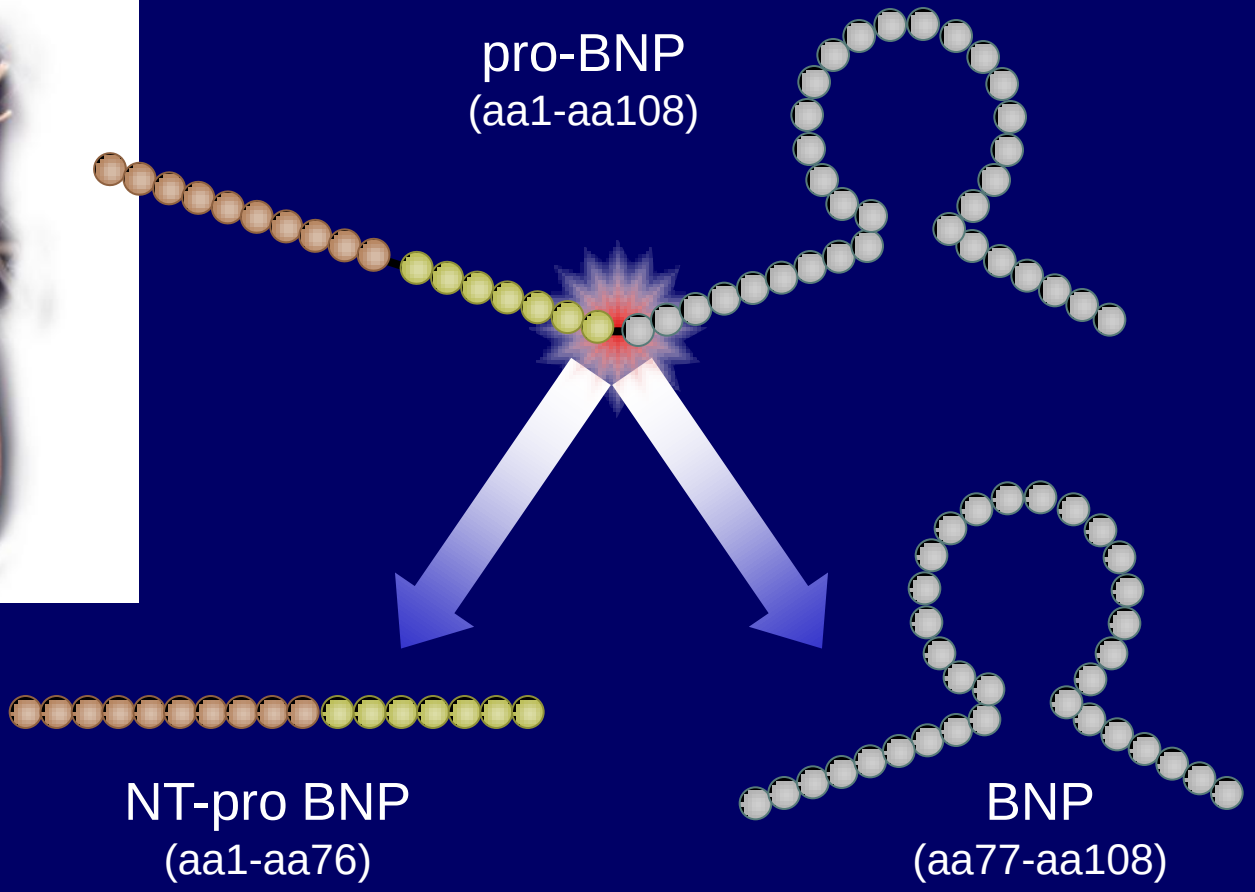


Proč inhibujeme hyperaktivovaný syst. RAA?

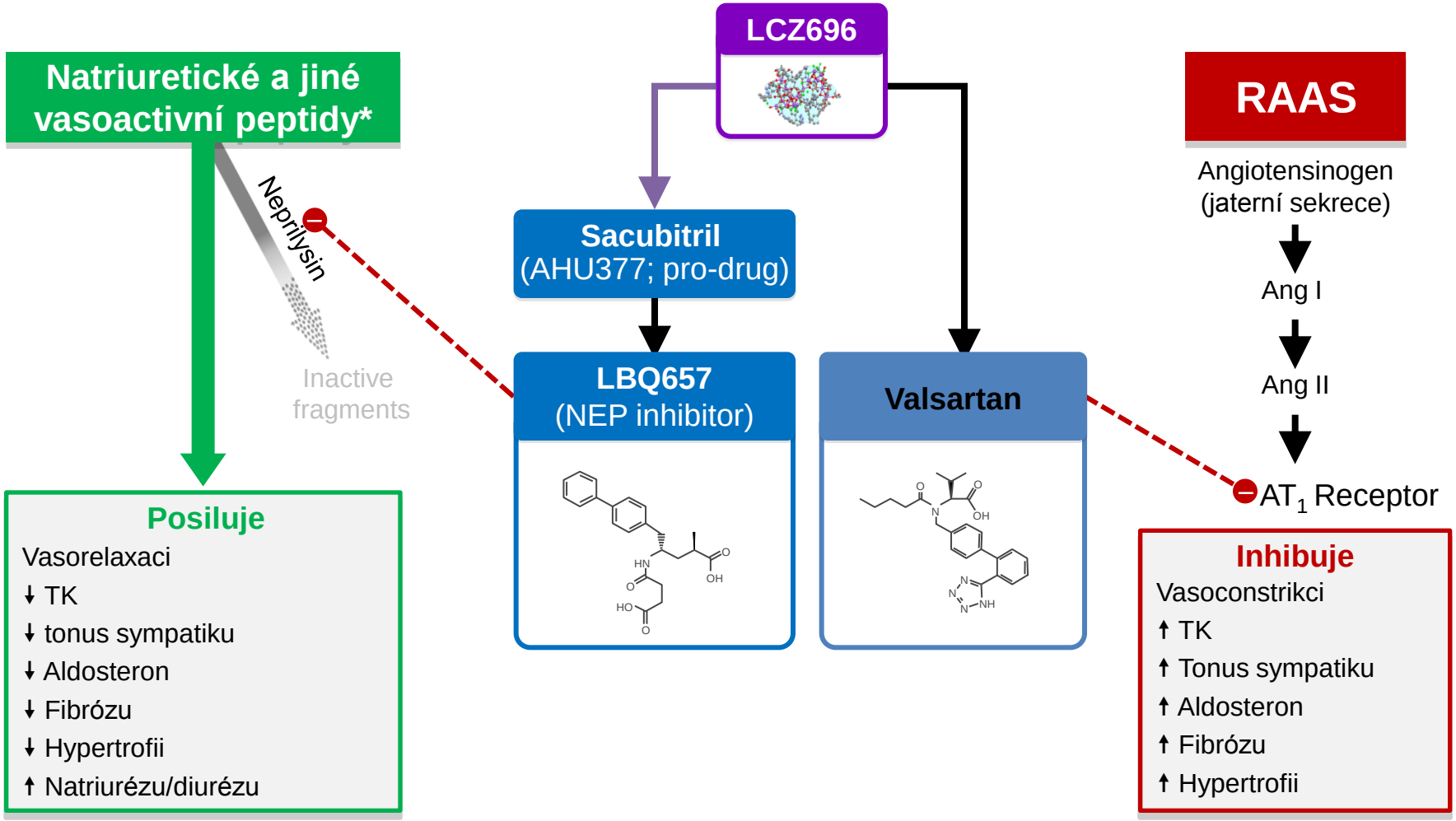
- pokles krevního tlaku
- zábrana remodelace srdce
- zábrana hypertrofie srdce i cév
- zlepšení inzulinové senzitivity (DM)
- snížení proteinurie
- antiarytmický efekt (FiSi)
- snížení morbidity i mortality

