ΕΤΗΣΙΟ ΣΥΝΕΔΡΙΟ ΕΝΩΣΗΣ ΠΝΕΥΜΟΝΟΛΟΓΩΝ ΕΛΛΑΔΑΣ
30 ΜΑΪΟΥ – 2 ΙΟΥΝΙΟΥ 2019, ΞΕΝΟΔΟΧΕΙΟ ROYAL OLYMPIC, ΑΘΗΝΑ

Καρδιά και πνεύμονας

Οξεία καρδιακή ανεπάρκεια

Βασιλική Μπιστόλα

Καρδιολόγος

Μονάδα Καρδιακής Ανεπάρκειας

Β' Παν/κη Καρδιολογική Κλινική

ΠΓΝ Αττικό

National and Kapodistrian UNIVERSITY OF ATHENS

ATTIKON

Disclosures

- Honoraria: Novartis, Servier, Winmedica

Παρουσίαση περιστατικού

- Γυναίκα 72 ετών προσέρχεται στο ΤΕΠ με δύσπνοια προσφάτου ενάρξεως (48 ώρες).

- Αναφερόμενη ιογενής συνδρομή τις προηγούμενες ημέρες με συνοδό απόχρεμψη.

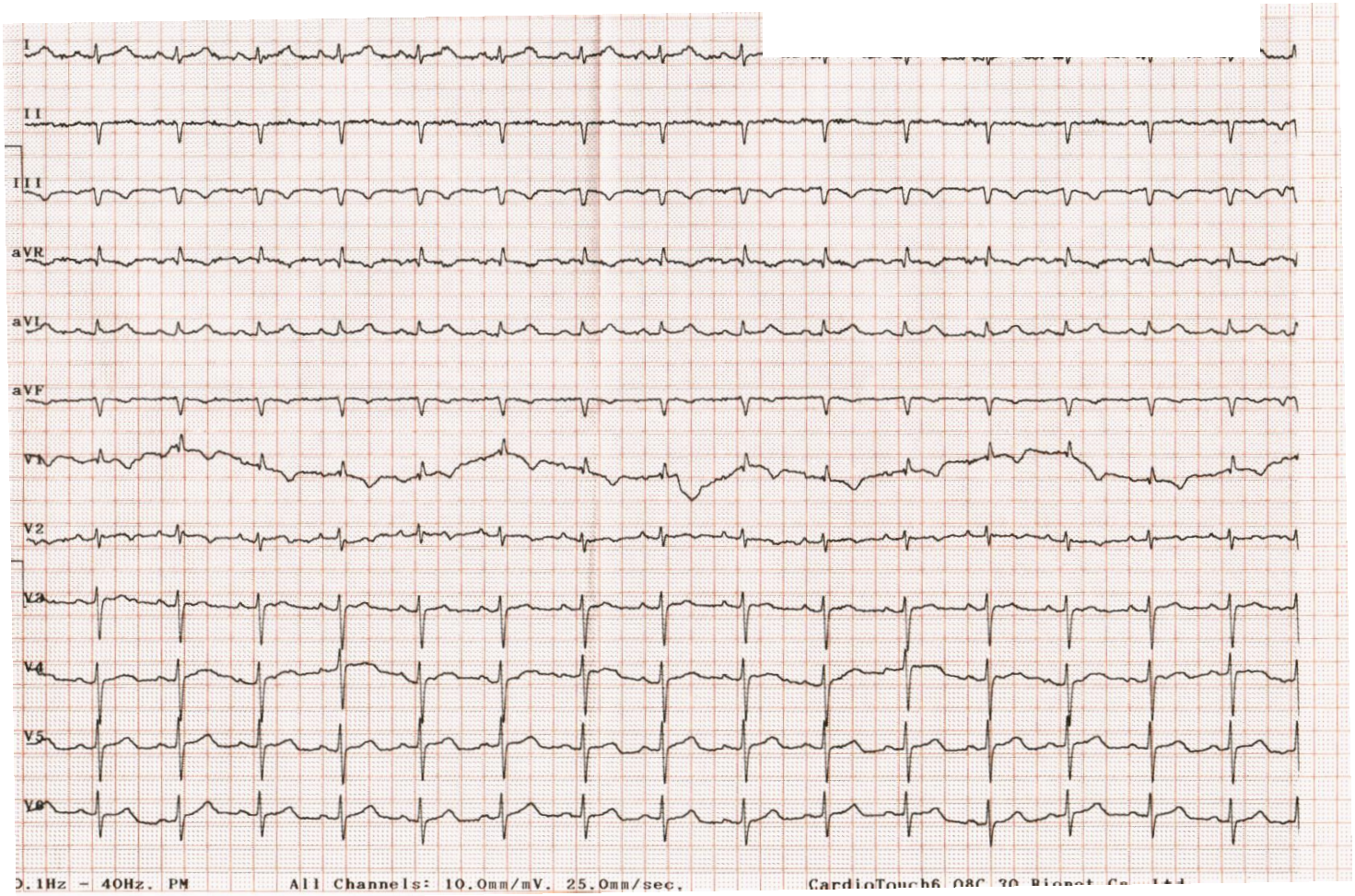
Ατομικό αναμνηστικό

- Χρόνια αποφρακτική πνευμονοπάθεια
- Προ 3ετίας PCI LCX
- Προ έτους σπινθηρογράφημα μυοκαρδίου αρνητικό για ισχαιμία
- Αρτηριακή υπέρταση
- Τέως καπνίστρια

Κλινική εξέταση

- Καρδιά: S1, S2, ρυθμικοί, ταχείς
- Πνεύμονες: Υγροί ρόγχοι στις βάσεις, παράταση εκπνοής, και εκπνευστικός συριγμός
- BP 180/95mmHg, HR 110bpm (SR)
- Ήπια οιδήματα κάτω άκρων
- Αέρια αρτηριακού αίματος: pH=7.35, pCO_2=45mmHg, pO_2=56mmHg, spO_2=89%

ΗΚΓ ΤΕΠ



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ΠΑΝΕΠΙΣΤΗΜΙΑΚΟ ΓΕΝΙΚΟ ΝΟΣΟΚΟΜΕΙΟ
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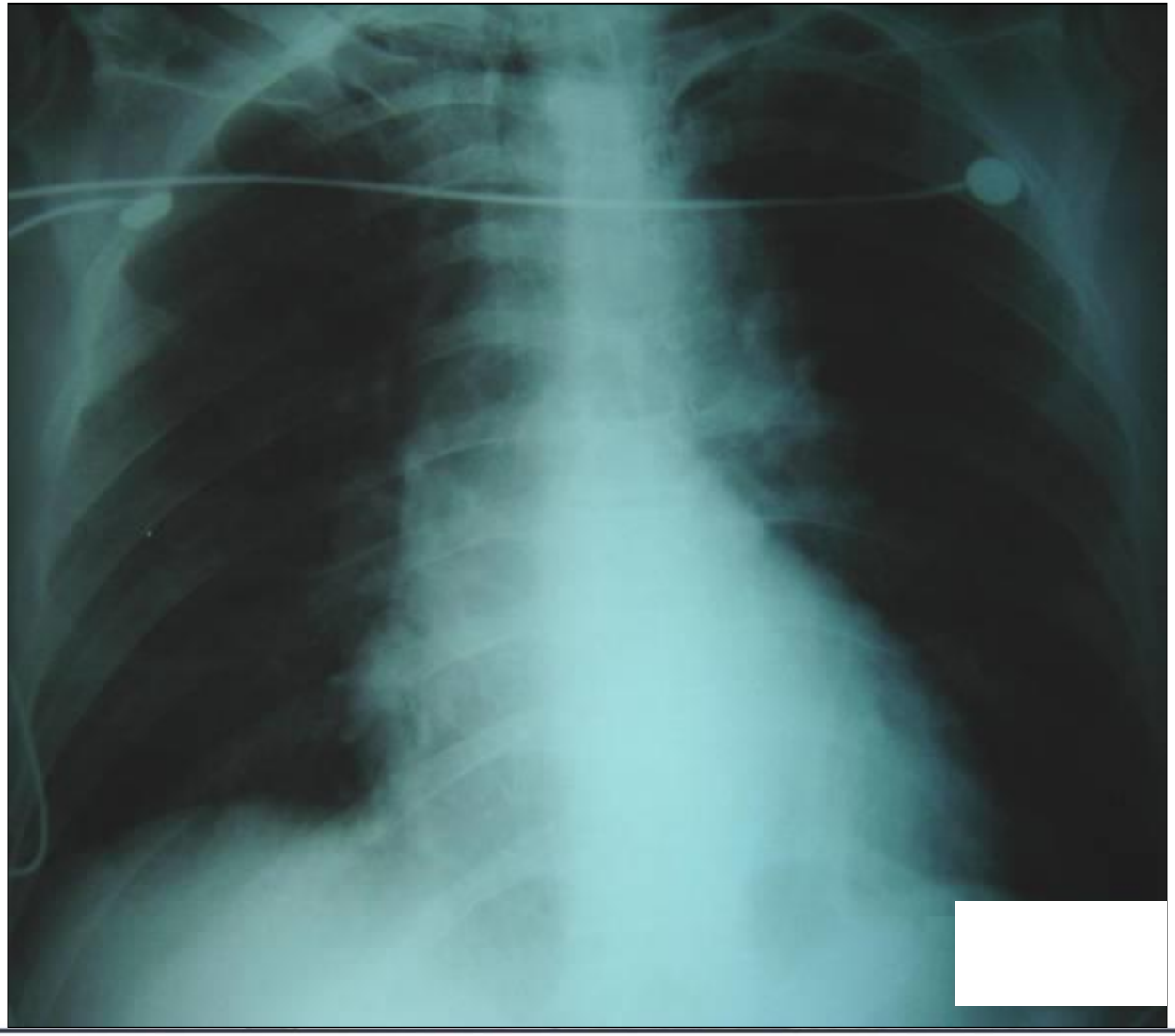


Εργαστηριακές εξετάσεις

Ht	39.5	CKMB	12	**CRP**	**18**
Hb	12.9	SGOT	46	Total Prot	6.5
WBC	**10.100**	SGPT	30	Albumine	3.9
PLT	315	ALP	104	**hsTroponin**	**24**
Glucose	110	γGT	146	**NTproBNP**	**2600**
Urea	50	TChol	145		
Creatinine	1.1	HDL	53	eGFR	52
K	4.8	LDL	77	D-dimers	200
Na	138	TG	77		
CPK	92	LDH	443		

Αγωγή προ της εισόδου

- Tb Carvedilol 6.25mg S:1x2
- Tb Ramipril 5mg S:1x2
- Tb Amlodipine 5mg S:1x1
- Tb Salospir 100mg S:1x1
- Tb Simvastatin 20mg S:1x1
- Inh Budenoside 200μg S:2x2

Διάγνωση:

- Παρόξυνση χρόνιας αποφρακτικής πνευμονοπάθειας?

