Why is My Patient Short of Breath; What am I Going to do About It?

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Conflict of Interest Statement

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Past Chair, CPFM Respiratory Medicine, CFPC

Medical Director, Central LHIN COPD Education Clinic

Regional Primary Care Lead, for the Central Regional Cancer Program.

Perceive no conflict of interest with giving this presentation, but present the following companies that I have worked with or consulted for:

Astra Zeneca, Behring Boehringer Ingeleheim, Cipla Covis, Eisai, GSK, Merck Frosst, Novartis, Pfizer, Sanofi Genzyme, Teva, Trudel and Valeo.

In addition, I am on Health Canada committee for Section of Allergy and Respiratory Therapeutics

Disclosure of Financial Support

- **Grant provided to the FPAGC by Boehringer Ingelheim to create this talk in 2020, in a hands-off fashion**

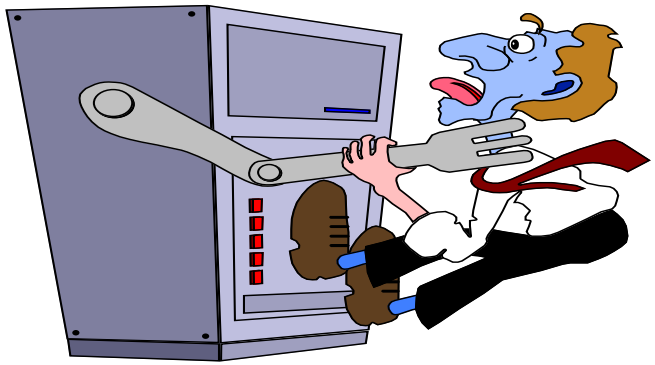
- **<u>Potential for conflict(s) of interest</u>:**
- **Products made for Asthma, COPD DVT, PE and CHF are made by multiple companies that the speaker may have a relationship with**

Mitigating Potential Bias

- Emphasis will be on the illnesses described and where relevant, on drug classes only, not individual company medications
- I was careful to be inclusive of multiple drugs in drug classes and mostly discussed medication classes and indications.

GOALS

- Define causes of dyspnea
- Define work up for a dyspneic patient
- Review COPD and CHF and how to manage when both are present in the same patient
- Review the management of thromboembolic disease
- Review therapeutic options for these conditions causing dyspnea



Part I:
Dyspnea The Basics

Dyspnea – from Latin ‘dyspnoea’

- Dyspnea (also SOB, **air hunger**)
- Subjective symptom of *breathlessness*.
- Normal in heavy exertion
- Pathological if it occurs in unexpected situations.

Take Dyspnea seriously!!

Dyspnoea upon hospital admission: listen to the bird of ill omen!

Maxens Decavèle, Thomas Similowski

European Respiratory Journal 2021 58: 2100988; **DOI:** 10.1183/13993003.00988-2021

Article | Figures & Data | Info & Metrics | PDF

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Extract

Dyspnoea, namely a patient's complaint of difficult, disturbing or distressing breathing, is a symptom that prompts physicians to undertake diagnostic and therapeutic procedures in order to identify and correct causative pathophysiological abnormalities. Dyspnoea can become self-perpetuating (a syndrome) when it persists despite mechanistic treatments. It then justifies symptomatic management to alleviate suffering [1, 2]. Above all, dyspnoea is an experience that changes patients' lives and requires holistic approaches [3]. In this issue of the *European Respiratory Journal*, STEVENS *et al.* [4], from a group that has achieved outstanding progress in the understanding of dyspnoea [5], spotlight yet another aspect of dyspnoea. They highlight its value as a very generic warning sign of impending doom: in unselected patients admitted to hospital, the "mere fact" of reporting dyspnoea is associated with an increased risk of hospital mortality. This risk increases with increasing intensity of dyspnoea.

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What is respiratory distress?

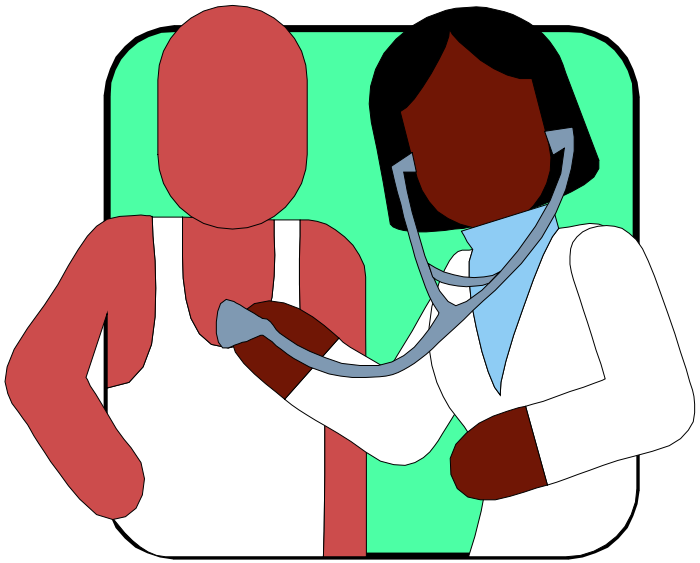
- Vague term meaning “not breathing well”. A constellation of signs including:
  - Using accessory muscles of respiration
  - Tachypnea
  - Gasping
  - Panting
  - Restlessness
  - Sometimes, also confusion (hypoxemia)
  - Somnolence (hypercarbia)

Case

- John is a 75 year old man with longstanding dyspnea
- **Worsening the last 3 months**
- Past smoker, forty pack years
- Quit three years ago with MI
- Occasional cough
- Remote PE at age 30 with a meniscectomy of his knee, anticoagulated for six months
- Meds: ASA, Betablocker, ACE, Statin, Antidepressant

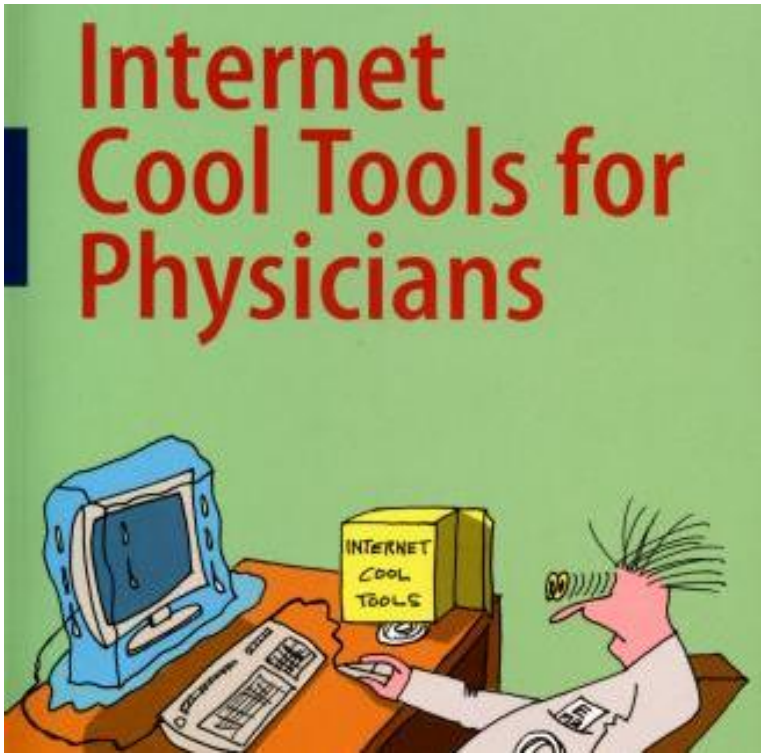
Tools to evaluate dyspnea

- Suspicion / Clinical knowledge. “If you don’t think of it, you will never find it.”
- History
- Physical Exam including
  - Vital Signs, pulse ox, PEF, Pulsus paradoxicus
- Formal Studies



What other tools?

- Spirometry
- ABG (acute presentations)
- Other blood tests
- CXR
- EKG
- CT
- Echocardiogram



Vital Signs

- What are the baseline VS?
- Normal vs Stable
- How do they change over time?
- What does this tell you?
- Pulsus Paradoxicus

Causes of Dyspnea

- Four General Categories:
  - Cardiac
  - Pulmonary
  - Mixed cardiac and pulmonary
  - Non-cardiac, non-pulmonary

Common specific disease entities

Metabolic

- Anemia
- Hypothyroidism
- Hyperthyroidism
- Metabolic acidosis

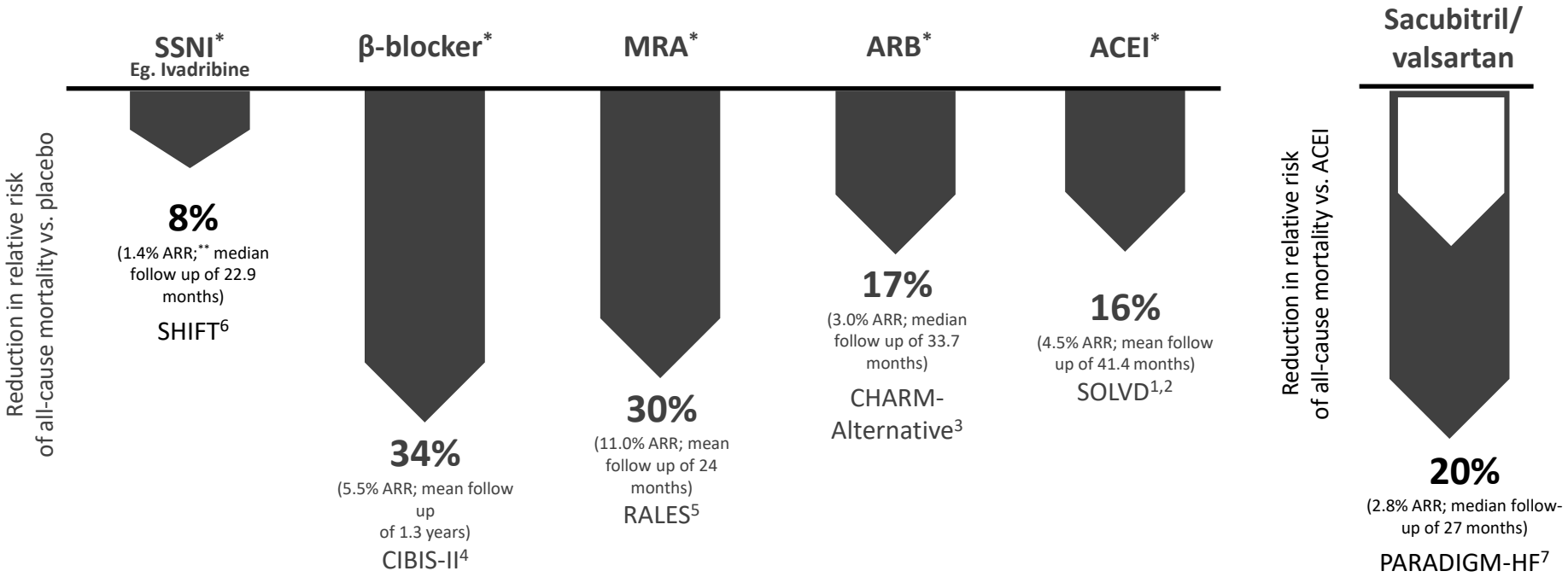
Other

- Mechanical obstruction
- Chest wall
- Psychogenic
- Central (brain related)

Part II:
COPD and CHF

Treatment of CHF

Pharmacotherapy: Therapies that Improve Overall Survival



ACEI=angiotensin-converting-enzyme inhibitor; ARB=angiotensin receptor blocker; ARNI=angiotensin II receptor blocker neprilysin inhibitor; HF=heart failure; HFrEF=heart failure with reduced ejection fraction; LVEF=left ventricular ejection fraction; MRA=mineralocorticoid receptor antagonist; SSNI = selective sinus nod inhibitor

*On top of standard therapy at the time of the study (except in CHARM-Alternative where background ACEI therapy was excluded) patient populations varied between trials and as such relative risk reductions cannot be directly compared. SOLVD (Studies of Left Ventricular Dysfunction), CIBIS-II (Cardiac Insufficiency Bisoprolol Study II) and RALES (Randomized Aldactone Evaluation Study) enrolled chronic HF patients with LVEF≤35%. CHARM-Alternative (Candesartan in Heart failure: Assessment of Reduction in Mortality and Morbidity) enrolled chronic HF patients with LVEF≤40%; SHIFT (Systolic Heart failure treatment with the If inhibitor Ivabradine Trial) enrolled patients with chronic moderate to severe HF and LVEF≤35%; PARADIGM-HF (Prospective comparison of ARNI with ACEI to Determine Impact on Global Mortality & morbidity in HF) enrolled chronic HF patients with LVEF≤40% (changed to LVEF≤35% by protocol amendment in December 2010); **Not statistically different from placebo

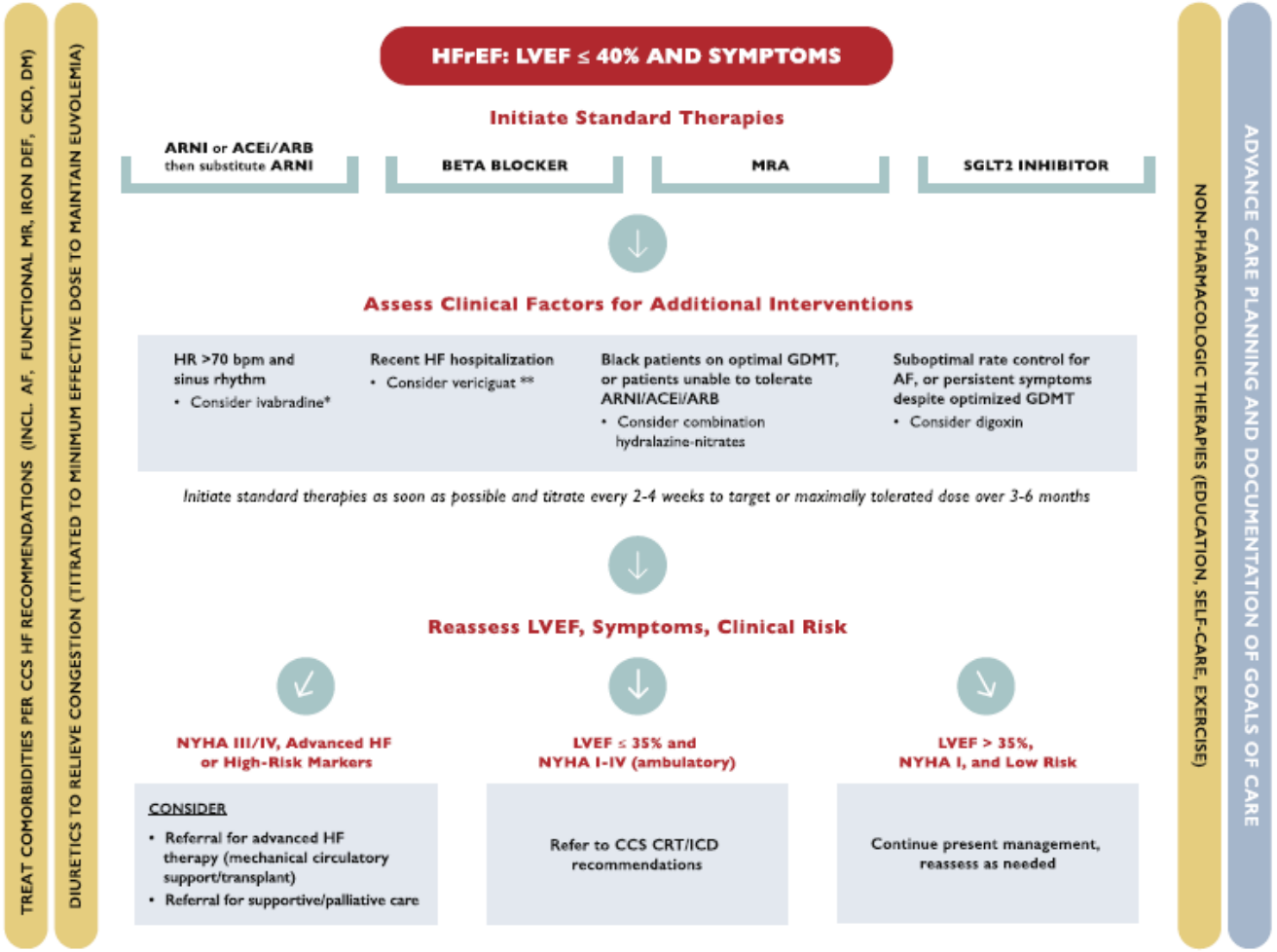
1. McMurray *et al. Eur Heart J* 2012; 33:1787-847. 2. SOLVD Investigators. *N Engl J Med* 1991; 325:293-302. 3. Granger *et al. Lancet* 2003; 362:772-6. 4. CIBIS-II Investigators. *Lancet* 1999; 353:9-13. 5. *Pitt et al. N Engl J Med* 1999; 341:709-17. 6. Swedberg *et al. Lancet* 2010; 376:875-85. 7. McMurray *et al. N Engl J Med* 2014; 371:993-1004.

ELSEVIER

Canadian Journal of Cardiology 37 (2021) 531–546

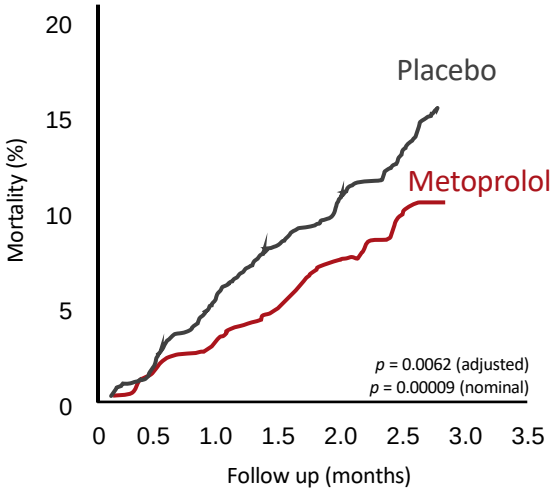
Society Guidelines

CCS/CHFS Heart Failure Guidelines Update: Defining a New Pharmacologic Standard of Care for Heart Failure With Reduced Ejection Fraction

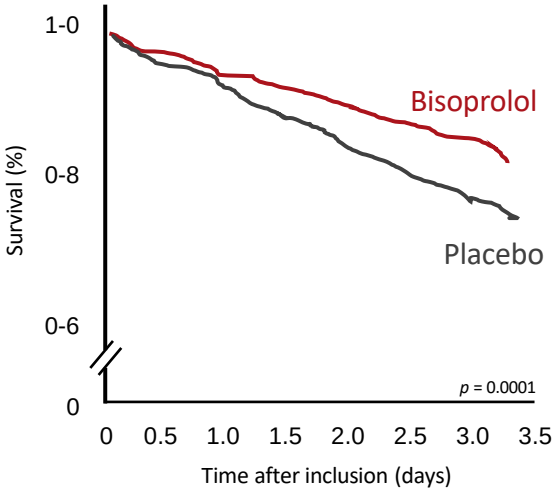


Key Evidence for ß-blockers in HF

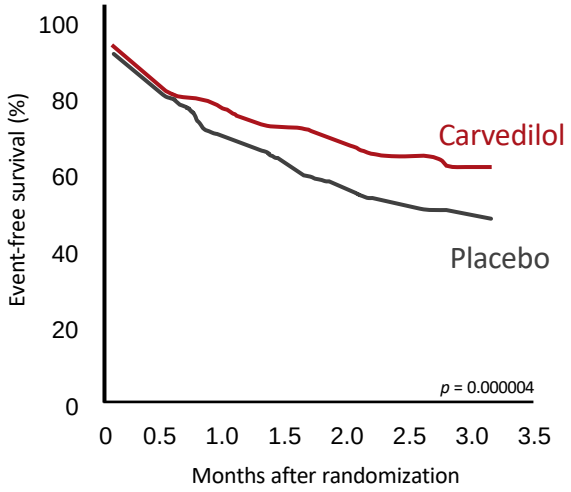
MERIT-HF
Cumulative percentage of total mortality*



CIBIS II
Survival curves**



COPERNICUS
Time to death or hospitalization***



*MERIT-HF Study Group. *Lancet* 1999; 353:2001-7.
**CIBIS II Investigators. *Lancet* 1999; 353:9-13.
*Arnold JM et al. *Can J Cardiol* **Packer M *et al. Circulation* 2002; 106:2194-9.
2006; 22(1):23-45.

How to start B-blockers?