B-type Natriuretic Peptide (BNP)





LCZ696 sooučasně inhibuje NEP (via LBQ657) a blokuje AT_1 receptor (via valsartan)

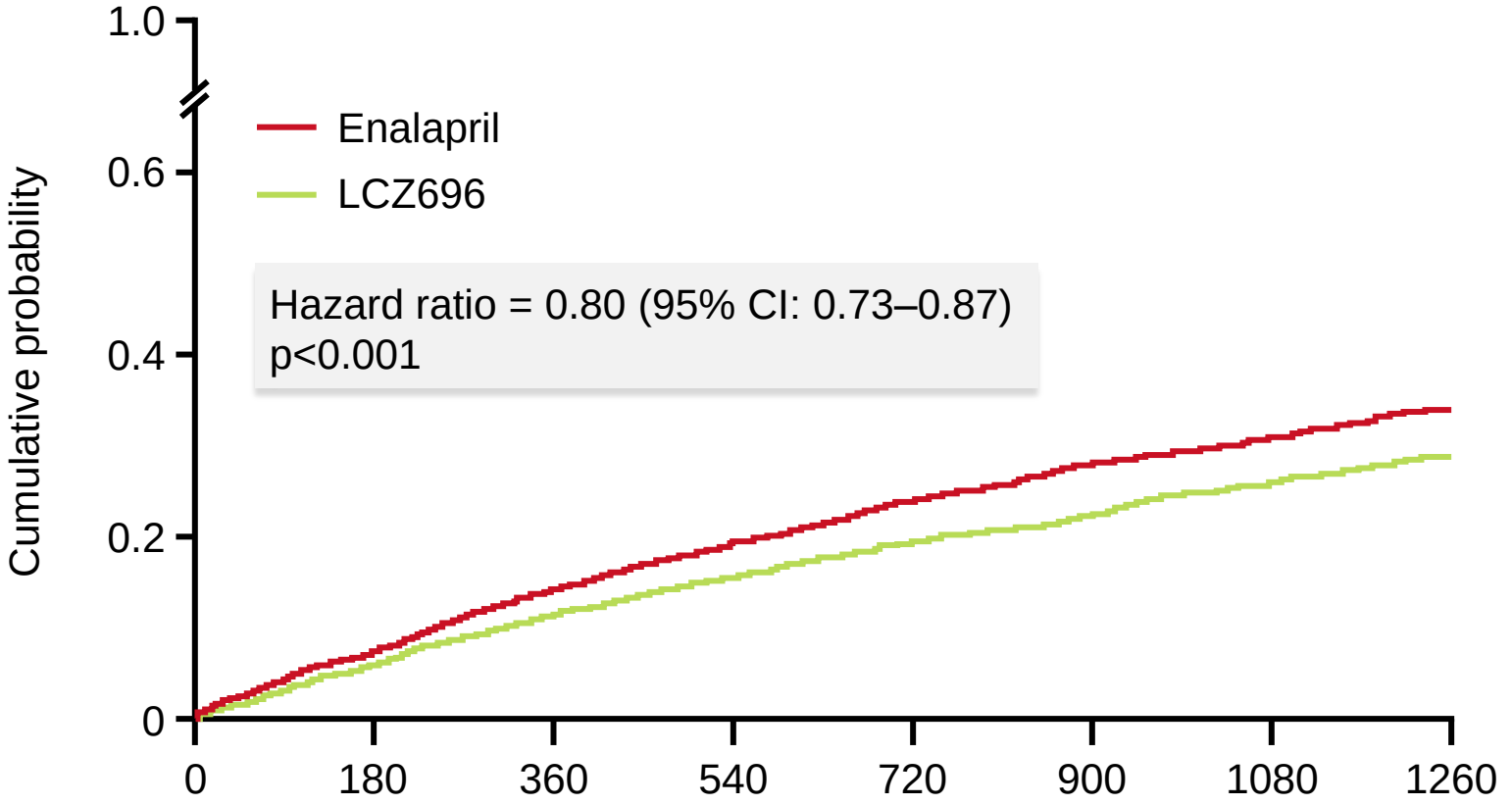


*Neprilysin substrates listed in order of relative affinity for NEP: ANP, CNP, Ang II, Ang I, adrenomedullin, substance P, bradykinin, endothelin-1, BNP

Levin et al. N Engl J Med 1998;339:321–8; Nathisuwan & Talbert. Pharmacotherapy 2002;22:27–42;
Schrier & Abraham N Engl J Med 2009;341:577–85; Langenickel & Dole. Drug Discov Today: Ther Strateg 2012;9:e131–9;
Feng et al. Tetrahedron Letters 2012;53:275–6

Primární endpoint:

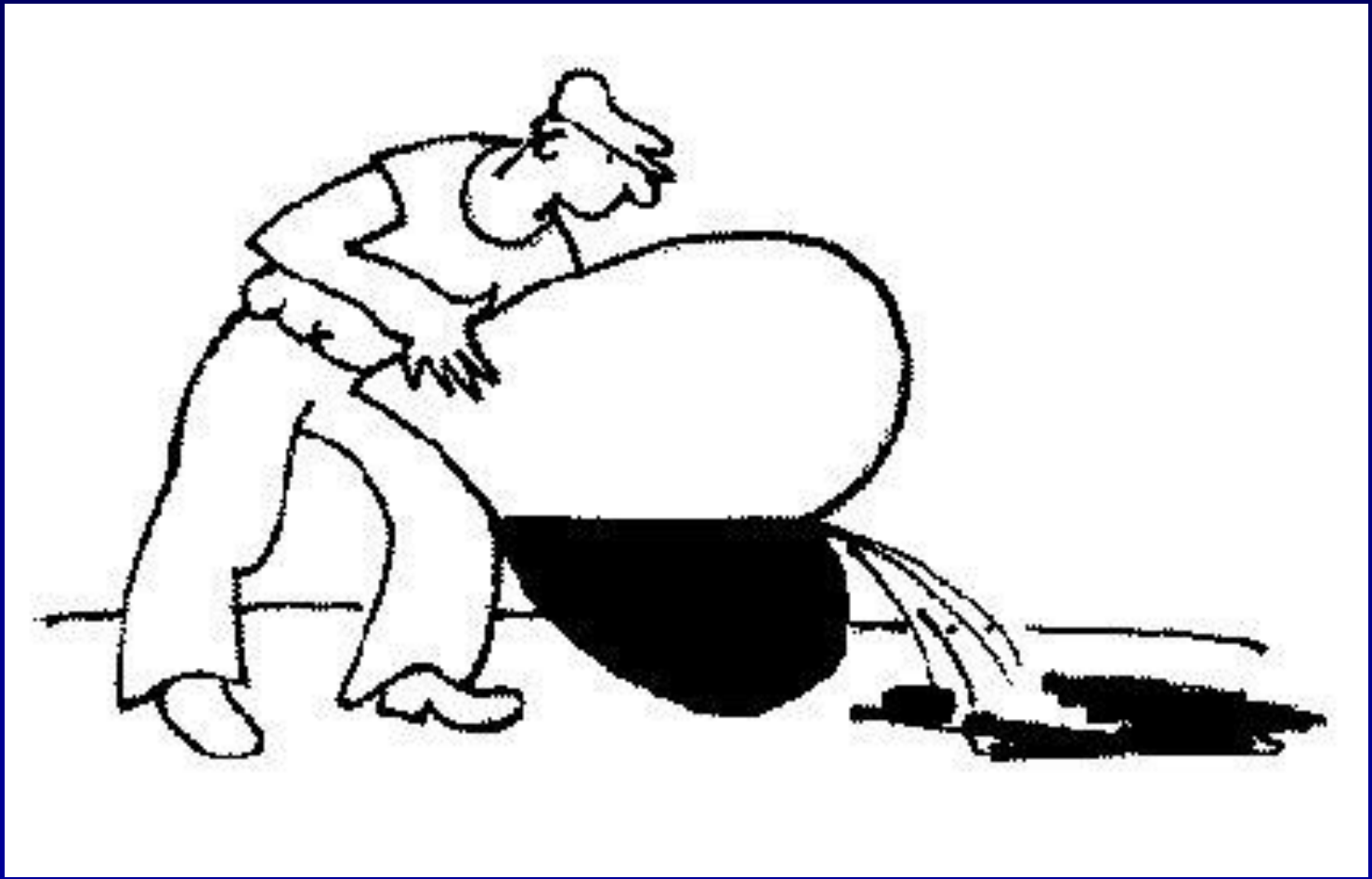
KV úmrtí nebo první hospitalizace pro SS



