

第六届北京协和呼吸病学峰会 暨第二届ACCP呼吸与危重症最新进展临床峰会

Proceedings of the 6th Peking Union Conference on Respiratory Medicine
and the Second ACCP-China Annual Conference Series
on Pulmonary and Critical Care in Beijing

论文集

蔡柏蔷 肖 毅 主编

中国协和医科大学出版社

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会议 PPT

Improving Patient Care Through Education

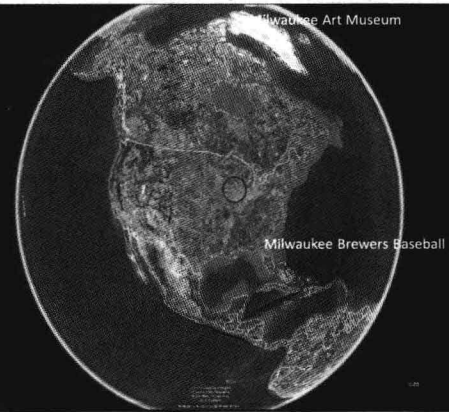
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David D. Gutterman, MD, FCCP
ACCP President



Milwaukee, Wisconsin



Milwaukee, Wisconsin

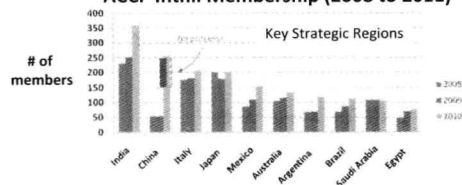


ACCP Overview

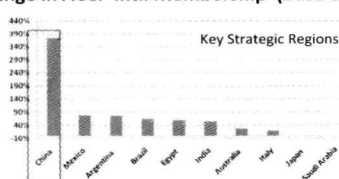
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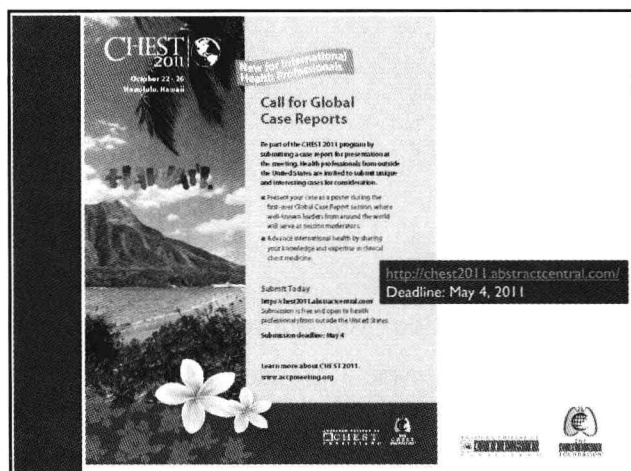
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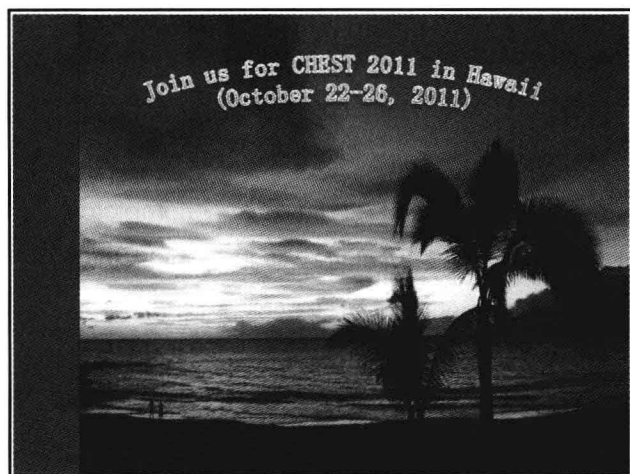
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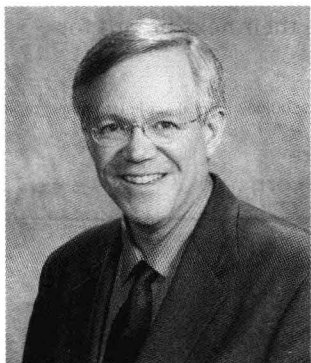
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个人简介