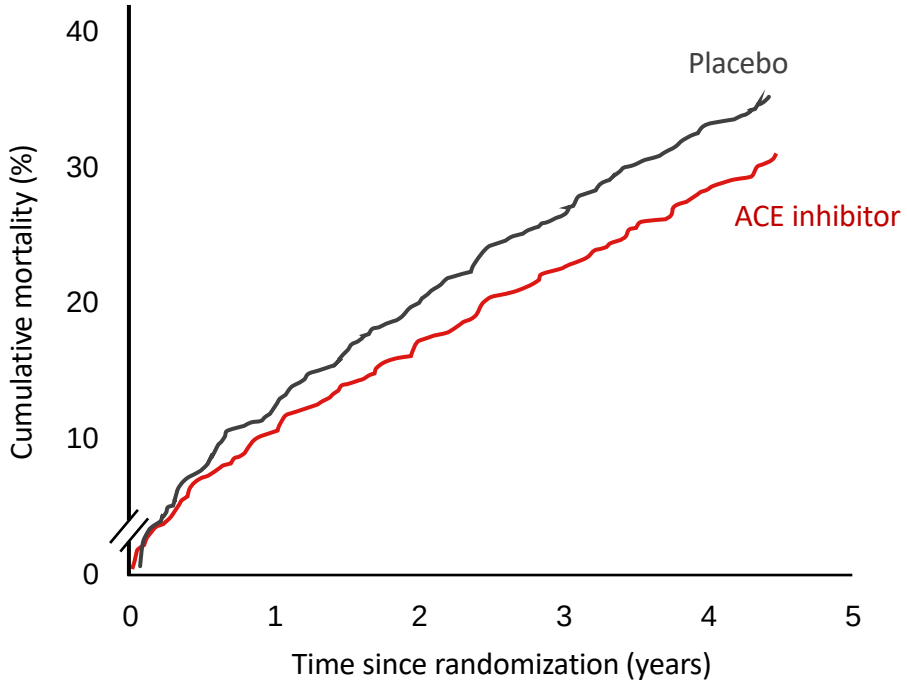
Initiating Beta-Blockade for HF in the Community Setting

- Beta-blockers can be initiated together with ACEIs at low doses, but increasing at not more often than every 2 weeks[a]
- Because they slow the heart to allow it to pump more efficiently, it is important to ensure the heart rate does not drop too quickly
- A "start low, go slow" strategy should be used[a]
- Start with a low dose and increase not more often than every 2 weeks[a]
- Monitor HF, blood pressure, and clinical status[a]
- Beta-blockers (cardioselective) should not be withheld because of age or in the presence of PVD, erectile dysfunction, diabetes, interstitial lung disease, or COPD[b]

a. NICE Guidelines. Chronic heart failure in adults; b. Ponikowski P, et al. *Eur Heart J*. 2016;18:891-975.

Clinical Effects of ACEIs on HF

Overview of Five Trials
(SAVE, AIRE, TRACE, SOLVD prevention, SOLVD treatment)



Large, prospective, randomized trials have consistently demonstrated a significant reduction in mortality

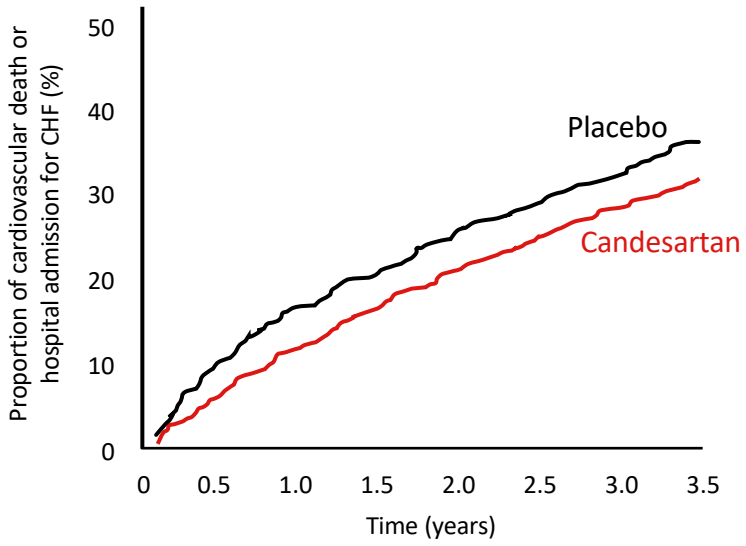
OVERALL, ACE INHIBITORS REDUCED RISK OF DEATH BY 20%
(p <0.0001)

ACE=angiotensin-converting-enzyme
Flather MD *et al. Lancet* 2000; 355(9215):1575-81.

Key Evidence for ARBs in HF

CHARM

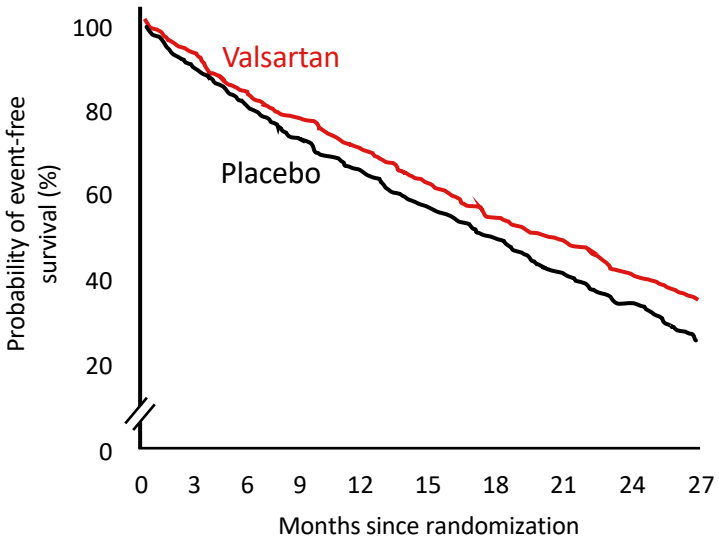
Proportion of patients with CV death or hospital admission for CHF*



Val-HeFT

Probability of freedom from combined endpoint**

(All-cause mortality, cardiac arrest with resuscitation, hospitalization for worsening HF, or therapy with intravenous inotropes or vasodilators)



*Pfeffer MA *et al. Lancet* 2003; 363:759-66.
**Cohn JN *et al. N Engl J Med* 2001; 345:1667-75.

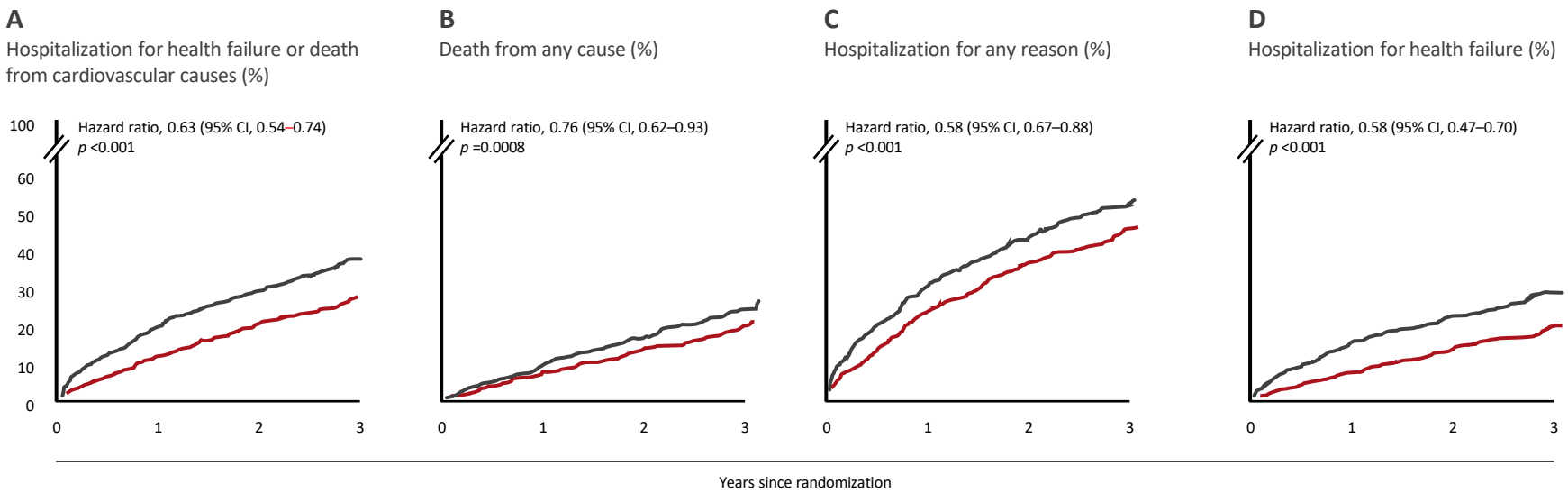
How to Start an ACE/ARB?

Initiating ACEI/ARB for HF in a Community Setting

- Start ACEI therapy at low dose and titrate upward not more than every 2 weeks
- If patients are tolerating this well and there is no symptomatic hypotension, continue uptitration
- Kidney function, including serum electrolytes, should be checked
  - Prior to treatment initiation
  - 1-2 weeks after the drug is started
  - After any dose titration to monitor for kidney dysfunction
- Once the ACEI/ARB target or maximum dose is reached
  - Monitor monthly for 3 months, then every 6 months and at any time the person becomes acutely unwell

NICE Guidelines. Chronic heart failure in adults.

EMPHASIS-HF: MRAs in HF: NYHA class II heart failure with Ejection Fraction ≤ 35%



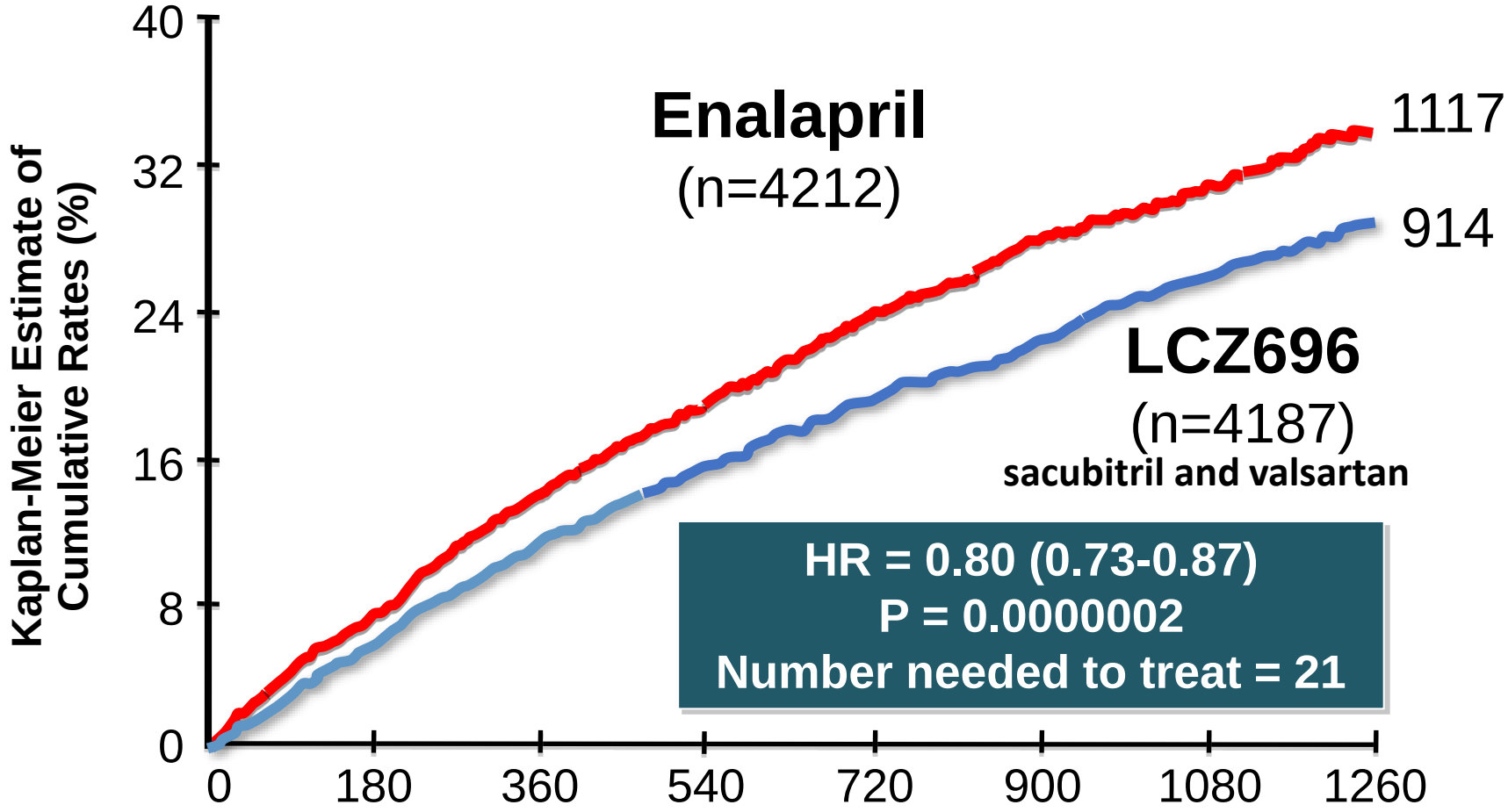
No. at risk

Placebo	1373	848	512	199	1373	947	587	242	1373	742	403	146	1373	848	512	199
Eplerenone	1364	925	562	232	1364	972	625	269	1364	795	451	179	1364	925	562	232

Placebo
Eplerenone

Zannad F *et al. N Engl J Med* 2011; 364(1):11-21.

PARADIGM-HF: Cardiovascular Death or Heart Failure Hospitalization (Primary Endpoint)



Patients at Risk

LCZ696	4187	3922	3663	3018	2257	1544	896	249
Enalapril	4212	3883	3579	2922	2123	1488	853	236

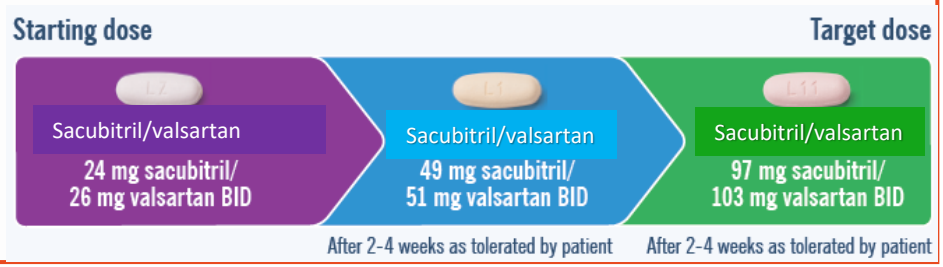
Initiating and Titrating Sacubitril/Valsartan to Target Dose

Stop ACE/ARB inhibitor therapy for a 36-hour washout

Sacubitril/valsartan must not be started until 36 hours have passed following discontinuation of ACE inhibitor therapy

Patients with:

- Prior ACE inhibitor or ARB at less than guideline-recommended doses
- Risk for hypotension (≥75 years old, low SBP)
- Moderate hepatic impairment (Child-Pugh B)



Patients with:

- Prior ACE inhibitor or ARB at guideline-recommended doses

Sacubitril/valsartan should only be initiated in clinically stable patients whose baseline systolic blood pressure, serum potassium and renal function are at acceptable levels.
If patients experience tolerability issues, e.g., symptomatic hypotension or hyperkalemia, consideration should be given to temporary down-titration or treatment interruption of sacubitril/valsartan

Practical Tips: Co-managing a Patient on Sacubitril/Valsartan

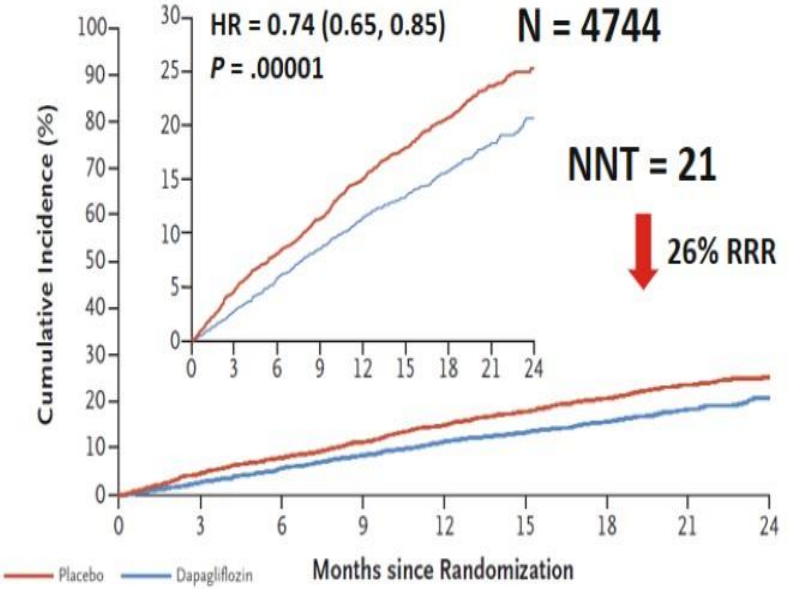
	**Replace ACE or ARB in the management of HFrEF**	*Leverage both suppression of RAAS pathway and increased NP levels*
	**Do watch K/Cr**	• *Cut down if hyperkalemia or significant rise in Cr occurs* • *Recall that with ACE/ARB/diuretic or sac/val – ↑ Cr of 30% is acceptable**
	**Do follow BP**	*Anticipate BP lowering effect*
	**Do NOT start an ACE when already on Sac/val**	*Risk of angioedema with combined use*
	**Do NOT start an ARB when already on Sac/val**	*Redundant, as there is ARB already in it*

*Howlett JG. The Canadian Cardiovascular Society HF companion; *Can J Cardiol* 2015; 1-15.
Individual product monographs.

DAPA-HF Trial
Design and Main Results

- **Key inclusion criteria:**
  Symptomatic HF; EF ≤ 40%; NT-proBNP ≥ 600 pg/mL (if hospitalized for HF within last 12 months, ≥ 400 pg/mL; if Afib, ≥ 900 pg/mL)

Results: Dapagliflozin significantly reduces the primary endpoint of CV death or worsening of HF (HHF or urgent HF visit)

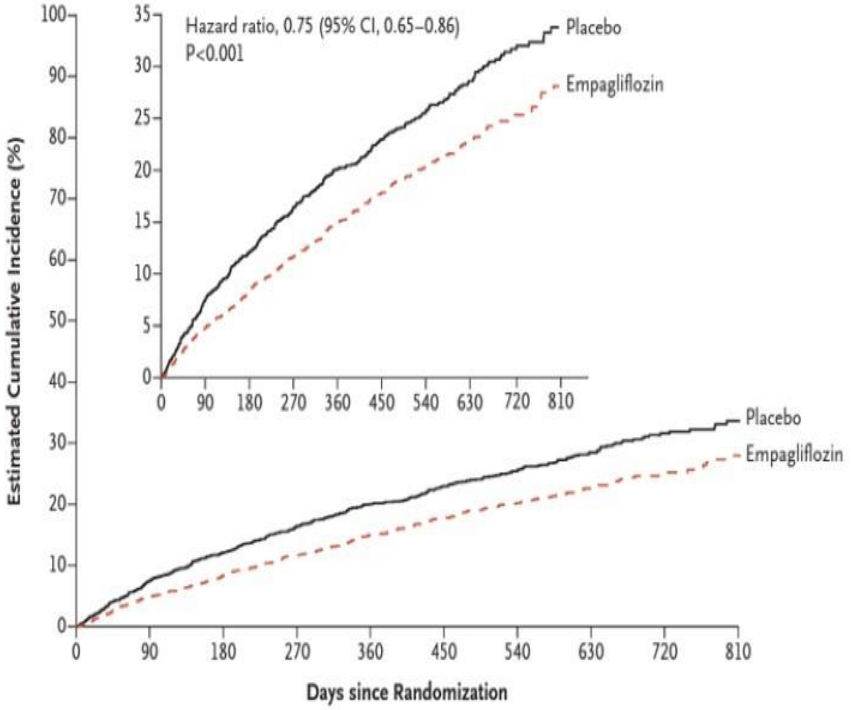


No. at Risk									
Placebo	2371	2258	2163	2075	1917	1478	1096	593	210
Dapagliflozin	2373	2305	2221	2147	2002	1560	1146	612	210

McMurray JJ, et al. *N Engl J Med.* 2019;381:1995-2008; McMurray JJ, et al. *Eur J Heart Fail.* 2019;21:665-675.

EMPEROR-Reduced Primary Endpoint

Results: Empagliflozin significantly reduced the primary endpoint (CV death or HHF)



RRR 25%

ARR 5.2%

NNT = 19

Packer M, et al. *N Engl J Med.* 2020;383:1413-1424.