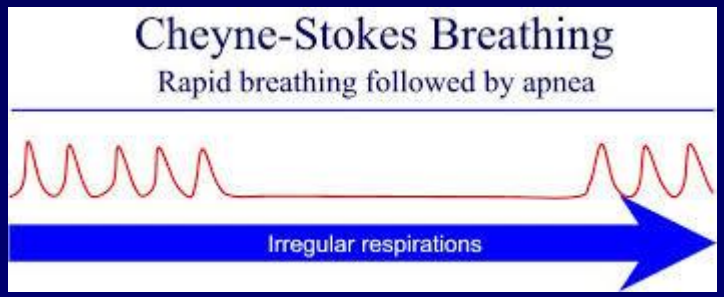
No at risk								
LCZ696	4187	3922	3663	3018	2257	1544	896	249
Enalapril	4212	3883	3579	2922	2123	1488	853	236

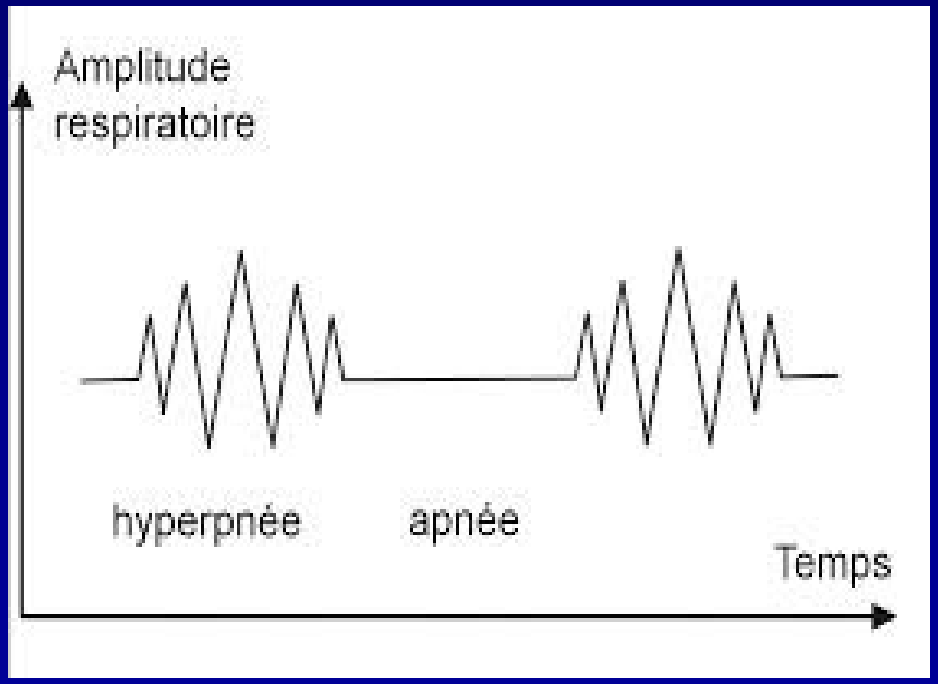
McMurray, et al. N Engl J Med 2014; ePub ahead of print: DOI: 10.1056/NEJMoa1409077.