- Απορρύθμιση καρδιακής ανεπάρκειας?

HF AND COPD: EPIDEMIOLOGY-PATHOPHYSIOLOGY

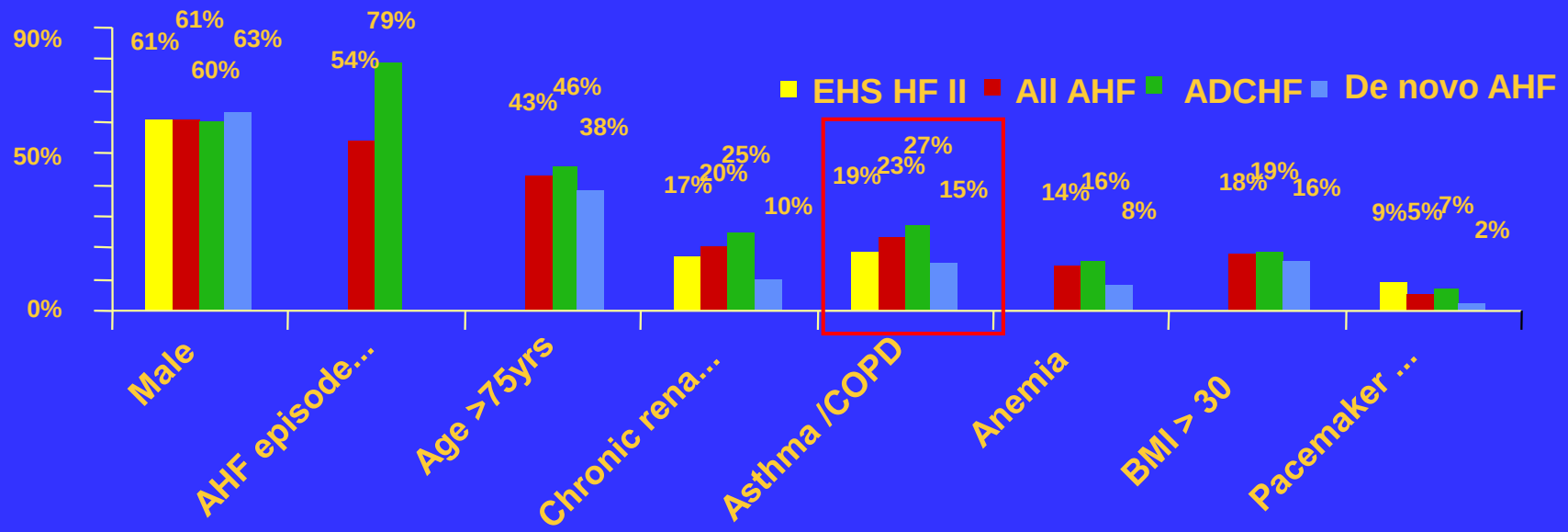
Epidemiology

- Prevalence of COPD in CHF is 20-33% and CAD is 40-47% (Medicare, Danish Diamond studies).

Clinical interaction

- Obstructive pattern: acute HF
- Restrictive pattern: chronic HF (reduced lung volume due to cardiomegaly and alveolar and interstitial fluid, development of interstitial fibrosis, changes of lung compliance, weakness of the respiratory muscles)
- COPD causes HF exacerbation: increased pre-load, increased after-load, neurohormonal/inflammatory activation

Co-morbidities in ALARM-HF survey (n=4953)



Intensive Care Med 2011;37(4):619-26

Limited Reliability of Physical Examination in Heart Failure

- Prospectively compared physical signs with hemodynamic measurements in 50 hospitalized patients
- **Rales, edema, jugular venous pulse elevation absent in 18 of 43 patients with pulmonary capillary wedge >24mmHg**
- **Sensitivity 58%, Specificity 100%**

Stevenson and Perloff. JAMA. 1989;261:884-888.

How Good are Existing Tools for Diagnosing Heart Failure?

	Sensitivity	Specificity	Accuracy
Hx of HF	62	**94**	80
Dyspnea	56	53	54
Orthopnea	47	**88**	72
Rales	56	80	70
S3	20	**99**	66
JVD	39	**94**	72
Edema	67	68	68

In ED, clinical misdiagnosis occurs in 25-50% of patients presenting with decompensating HF **(Agency for Health Care and Research 1994)**

Dao Q *et al. J Am Coll Cardiol* 2001;37:379-85.

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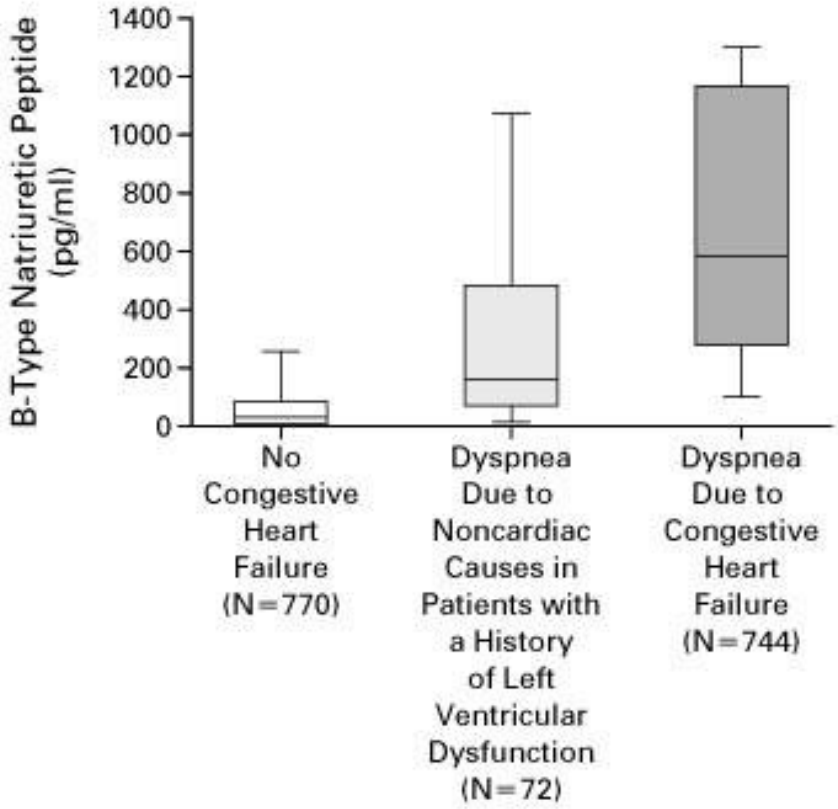
Rapid Measurement of B-Type Natriuretic Peptide in the Emergency Diagnosis of Heart Failure



Maisel A, et al. N Engl J Med 2002; 347:161-167

Regarding applied diagnostic measurements

Recommendations	Class	Level
Upon presentation a measurement of plasma natriuretic peptide level (BNP, NT-proBNP or MR-proANP) is recommended in all patients with acute dyspnoea and suspected AHF to help in the differentiation of AHF from non-cardiac causes of acute dyspnoea.	I	A
At admission in all patients presenting with suspected AHF, the following diagnostic tests are recommended:		
a. 12-lead ECG;	I	C
b. chest X-ray to assess signs of pulmonary congestion and detect other cardiac or non-cardiac diseases that may cause or contribute to the patient's symptoms;	I	C
c. the following laboratory assessments in the blood: cardiac troponins, BUN (or urea), creatinine, electrolytes (sodium, potassium), glucose, complete blood count, liver function tests and TSH.	I	C
Echocardiography is recommended immediately in haemodynamically unstable AHF patients and within 48 hours when cardiac structure and function are either not known or may have changed since previous studies.	I	C

European Heart Journal (2016) **37**, 2129-2200 - doi:10.1093/eurheartj/ehw 128

Causes of elevated concentrations of natriuretic peptides

Cardiac	Heart failure
	Acute coronary syndromes
	Pulmonary embolism
	Myocarditis
	Left ventricular hypertrophy
	Hypertrophic or restrictive cardiomyopathy
	Valvular heart disease
	Congenital heart disease
	Atrial and ventricular tachyarrhythmias
	Heart contusion
	Cardioversion, ICD shock
	Surgical procedures involving the heart
	Pulmonary hypertension

Non-cardiac	Advanced age
	Ischaemic stroke
	Subarachnoid haemorrhage
	Renal dysfunction
	Liver dysfunction (mainly liver cirrhosis with ascites)
	Paraneoplastic syndrome
	Chronic obstructive pulmonary disease
	Severe infections (including pneumonia and sepsis)
	Severe burns
	Anaemia
	Severe metabolic and hormone abnormalities (e.g. thyro-toxicosis, diabetic ketosis)

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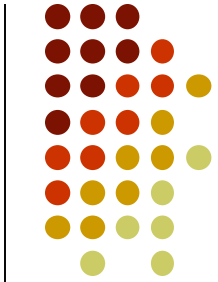
Clinical conditions with congestion and low NP levels



- Acute pulmonary edema due to acute mitral regurgitation
- Flash pulmonary edema in patients with preserved EF
- Acute pulmonary edema in patients with mitral stenosis
- Constrictive pericarditis without intrinsic heart disease
- Obesity
- Left atrial tumors