What about heart failure with PRESERVED Ejection Fraction (Ej Fr>40%)

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 OCTOBER 14, 2021 VOL. 385 NO. 16

Empagliflozin in Heart Failure with a Preserved Ejection Fraction

S.D. Anker, J. Butler, G. Filippatos, J.P. Ferreira, E. Bocchi, M. Böhm, H.-P. Brunner–La Rocca, D.-J. Choi, V. Chopra, E. Chuquiure-Valenzuela, N. Giannetti, J.E. Gomez-Mesa, S. Janssens, J.L. Januzzi, J.R. Gonzalez-Juanatey, B. Merkely, S.J. Nicholls, S.V. Perrone, I.L. Piña, P. Ponikowski, M. Senni, D. Sim, J. Spinar, I. Squire, S. Taddei, H. Tsutsui, S. Verma, D. Vinereanu, J. Zhang, P. Carson, C.S.P. Lam, N. Marx, C. Zeller, N. Sattar, W. Jamal, S. Schnaidt, J.M. Schnee, M. Brueckmann, S.J. Pocock, F. Zannad, and M. Packer, for the EMPEROR-Preserved Trial Investigators*

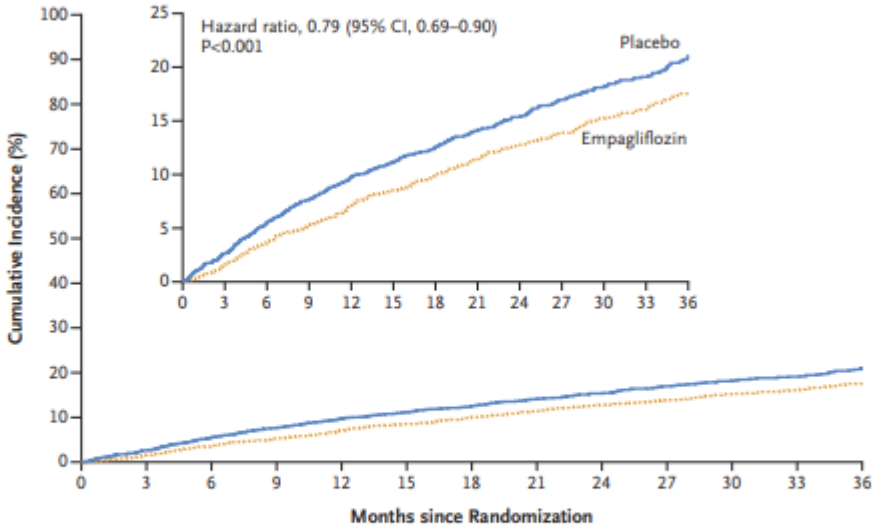


Figure 1. Primary Outcome, a Composite of Cardiovascular Death or Hospitalization for Heart Failure.

The estimated cumulative incidence of the primary outcome in the two groups is shown. The inset shows the same data on an expanded y axis.

Cardiology Societies Guidelines Regarding the Use of SGLT2 Inhibitors in Patients With T2D

2019 ACC/AHA Guideline on the Primary Prevention of Cardiovascular Disease[a]

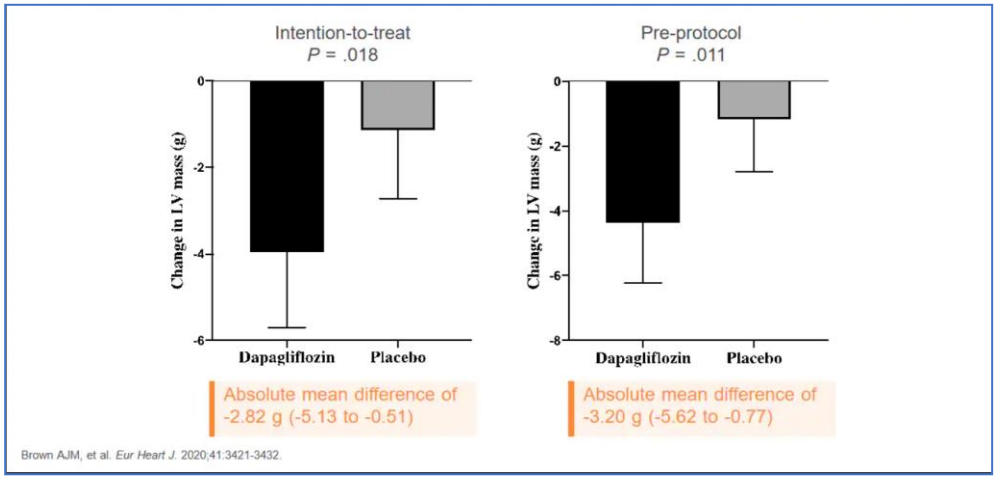
"SGLT2 inhibitor or GLP-1 RA as an early add-on to metformin in patients with T2D and CV risk factors ***for primary prevention of CVD****."*

2019 ESC Guidelines on diabetes, pre-diabetes, and cardiovascular diseases developed in collaboration with the EASD[b]

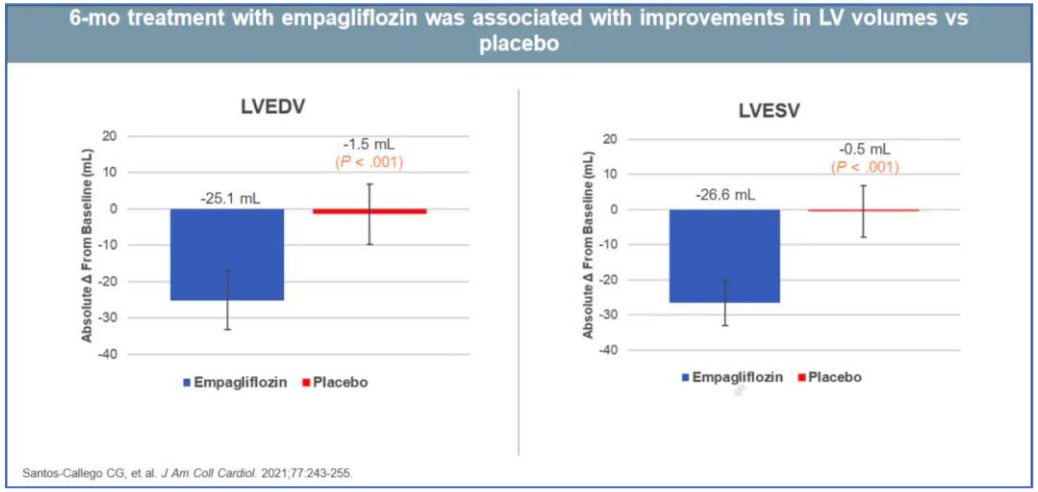
"For the first time, we have evidence from several CVOTs that indicate ***CV benefits*** *from the use of SGLT2 inhibitors and GLP1-RAs in patients with* ***CVD, or at very high/high CV risk****"*

a. Arnett DK, et al. *J Am Coll Cardiol* 2019;74:1376; b. Cosentino F, et al. *Eur Heart J.* 2020;41:255.

Mechanism of improved heart failure results?



DAPA-LVH
Effect on LV Mass (Primary Endpoint)



EMPA-TROPISM
Principal Findings

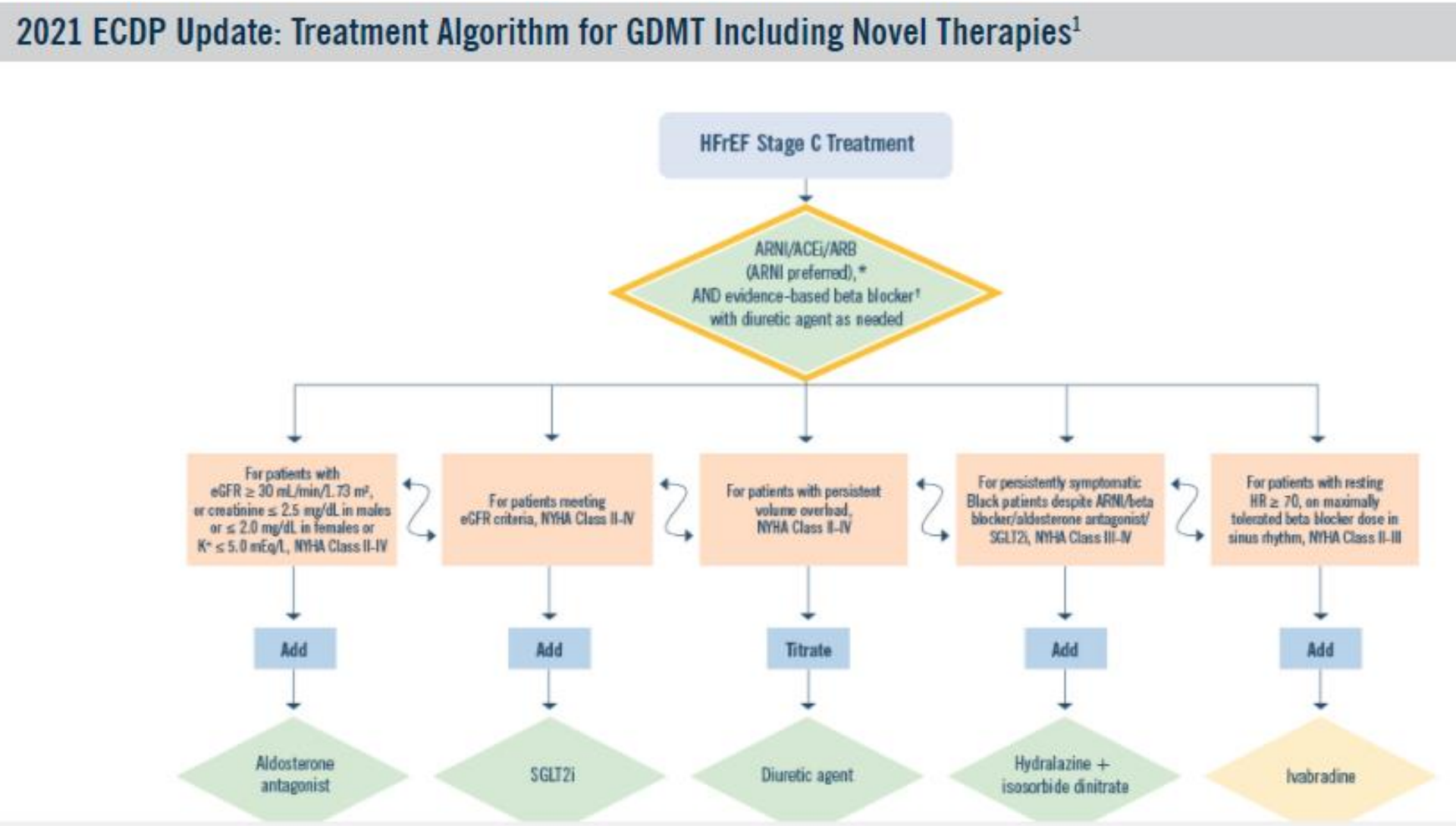
Significant Reversal of LV remodelling!

Summary: Heart Failure with Reduced Ejection Fraction Activates…

Neurohormonal System	Effect on HF	Target Medication Classes	Effect
Sympathetic	Worsens	Beta blockers	Reduces sympathetic overdrive
RAAS	Worsens	ACEI ARB	Blocks RAAS
Natriuretic peptide system	Improves	Neprilysin Inhibitor	Augments NPS
Other	Diuretic effect? Sodium Hydrogen exchange Ketones as metabolic substrate for the heart!	SGLT2 inhibitor	Decreases mortality Preserves renal function

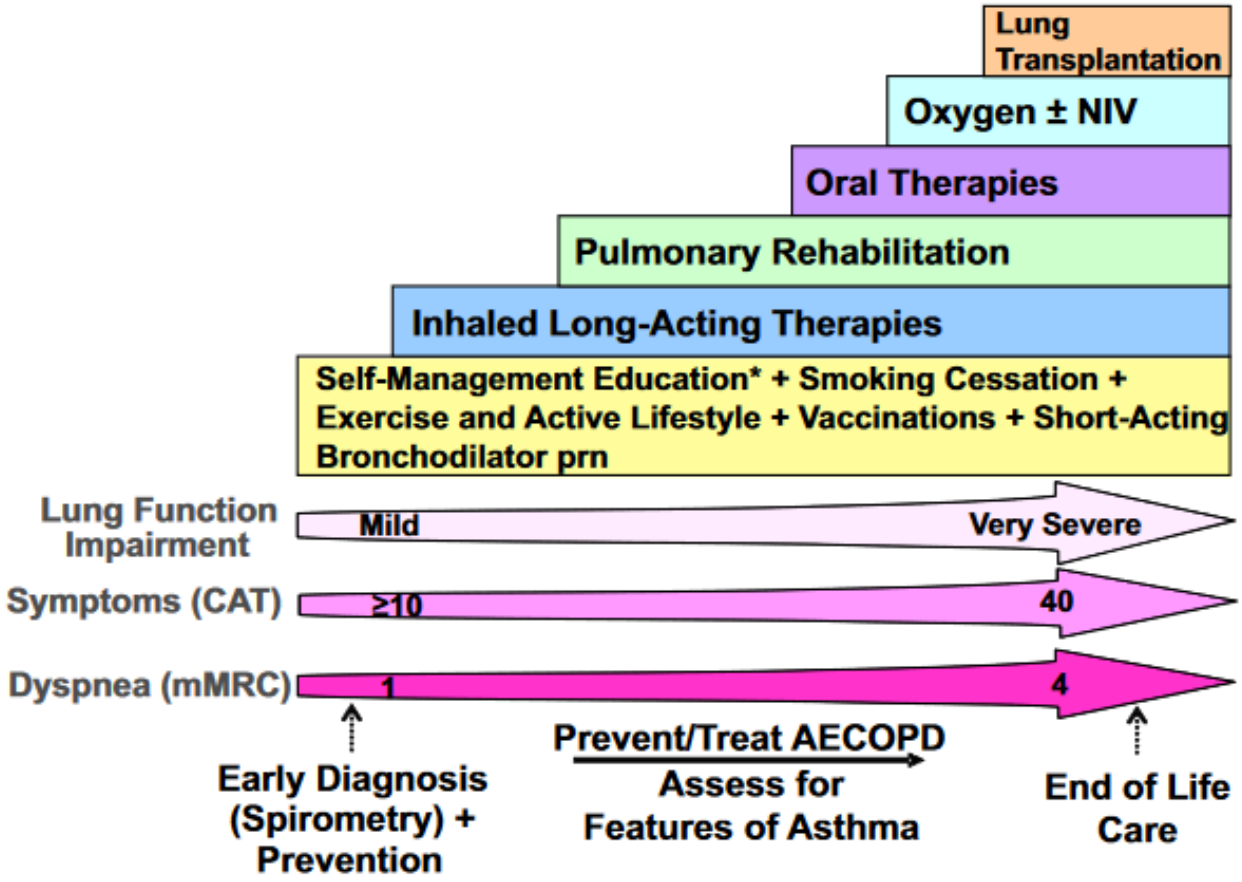
Note
– NEP inhibitor must be combined with RAAS inhibition
– RAAS inhibition must be done via ARB (increase angioedema if NEP inhibitor + ACE)

And there are further steps, getting a bit complicated, though….



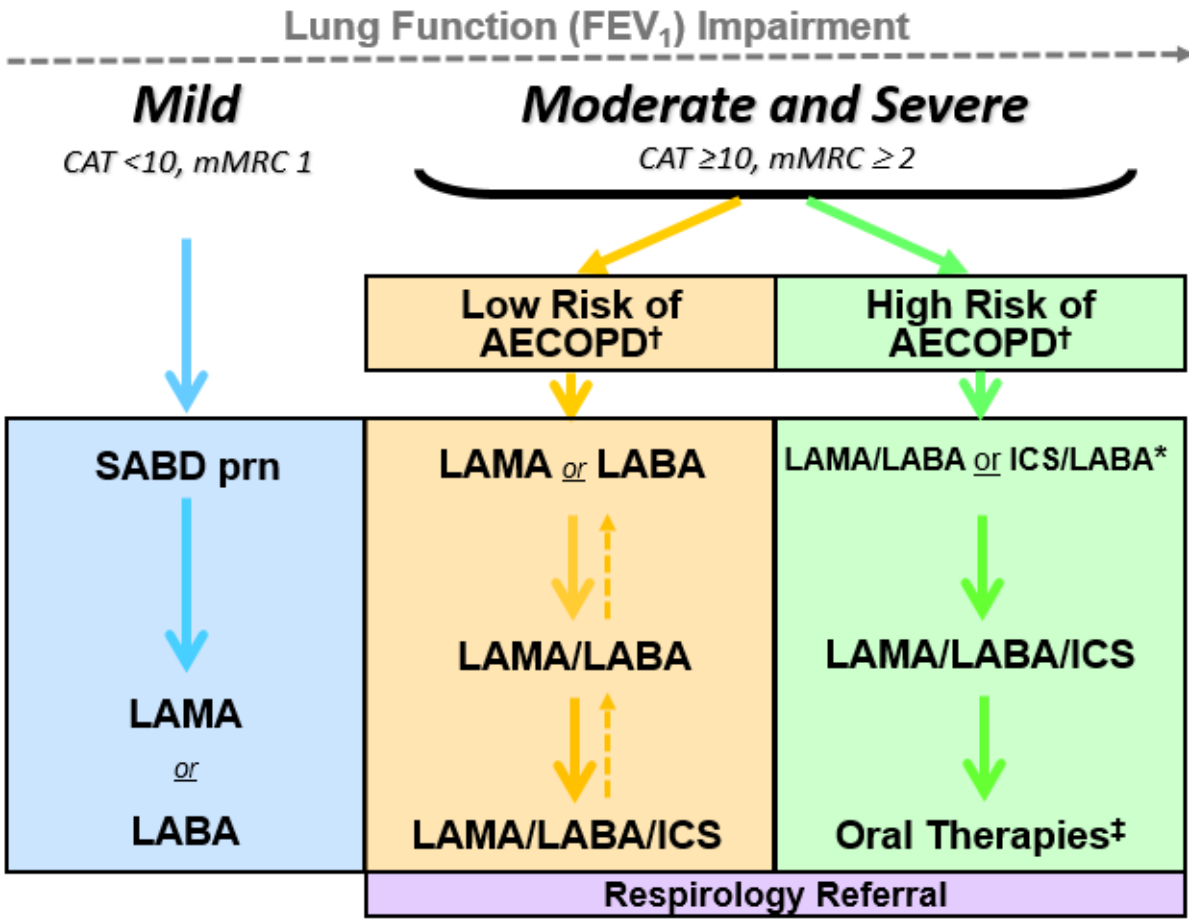
Treatment of COPD

Comprehensive Management of COPD CTS 2019 Update



Bourbeau J et al. CTS position statement: Pharmacotherapy in patients with COPD - An update. Canadian Journal of Respiratory, Critical Care, and Sleep Medicine 1(4): 222-241; Oct 2019; Figure 1

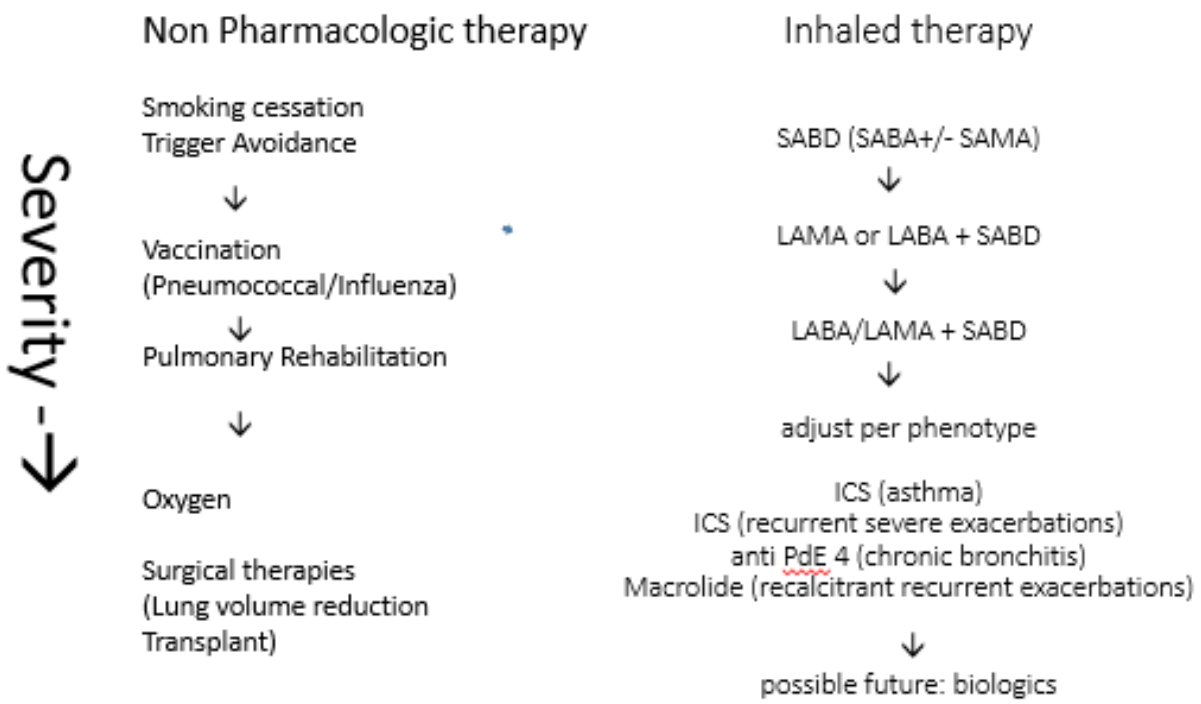
2019 CTS Position Statement on COPD Pharmacotherapy



Bourbeau, J., et al, Canadian Thoracic Society Clinical Practice Guideline on pharmacotherapy in patients with COPD – 2019 update of evidence, CJRCCSM, 3.4, 210-232

Easier approach?

Kaplan A. What is new in COPD in 2017?, Canadian Jounral of Geriatrics



COPD vs CHF


COPD
CHRONIC AIRFLOW LIMITATION
• Easily Fatigued
• Frequent Respiratory Infections
• Use of Accessory Muscles to Breathe
• Orthopneic
• Wheezing
• Pursed-Lip Breathing
• Chronic Cough
• Barrel Chest
• Dyspnea
• Prolonged Expiratory Time
• Bronchitis - Increased Sputum
• Digital Clubbing
• Cor Pulmonale
• Thin in Appearance

Differentiating COPD and HF

- COPD and HF commonly co-exist
- Often difficult to differentiate clinically due to similarities in clinical presentation
- May be complicated in the face of an acute exacerbation of either disease state
  - Mixed pattern of COPD exacerbations with acute pulmonary edema is common

Adapted from: Wang CS, et al. JAMA 2005;294:1944-56; GOLD. Update January 2014.
Available at: http://www.goldcopd.org/guidelines-global-strategy-for-diagnosis-management.html
Accessed July 16, 2014.