David D. Gutterman, MD

Dr. Gutterman is the Northwestern Mutual Professor of Cardiology. He is involved actively in clinical practice, supervises an NIH funded research laboratory, and provides senior administrative oversight of research administration at the Medical College of Wisconsin. He has served in a leadership role in a variety of national and international cardiovascular scientific organizations. He is currently the president of the American College of Chest Physicians, and has served as chair of the American Physiological Society's Cardiovascular Section, and as chair of the American Heart Association Scientific Publishing Committee. Dr. Gutterman is the Senior Associate Dean for Research at the Medical College of Wisconsin. He received an M. D. degree from the University of North Carolina, and completed residency training in internal medicine and fellowship in cardiology at the University of Iowa where he joined the faculty in 1987. Dr. Gutterman was promoted to Professor of Medicine in 1998 prior to moving to the Medical College of Wisconsin.

Dr. Gutterman's investigative interests focus on regulation of human vascular reactivity both at the fundamental and translational research levels. His research efforts span basic and clinical science related to vascular health and disease. His laboratory is one of only 2 worldwide that examines the ability of small blood vessels to control blood flow to the human heart. Dr. Gutterman has also undertaken studies to examine the earliest changes that occur in the development of atherosclerosis (clinical endothelium dysfunction) and has used this technique to examine the beneficial and detrimental roles of various exercise regimens and of dieting on cardiovascular health. Dr. Gutterman had authored more than 100 articles and reviews related to cardiovascular function and pathophysiology.

Peking University Medical College, Beijing

**Cardiovascular Complications
of Sleep Apnea**

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April 2011

Disclosures: None

Milwaukee, Wisconsin

Green Bay Packers
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Medical College of Wisconsin, Milwaukee

Cardiovascular Disease and Sleep Apnea

Objectives

- Define epidemiology of cardiovascular disease in sleep disordered breathing
- Examine prognosis and management of SDB and CVD

Address the following questions:

1. Which cardiovascular complications are associated with sleep disordered breathing (SDB)? How common are they?
2. Does treating SDB influence cardiovascular disease?
3. What are the clinical practice implications for patient management?



Cardiovascular Disease and Sleep Apnea

Epidemiology

SDB prevalence in US: 15 million adults (24% for men, 9% for women) (in Japan, men=3%, women=0.5%)

1 in 5 has some degree of OSA (AHI>5/hour)

1 in 15 with mod-severe OSA (AHI>15/hour)

only ~15% are referred for evaluation

CSA Prevalence in general population is 0.5-2%, more in older population, diabetes, CHF

CVD Prevalence (MI, angina, CHD) in US =6.5%, with 4% MI

Both SDB and CVD are relatively common occurrences



Association between Sleep Disordered Breathing & Cardiovascular Disease

Cardiovascular conditions associated with SDB

- **Congestive Heart Failure**
 - Systolic Dysfunction
 - Diastolic Dysfunction
- **Stroke**
- **Hypertension**
 - Left Ventricular Hypertrophy
 - Pulmonary Arterial Hypertension
- **Arrhythmias**
 - Tachyarrhythmias
 - Ventricular Tachycardia and Fibrillation;
 - Sudden Cardiac Death
 - Atrial Fibrillation
- Bradycardias



Association between Sleep Disordered Breathing & Cardiovascular Disease

Cardiovascular conditions associated with SDB

Congestive Heart Failure:

- "45/45 rule": SDB is present in 45% of patients with LVEF<45%
- In CHF, most SDB is Central Sleep Apnea/Cheyne-Stokes Respiration, although 11-37% is OSA
- higher frequency of OSA in diastolic dysfunction (~50%)

Somers et al. *Circulation* 2008
Yaggi et al. *NEJM* 2005

Minoguchi et al. *AJRCCM* 2007
Shahar et al. *AJRCCM* 2001

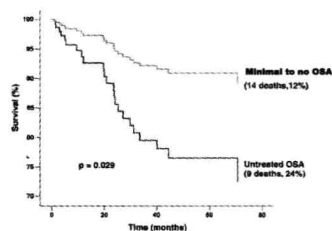


Does OSA Influence Survival in CHF?