Thygesen K et al. Eur Heart J 2011;eurheartj.ehq509

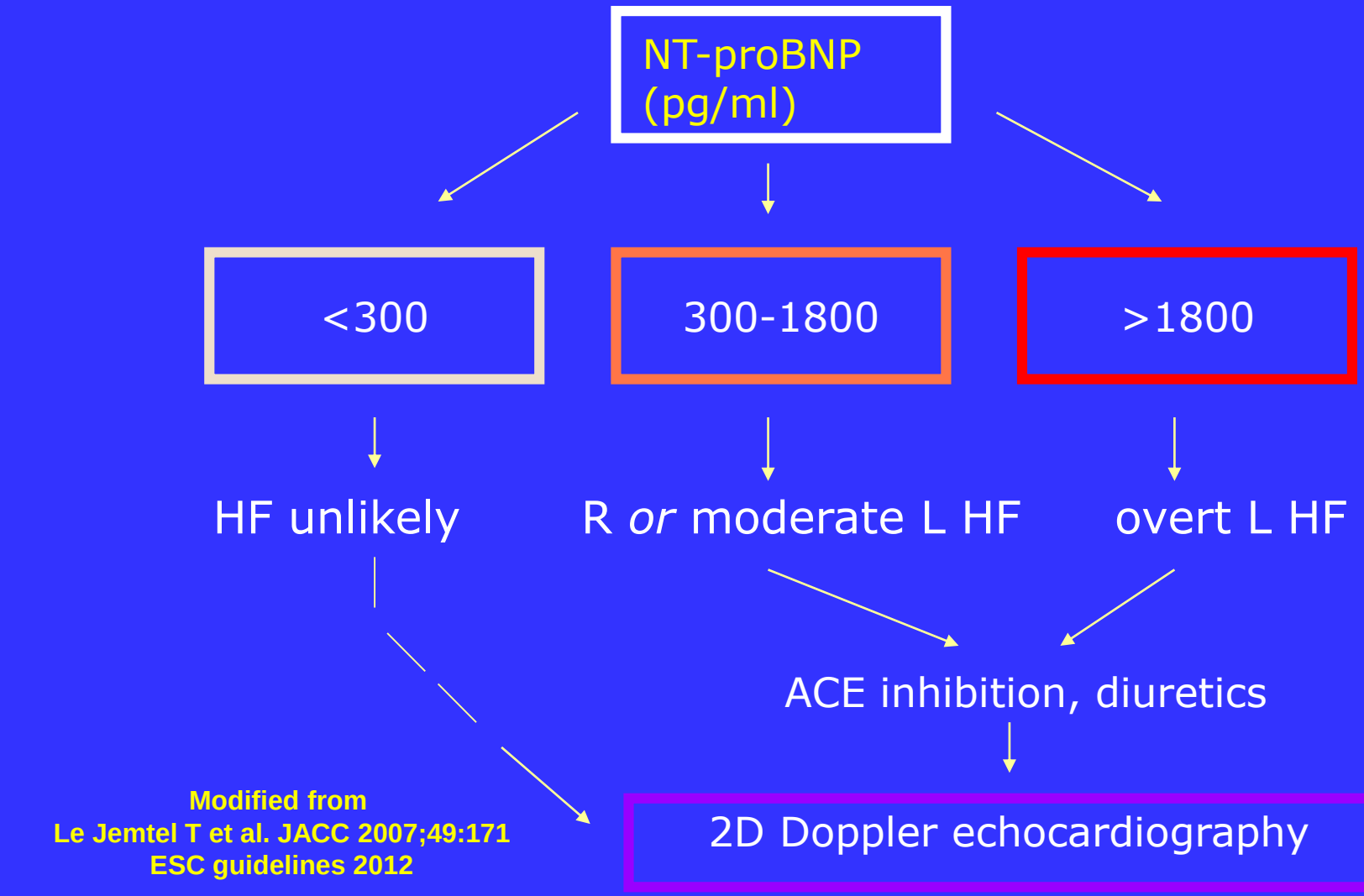
Optimal cutoffs of natriuretic peptides for AHF diagnosis

	BNP (pg/mL)	NT-proBNP (pg/mL)
AHF unlikely	<100	<300
AHF likely		
Age <50 years	>400	>450
Age 50–75 years	>400	>900
Age >75 years	>400	>1800
BMI >30 kg/m^2	>800	No correction needed
eGFR <60 mL/min/1.73 m^2	>200	>1200

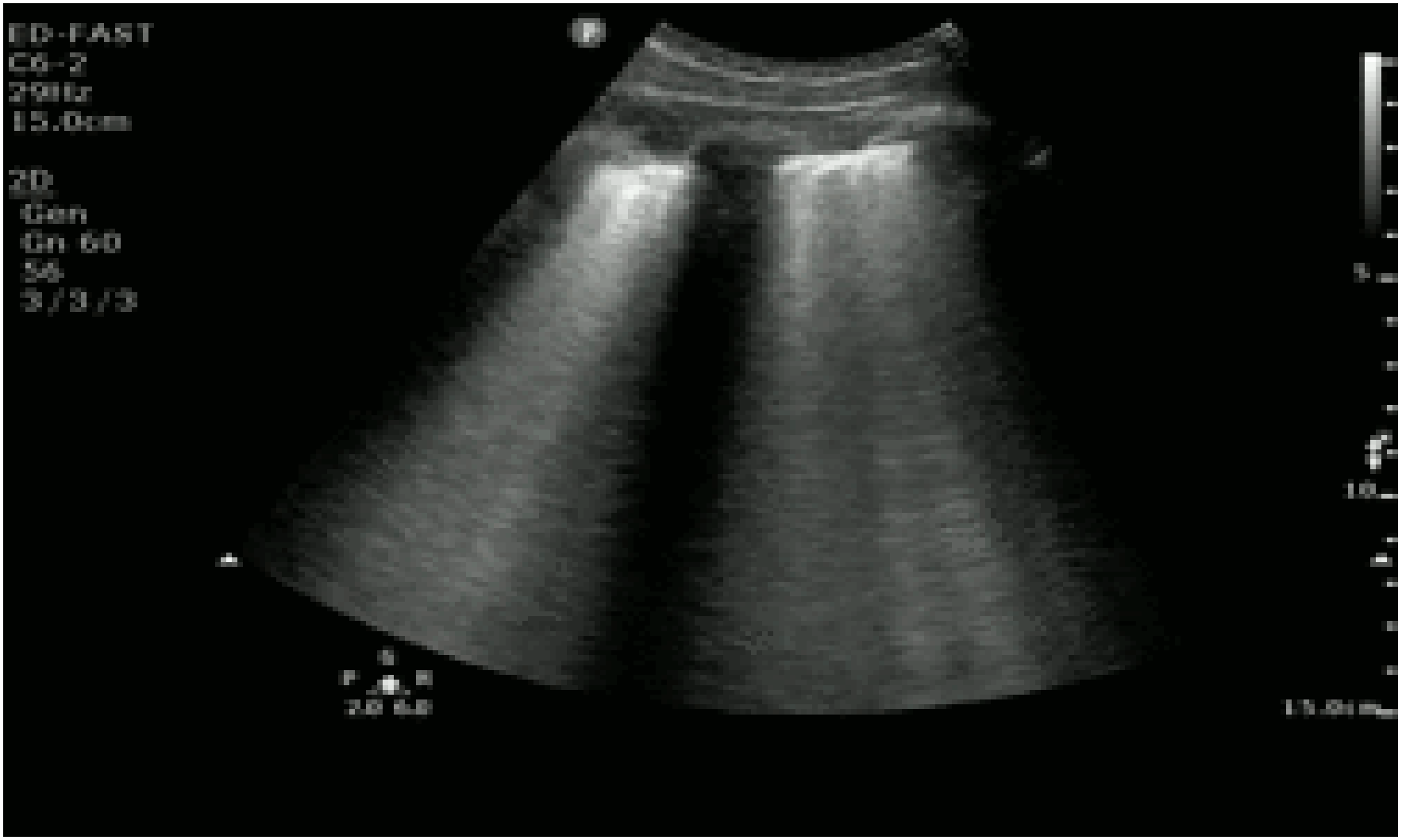
AHF, acute heart failure; BMI, body mass index; BNP, brain natriuretic peptide; eGFR, estimated glomerular filtration rate; NT-proBNP, N-terminal probrain natriuretic peptide.

Maisel et al. Eur J Heart Fail 2008
Kim & Januzzi. Circulation 2011

Evaluation of HF during COPD exacerbation

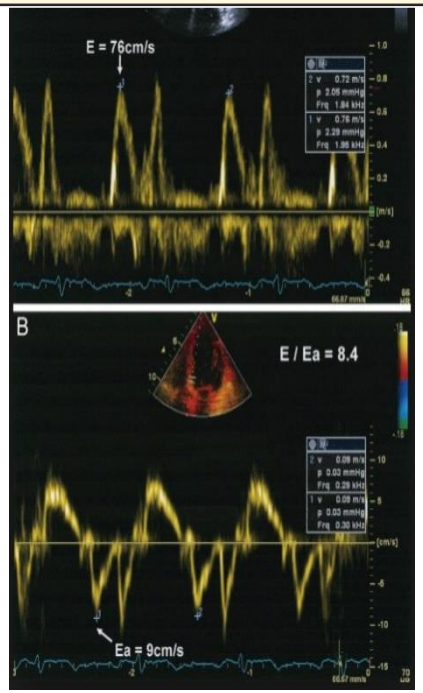


Modified from
Le Jemtel T et al. JACC 2007;49:171
ESC guidelines 2012

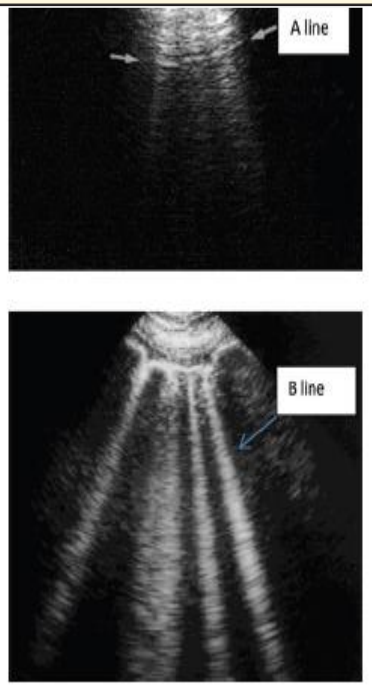

ED-FAST
C6-2
29Hz
15.0cm
2D
Gen
Gn 60
56
3/3/3

Evaluation of filling pressures and congestion

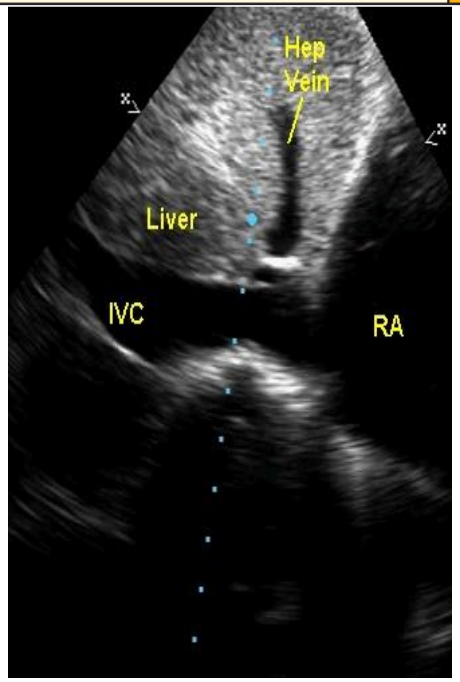
Thoracic ultrasound may be considered for the confirmation of pulmonary congestion and pleural effusion in patients with AHF.	IIb	C
Ultrasound measurement of inferior vena cava diameter may be considered for the assessment of volaemia status in patients with HF.	IIb	C



Elevated LV filling pressure
E/e' >13



B-lines / comets
Pleural fluid



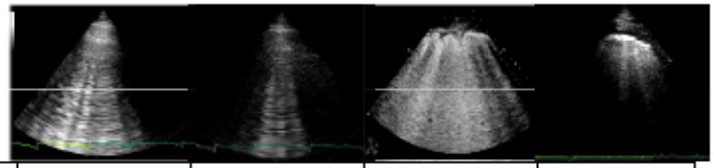
(Clearly) elevated CVP:
IVC diameter >20 mm &
Collapsibility index < 0.5