Features Useful in Diagnosing HF in Patients with Dyspnea

- Historical Features
  - Heart failure
  - Myocardial infarction
  - Coronary artery disease
- Symptoms
  - Paroxysmal nocturnal dyspnea
  - Orthopnea
  - Dyspnea on exertion
- Physical Examination
  - Listening for a third heart sound (ventricular filling gallop)
  - Jugular venous pressure assessment
  - Auscultating for rales and wheezing
  - Auscultating for a murmur
  - Assessing the legs for edema
- Chest Radiograph
  - Pulmonary venous congestion
  - Interstitial edema
  - Cardiomegaly
  - Pleural effusion(s)
- Electrocardiogram Findings
  - Atrial fibrillation
  - An abnormal result
- B-type Natriuretic Peptide (BNP)
  - Reduced likelihood of HF with BNP <100 pg/mL

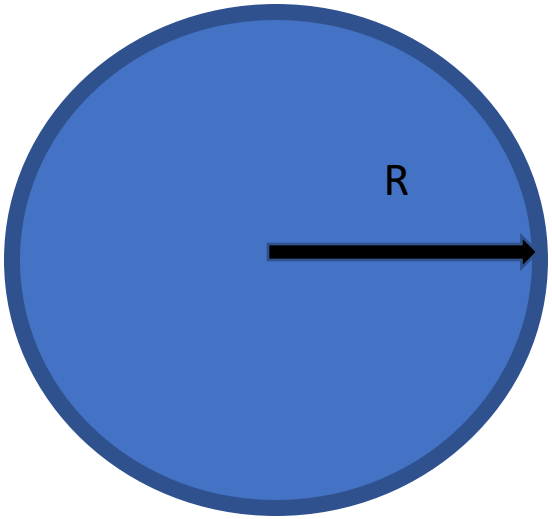
- Adapted from Wang CS, et al. JAMA 2005;294:1944-56.

Differentiating COPD and HF

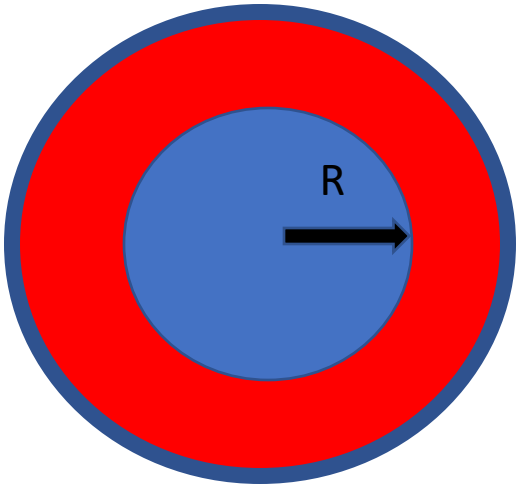
- Diagnostic tools may help to differentiate these disease entities:
  - Spirometry useful when the patient's volume status is optimized
    - During acute HF exacerbations, diagnostic accuracy may be limited
  - Echocardiography may be helpful to rule out the presence of systolic or diastolic dysfunction, however:
    - Poor visualization in 10–30% of patients
    - Concomitant AF precludes accurate assessment of diastolic function
  - Consider BNP/nT-pro-BNP to rule out the presence of HF
    - Has good negative predictive value

BNP: B-type natriuretic peptide (BNP); nT-pro-BNP: N-terminal pro b-type natriuretic peptide
Adapted from: Wang CS, et al. JAMA 2005;294:1944-56; GOLD. Update January 2014.
Available at: http://www.goldcopd.org/guidelines-global-strategy-for-diagnosis-management.html
Accessed July 16, 2014.

Flow related to radius^3



No swelling or water in Airway lumen



Swelling or water in Airway lumen

A must read article!

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CASE-BASED LEARNING

A woman with breathlessness: a practical approach to diagnosis and management

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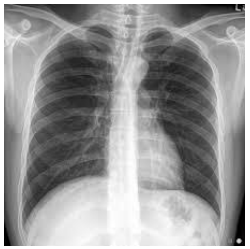
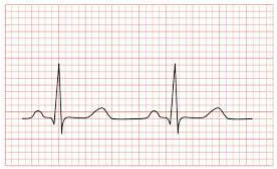




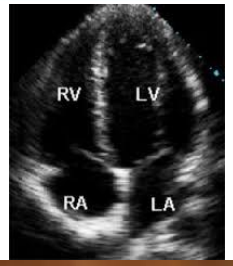


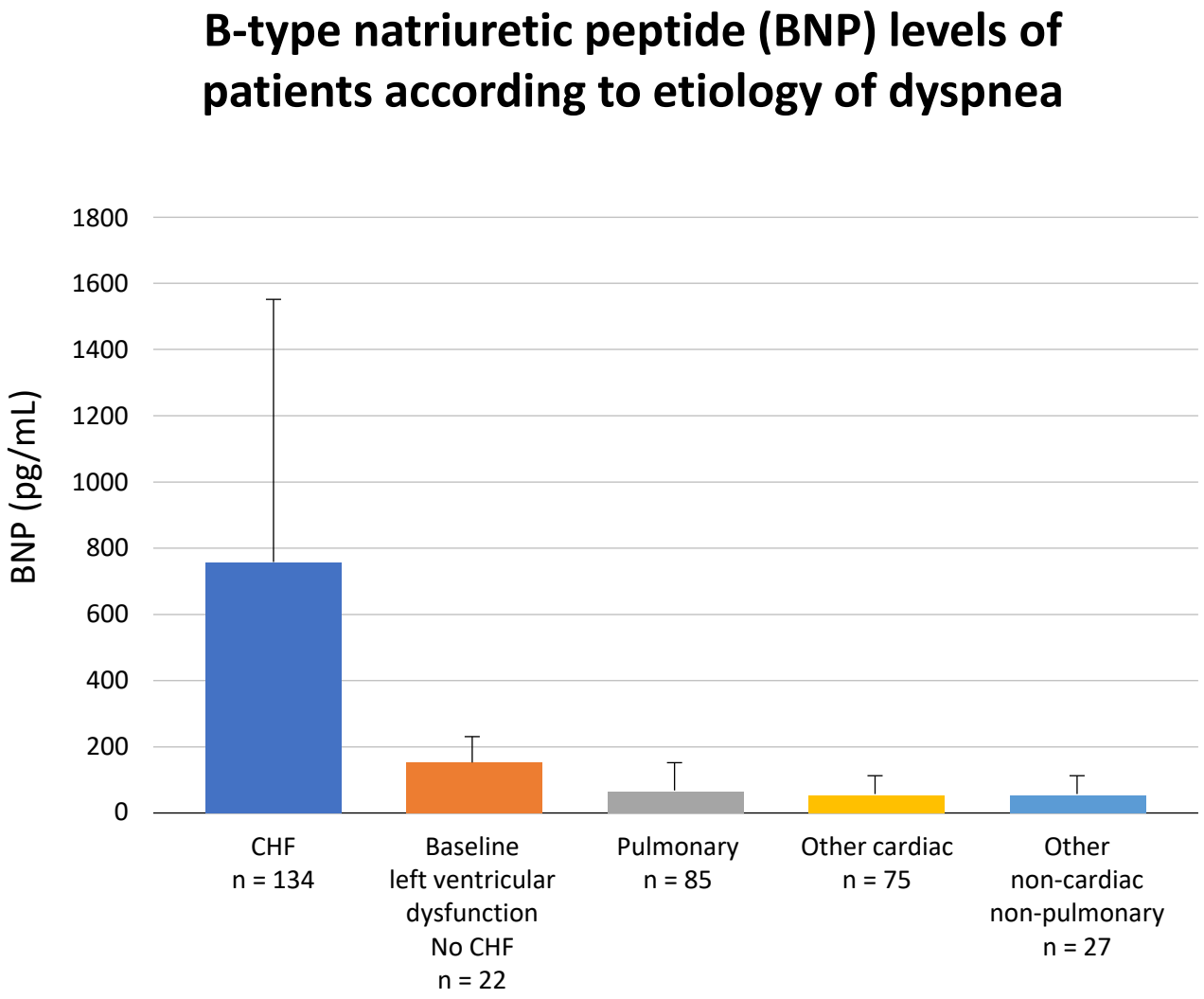
Table 1. Investigation of COPD and CHF

Investigation	Result in COPD	Result in CHF	Notes about the overlap
CXR	Hyperinflation. Vascular remodelling	Cardiomegaly, Vascular redistribution, Alveolar shadowing	Pulmonary vascular remodelling in those with COPD can mimic (upper lobe venous diversion[77]) or mask pulmonary oedema (asymmetric, regional, and reticular patterns),[10,78] Chest hyperinflation will falsely reduce the cardiothoracic ratio
Electrocardiography (ECG)	Cor pulmonale results in a range of ECG abnormalities including right bundle branch block and right ventricular hypertrophy	Check for rhythm disturbances and signs of ischaemic heart disease	The presence of ECG signs of cor pulmonale are indicative of a poor prognosis
Echocardiography	Assess cardiac function	Systolic vs diastolic function, Valvular issues	Acoustic windows may be impeded by air trapping in pulmonary disease, affecting quality of images as often as 10% in stable primary care patients with COPD(viii), 35% in severe disease,[79] and 50% in very severe airflow obstruction.[80]
B-type natriuretic peptide (BNP)	In stable disease, should be <100, but BNP levels can be increased in patients with COPD (and many other conditions)[81]	BNP is secreted by the left ventricle (LV) in response to volume elevated LV pressure; will differentiate cardiac from pulmonary cause of dyspnea,[82] especially for excluding CHF in subjects with acute dyspnoea.[83]	Normal levels exclude CHF, but raised levels can have many causes. Cor pulmonale is associated with an intermediate elevation of BNP,[84] typically ranging from 100 to 500 pg/mL. Levels <100 and >500 pg/mL have high negative and positive predictive values, respectively, for HF. See Figure 1
Spirometry	Obstruction. Diagnostic is post BD ratio of FEV_1/FVC <70%	Interstitial and alveolar oedema cause compression and obstruction of the airways in patients with decompensated CHF,[85,86] contrasting with restrictive defects when CHF is stable	Potential misdiagnosis and overestimation of COPD severity. With diuresis, mean FEV_1 improves by up to 35% and often returns to normal;[85]

Echocardiography

- TEE = transesophagyl echocardiogram (TEE) is > 90% sensitive for large clots, very specific.
  - Requires sedation and very specialized service
- TTE = TransThoracic echocardiogram: aortic dissection, cardiac tamponade, acute valvular lesion, EJECTION Fraction.
  - This, we can do routinely

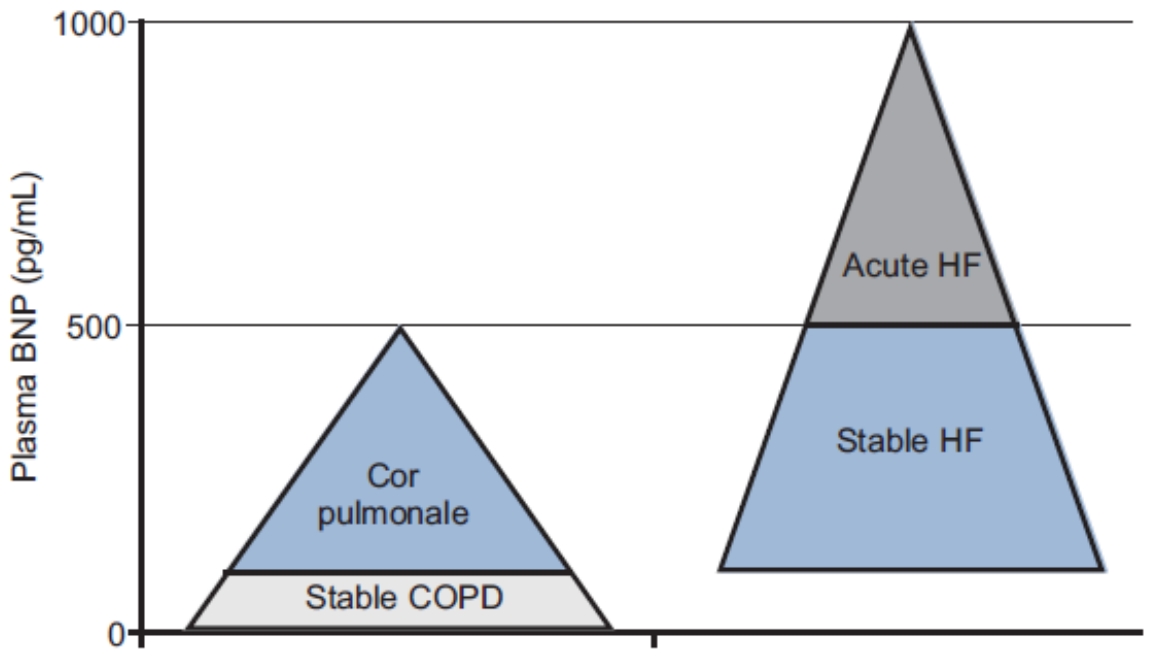
Utility of BNP in Differentiating HF from Lung Disease in Patients Presenting with Dyspnea



Adapted from Morrison LK, et al. J Am Coll Cardiol 2002;39:202-9.

BNP in CHF?

Figure 1. BNP levels in COPD vs CHF (reproduced with permission[12])



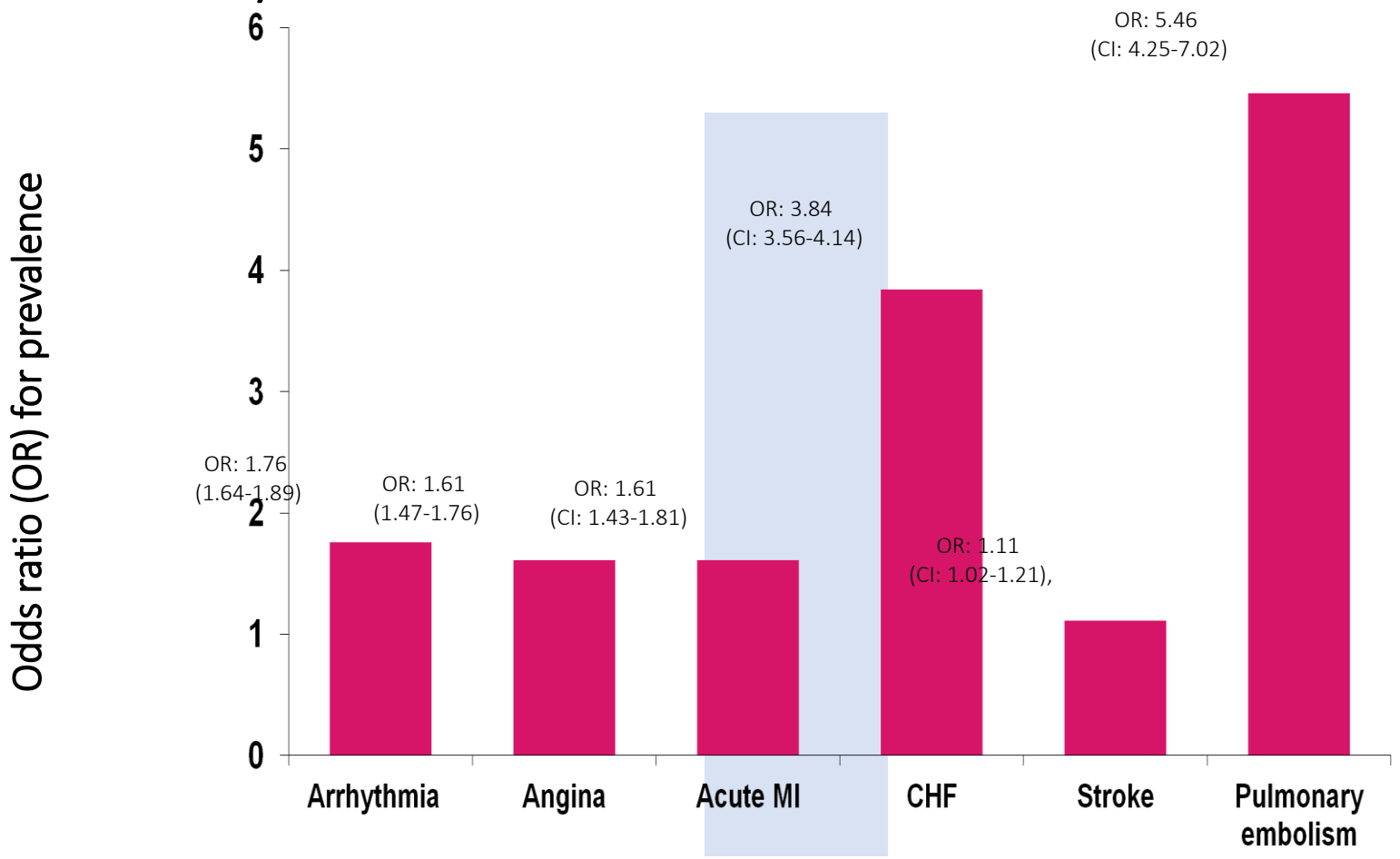
Part III:
What about COPD **AND** CHF?!!

Cardiovascular disease and COPD: dangerous liaisons?

Klaus F. Rabe[1,2], John R. Hurst[3] and Samy Suissa[4,5]

CrossMark

CV Comorbidities are Common in COPD, Particularly HF

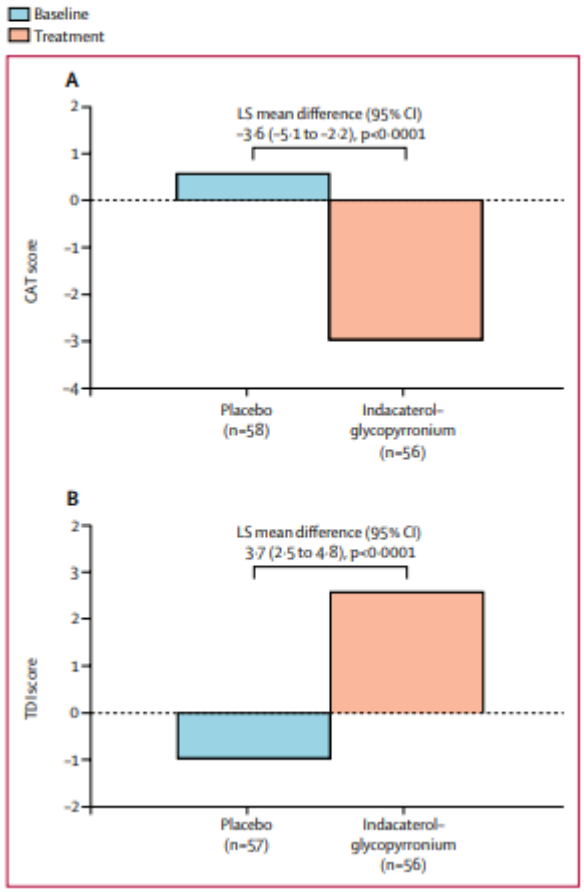


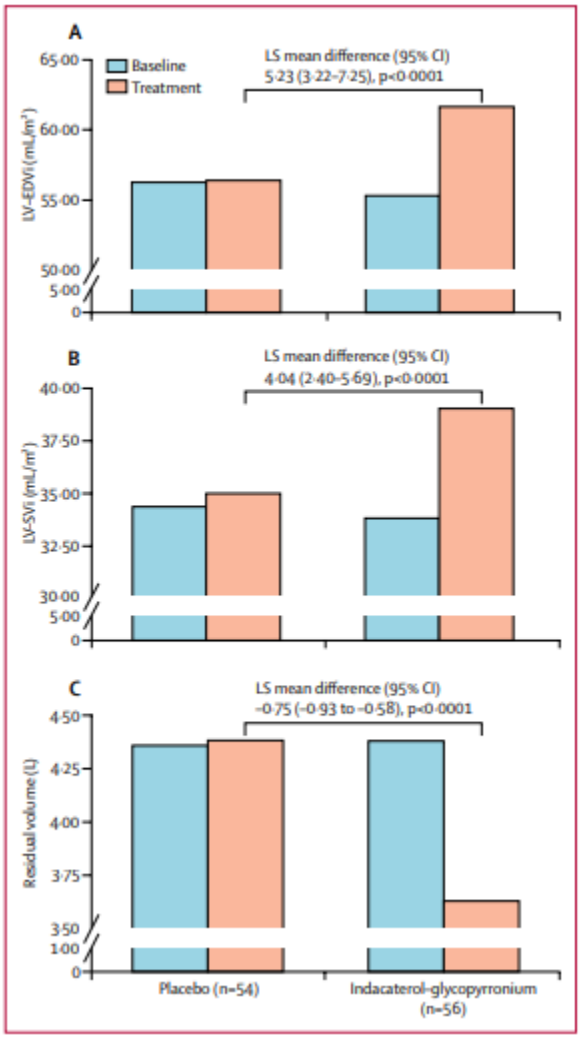
Retrospective cohort study of longitudinal healthcare databases maintained by the government of Saskatchewan, Canada. Subjects were persons age ≥40 years with COPD (n = 11,493). Each subject was matched by age and sex to two controls without COPD or asthma.
CV: cardiovascular; CHF: congestive heart failure; MI: myocardial infarction; CI: confidence interval
Adapted from Curkendall SM, et al. Ann Epidemiol 2006;16:63-70.