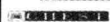
- 164 patients with CHF
- Polysomnography showed OSA or (minimal to no SA)
- baseline cardiovascular characteristics similar in those with OSA/M-NSA
- f/u for 2.9±2.2 years

• Severe OSA associated with higher mortality (8.7 deaths/100 pt-years) than mild or no OSA (4.2)

• CPAP treatment of severe OSA tended to reduce mortality (p=0.07)



- OSA increases mortality in CHF;
- CPAP might reduce mortality



Wang et al. *JACC*, 2007



Association between Sleep Disordered Breathing & Cardiovascular Disease

Cardiovascular conditions associated with SDB

CHF:

- SDB is present in 45% of patients with LVEF<45%
- In CHF, most SDB is CSA, although 11-37% is OSA
 - rare daytime somnolence
 - male > female prevalence
 - higher frequency of OSA in diastolic dysfunction (~50%)

Stroke:

- In patients with stroke, 17% have OSA, 21% with CSA
- up to 62% of TIAs are associated with SDB
- conversely, 25% of those with moderate OSA have MRI-proven CVA vs. 6% of control group without SDB



Somers et al. *Circulation* 2008
Yaggi et al. *NEJM* 2005

Minoguchi et al. *AJRCCM* 2007
Shahar et al. *AJRCCM* 2001



Risk of Stroke or Death in OSA (primary risk)

Observational cohort study of subjects referred for SDB
Control: AHI<2 (n=325)
OSA: AHI≥35 (n=697)

After adjusting for other factors, baseline OSA was the ONLY factor that predicted stroke-free survival.

OSA predicts increased risk of CVA or death of any cause (~2X higher), independent of other risk factors

Covariate	Unadjusted Hazard Ratio (95% CI)	Adjusted Hazard Ratio (95% CI)
Age (yr)	1.07 (1.06-1.11)	1.06 (1.06-1.11)
Male sex	0.99 (0.62-1.68)	0.79 (0.49-1.27)
Race		
White (reference group)	1.00	1.00
Black	0.46 (0.19-2.33)	0.48 (0.19-2.40)
Other	0.41 (0.14-1.08)	0.44 (0.14-2.05)
Diabetes mellitus	0.93 (0.47-1.92)	0.99 (0.49-2.02)
Current smoker	1.21 (0.60-2.44)	1.46 (0.73-2.93)
Current consumption of alcohol	1.01 (0.66-1.52)	0.94 (0.75-1.18)
Dyslipidemia	1.54 (1.02-2.36)	1.71 (0.76-2.26)
Atrial fibrillation	1.56 (0.78-3.12)	0.91 (0.45-1.83)
Hypertension	1.04 (0.64-1.68)	1.04 (0.65-1.65)
Hypertension	1.45 (0.95-2.23)	1.73 (0.73-2.40)
Cardiovascular disease	2.24 (1.49-3.36)	2.47 (1.27-4.85)

*Hazard ratios were adjusted for all other covariates in the model. CI denotes confidence interval.

***SDB is independent risk factor for mortality after CVA (secondary risk)**
***5% increase in total mortality for each unit increase in AHI**

Yaggi et al. NEJM 2005

Parra et al. EurRespir J 2004

Association between Sleep Disordered Breathing & Cardiovascular Disease

Cardiovascular conditions associated with SDB

HTN:

- Among those with SDB, 50% have HTN;
- Similarly, in mainland China, 56.2% of those with SDB have HTN
- conversely 30% of those with HTN have SDB (only 15% are diagnosed).
- In refractory HTN (3 drugs), 83% have OSA
- OSA is an independent risk factor for HTN

Chen, He. Chin J TubercRespir Dis 2007
Somers et al. Circulation 2008
Sorajja et al. CHEST 2008

Do Patients with SDB Develop Hypertension?