Lung ultrasound: a new tool for the cardiologist

Table 1 Scoring of B-lines

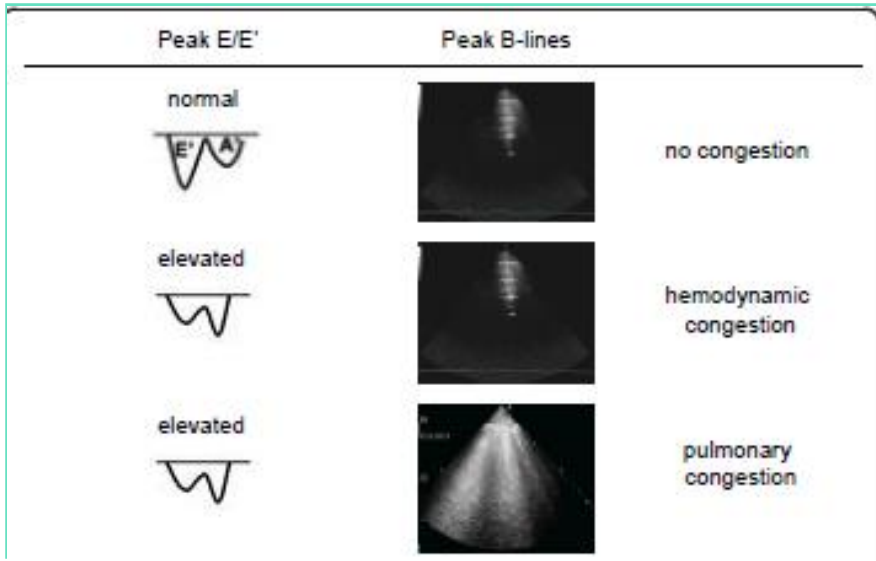
Score	Number of B-lines	Extravascular lung water
0	≤ 5	No sign
1	6 - 15	Mild degree
2	16 - 30	Moderate degree
3	> 30	Severe degree

(Modified from Picano et al, 2006 [16]).



	Acute cardiogenic pulmonary edema	Chronic heart failure	ALI/ARDS	Pulmonary fibrosis
Clinical setting	acute	chronic	acute	chronic
B-lines number	++++	+/++/+++	++++	+/++/+++
B-lines distribution	multiple, diffuse, bilateral (white lung)	multiple, diffuse, bilateral, following decubitant regions (black and white lung)	non-homogeneous distribution, presence of spared areas	more frequently posterior at lung basis
Other LUS signs	pleural effusion	pleural effusion	pleural effusion, pleural alterations, parenchymal consolidations of various size	pleural thickening
Echocardiogram	abnormal	abnormal	likely normal	likely normal

ALI = acute lung injury; ARDS = acute respiratory distress syndrome; LUS = lung ultrasound.



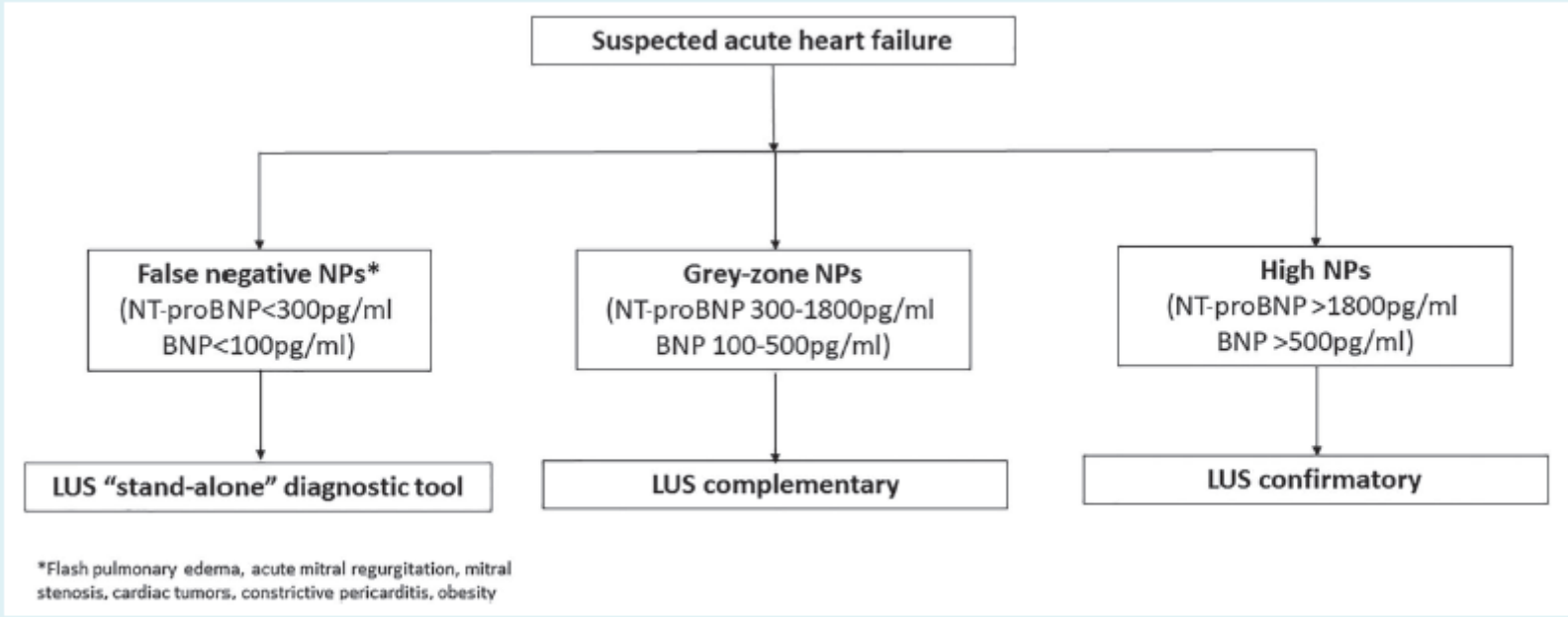


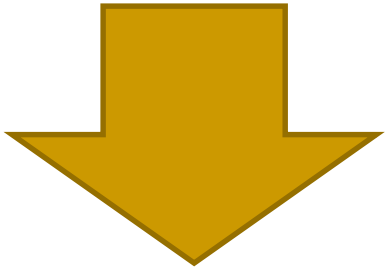
Figure 1 Suspected acute heart failure in the emergency department. Proposed diagnostic algorithm combining lung ultrasound (LUS) and natriuretic peptides (NPs). BNP, B-type natriuretic peptide; NT-proBNP, N-terminal pro-B-type natriuretic peptide.

Vasiliki Bistola[1], Eftihia Polyzogopoulou[2], Ignatios Ikonomidis[1], and John Parissis[1,2]

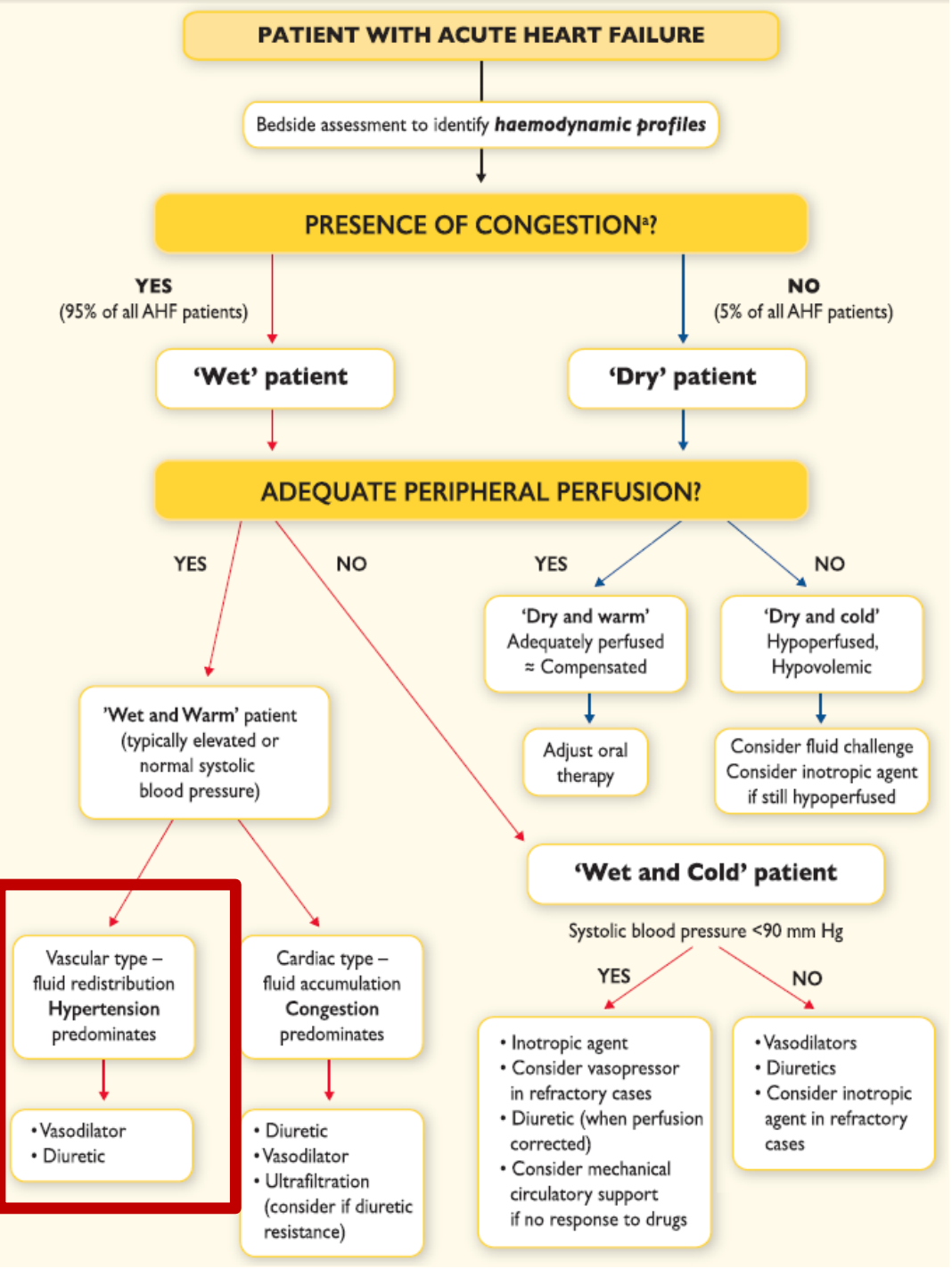
European Journal of Heart Failure (2019)
doi:10.1002/ejhf.1414

Διάγνωση:

- Απορρύθμιση καρδιακής ανεπάρκειας



- Εισαγωγή στην καρδιολογική κλινική



Management of patients with acute heart failure based on clinical profile during an early phase

HEART FAILURE ASSOCIATION OF THE ESC

INTRAVENOUS DIURETICS IN AHF

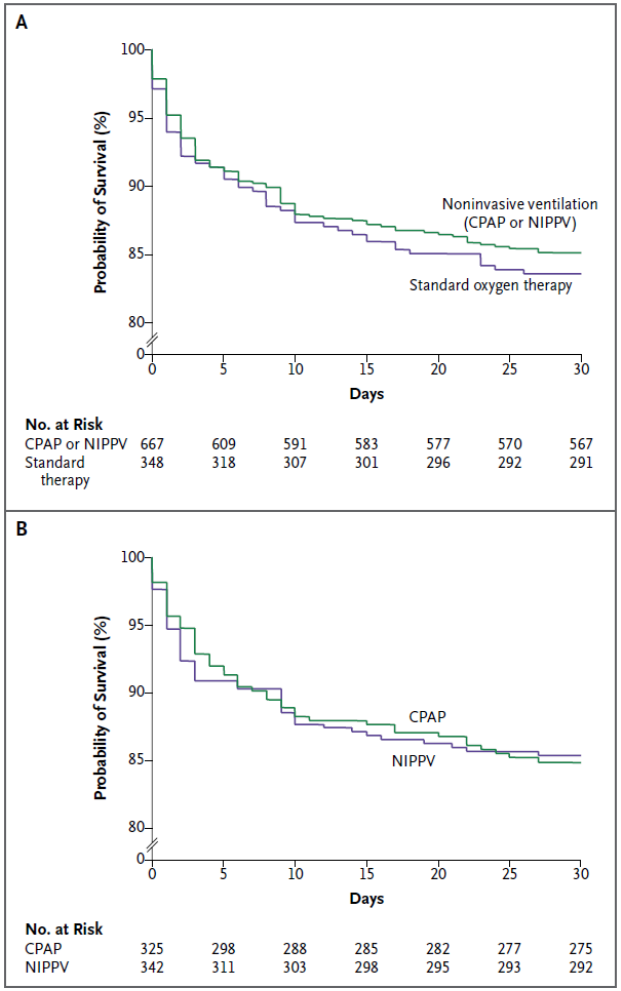
- High doses of loop diuretics should be avoided. High doses of these agents can cause excessive reduction of preload (risk of hypotension) and metabolic alkalosis (risk of hypoventilation and hypercapnia)

The management of patients with acute heart failure: oxygen therapy and ventilatory

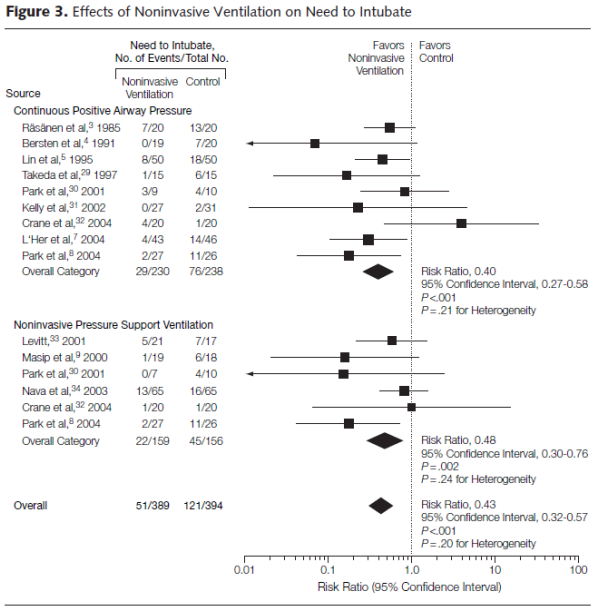
Recommendations	Class	Level
Monitoring of transcutaneous arterial oxygen saturation (SpO_2) is recommended.	I	C
Measurement of blood pH and carbon dioxide tension (possibly including lactate) should be considered, especially in patients with acute pulmonary oedema or previous history of COPD using venous blood. In patients with cardiogenic shock arterial blood is preferable.	IIa	C
Oxygen therapy is recommended in patients with AHF and SpO_2 <90% or PaO_2 <60 mmHg (8.0 kPa) to correct hypoxaemia.	I	C
Non-invasive positive pressure ventilation (CPAP, BiPAP) should be considered in patients with respiratory distress (respiratory rate >25 breaths/min, SpO_2 <90%) and started as soon as possible in order to decrease respiratory distress and reduce the rate of mechanical endotracheal intubation. Non-invasive positive pressure ventilation can reduce blood pressure and should be used with caution in hypotensive patients. Blood pressure should be monitored regularly when this treatment is used.	IIa	B
Intubation is recommended, if respiratory failure, leading to hypoxaemia (PaO_2 <60 mmHg (8.0 kPa)), hypercapnia ($PaCO_2$ >50 mmHg (6.65 kPa)) and acidosis (pH <7.35), cannot be managed non-invasively.	I	C

European Heart Journal (2016) **37**, 2129-2200 - doi:10.1093/eurheartj/ehw 128

Noninvasive Ventilation in acute heart failure



- **Neutral effects of NIV on mortality**
- **Decreases respiratory distress and rate of mechanical endotracheal intubation**
- **Bi-level PPV also improves minute ventilation and is especially useful in patients with hypercapnia, most typically COPD patients**



Gray, A et al. N Engl J Med 2008;359:142-51
Masip, J et al. JAMA. 2005;294:3124-3130

Αγωγή εισόδου

- Ρινικός καθετήρας χορήγησης O2 (2lt/min)
- ΕΦ Νιτρώδη
- ΕΦ Φουροσεμίδη 20mg x2
- Neb Ipratropium bromide 500μg x4
- Tb Carvedilol 6.25mg x2→ διακοπή λόγω βρογχόσπασμου
- Έναρξη ιβαμπραδίνης 5mgx2
- Tb Salospir 100mg x1
- Tb Simvastatin 20mg x1
- ΗΧΜΒ (προφυλακτική θεραπεία ΕΦΘ)
- **PCT 0.03**

Prescription and short term effects of acute respiratory therapies in acute heart failure

- 164,494 ADHF hospitalizations in 2009-2010 US
- Acute respiratory therapy (ART) is administered to >50% of patients hospitalized with ADHF.
- Treatment with ART during the first 2 hospital days was associated with higher odds of adverse outcomes.

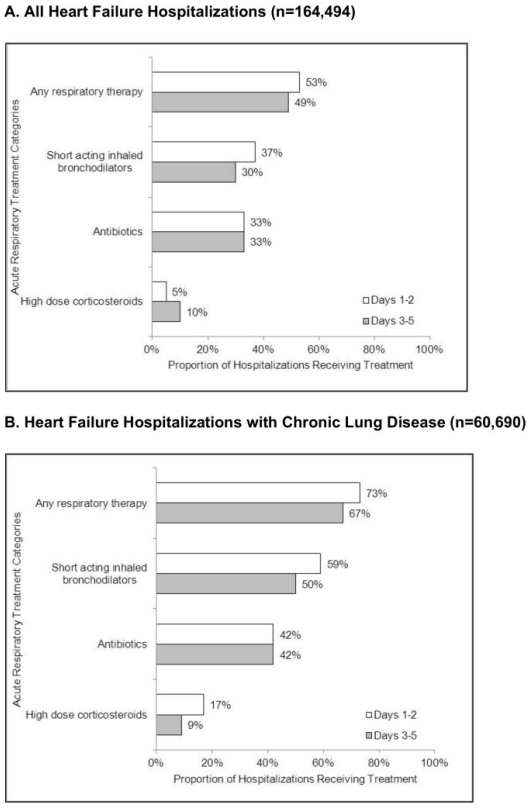


Table 2. Odds Ratios for Adjusted Outcomes by Treatment During the First 2 Hospital Days for All Heart Failure Hospitalizations.

Treatment During First 2 Hospital Days	Adjusted Outcomes by Treatment Group for All Heart Failure Hospitalizations (N=164,494)		
	Odds Ratio (95% CI)		
	In-Hospital Mortality	ICU admission	Late Intubation
HF Only	--	--	--
HF + Bronchodilators	1.56 (1.41, 1.73)	1.27 (1.22, 1.33)	1.35 (1.24, 1.46)
HF + Antibiotics (± Bronchodilators)	1.74 (1.60, 1.88)	1.74 (1.68, 1.80)	1.76 (1.65, 1.88)
HF + Corticosteroids (± Bronchodilators)	1.40 (1.16, 1.71)	1.44 (1.33, 1.55)	1.33 (1.15, 1.54)
HF + Antibiotics + Corticosteroids (± Bronchodilators)	2.04 (1.78, 2.34)	2.03 (1.91, 2.16)	1.85 (1.65, 2.07)

HF: heart failure; ICU: intensive care unit.

doi: 10.1371/journal.pone.0078222.t002

Table 3. Odds Ratios for Adjusted Outcomes by Treatment During the First 2 Hospital Days for Heart Failure Hospitalizations with Chronic Lung Disease.

Treatment During First 2 Hospital Days	Adjusted Outcomes by Treatment Group for Heart Failure Hospitalizations with Chronic Lung Disease (N=63,690)		
	Odds Ratio (95% CI)		
	In-Hospital Mortality*	ICU admission	Late Intubation
HF Only	--	--	--
HF + Bronchodilators	1.24 (1.06, 1.45)	1.14 (1.07, 1.22)	1.11 (0.99, 1.25)
HF + Antibiotics (± Bronchodilators)	1.34 (1.17, 1.54)	1.71 (1.61, 1.81)	1.51 (1.36, 1.67)
HF + Corticosteroids (± Bronchodilators)	1.33 (1.04, 1.68)	1.32 (1.20, 1.46)	1.16 (0.97, 1.38)
HF + Antibiotics + Corticosteroids (± Bronchodilators)	1.63 (1.36, 1.95)	1.87 (1.73, 2.02)	1.60 (1.40, 1.83)