Treating COPD will help heart function

Effect of lung deflation with indacaterol plus glycopyrronium on ventricular filling in patients with hyperinflation and COPD (CLAIM): a double-blind, randomised, crossover, placebo-controlled, single-centre trial

Jens M Hohlfeld, Jens Vogel-Claussen*, Heike Biller, Dominik Berliner, Korbinian Berschneider, Hanns-Christian Tillmann, Simone Hiltl, Johann Bauersachs, Tobias Welte*





COPD drugs in CHF?

- SABA– tachycardia
- LABA– no issue
- Theophylline: tachycardia and arrhythmia
- LAMA
  - Singh article said issues, but
  - Uplift has proven OK!

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CV drugs in COPD

- ACE/ARB good to reduce pulmonary

  vasoconstriction
- Careful with diuretics and metabolic alkalosis
  - (theoretically causing decreased resp drive)
- Beta blockers?

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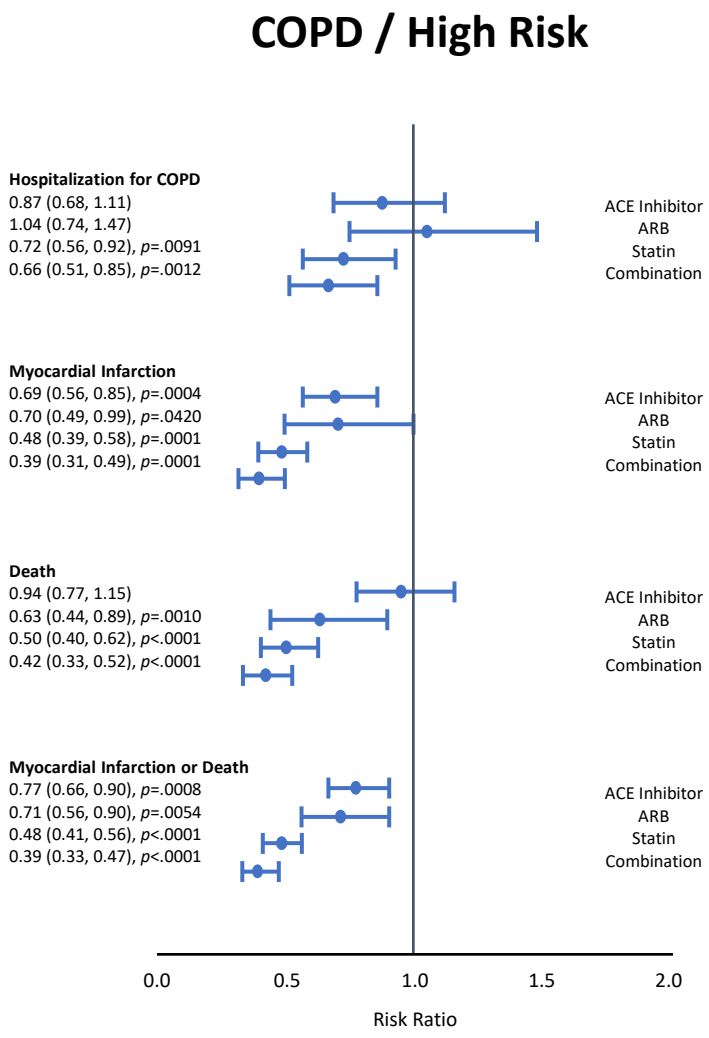
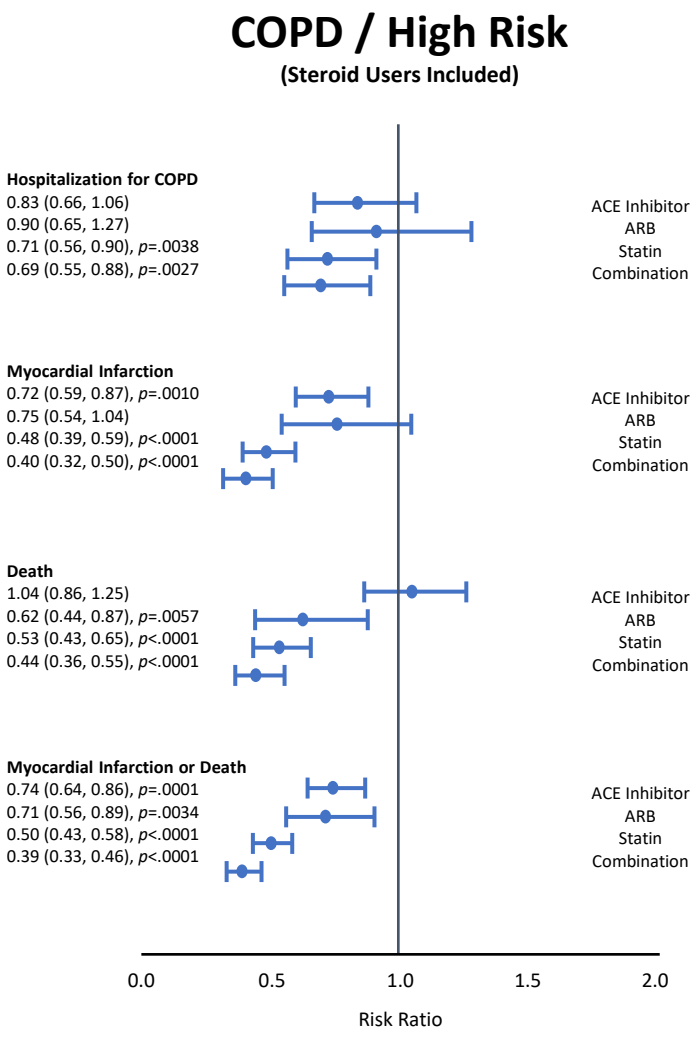
Reduction of Morbidity and Mortality with ACE-I, ARB and Statins in COPD





ACE-I: angiotensin-converting enzyme inhibitors; ARB: angiotensin receptor blocker
Adapted from Mancini GB, et al. J Am Coll Cardiol 2006;47:2554-60.

Statins may reduce mortality (esp if on ICS?)

Eur Respir J 2007; 29: 279–283
DOI: 10.1183/09031936.00106406
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Statin use is associated with reduced mortality in COPD

V. Søyseth, P.H. Brekke, P. Smith and T. Omland

ABSTRACT: **Patients with chronic obstructive pulmonary disease (COPD) have an increased risk of ischaemic heart disease (IHD). Statins reduce mortality and morbidity in IHD. It has been hypothesised that statin treatment is associated with reduced long-term mortality in patients with COPD.**

Using a retrospective cohort design, 854 consecutive patients (mean age 70.8 yrs; 51.5% female) with a diagnosis of COPD exacerbation were included in the study at discharge from a Norwegian teaching hospital.

Median follow-up was 1.9 yrs, during which 333 patients died. The crude mortality rate per 1,000 person-yrs was 110 in patients treated with statins, and 191 in patients not treated with statins. After adjustment for sex, age, smoking, pulmonary function and comorbidities, the hazard ratio (HR) for statin users *versus* statin nonusers was 0.57 (95% confidence interval 0.38–0.87). When subdividing statin users and statin nonusers into groups according to concomitant treatment with inhaled corticosteroids (ICS) the following HRs were found: 0.75 (0.58–0.98) for ICS only; 0.69 (0.36–1.3) for statins only; and 0.39 (0.22–0.67) for the combined treatment with statin and ICS compared with no such treatment.

Treatment with statins was associated with improved survival after chronic obstructive pulmonary disease exacerbation, while inhaled corticosteroids appeared to increase the survival benefit associated with statin use.

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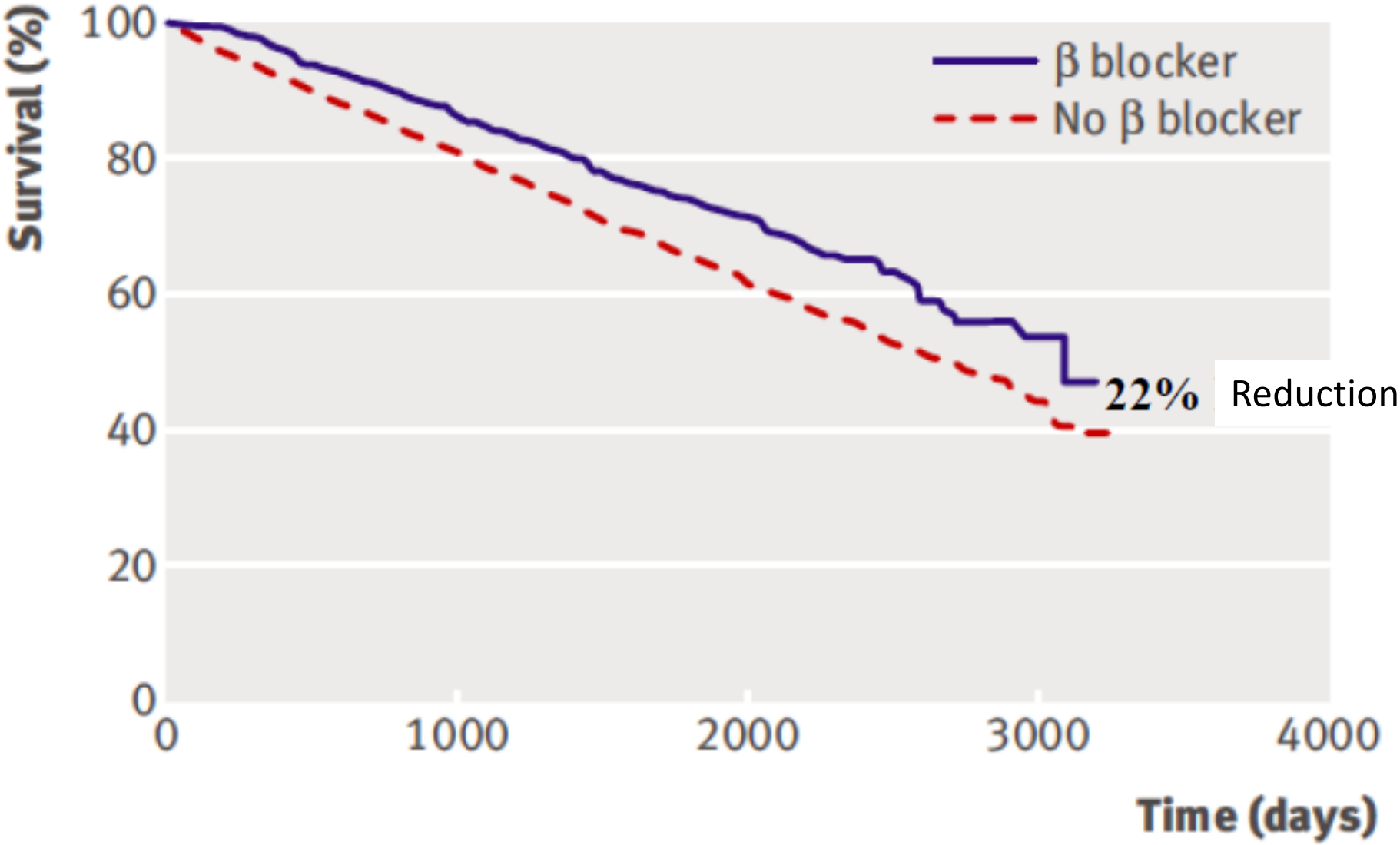
Received:
August 14 2006
Accepted after revision:
October 11 2006

SUPPORT STATEMENT

ß blocker reduce mortality in COPD



BMJ 2011;342:d2549

ß blocker effectiveindependent of COPD therapy

Characteristic	Adjusted hazard ratios (95% CI)†
Treatment groups*	
ICS+LABA+Tio+BB	0.28 (0.21 to 0.39)
ICS+LABA+Tio	0.43 (0.38 to 0.48)
ICS+LABA+BB	0.44 (0.31 to 0.62)
ICS+LABA	0.64 (0.57 to 0.74)
ICS+BB	0.48 (0.31 to 0.74)
ICS	0.69 (0.58 to 0.83)
ICS +Tio	0.61 (0.47 to 0.80)
LABA or Tio (no ICS)+BB	0.52 (0.36 to 0.76)
LABA or Tio (no ICS)	0.71 (0.59 to 0.84)
BB (no ICS)	0.65 (0.51 to 0.83)

Beta-Blockers Improve Survival in COPD

Association between beta-blockers and all-cause mortality in patients with COPD in observational cohort studies

Study	Population	N with COPD	Follow-up	Adjusted risk (±95% CI)
Sin et al. (2002)	HF	3834	Median 21 months	0.78 (0.63-0.95)
Staszewsky et al. (2007)	HF	628	Median 23 months	0.55 (0.37-0.82)
Hawkins et al. (2009)	MI	1258	Median 25 months	0.74 (0.68-0.80)
Gottlieb et al. (1998)	MI	41,814	2 years	0.60 (0.57-0.63)
Chen et al. (2001)	MI	10,988	1 year	0.86 (0.73-1.00)
Van Gestel et al. (2008)	Vascular disease	1205	Median 5 years	0.73 (0.60-0.88)
Au et al. (2004)	HTN	1966	2 years	0.57 (0.33-0.89)
Rutten et al. (2010)	COPD primary care	2230	7.2 years	0.68 (0.56-0.83)
Dransfield et al. (2008)	COPD exacerbation	825	—	0.39 (0.14-0.99)
Short et al. (2011)	COPD primary care	5977	Mean 4.35 years	0.78 (0.67-0.97)

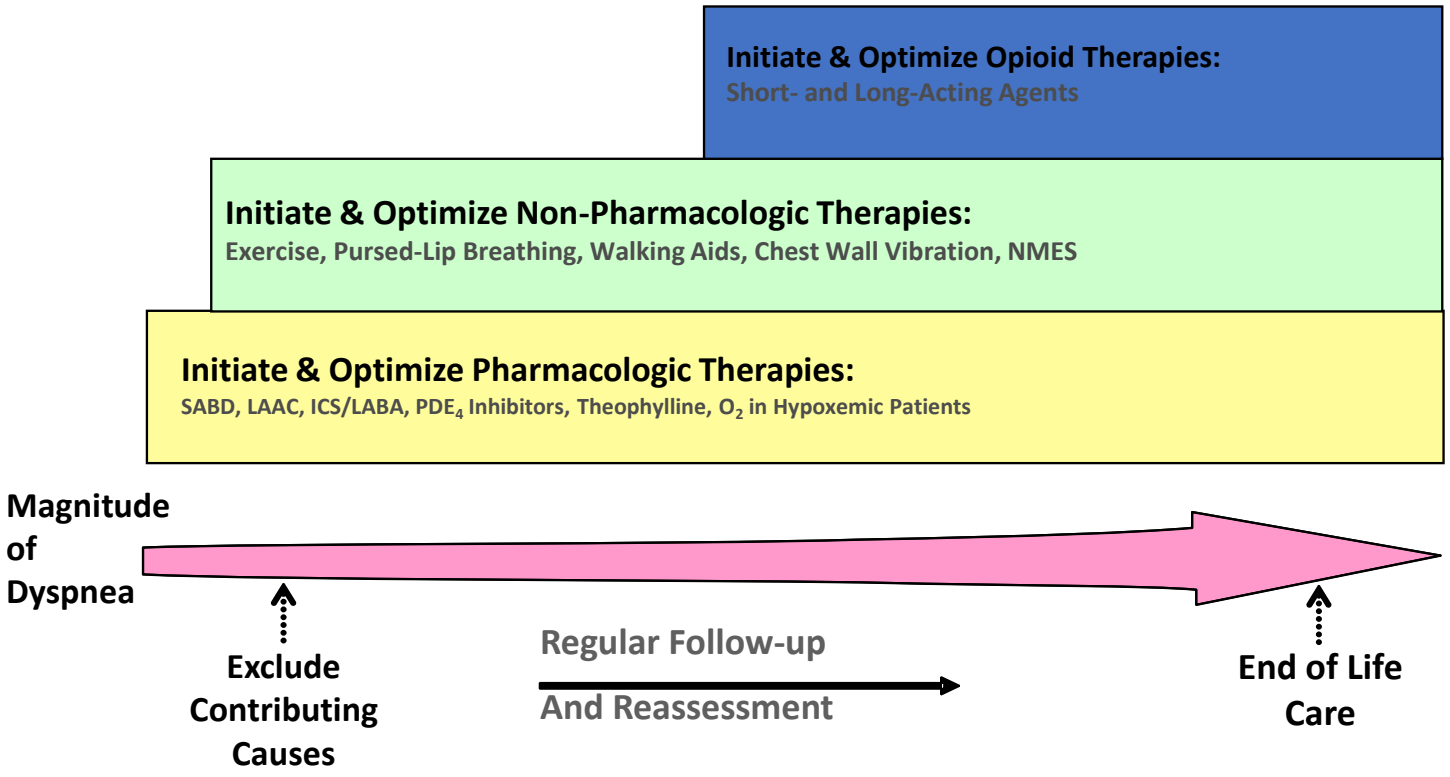
HF: heart failure; MI: myocardial infarction; HTN: hypertension; CI: confidence interval Adapted from Hawkins NM, et al. Eur Heart J 2013;34:2795-803.

And...STOP SMOKING

- Beyond the lung effects…
- Cigarette smoking can inhibit the effects of cardiovascular medications
  - Reverses the effect of beta-blockers
  - Blocks vasodilation of CCBs, ACE-Is, ARBs
  - Blocks the effect of antiplatelet drugs
    - ASA
    - Clopidogrel
  - Blocks the effect of lipid-lowering drugs by oxidizing LDL-C
  - Blocks the effect of nitroglycerin

CCBs: calcium-channel blockers; ACE-I: angiotensin-converting enzyme inhibitors; ARB: angiotensin receptor blocker;
ASA: acetylsalicylic acid; LDL-C: low-density lipoprotein cholesterol
Adapted from Leone A. Cardiol Res Pract 2011;2011:264894.

Comprehensive Approach to Management of Refractory Dyspnea in Advanced COPD



Marciniuk D, Goodridge D, Hernandez P, et al. Can Resp J 2011; 18:69-78.

A

Suggested Protocol for Managing Dyspnea with Opioids in Advanced COPD

- Initiate opioid therapy with oral immediate release morphine syrup – titrate slowly at weekly intervals over a 4 to 6 week period.
- Start therapy with morphine 0.5 mg orally twice daily for 2 days, and then increase to 0.5 mg orally every 4 hours while awake for remainder of week 1.
- If tolerated and indicated, increase to morphine 1.0 mg orally every 4 h while awake in week 2, increasing by 1.0 mg/week or 25% dosage increments/week until the lowest effective dose that appropriately relieves dyspnea is achieved.
- Once a stable dosage is achieved (i.e., no significant dose change for 2 weeks and dyspnea controlled), a sustained-release preparation at a comparable daily dose could be considered for substitution.
- If patients experience significant opioid-related side effects such as nausea or confusion, substitution of an equipotent dose of oral hydromorphine could be considered (1 mg hydromorphine = 5 mg morphine).
- Stool softeners and laxatives should be routinely offered to prevent opioid-associated constipation.

Consider Movantik?