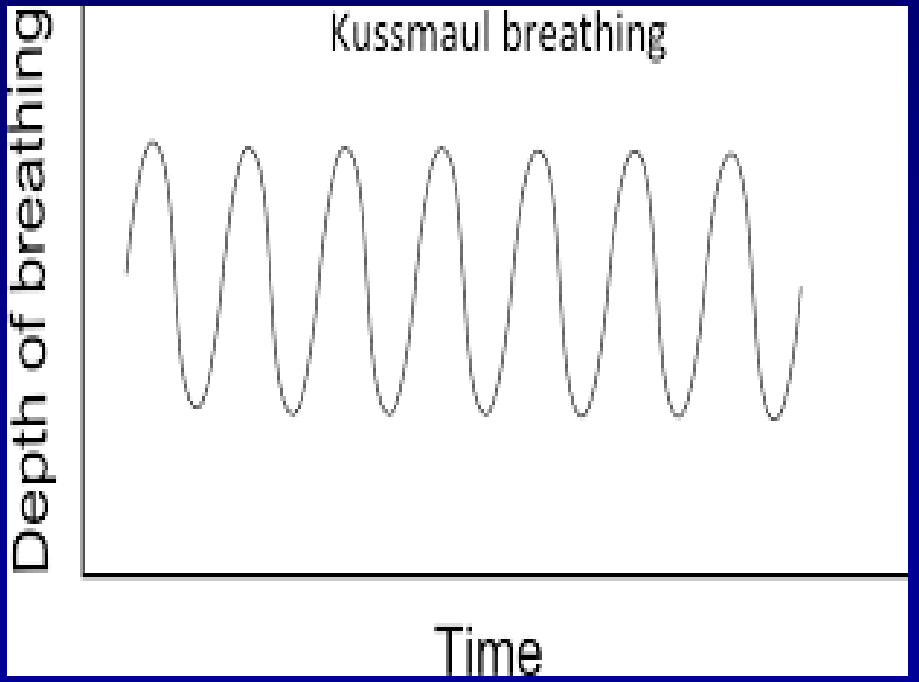
Terminální stadium



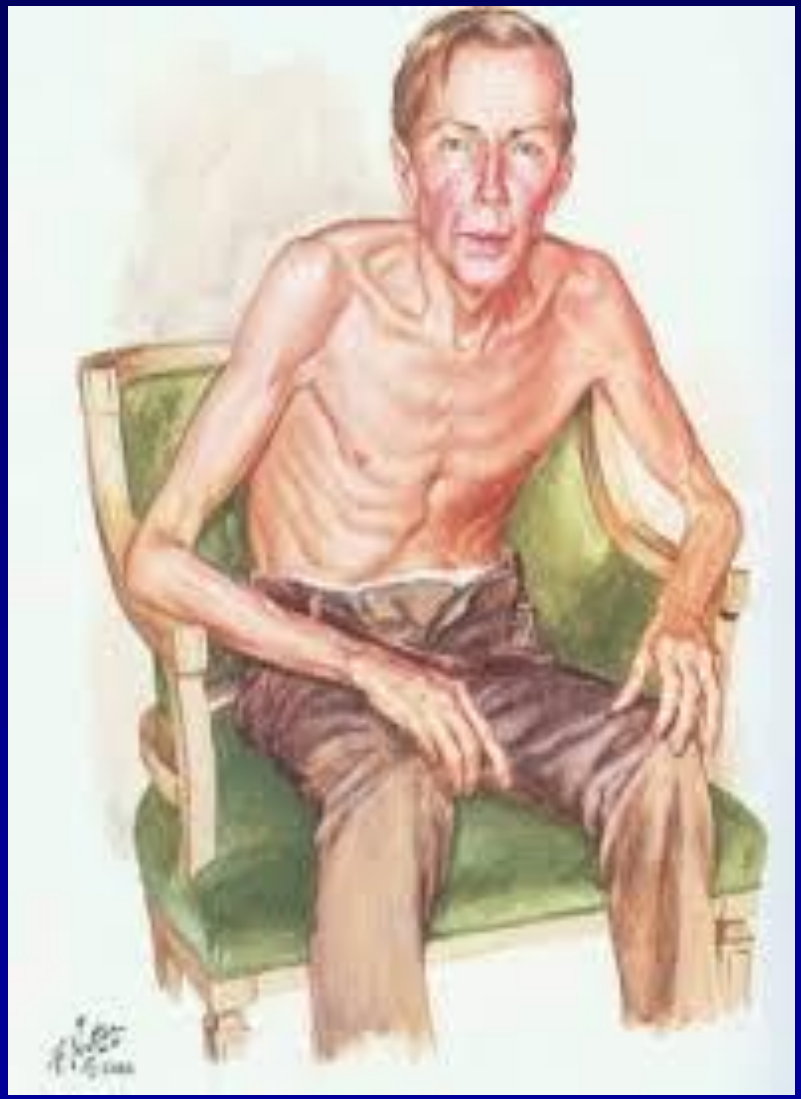
Dýchání


Cheyne-Stokes Breathing
Rapid breathing followed by apnea
Irregular respirations

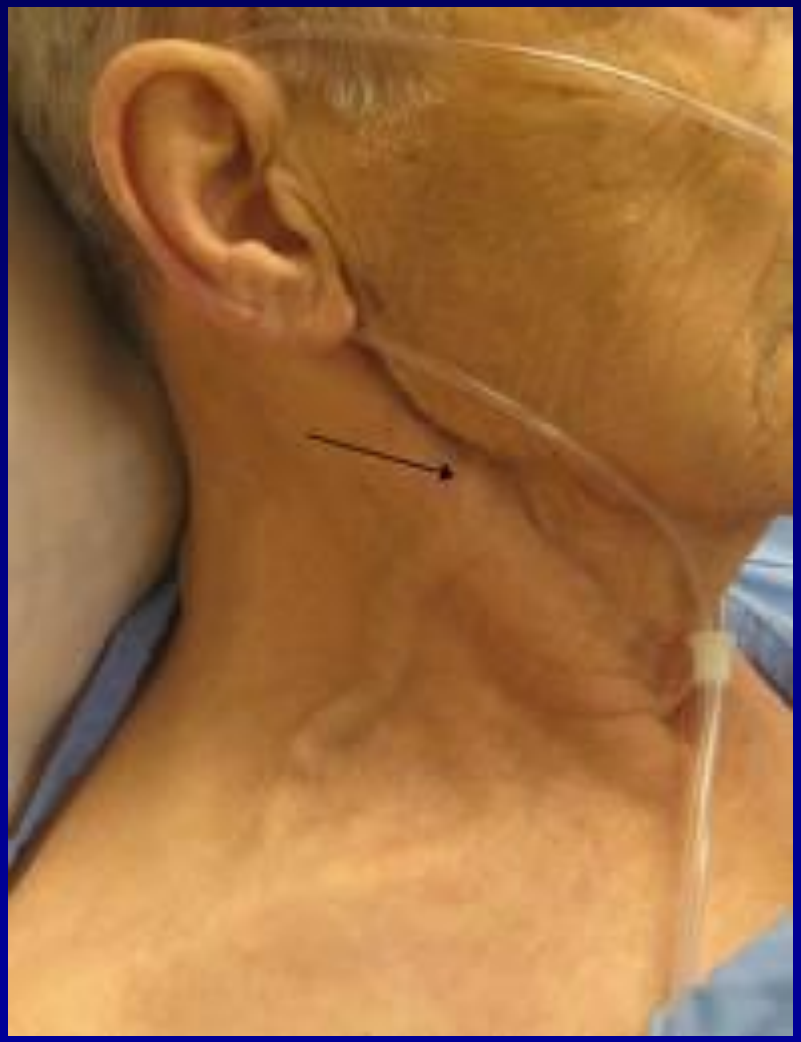

Amplitude respiratoire
hyperpnée
apnée
Temps