Wisconsin Sleep Cohort Study: n=709
8 year follow up for development of HTN
45% women
Avg. age = 45 years
BMI=29
Follow up for 4 years

TABLE 3. Adjusted Odds Ratios for Hypertension at a Follow-up of 8 Years, According to the Apnea-Hypopnea Index at Baseline *

Baseline Apnea-Hypopnea Index	Odds Ratio, Adjusted for Baseline Hypertension Status and Mortality Risk Factors (Age and Sex)	Odds Ratio, Adjusted for Baseline Hypertension Status and Mortality Risk Factors (Age and Sex) and Waist Circumference and Quotient Use
0-5	1.0	1.0
6-15	1.65 (1.35-2.03)	1.42 (1.14-1.76)
16-30	2.74 (1.82-4.12)	2.80 (1.29-6.19)
>30	4.44 (2.40-8.26)	4.47 (2.07-9.69)
P for trend	<0.001	0.002

*Hypertension was defined as a blood pressure of at least 160/90 mm Hg or the use of antihypertensive medications. Data on 688 follow-up sleep studies from 709 participants were analyzed. The odds ratio and confidence intervals were adjusted for the fact that the participants completed one follow-up sleep study. BMI denotes body mass index.

†This category served as the reference group.

‡P values are not the linear trend of the logistic regression coefficient (log of the odds ratios).

The presence of SDB was predictive of HTN within 4 years, even with mild disease and accounting for several other factors

Peppard et al. NEJM 2000

Link between OSA and Cardiovascular Disease

TABLE 1. UNIVARIATE RELATION OF THE APNEA-HYPOPNEA INDEX (AHI) TO VARIOUS CHARACTERISTICS AND PREVALENT CARDIOVASCULAR DISEASE

Variable (mean or %)	I	II	III	IV
Quartile range	0-1.3	1.4-4.4	4.5-11.0	>11.0
Age, yr (mean)	61	63	65	66
Race, % white	78	79	78	77
Sex, % female	70	57	49	35
Body mass index, kg/m ² (mean)	25.9	27.7	29.3	30.9
Smoking status, % never smoked	49	48	44	43
Total cholesterol, mg/dL (mean)	203	208	207	205
HDL cholesterol, mg/dL (mean)	54	50	48	46
Diabetes, %	6	9	11	15
Self-reported hypertension, %	30	35	42	46
Systolic blood pressure, mm Hg (mean)	126	130	132	133
Use of antihypertensive medications, % using	32	36	43	50
Prevalent cardiovascular disease, n (%)	183 (11)	205 (14)	303 (20)	357 (23)
Prevalent coronary heart disease, n (%)	134 (9)	166 (11)	248 (16)	290 (19)
Prevalent heart failure, n (%)	13 (0.9)	22 (1.5)	38 (2.5)	50 (3.3)
Prevalent stroke, n (%)	15 (2.3)	47 (3.1)	70 (4.6)	80 (5.3)

Definition of abbreviations: HDL = high-density lipoprotein.

*p<0.05 for linear trend

- Sleep Heart Health Study
- 6,424 patients with PSN; association study
- 1023 (16% overall) with self-reported Cardiovascular Disease, stratified by AHI

Shahar et al. Am J Respir Crit Care Med. 2001

OSA as Predictor of Cardiovascular Mortality in CAD

- *69 patients with CAD stratified by OSA +/-
- *5 year follow-up
- *No difference in age, gender, BMI, smoking, HTN, DM.