What if Restriction on Spirometry

- Not the lungs: Neurologic
- Outside the lungs: Chest wall
- Inside the lungs: Pleural
- Lungs: mass, interstitial lung disease

- Diagnosis:
- Imaging: CXR, CT
- Full PFTs
- Walk tests
- Blood gases

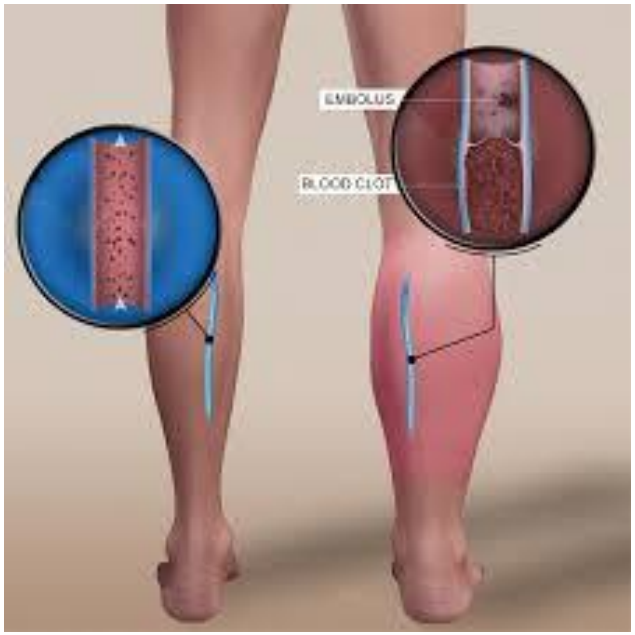
What if…

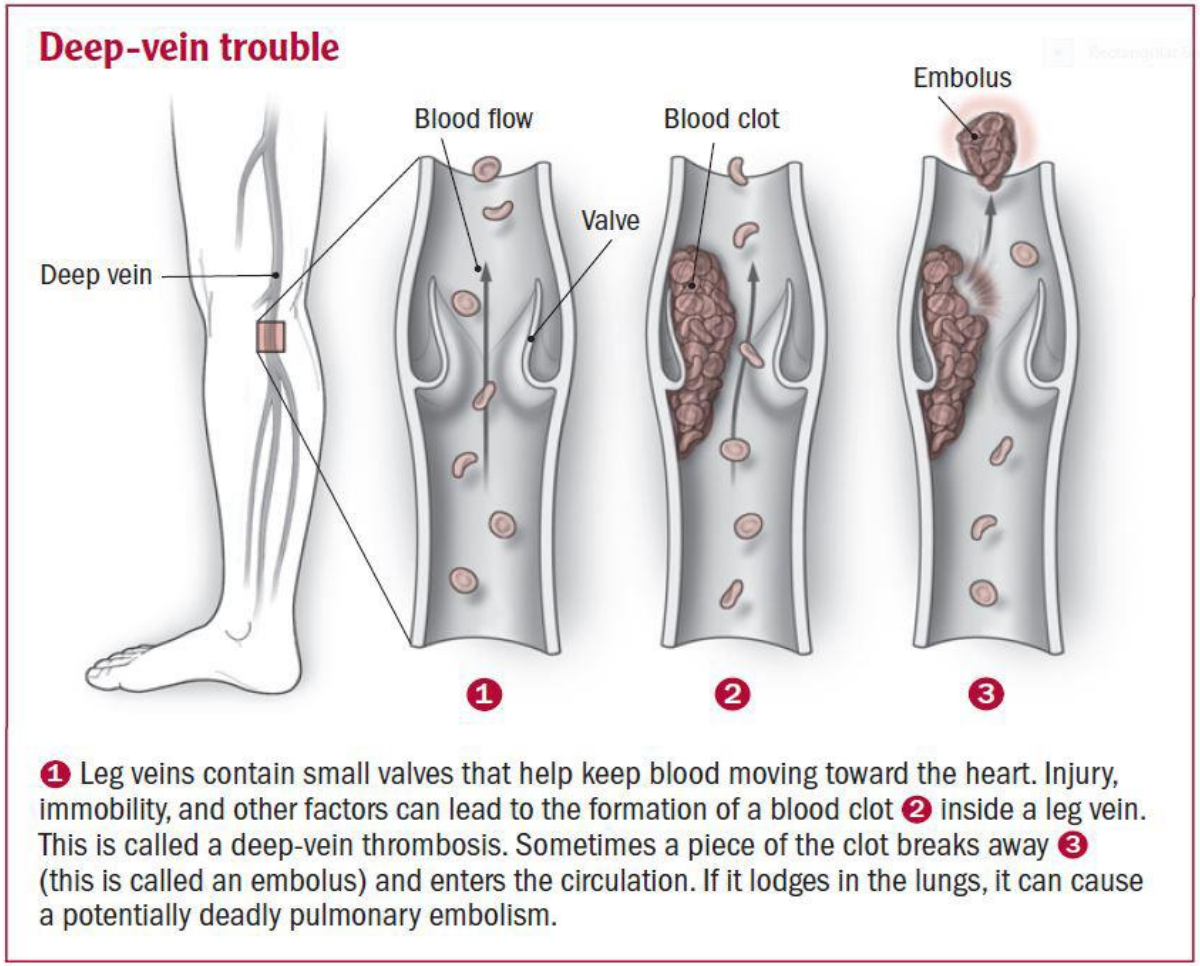
- Dyspneic
- Normal Echo
- Normal Spirometry
- Normal Hb
- What other clues?

Part IV: Thromboembolic Disease

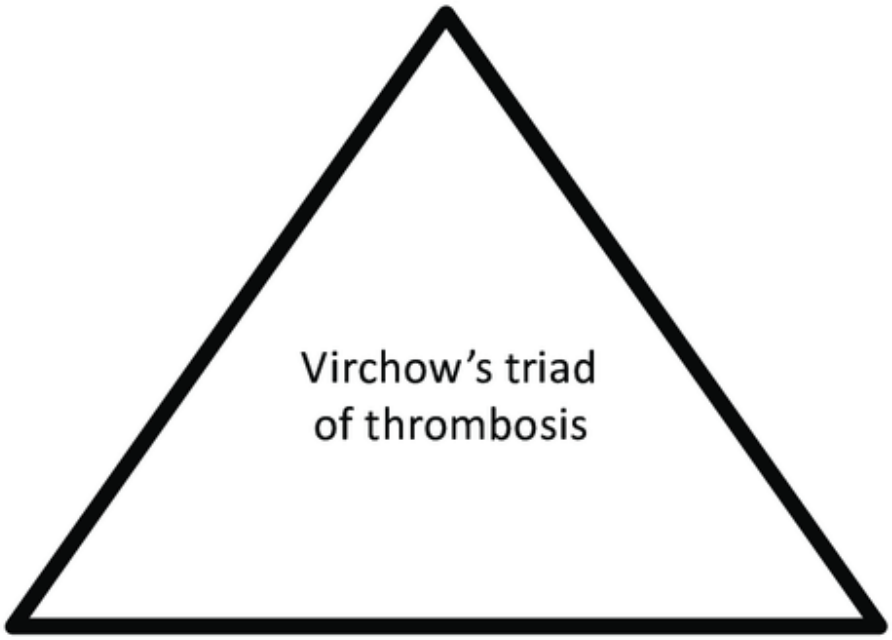
Pulmonary embolism

- At least 90% of pulmonary emboli originate from major leg veins.





Virchows Triad



Natural History of VTE

- 40-50% of pts with DVT develop PE, often “silent”
- PE presents 3-7 days after DVT
  - Fatal within 1 hour after onset of respiratory symptoms in 10%
  - Shock/persistent hypotension in 5-10% (up to 50% of patients with RV dysfunction)
- Most fatalities occur in untreated patients
- Perfusion defects completely resolve in 75% of all patients (who survive)

Diagnosis: Clinical Presentation

- Dyspnea, tachypnea, or pleuritic chest pain most common
  - Pleuritic pain = distal emboli →pulmonary infarction and pleural irritation
  - Isolated dyspnea of rapid onset= central PE with hemodynamic sequlea
  - Retrosternal angina like sxs= RV ischemia
- Syncope=rare presentation, but indicates severely reduced hemodynamic reserve
- Symptoms can develop over weeks, may be non-specific!!
- In pts with pre-existing CHF or COPD, worsening dyspnea may indicate PE

- This study showed a 25% prevalence of PE in patients with COPD hospitalized for severe exacerbation of unknown origin

Risk factors:
Recent surgery
Hx of thrombo-embolic disease
Malignancy hx
Decrease in $PaCO_2$

Clinical Diagnosis of PE

- In summary, clinical signs, symptoms and routine tests do not allow for the exclusion or confirmation of acute PE but may increase the index of its suspicion
- Consider PE in cases of unexplained tachycardia or syncope

Diagnosis-Probability Assessment

- Implicit clinical judgement is fairly accurate: “Do you think this patient has a PE?”
- Validated prediction rules standardize clinical judgement
    - Wells
    - Geneva

Modified Wells criteria: clinical assessment for pulmonary embolism

Clinical symptoms of DVT (leg swelling, pain with palpation)	3.0
Other diagnosis less likely than pulmonary embolism	3.0
Heart rate >100	1.5
Immobilization (≥3 days) or surgery in the previous four weeks	1.5
Previous DVT/PE	1.5
Hemoptysis	1.0
Malignancy	1.0
Probability	**Score**
Traditional clinical probability assessment	
High	>6.0
Moderate	2.0 to 6.0
Low	<2.0
Simplified clinical probability assessment*	
PE likely	>4.0
PE unlikely	≤4.0

Proportion with PE

65%

30

10%

Data from van Belle, A, et al. JAMA 2006; 295:172.

Diagnosis:Other tests

- Most patients with PE have a normal pulse oximetry
- A-a gradient is insensitive and non-specific

Diagnosis: Chest X-Ray

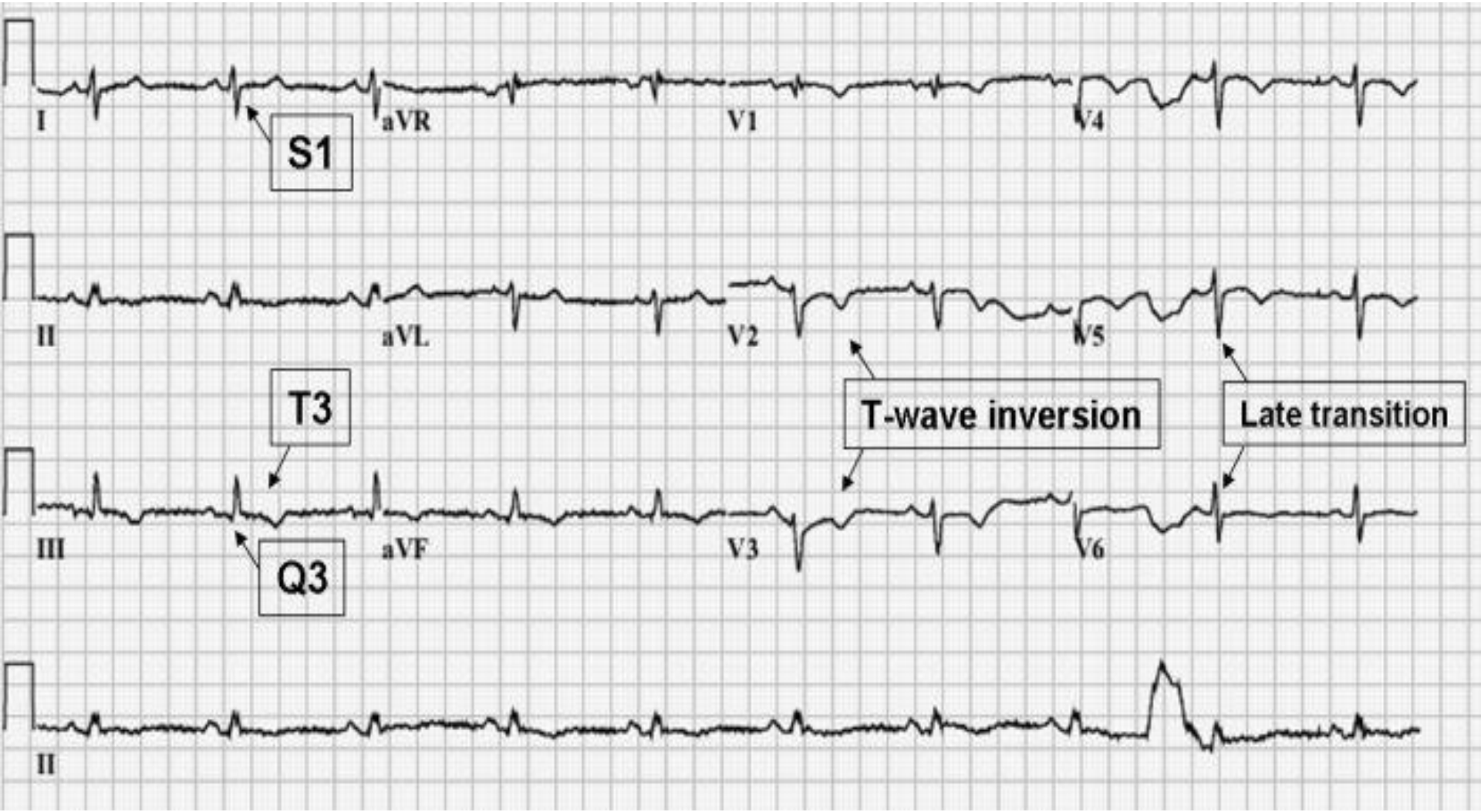
- Usually abnormal, but non-specific
- Study of 2,322 patients with PE:
  - Cardiac enlargement (27%)
  - Normal (24%)
  - Pleural effusion (23%)
  - Elevated hemidiaphragm (20%)
  - Pulmonary artery enlargement (19%)
  - Atelectasis (18%)
  - Parenchymal pulmonary infiltrates (17%)

Chest Radiographs in Acute Pulmonary Embolism: Results From the International Cooperative Pulmonary Embolism Registry. Chest July 2000 118:3338; 10.1378/chest.118.1.33

Diagnosis: ECG

- Usually non-specific ST/T waves changes and tachycardia
- RV strain patterns suggest severe PE
  - Inverted T waves V1-V4
  - QR in V1
  - Incomplete RBBB
  - S1Q3T3

S1Q3T3 and T wave changes



Ventilation / Perfusion Scanning (V/Q Scan)

- combined with clinical suspicion
- sensitivity is 85 - 90%
- positive predictive value depends on clinical suspicion
- More radiation than a CT-PE study.

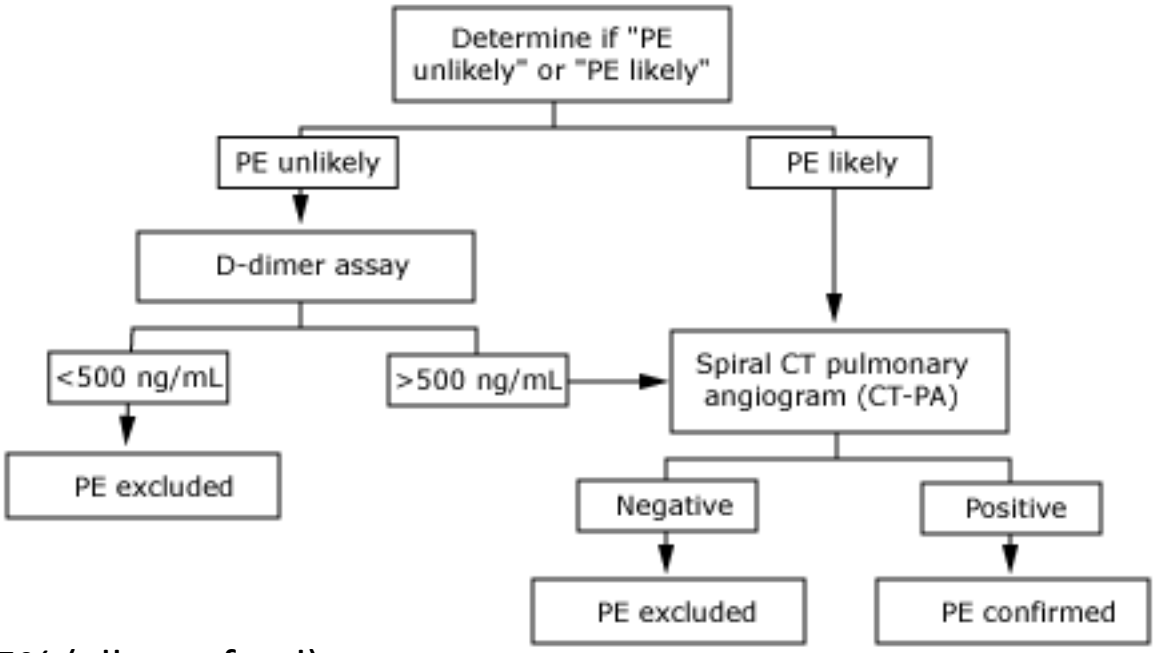
Diagnosis

- D-Dimer
  - Fibrin degradation product
  - ELISA tests are highly sensitive (>95%)
  - Non specific (~40%):
  - cancer, sepsis, inflammation increase d-dimer levels
- Reliability?
  - Negative result excludes PE safely in **PE-unlikely** patients (using Clinical probability scores)

Spiral CT

- Direct visualization of emboli.
- • Both parenchymal and mediastinal structures can be evaluated.
- • Offers differential diagnosis in 2/3 of cases with a negative scan.
- BUT…
- Dye load and large radiation dose
- Optimally used when incorporated into a validated diagnostic decision tree

CT-based diagnostic strategy used in patients with suspected pulmonary embolism



3 month VTE rate

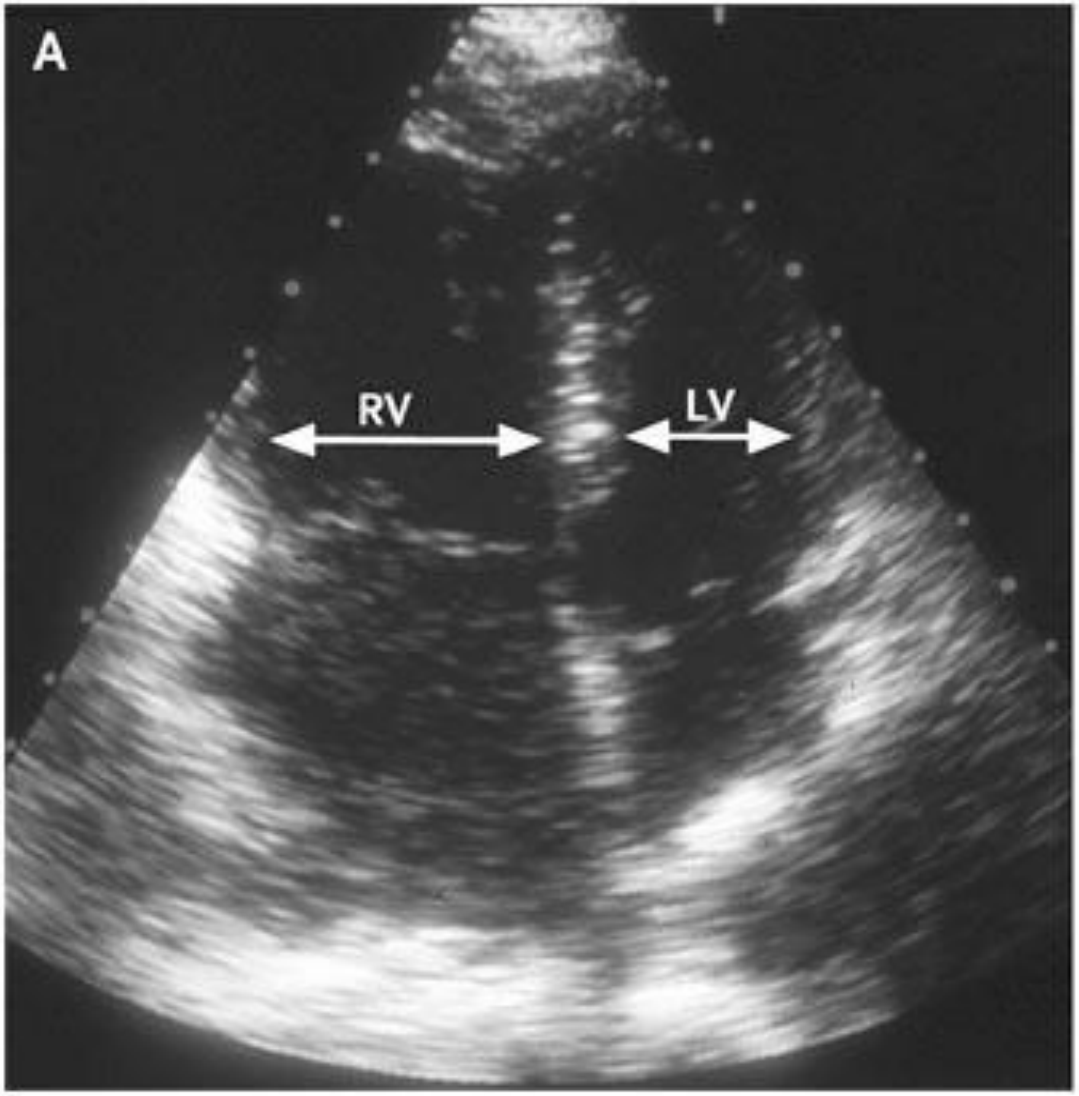
0.5% (all non fatal)

1.3%

Adapted from van Belle, A, et al. JAMA 2006; 295:172.

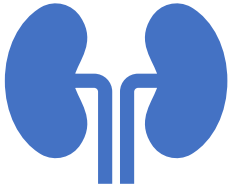
This algorithm allowed for a management decision in 98% of patients presenting with symptoms suggestive of PE

Echocardiography-RV Dilation



Diagnosis- Summary

History and physical examination



Then 1,2,3 approach:

Clinical decision score

D-Dimer test

Chest CT

(V/Q scan remains a valid option for patients with contraindication to CT)

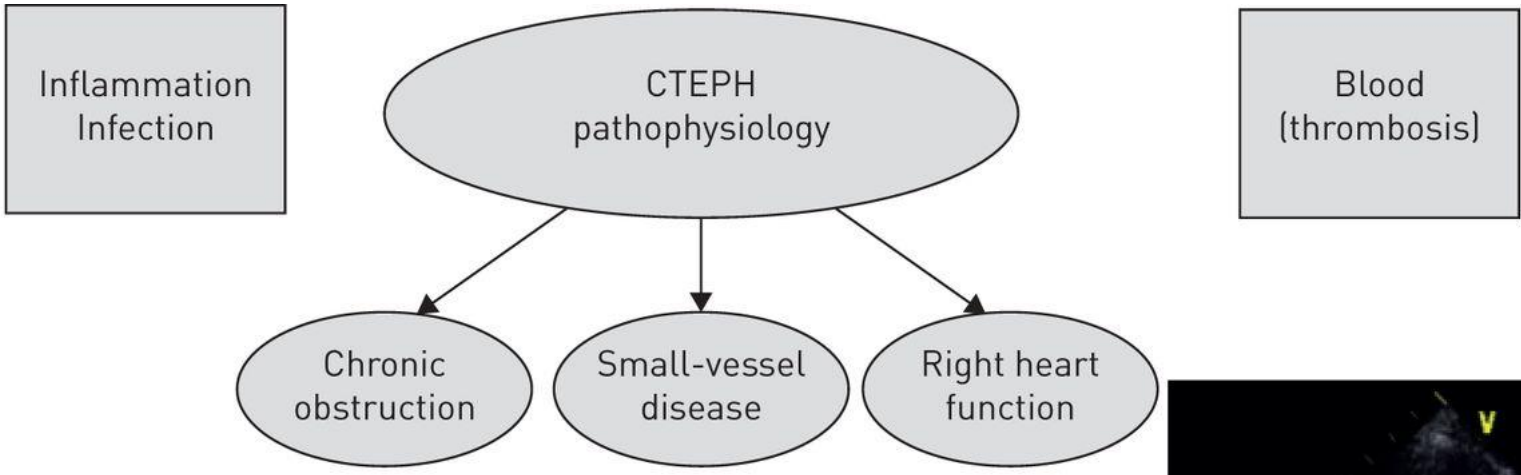
What if…

- Dyspneic
- Normal Echo
- Normal Spirometry
- Normal Hemoglobin
- What other clues?