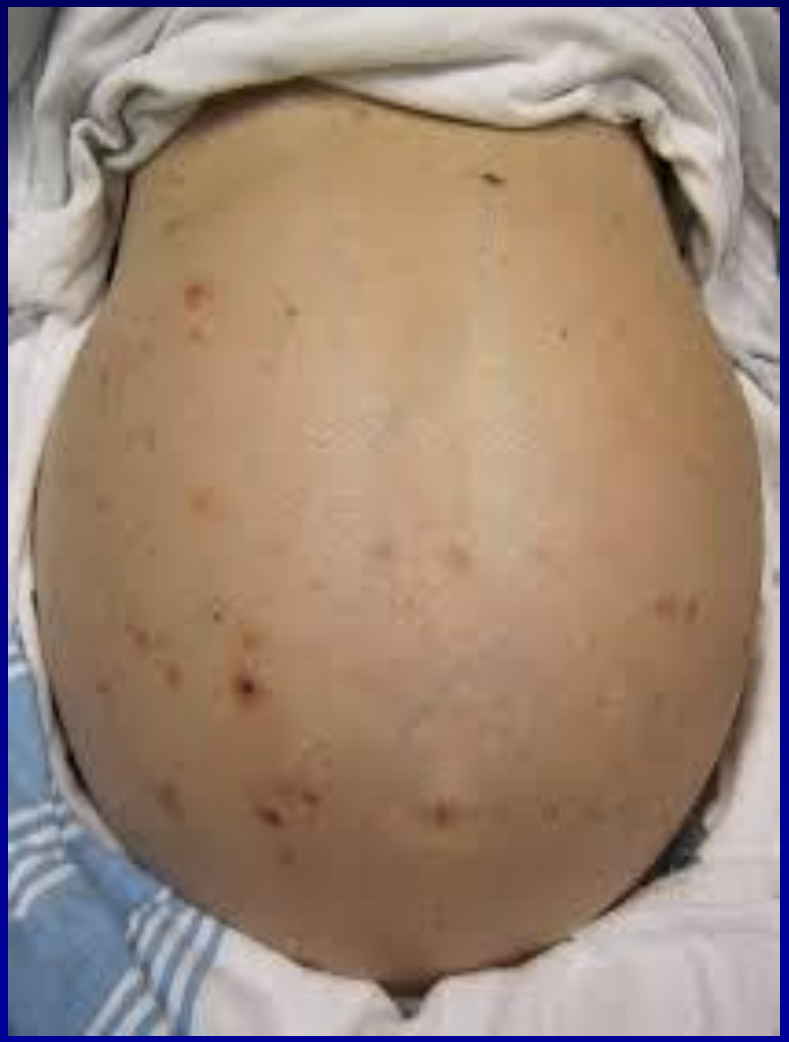

Kussmaul breathing
Depth of breathing
Time

Kachexie a dušnost

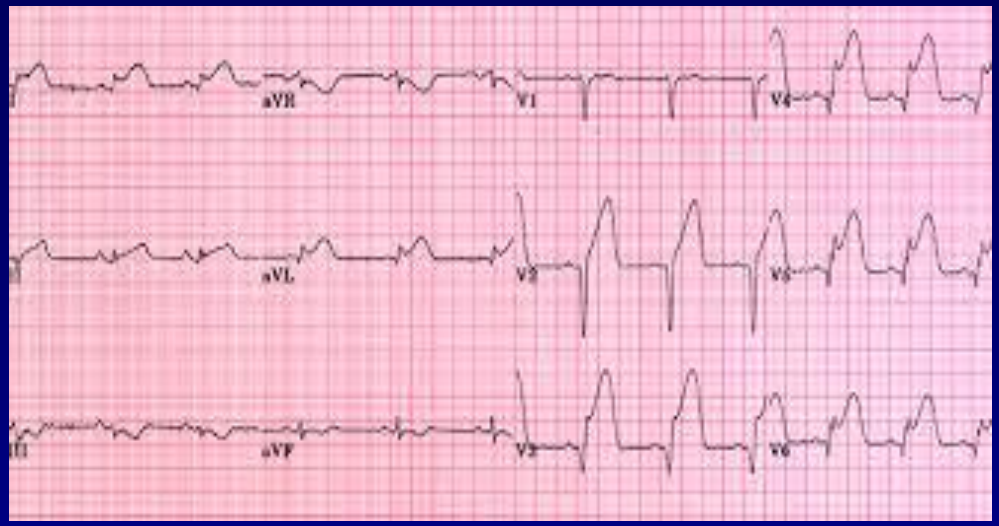


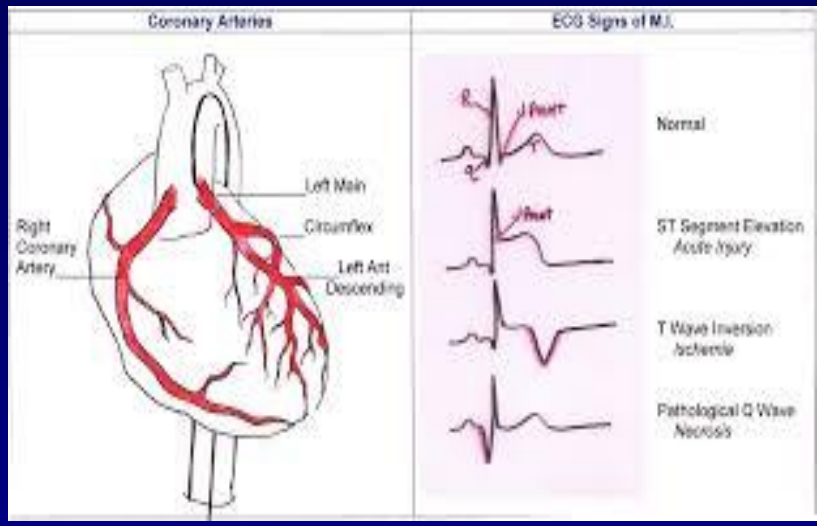
Náplň žil, ascites

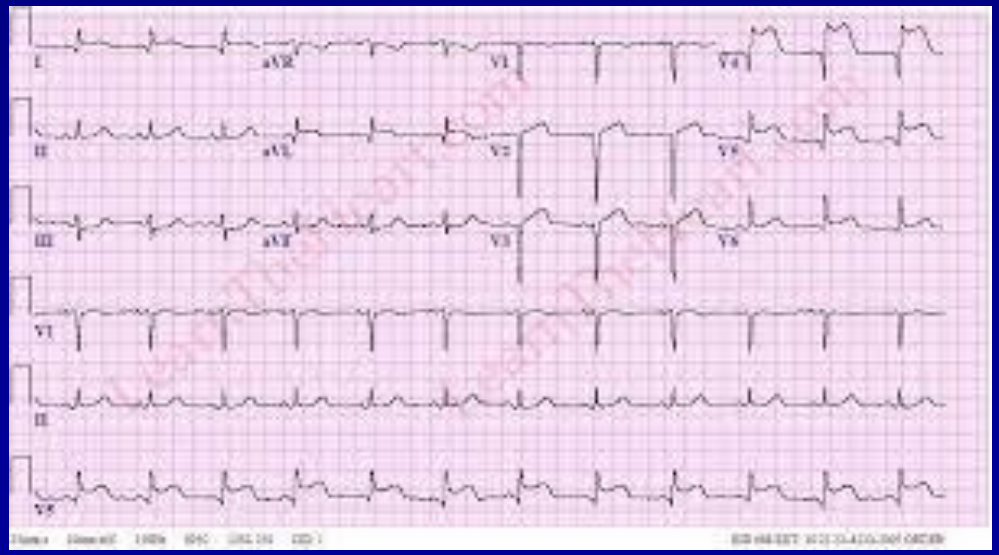


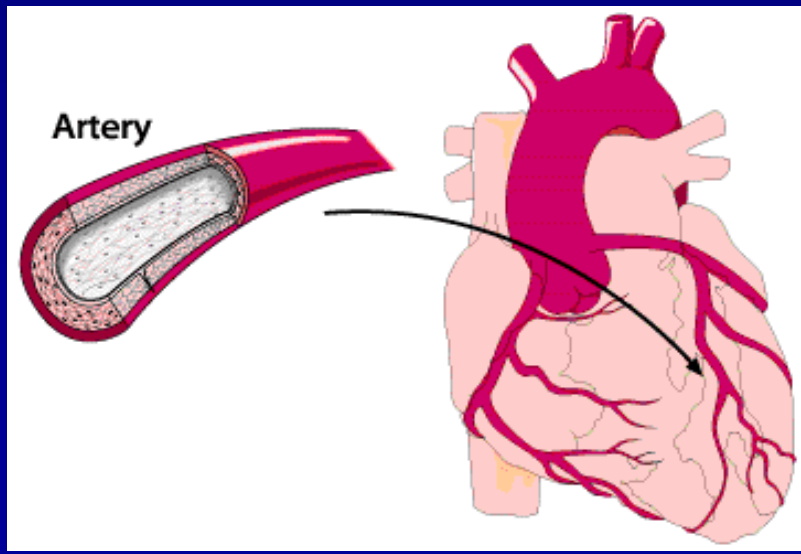


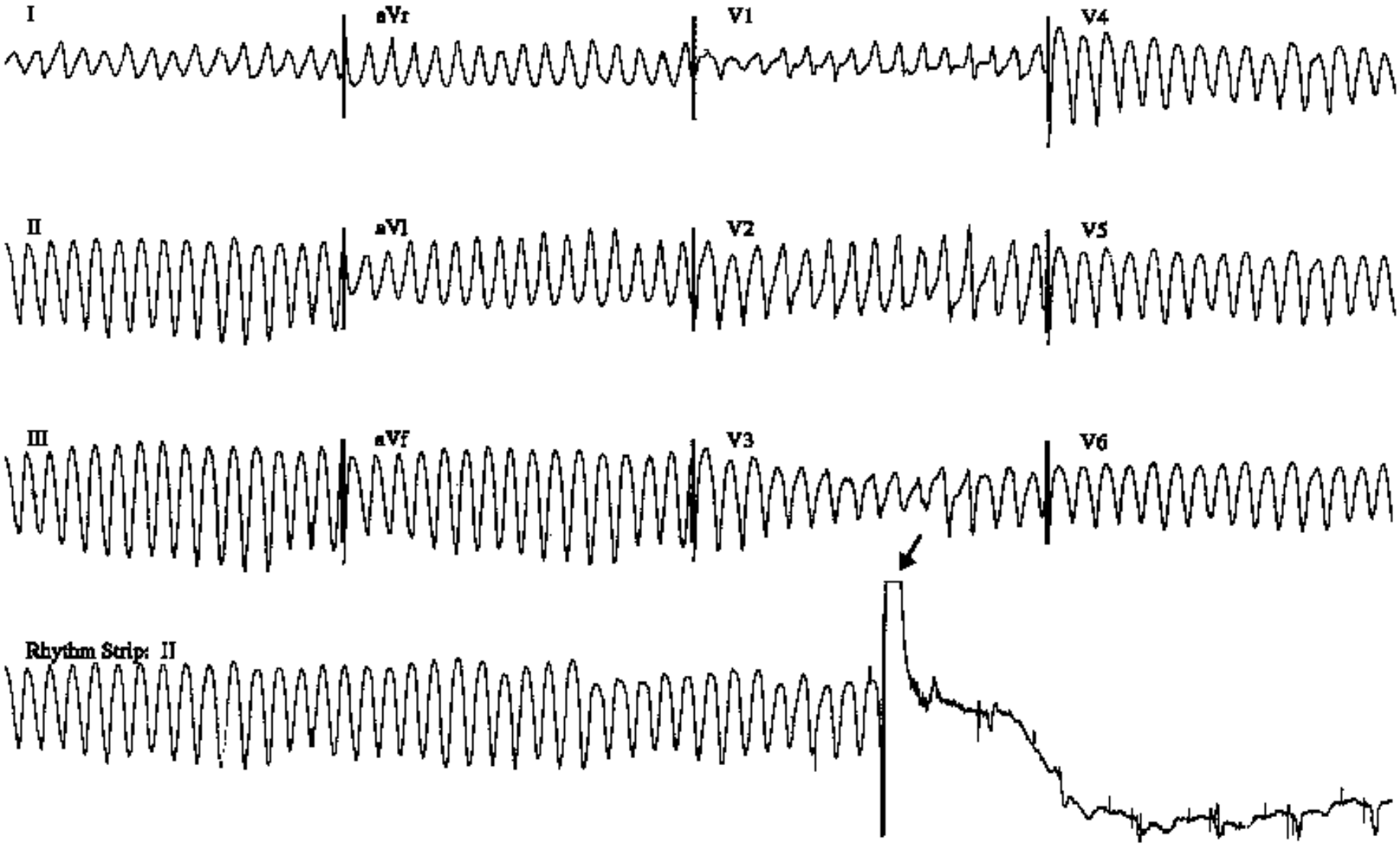