Variable	OSA (+) (n = 16)	OSA (-) (n = 43)	p Values
Myocardial infarction, n (%)	5 (31.3)	8 (18.6)	NS
Stroke, n (%)	1 (6.3)	5 (11.6)	NS
Mortality, n (%) (all cardiovascular)	6 (37.5)	4 (9.3)	0.018

Definition of abbreviations: NS = not significant.
*Comparison of groups by Fisher exact test (two-tailed).

Cardiovascular mortality is 4 fold greater in CAD patients who also have OSA

Peker et al. AJRCCM 2000

Association between Sleep Disordered Breathing & Cardiovascular Disease

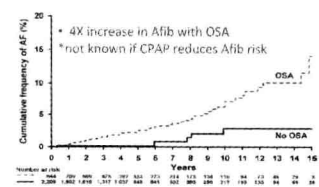
Cardiovascular conditions associated with SDB

Arrhythmias:

- In patients with Afib, 49% have OSA vs. 33% in a control group presenting to a general cardiology practice (conversely in patients with SA, 20% have atrial fibrillation)
- After cardioversion for Afib., persistent OSA doubles chance of recurrent afib to 80%

*3542 adults < 65 y.o.

- *PSN at baseline
- *New onset AF over average of 4.7 years follow-up



Bradley et al. Lancet 2009
Garrigue et al. Circulation 2007

Gami et al. JACC 2007
Caples et al. Prog. Card. Dis. 2009

Association between Sleep Disordered Breathing & Cardiovascular Disease

Cardiovascular conditions associated with SDB

Arrhythmias:

- In patients with Afib, 49% have OSA vs 33% control group presenting to a general cardiology practice
- OSA is risk factor for Afib even correcting for other risk factors (in patients <65 years of age) Association much stronger for CSA
- nocturnal arrhythmias in 50% of OSA (vs. 10% without): NSVT, 2nd degree AVB, PVC's
- 59% of patients with pacemakers have SDB (improved with Chronic Resynchronization Therapy (biventricular pacing))
- It is controversial if there is an increased prevalence of bradyarrhythmias, but when present, they do resolve with treatment of OSA

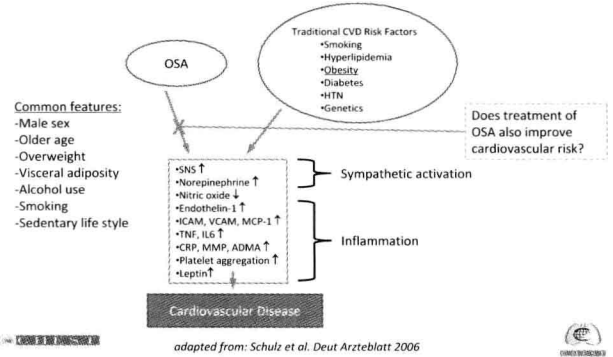


Bradley et al. Lancet 2009
Garrigue et al. Circulation 2007

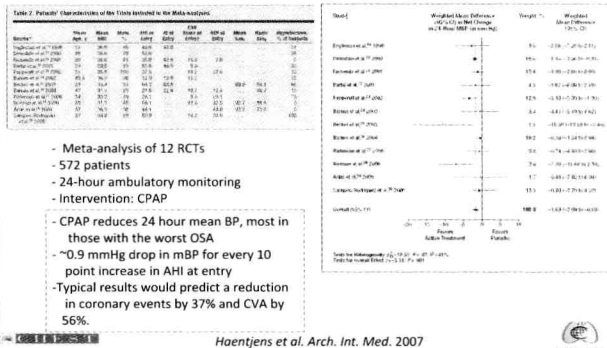
Gami et al. JACC 2007
Caples et al. Prog. Card. Dis. 2009