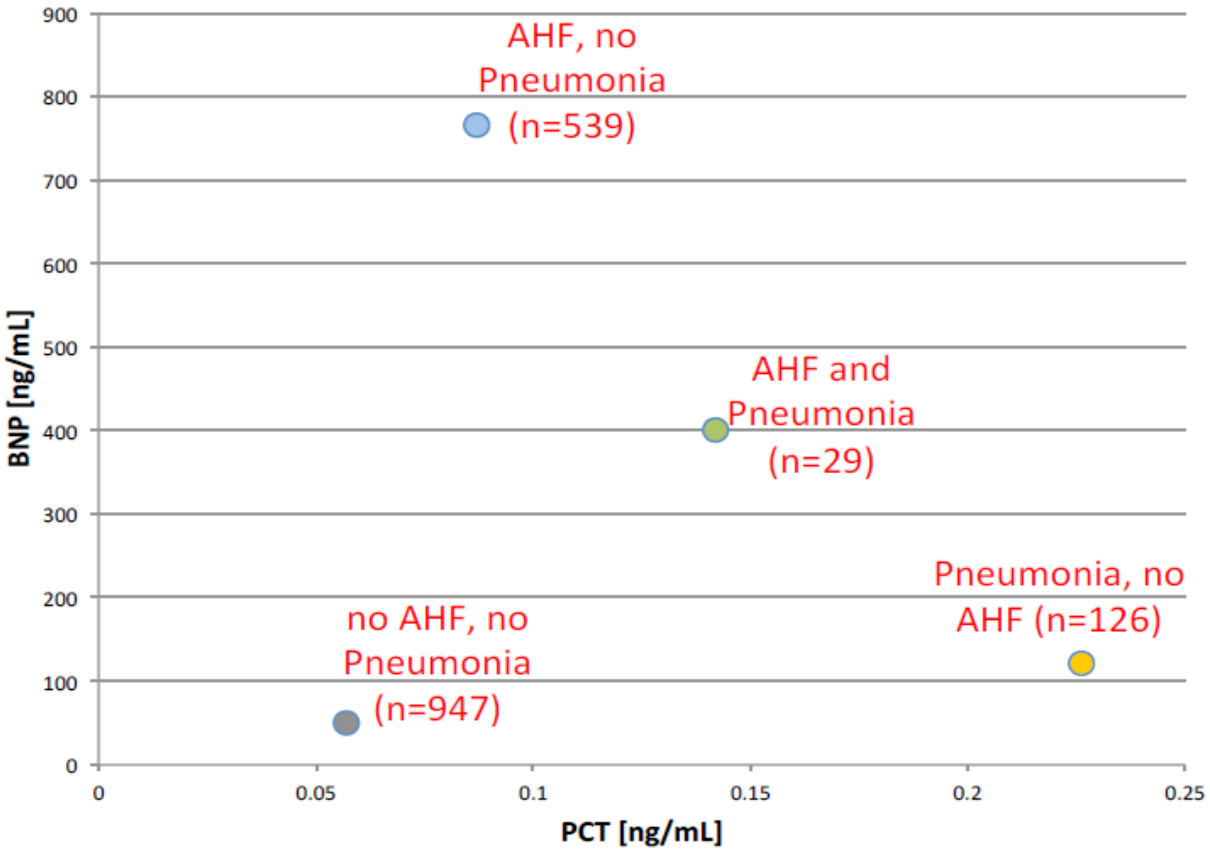
Dharmarajan K, et al. PLoS ONE 2013 8(10): e78222.

ALARM sub-analysis: Differences in clinical presentation and precipitating factors between patients with vs without COPD

Clinical characteristics	COPD	Non COPD	p
Clinical presentation			<0.001
Acutely decompensated chronic HF	77.2%	60.1%	
Acute de novo HF	22.8%	39.9%	
Precipitating factors			<0.001
Acute coronary syndrome	35.5%	36.7%	0.5
Arrhythmias	31.5%	26%	<0.001
Poor compliance	16.3%	12.5%	0.001
Infection	25.0%	14%	<0.01
Drug-induced	2.4%	3.2%	0.1
Post-surgical	3.9%	3.1%	0.2
Valvular heart disease	13.1%	12.9%	0.8
Systolic blood pressure (mmHg)			
< 100	24.5	28.6	0.04
101-120	18.6	17.8	
121-159	27.6	24.8	
>160	29.4	28.8	

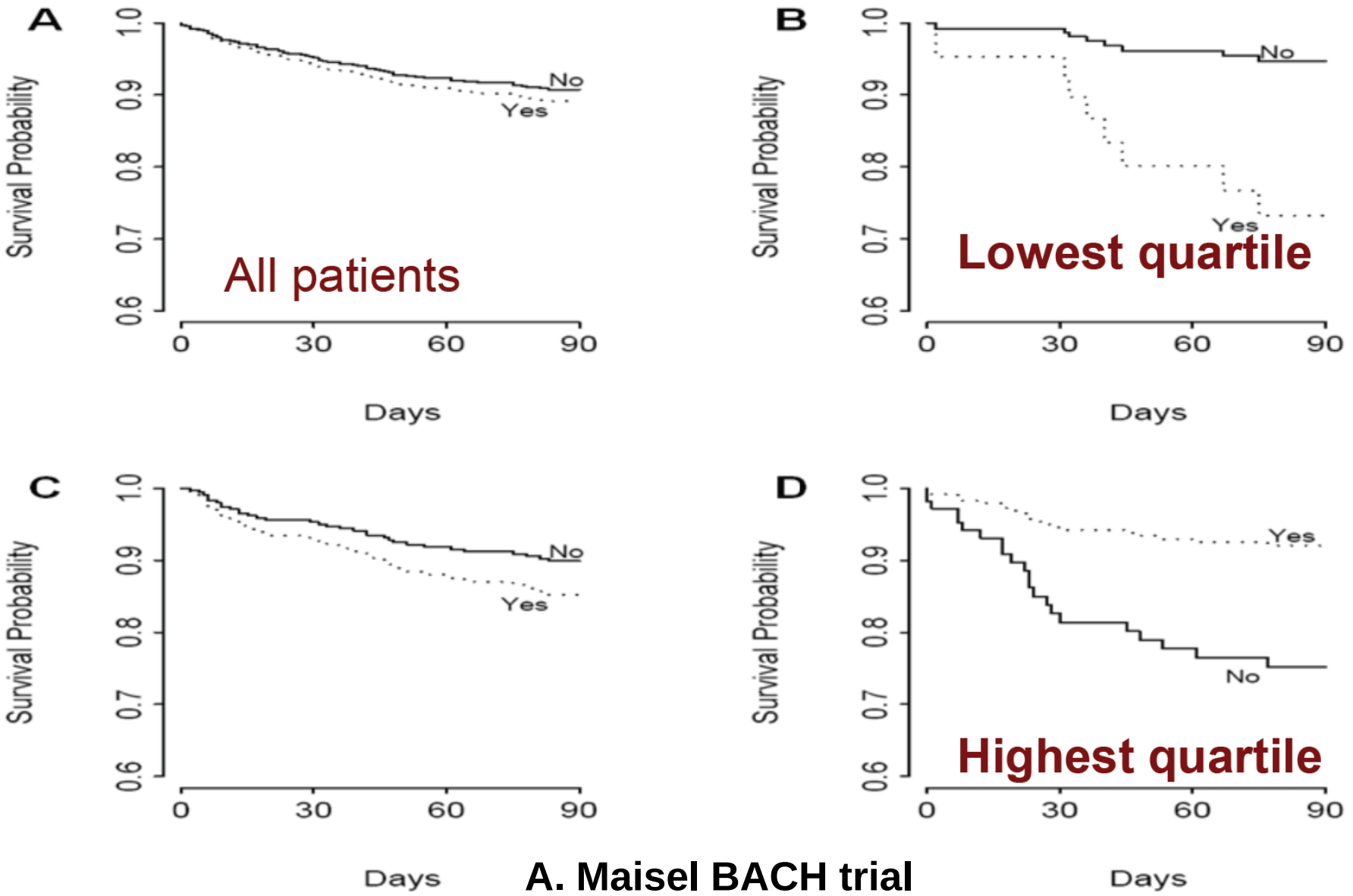
Parissis et al. Clin Res Cardiology 2014

BACH TRIAL: Combining BNP and PCT in differential diagnosis of dyspnea



Maisel et al EJHF in press

Antibiotic treatment (yes or no) and all cause mortality within 90 days



A. Maisel BACH trial

Υπερηχοκαρδιογράφημα

- LVED=56mm
- IVSd=11mm
- EF=45%, διάχυτη έκπτωση της συστολικής απόδοσης και επιπρόσθετη υποκινησία κατωτέρου τοιχώματος
- LA=45mm
- IVC=23mm
- Ήπια MR & TR, RVSP=45mmHg
- E/e ratio: 16

Κλινική πορεία

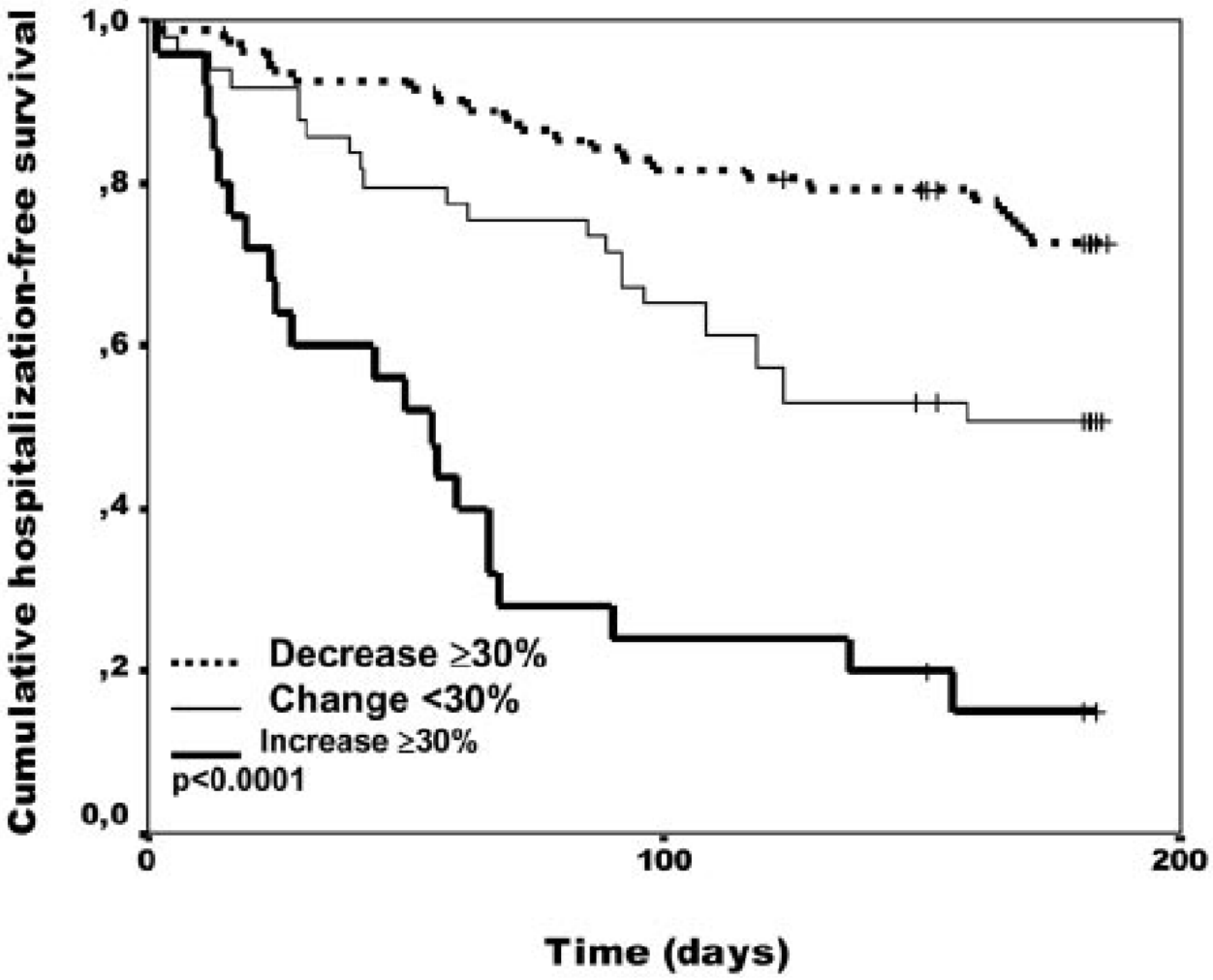
- 24 ώρες μετά την εισαγωγή: κλινική βελτίωση
- Αντικατάσταση καρβεντιλόλης με μικρή δόση βισοπρολόλης (β1 εκλεκτικός)
- Titropium bromide 2 inh X 2
- NT-proBNP εξόδου (5 ημέρες αργότερα): 1300 pg/ml
- Επανέλεγχος στο ιατρείο καρδιακής ανεπάρκειας (στόχος NT-proBNP <1000 pg/ml)