EKG změny


aVR
V1
V4
aVL
V2
V5
aVF
V3
V6

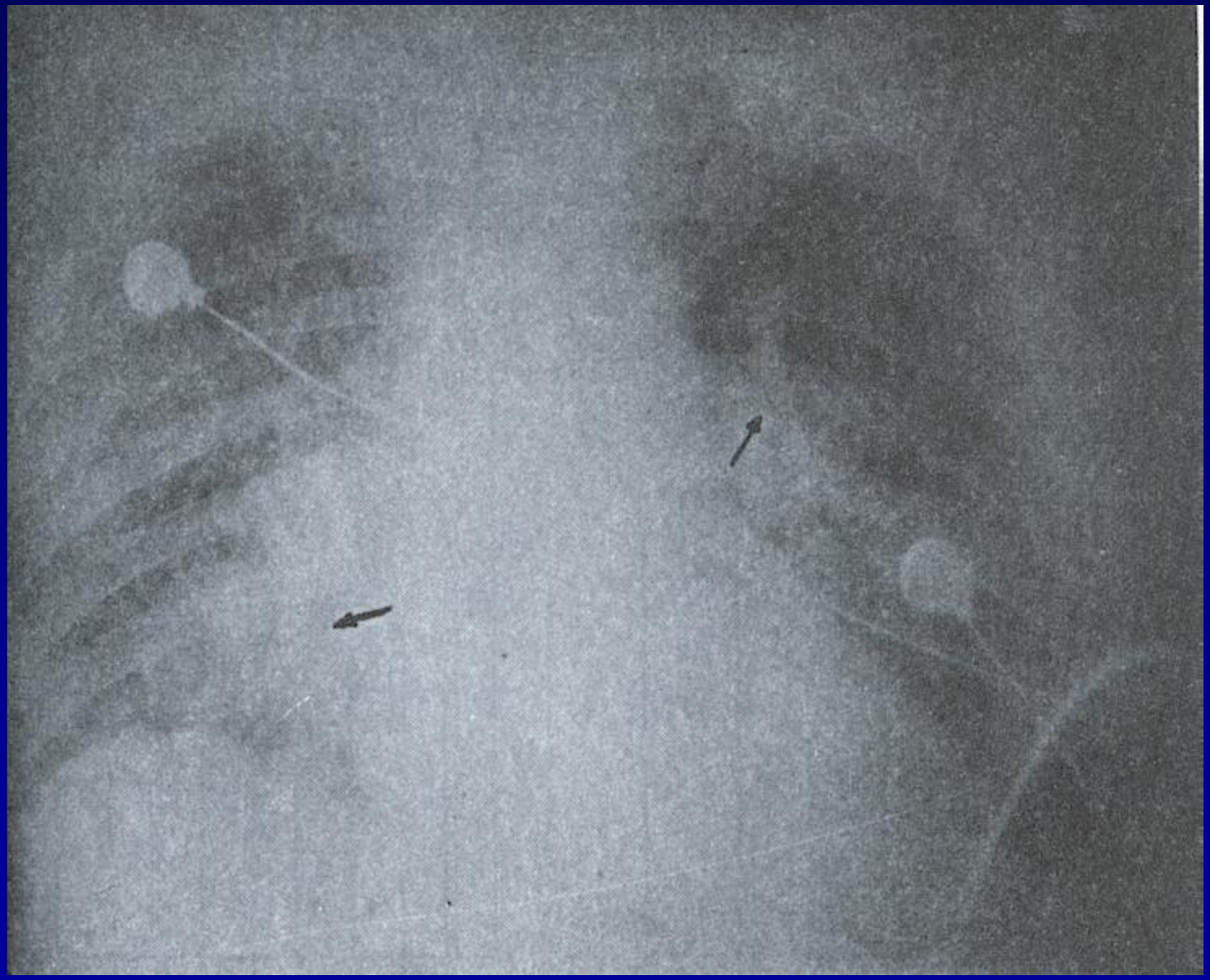

Coronary Arteries
ECG Signs of M.I.
Left Main
Circumflex
Right Coronary Artery
Left Ant Descending
Normal
ST Segment Elevation
Acute Injury
T Wave Inversion
Ischemia
Pathological Q Wave
Necrosis