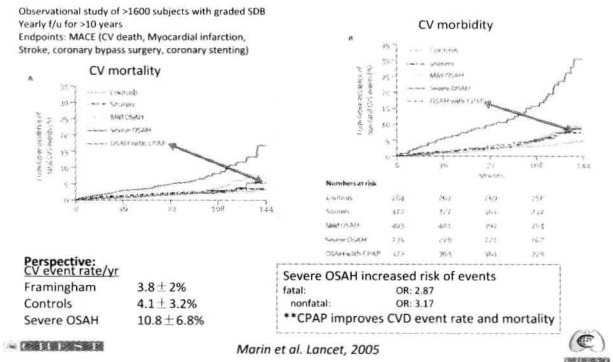
Pathophysiological Link Between OSA and CVD



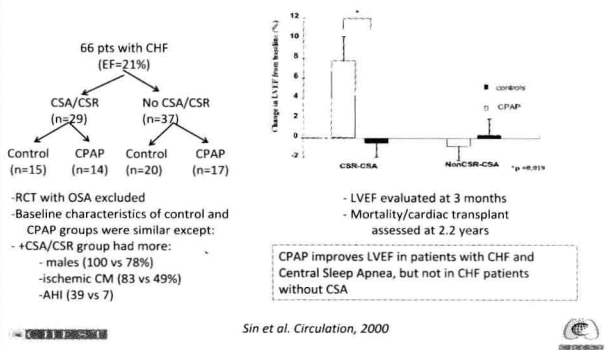
Does Treatment of OSA Improve HTN – a meta-analysis



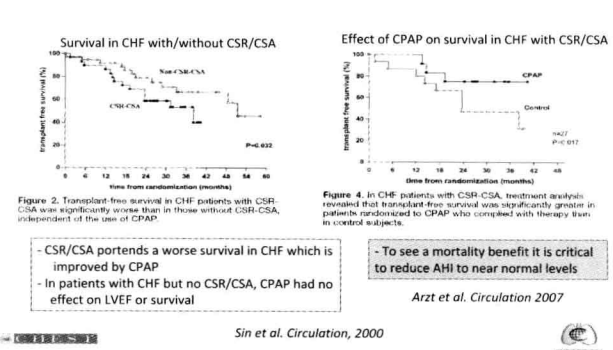
Does Treatment of OSA Reduce Cardiovascular Events?



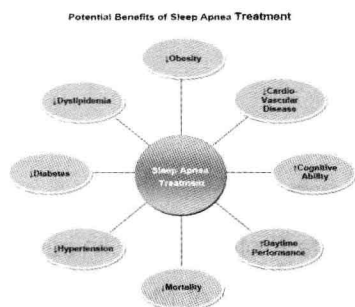
Does Treatment of CSA Improve Ventricular Function Severe Heart Failure?



Does Treatment of CSA Improve Survival in Severe Heart Failure?



Systemic Benefits of Treating Sleep Apnea



Jean-Louis et al. J Clin Sleep Med 2008

Summary and Recommendations

Sleep Apnea is pathophysiologically and clinically linked to Cardiovascular Diseases including

- stroke
- arrhythmias (Sudden Death, atrial fibrillation, PVC's, NSVT)
- atherosclerosis
- hypertension
- heart failure

A variety of observational studies (although not all) show that treatment of sleep disordered breathing, improves cardiovascular risk factors and reduces major adverse cardiovascular events

Clinical Implications and Caveats

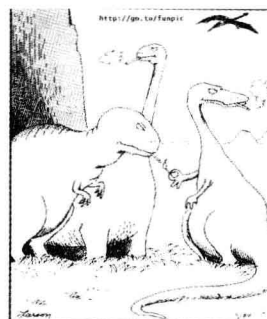
- Pts. with OSA and HTN should be aggressively treated for risk factors and screening for CAD considered. (43% will have significant disease)
- CPAP for treatment of CSA in CHF is not yet well-established. A successful titration to reduce AHI to near normal values may be necessary to improve mortality.
- Snoring alone does not increase risk of cardiovascular disease.