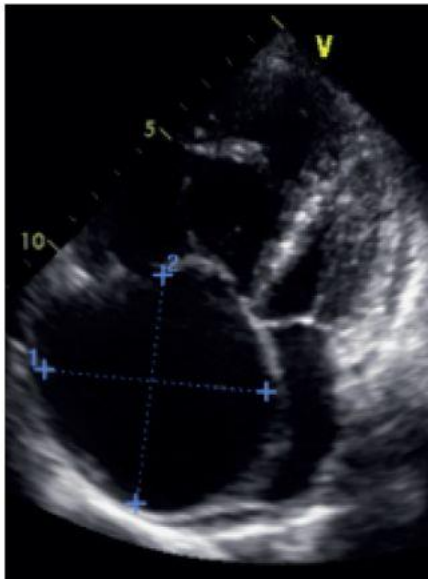
History!

- Hx of Pulmonary Embolism
- Hx of use of diet pills
- Family History
- Cough? Sputum?

Chronic Thromboembolic Hypertension



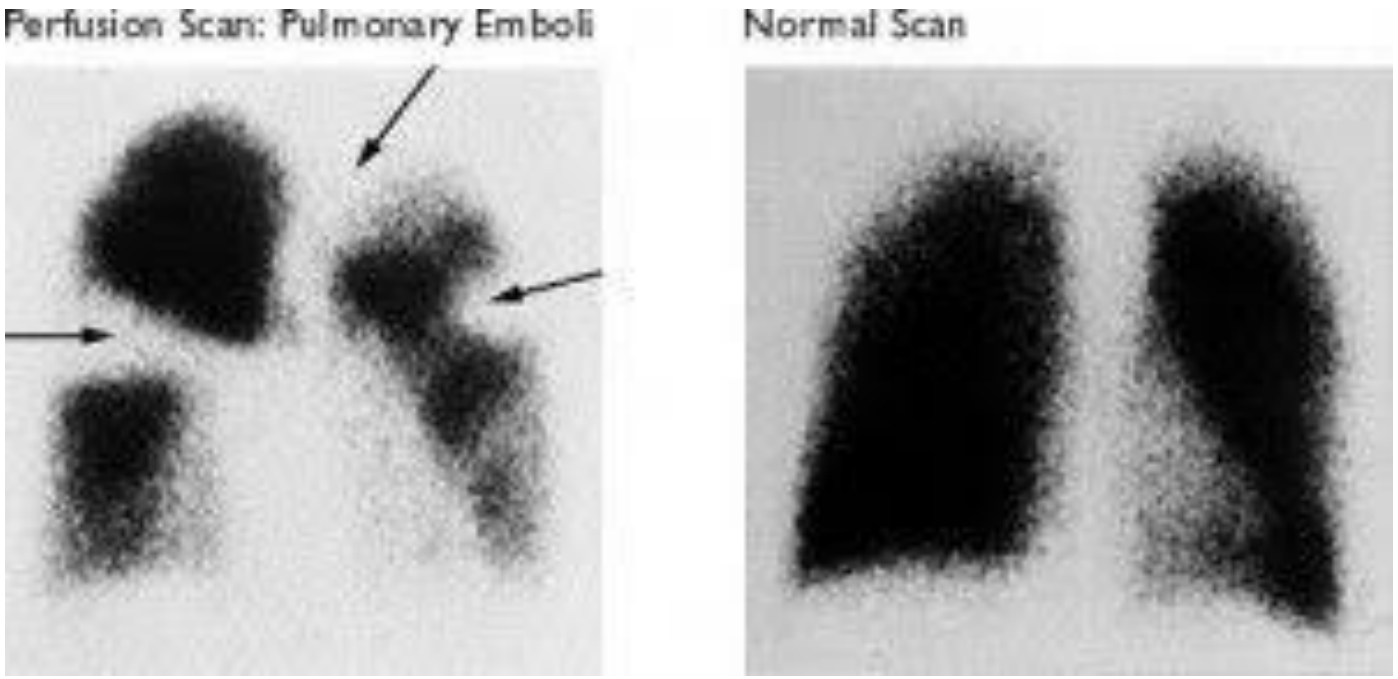
Residual PH
arteriopathy
Venous/capillary
disease



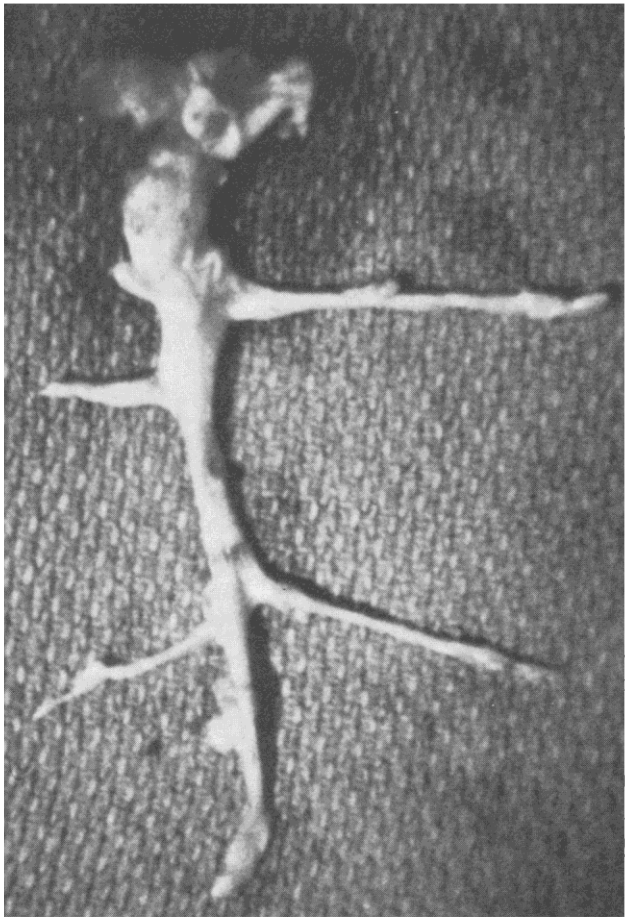
Red flags

Dyspnea presenting after a remote history of PE
Underlying malignancy
Splenectomy
Ventriculoatrial shunts
Chronic indwelling infected IV lines
Chronic inflammatory diseases (i.e. IBD, CTD, RA, SLE)
Myeoloproliferative syndromes

Best diagnostic test is?



Chronic clot



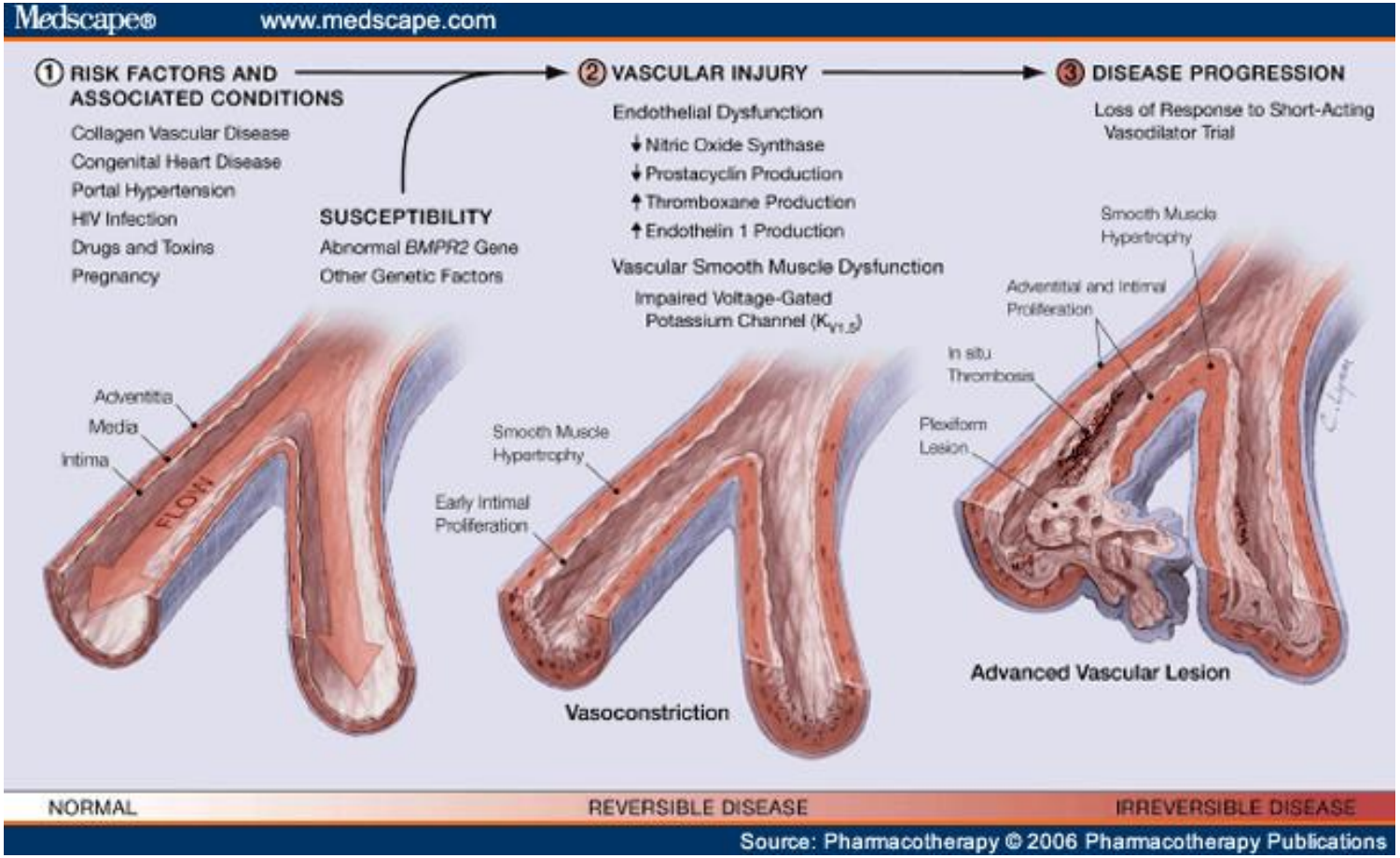
Ann Surg 1977; 15 (7): 699-711

Pulmonary Hypertension

- Pulmonary hypertension (PH) is characterized by elevated pulmonary pressure and secondary right ventricular failure
- It is a life-threatening condition
- Poor prognosis if untreated
- Definition is based upon ***right heart catheterization*** PH
  - MAP (mean pulmonary artery pressure) > 25 mmHg at rest

PHA Pulmonary Hypertension Association
Empowered by hope

Pulmonary Arterial Hypertension



ACCF/AHA 2009 Expert Consensus Document on Pulmonary Hypertension
ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension

PAH - Manifestation

- Initially symptoms
  - –Exertional dyspnea
  - –Lethargy, and fatigue
  - –Palpitation and lightheadedness
- Advanced symptoms
  - –Right ventricular failure develops
  - –Exertional chest pain
  - –Exertional syncope

Investigations – done by PCP

- ECG
- Basic blood tests
- CXR
- PFT and DLCO (may be first clue)
- Metacholine test
- Echo (honestly, it's the first clue)
  - Estimation of RVSP
  - Tricuspid regurgitation velocity
  - Tricuspid regurgitation gradient
  - RV dilatation

Labwork to consider

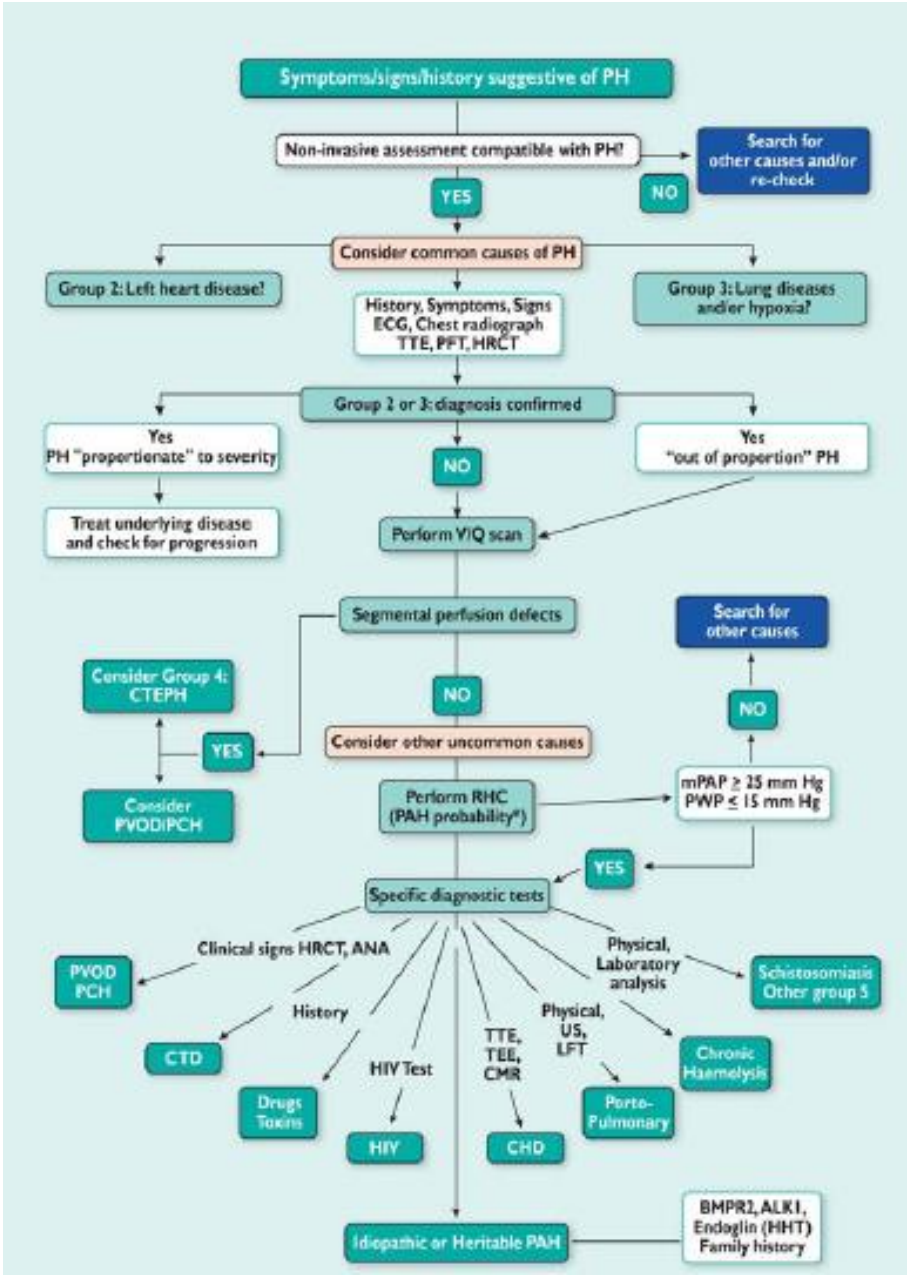
Blood tests

- –HIV
- –CTD
- –Hepatitis
- –Lupus anticoagulant, anti-phopholipids, anticardiolopin
- –Troponin
- –BNP

Investigations – done by PCP/local specialists

- Cardiac stress test
  - Regular vs nuclear
- Abdo US
- Sleep study
- CT chest
- Left heart cath (coronary angiogram)
- 6-MWT (6-minute walk test)

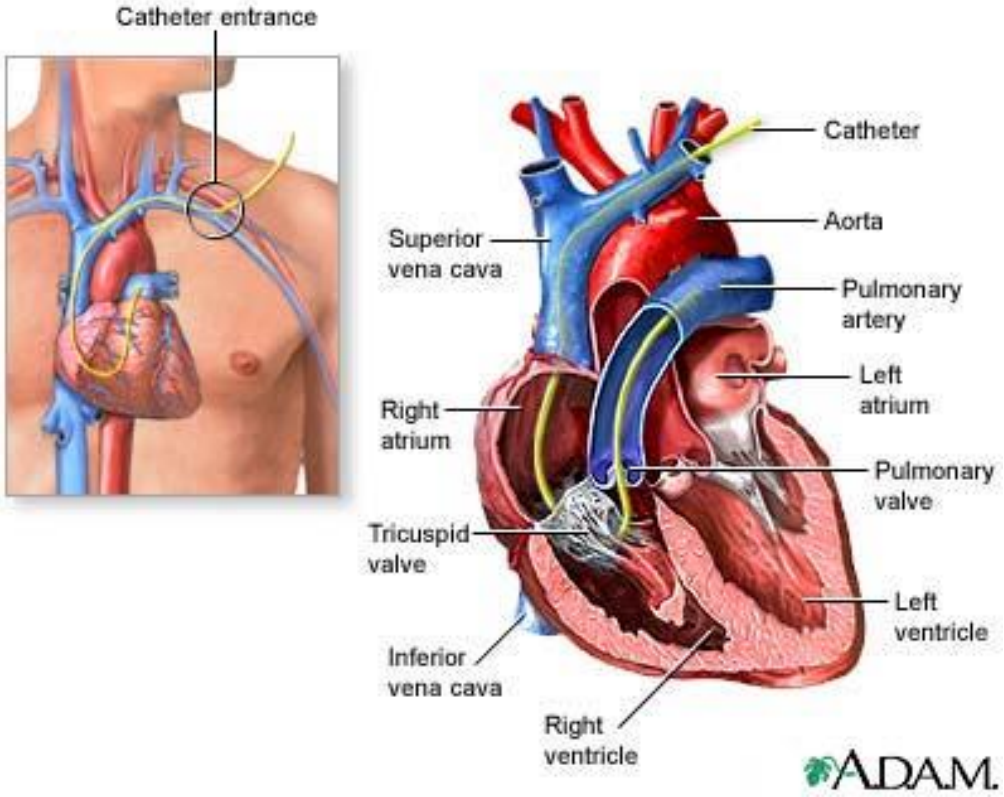
Diagnostic algorithm



ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension

Gold standard test

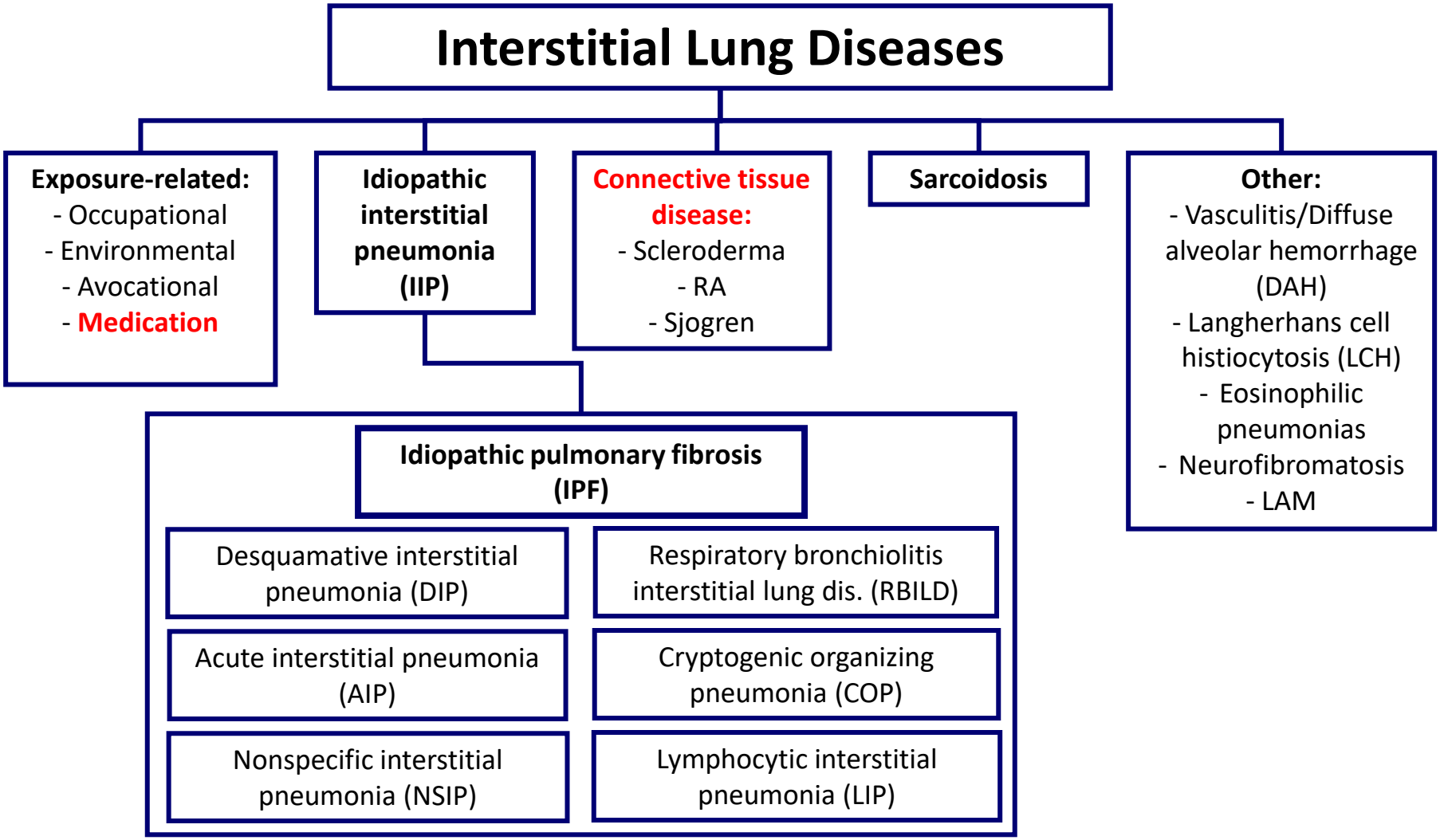
Right heart cath



What if you hear crackles?