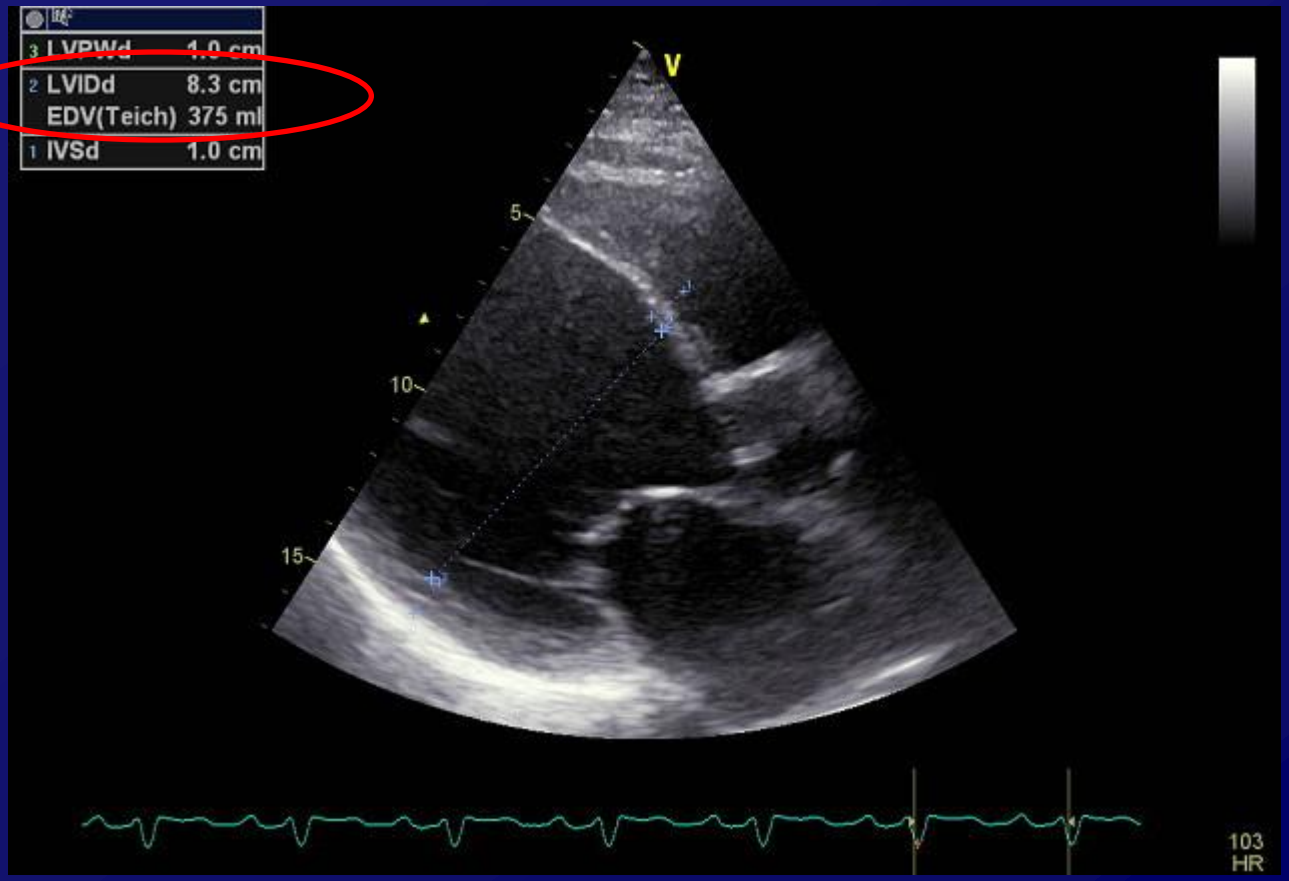

Artery


I
aVr
V1
V4
II
aVl
V2
V5
III
aVf
V3
V6
Rhythm Strip: II

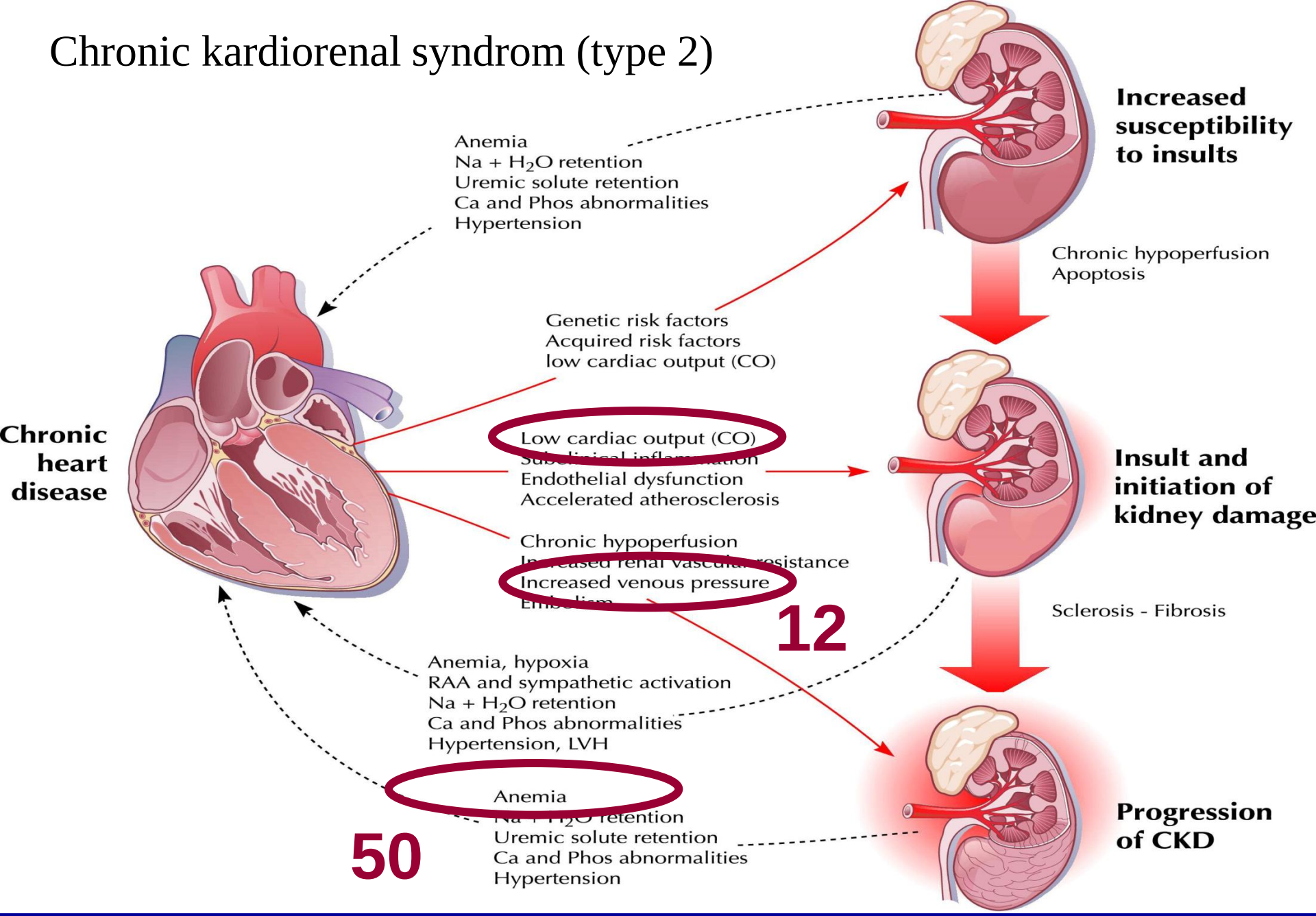
Alveolární plicní edém, KTR 0,61



EF 20%, dilatovaná LK



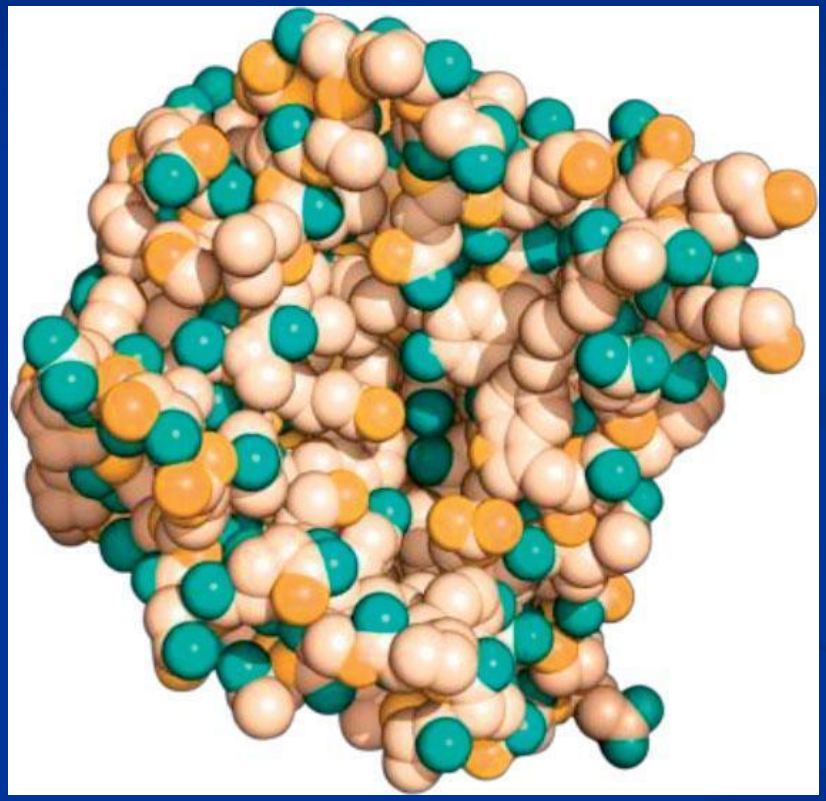
Chronic kardiorenal syndrom (type 2)



Ronco C, et al. Cardiorenal syndrome. *JACC* 2008; 52:1527-1539.