Prognosis of HF patients with COPD

- **COPD strongly predicts hospitalisation rate and duration and non-cardiovascular mortality**
- **5-year mortality as high as 69% (58% in patients without COPD)**
- **Respiratory infections associated with cardiac decompensations in 10-16% admissions**

Nieminen MS. Eur Heart J 2006;27:2725-2736

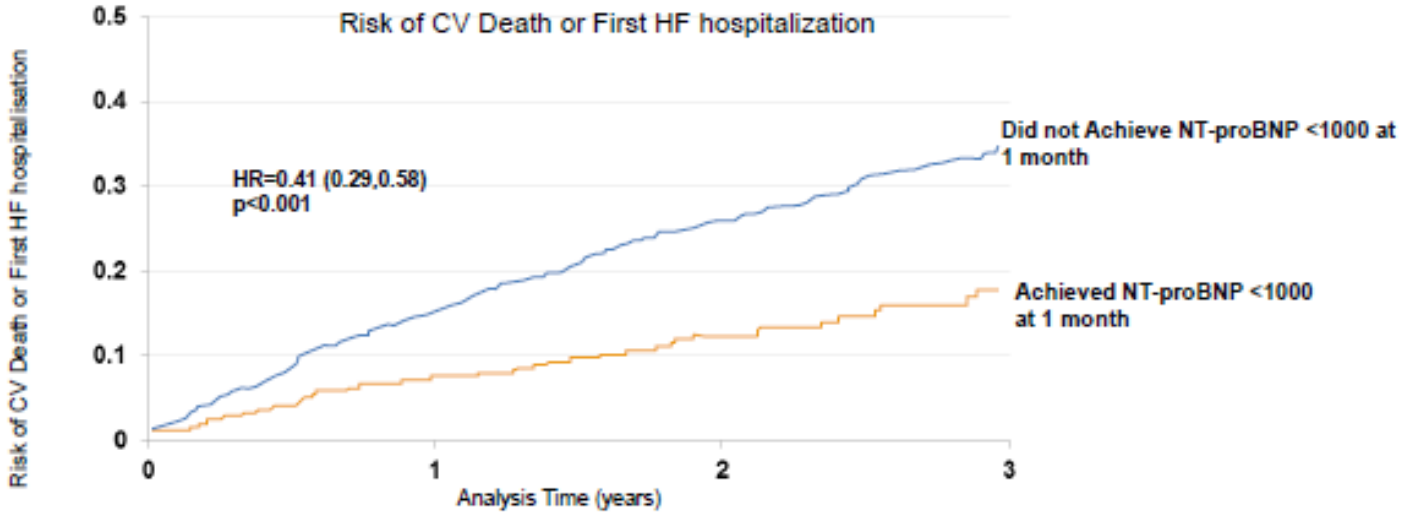
Variation of in-hospital NT-proBNP levels strongly predicts adverse prognosis in ADHF patients



Bettencourt et al. Circulation. 2004;110:2168

Relationship of NT-proBNP and Cardiovascular Events (A post hoc analysis of PARADIGM-HF Study)

Reduction in NT-proBNP Following HF Treatment is Associated with Reduction in CV Death and HF hospitalization (Post hoc Analysis)



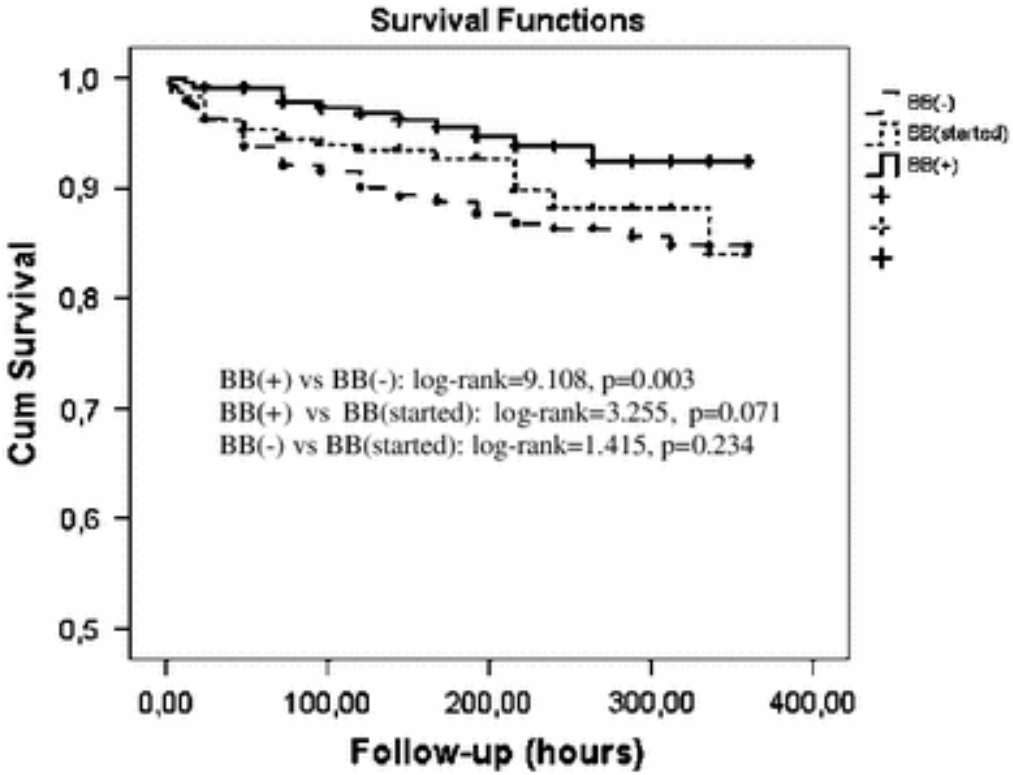
Achieving levels of NT-proBNP <1000 as early as 1 month after randomization to HF therapy was associated with a significant reduction in risk of CV death or first HF hospitalization

This was a post hoc analysis of PARADIGM-HF Study. Analytic variability (imprecision of the test) and biological variability (expected variability within the subject over time) may influence the accuracy of a predictive value of a change in biomarkers. The change from baseline data should therefore be interpreted in light of the influence of the biological variability known to be present in HFrEF patients.
NT-proBNP N-terminal pro–brain natriuretic peptide. HF, Heart Failure. CV, Cardiovascular

233.Zile MR, et al. *J Am Coll Cardiol.* 2016;68(22):2425–2436.

In-hospital survival of patients with AHF and COPD according to BB therapy status

BB (+) vs (−) before admission
BB started during index hospitalization



Parissis, J.T., Andreoli, C., Kadoglou, N. et al. Clin Res Cardiol (2014) 103: 733.

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Impact of COPD on the Mortality and Treatment of Patients Hospitalized With Acute Decompensated Heart Failure

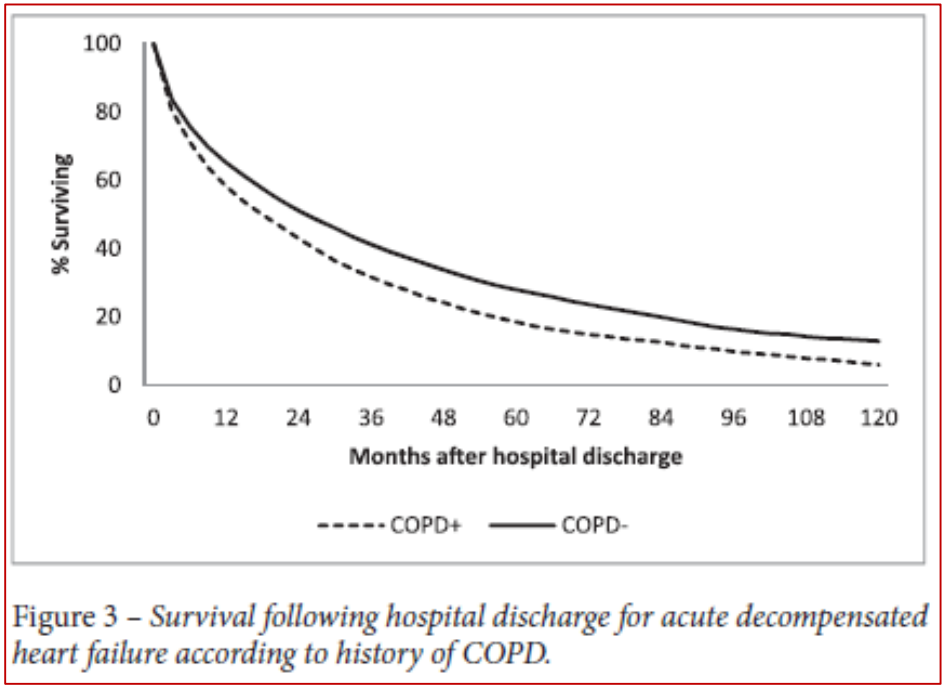

Figure 3 – *Survival following hospital discharge for acute decompensated heart failure according to history of COPD.*

- In-hospital death similar for patients with and without COPD.
- Patients with COPD had higher 1- & 5 year mortality after discharge

TABLE 2] Heart Failure-Specific Discharge Medications in Patients Hospitalized With Acute Decompensated Heart Failure According to History of COPD