- Echo/CXR to see if heart failure
- Then what?

Classification of Interstitial Lung Diseases



Am J Respir Crit Care Med 2002;165:277–304.

Drug-induced lung disease

- At least 150 different agents are recognized as potentially causing pulmonary disease[1]
- Drugs that may cause pulmonary fibrosis include (most common bolded)[2]:

Chemotherapeutic and immunosuppressive	Anticonvulsant, antipsychotic, antidepressant	Cardiovascular	Antimicrobial	Antimetabolic
Bleomycin	Carbamazepine	**Amiodarone**	**Nitrofurantoin**	**Methotrexate**
Busulfan		Flecainide	Amphotericin B	Leflunomide
Chlorambucil			Tetracycline	
Cyclophosphamide				
Rituximab				
Melphalan				
Nitrosoureas				
Paclitaxel				
Procarbazine				

1. www.pneumotox.com; 2. Ozkan M et al. *Cleve Clin J Med*. 2001;68(9):782–5, 789–795.

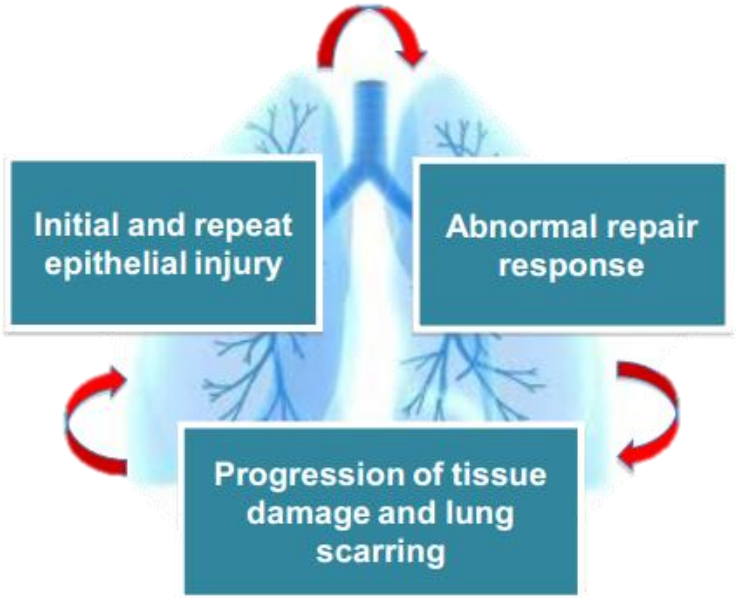
Common clinical presentation of IPF

- Older age (>60 years)
- Male gender
- Smoking common, in 60–70%
- Symptoms:
  - Progressive exertional dyspnea
  - Non-productive cough
- Signs:
  - Bilateral inspiratory Velcro-like crackles
  - Clubbing of fingers
- Tests:
  - Abnormal pulmonary function test indicating restriction and impaired gas exchange

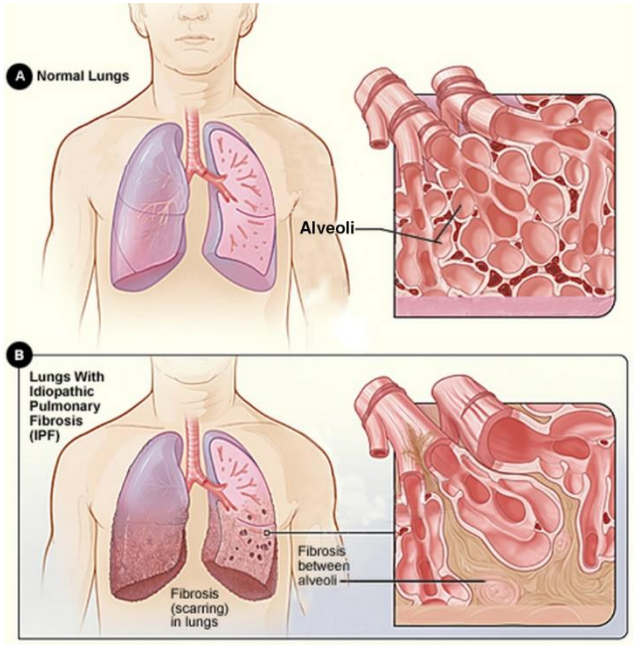
Raghu G et al. *Am J Respir Crit Care Med*. 2011;183(6):788–824.

IPF: abnormal wound healing leads to irreversible fibrosis

- Three stages of pathogenesis[1]:



- What initiates this process is unknown

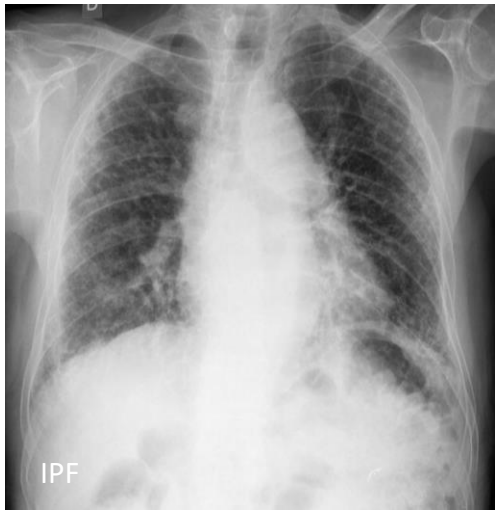


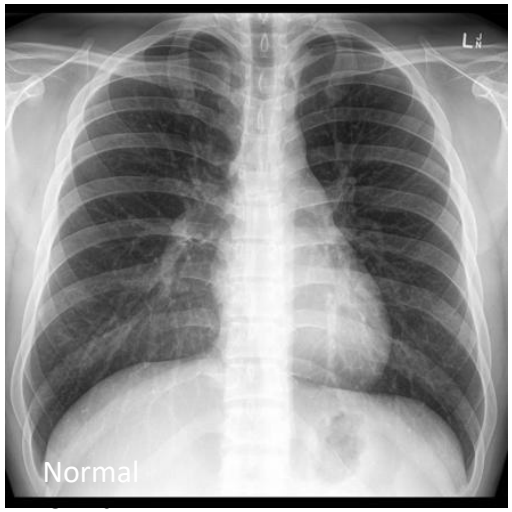
- Fibrosis distinguishes IPF from other ILDs, which are mainly inflammatory[2]
- Distorted alveolar–capillary barrier architecture leads to impaired gas exchange, which limits routine physical activity[3]

1. Travis WD et al. *Am J Respir Crit Care Med*. 2013; 188(6):733–748.
2. Raghu G et al. *Am J Respir Crit Care Med*. 2011;183(6):788–824.
3. Meltzer EB, Noble PW. *Orphanet J Rare Dis*. 2008;3:8–22.

Chest X-ray in IPF

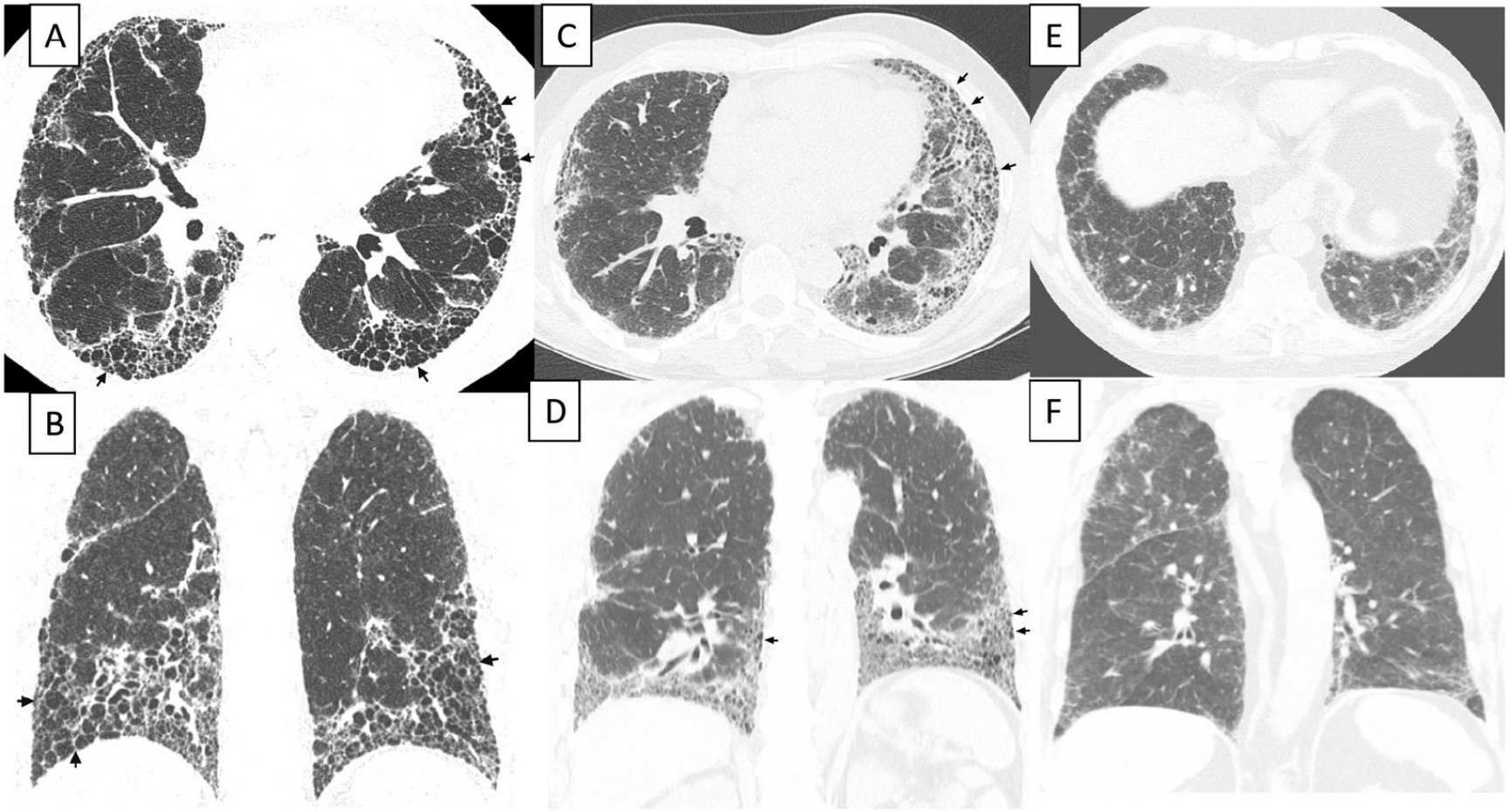
- Non-specific findings
- Symmetric peripheral, bibasilar reticular markings
- Low lung volumes
- Heart borders often hazy or poorly defined
- ~10% patients - normal chest X-rays





Oldham JM. *Respiratory Med*. 2014;108(6):819-829.
Meltzer EB. *Orphanet J Rare Dis*. 2008;3:8.

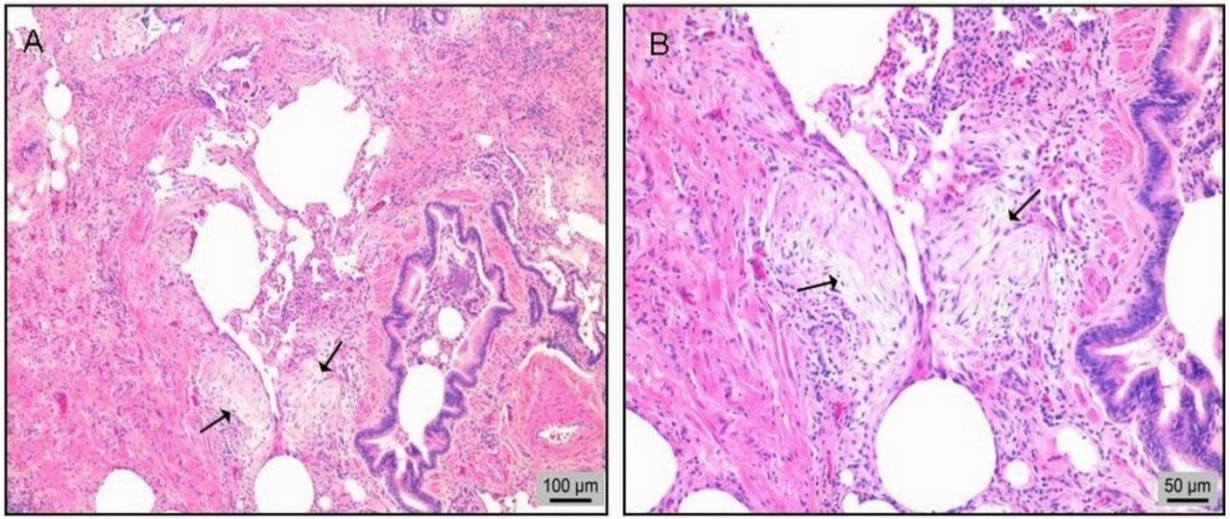
HRCT in IPF



7. Raghu G et al. *Am J Respir Crit Care Med*. 2011;183(6):788-82.

Lung biopsy can confirm IPF diagnosis when HRCT is not conclusive

IPF lung **IPF lung (higher magnification)**



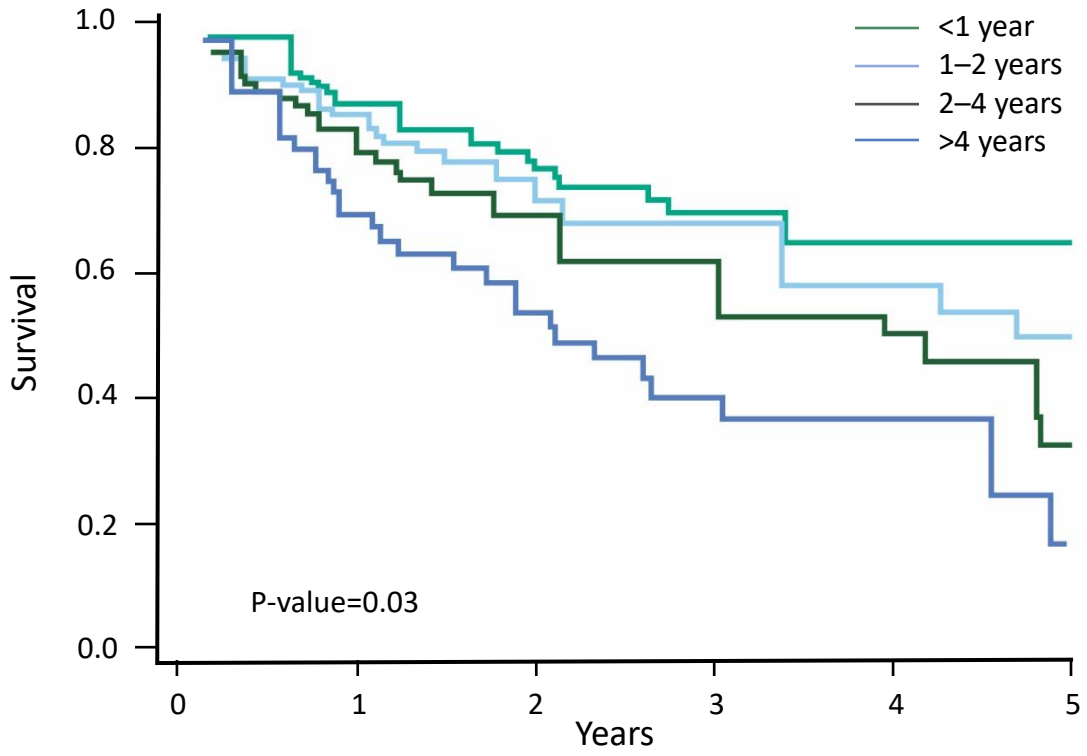
IPF diagnosis can be confirmed by histopathology, if all four UIP criteria are met:

1. Marked fibrosis ± honeycombing in a predominantly subpleural/paraseptal distribution
2. Presence of patchy involvement of lung parenchyma by fibrosis (left, arrows)
3. Presence of fibroblast foci (right, arrows)
4. Absence of features suggesting an alternate diagnosis

Raghu G et al. *Am J Respir Crit Care Med*. 2011;183(6):788–824.

Importance of early referral to a specialty centre

- Delayed access to a tertiary care centre is associated with a higher mortality rate, independent of disease severity



Lamas DJ et al. *Am J Respir Crit Care Med*. 2011;184(7):842–847.

And do NOT forget for everyone!

- Exercise
- Weight optimization
- Oxygen for those meeting criteria
  - Albeit usually NOT a treatment for dyspnea
- Vaccinate
  - Pneumonia
  - Influenza
  - Pertussis
  - Covid
  - Shingles

Summary: Dyspnea

Re-evaluate history, look for subtle clues in history and exam

Pulmonary, Cardiovascular or Other?

Conditions can co-exist

Additive treatment strategies

Basics: vaccinate, smoking cessation, exercise, educate

Optimization of outcomes not the same as symptoms:

- ICS in appropriate COPD patients
- SGLT2, Sacubitril/valsartan in CHF patients

Proposed Primary Care Approach to Assessing Adults with Chronic Dyspnea

airways group

First steps

To aid in diagnosis/referral/initial treatment

- ❑ **History**
- ❑ Consider duration of symptoms
- ❑ Review Red Flags (see below)
- ❑ Review medical history, including medications:
  - ❑ Smoking/Lung exposures
  - ❑ CV risk and history (CAD, BP)
  - ❑ Occupational/environmental issues or travel exposure?
  - ❑ PE risk: Virchows triad
  - ❑ Sputum
  - ❑ Constitutional symptoms
- ❑ Perform **physical exam**

Tests

Investigations

May do multiple in sequence or as a group in primary care mostly, some requiring referral/hospital settings

Primary Care

- ❑ **Oximetry**
- ❑ **Peak flow in office**
- ❑ **Spirometry**
- ❑ **CXR**
- ❑ **ECG**
- ❑ **Echocardiogram**
- ❑ **Routine labs: CBC, TSH, Bicarb**

Primary or Secondary care

- ❑ **Special labs: proBNP, D-dimer, FENO**
- ❑ **Tests for cardiac ischemia**
- ❑ **Full PFTs**
- ❑ **Walk test**
- ❑ **Blood gases**

Institute therapy

While patient waits to be seen by specialist

-follow up

- ❑ Consider other potential causes / additional investigations
- ❑ Consider possibility of >1 cause
- ❑ Assess adherence to treatment of potential underlying conditions
- ❑ Re-evaluate patient for (subtle) symptoms
- ❑ Continue to support your patient through their journey

Red flags for acute issues

- Hemoptysis
- Smoker >45 years with new cough, cough change or coexisting voice disturbance
- Prominent dyspnea, especially at rest or at night
- Hoarseness
- Systemic symptoms, including fever, weight loss, peripheral edema with weight gain
- Trouble swallowing while eating or drinking
- Vomiting
- Recurrent pneumonia
- Chest pain
- Calf pain

Potential causes

- Asthma
- COPD
- Cardiac ischemia
- Infection
- Interstitial lung disease
- PE/CTEPH
- Cardiomyopathy
- HFpEFr
- HFrEFr
- Pulmonary Hypertension

Additional investigations (depending on access and situation)

- Further Cardiac workup (Holter monitoring, Stress Echo)
- HRCT
- Ventilation perfusion lung scan
- Bronchial provocation
- Bronchoscopy
- 24-hour oesophageal pH monitoring
- Coronary Angiogram left vs right sided
- Lung biopsy

Kaplan, 2021 work in progress


HOUSE M.D.
"You can think I'm wrong,
but that's no reason to quit thinking."
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