NGAL

(Neutrophil gelatinase associated lipocalin)



Protein evaluaeted in serum or urine

Cardio-hepatic syndrom
SURVIVE study 1134 pacientů

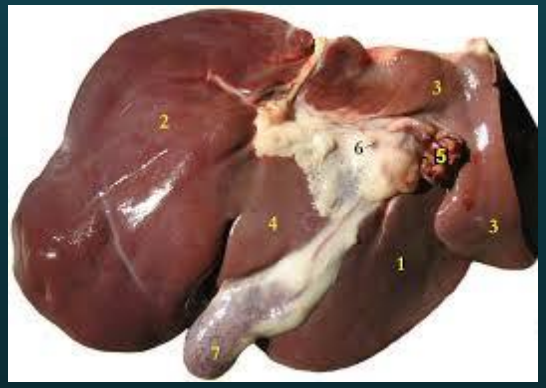
EUROPEAN SOCIETY OF CARDIOLOGY®

European Heart Journal (2013) **34**, 742–749
doi:10.1093/eurheartj/ehs332

CLINICAL RESEARCH
Heart failure/cardiomyopathy

Liver function abnormalities, clinical profile, and outcome in acute decompensated heart failure

Maria Nikolaou[1,2,3], John Parissis[3], M. Birhan Yilmaz[1,15], Marie-France Seronde[1,2,4], Matti Kivikko[5,6], Said Laribi[1,2,7], Catherine Paugam-Burtz[2,8], Danlin Cai[9], Pasi Pohjanjousi[6], Pierre-François Laterre[10], Nicolas Deye[1,11], Pentti Poder[12], Alain Cohen-Solal[1,2,13], and Alexandre Mebazaa[1,2,14]*



Post hoc analýza studie SURVIVE

	Normal AP	Abnormal AP	*P*-value	Normal ALT and AST	Abnormal ALT and/or AST	*P*-value
Peripheral oedema (%)	65.4	78.8	<0.001	71.2	63.2	0.005
Ascites (%)	17.3	31.7	<0.001	22.2	17.1	0.047
Cold extremities (%)	20.7	25.8	0.094	19.6	25.6	0.021
AMI	18.6	11.3	0.002	8.8	31.1	<0.001
LVEF	24.0	23.3	0.054	24.1	23.5	0.070
Tricuspid regurgitation	46.0	53.3	0.049	51.7	40.4	<0.001

Factors predicting abnormal liver tests

AP

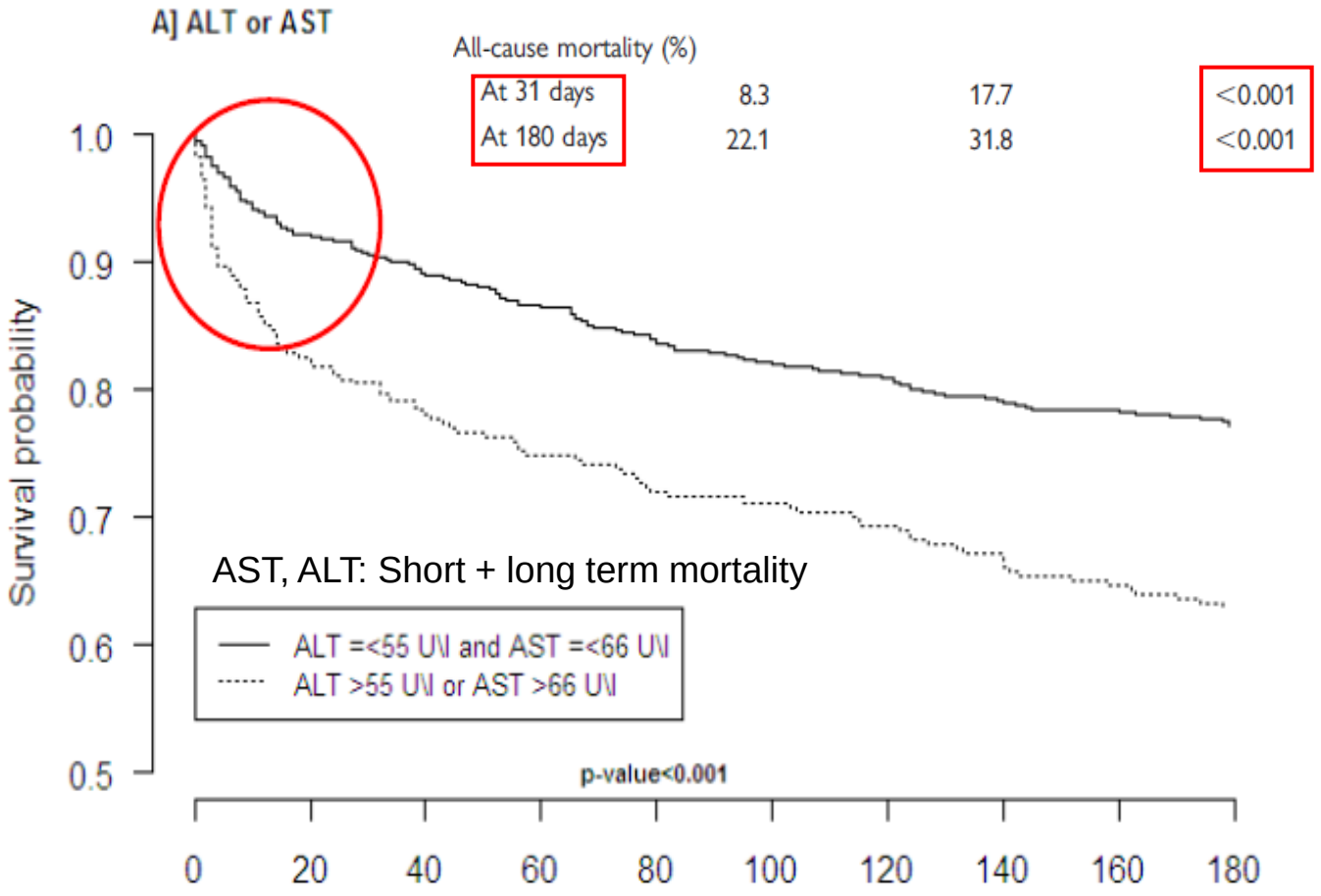
	OR	Lower CI	Upper CI	*P*-value
Ascites (yes/no)	1.808	1.276	2.561	0.002
Peripheral oedema (yes/no)	1.724	1.276	2.561	0.001
Diabetes mellitus (yes/no)	1.460	1.067	1.998	0.018
BNP (pg/mL per 100)	1.026	1.014	1.032	<0.0001

ALT/AST

	OR	Lower CI	Upper CI	*P*-value
Acute MI during current admission (yes/no)	3.138	2.010	4.899	<0.0001
BNP (pg/mL per 100)	1.020	1.011	1.028	<0.0001
HR (b.p.m.)	1.018	1.010	1.026	<0.0001
SBP (mmHg)	0.990	0.983	0.998	0.010
Beta-blocker at admission (yes/no)	0.690	0.526	0.905	0.007

Nikolaou M et al Eur Heart J 2013

6-month mortality as a function of liver cytolysis



Nikolaou M et al Eur Heart J 2013

Děkuji Vám za pozornost

Rytmus života 2015: 38:42-3