Medication	With COPD (n = 3,223)	Without COPD (n = 5,821)	*P* Value
ACE-I/ARB	1,596 (49.5)	3,157 (54.2)	<.001
β-Blockers	1,271 (39.4)	3,251 (55.9)	<.001
Diuretics	2,701 (83.8)	4,676 (80.3)	<.001
Nitrates and hydralazine	36 (1.1)	48 (0.82)	.17

Fisher, K , CHEST 2015; 147 (3): 637 - 645

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The EuroHeart Failure Survey programme - Predictors of under-prescription of beta-blockers

Beta-blockers	
Factor	
Respiratory/pulmonary disease	0.35 (0.30 to 0.40)
Speciality at admission (Cardiology vs GIM, for being Cardiology)	2.69 (2.37 to 3.31)
IHD	2.63 (2.32 to 2.99)
Age group (>70)	0.55 (0.49 to 0.61)
Gender (being male)	1.16 (1.05 to 1.29)

Komajda M, et al. Eur Heart J 2003;24:464–474

Heart Failure and Chronic Obstructive Pulmonary Disease

The Quandary of Beta-Blockers and Beta-Agonists

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Table 1 Properties of Beta-Blockers Approved for the Treatment of HF

Beta-Blocker	$Beta_1$-Selectivity (Ref. #)	Alpha-Antagonism	Intrinsic Sympathomimetic Activity	Lipid Solubility	Route of Elimination	Half-Life (h)
Carvedilol (53)	1	+	—	Moderate	Hepatic	7–10
Metoprolol tartrate (54)	40	—	—	Moderate	Hepatic	3–7
Metoprolol succinate (57)	40*	—	—	Moderate	Hepatic	20
Bisoprolol (55)	75	—	—	Low	Hepatic/Renal	10–12
Nebivolol (56)	>300	—	—	High	Hepatic	12–19

Dashes indicate that the property is not present. *The clinical cardioselectivity of metoprolol succinate controlled release/extended release is much higher than that of metoprolol tartrate because of the even beta-blockade achieved with this formulation avoiding peaks and troughs (57).
HF = heart failure.

JACC Vol. 57, No. 21, 2011
May 24, 2011:2127–38

Bisoprolol in patients with HF and moderate to severe COPD: a randomized controlled trial

Table 3 Effect of bisoprolol on pulmonary function

Mean ± SD	Bisoprolol ($n = 14$)	Placebo ($n = 13$)
FEV_1 (L)		
Baseline	1.37 ± 0.42	1.26 ± 0.42
Change	−0.07 ± 0.08*	0.12 ± 0.21
FEV_1 reversibility (L)		
Baseline	0.11 ± 0.08	0.22 ± 0.19
Change	−0.03 ± 0.09	−0.10 ± 0.08
FEV_1 post-salbutamol (L)		
Baseline	1.48 ± 0.40	1.48 ± 0.50
Change	−0.09 ± 0.10*	0.02 ± 0.15
Peak expiratory flow (L/min)		
Baseline	209 ± 60	216 ± 75
Change	−13 ± 17	12 ± 40
Vital capacity (L)		
Baseline	2.66 ± 0.91	2.56 ± 0.74
Change	−0.07 ± 0.19	0.10 ± 0.30
Residual volume (L)		
Baseline	2.85 ± 0.94	3.21 ± 1.09
Percent predicted normal value (%)	118 ± 29	138 ± 44
Change	0.06 ± 0.56	−0.04 ± 0.51
Total lung capacity (L)		
Baseline	5.51 ± 1.35	5.83 ± 1.25
Change	−0.01 ± 0.57	−0.02 ± 0.54

*$P < 0.05$ compared with placebo.

Table 4 Effect of bisoprolol on health status[a]

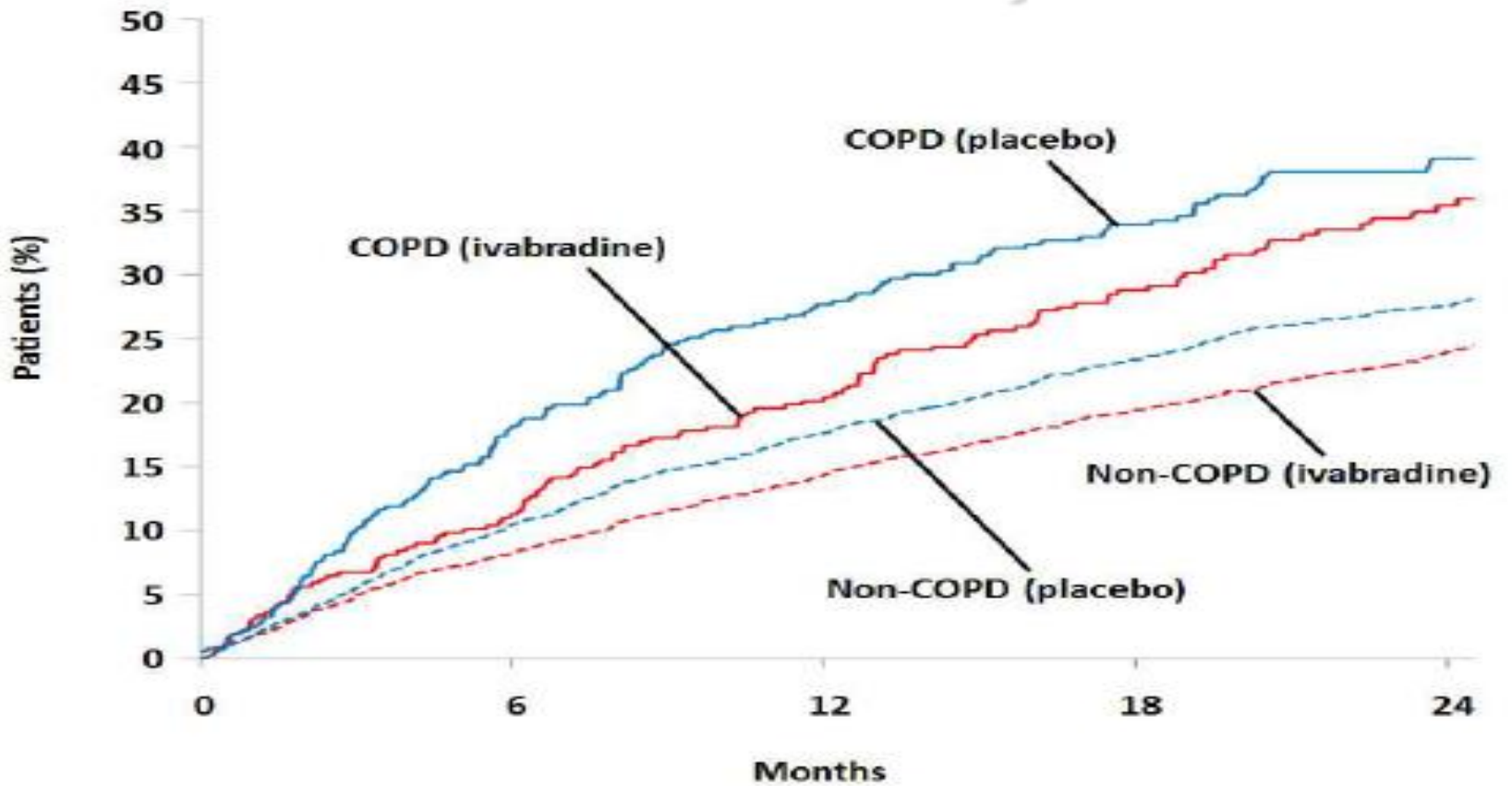
Mean ± SD	Bisoprolol ($n = 14$)	Placebo ($n = 13$)
SF-36 PCS		
Baseline	31.7 ± 7.5	32.6 ± 8.1
Change	2.6 ± 5.4	0.5 ± 4.5
SF-36 MCS		
Baseline	42.3 ± 9.9	38.6 ± 11.8
Change	0.8 ± 6.0	−0.3 ± 9.5
MLHFQ		
Baseline	49.4 ± 25.5	47.4 ± 21.0
Change	−2.5 ± 12.3	3.5 ± 11.1
CRQ total		
Baseline	3.94 ± 0.85	3.75 ± 0.95
Change	0.07 ± 0.64	−0.24 ± 0.68
CRQ dyspnoea		
Baseline	2.57 ± 0.81	3.08 ± 1.22
Change	0.51 ± 1.19	−0.14 ± 1.27

[a]For all scales, except MLHFQ, a positive change equates to an improvement; for MLHFQ, a negative change equates to improvement. SF-36, Short Form 36; PCS, physical component score; MCS, mental component score; MLHFQ, Minnesota Living with Heart Failure Questionnaire; CRQ, Chronic Respiratory Questionnaire.

Initiation of bisoprolol in patients with HF and concomitant moderate or severe COPD resulted in a mild reduction in FEV1. However, symptoms and quality of life were not impaired

Hawkins N. Eur J Heart Fail 2009;11:684–690

Effect of ivabradine on composite of CV death or HF hospitalization



Number at risk

COPD (placebo)	372	298	250	209	110
COPD (ivabradine)	358	312	266	216	124
Non-COPD (placebo)	2892	2570	2239	1852	979
Non-COPD (ivabradine)	2883	2616	2334	1957	1067

Tavazzi L, et al. *Eur Heart J*. 2013;34 (Abs. Suppl), 652

Limitations of drugs used for the treatment of COPD in HF patients

- Short-acting beta-2 adrenergic bronchodilators can cause tachycardia and increase myocardial oxygen consumption- These agents may increase risk of MI, mortality and hospitalizations (no efficient prospective data)
- Slow-acting agents (b2 agonists, anticholinergics,) may be an adequate alternative although there are not prospective mortality data.
- Oral steroids lead to water retention and increase risk of HF exacerbations- Inhaled steroids may be safer.
- Theophylline products may increase risk of arrhythmias

Rutten FH, et al. Eur J Heart Fail 2006;8:706-711

Anti-cholinergics and cardiac outcomes in COPD

N Engl J Med. 2008;359:1543–1554.

	UPLIFT[16]	
	TIO	PLAC
Enrolled	2987	3006
Withdrew	1099 (37%)	1358 (45%)
Duration	4 years	
Age	64	64
% men	75	74
Post-bronch FEV_1	47	47
Smokers	29	30
Smoking history (pack-year)	49	48
SGRQ	45.7	46.0
On ICS	62	62
All cause mortality	14.9%	16.5%
Cardiac death	26 (0.9%)	32 (1.1)

Take Home messages

- COPD is a frequent comorbidity in AHF with diagnostic and therapeutic implications
- Natriuretic peptides are used to discriminate dyspnea of cardiac vs non-cardiac origin
- Acute imaging with lung ultrasound complements NP testing, speeding up diagnosis
- Acute HF therapies are applied as in all HF patients
- Caution with high dose diuretics
- Acute inhaled respiratory therapies as needed but caution
- Selective b-blockers or ivabradine upon discharge