Summary and Recommendations

- Who should be screened for SDB (high risk groups):
 - CHF, CAD, HTN, atrial fibrillation, if with one of the following:
 - hypersomnolence
 - witnessed apneas
 - obesity
 - nocturnal angina
 - pacemaker patients
 - Sudden Cardiac Death episode during sleep

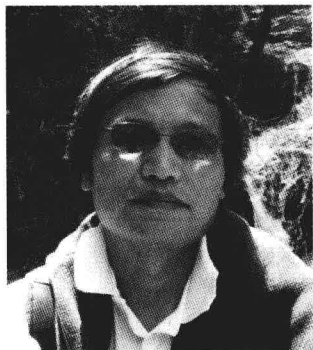
- Patients with OSA and HTN should be considered for treated with CPAP *independent of daytime sleepiness* (the anticipated 2-10 mmHg drop in BP translates to a 10-50% reduction in mortality and serious cardiovascular morbidity, based on pharmacological treatment of HTN). This is based on observational studies and conflicts with several smaller prospective studies.

The End



The Real Reason Dinosaurs Became Extinct

个人简介



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Dr. Renli Qiao is an Associate Professor of Clinical Medicine at Department of Medicine, Keck School of Medicine, University of Southern California in Los Angeles, California, USA. He received his MD in Shanxi Medical College and his PhD in Peking Union Medical College. He is one of the very few with quadruple Board Certifications in Internal Medicine, Pulmonary Diseases, Critical Care and Sleep Medicine. He is a Fellow and a member of Executive Committee of American College of Chest Physicians. His research interest is in pulmonary hypertension and phenotypic transdifferentiation of alveolar epithelial cells. He spends his spare time in Chinese writing and enjoys a wide readership with his series of “As a Doctor in America” which appears regular in an oversea internet-based magazine.

COPD and Pulmonary Hypertension

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Los Angeles, CA

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Night Skyline of Los Angeles

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Why discuss?

1. Pulmonary hypertension in COPD and ILD. Weitzenblum E et al. *SeminRespirCrit Care Med* 30:458-470, 2009
2. PHTN in pts with COPD. Barbera JA, Blanco I. *Drugs*. 1153-71, 2009
3. Rt heart function in COPD. MacNee W. *SeminRespirCrit Care Med*. 31:295-312, 2010
4. PHTN in COPD. Minai OA et al. *Chest*. 137:39S-51S, 2010

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Hypoxic Pulmonary Vasoconstriction



Von Euler, US



Liljestrand, G

(1946). "Observations on the pulmonary arterial blood pressure in the cat". *Acta Physiol. Scand* 12 (301-320)

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The Early Report of CorPulmonale

WHO. Chronic corpulmonale: a report of
an expert committee. WHO Tech Rep Ser.
213:35, 1961

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Harrison's Principles of Internal Medicine

Corpulmonale, often referred to as *pulmonary heart disease*, is defined as dilation and hypertrophy of the right ventricle (RV) in response to diseases of the pulmonary vasculature and/or lung parenchyma that are sufficient to cause **pulmonary hypertension**.

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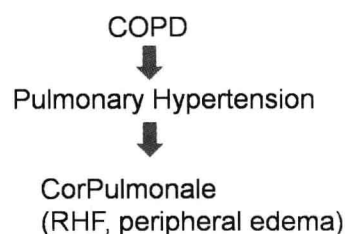
Harrison's Principles of Internal Medicine

Many of the signs that are encountered in cor pulmonale are also present in HF patients with a depressed EF, including:

1. tachypnea
2. **elevated JVD, hepatomegaly**
3. **peripheral edema.**

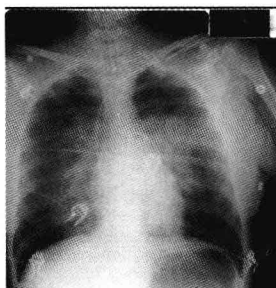
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The Natural History of COPD



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Pulmonary Arterial Catheterization



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Pulmonary HTN in COPD

- In COPD pts (120) evaluated for LVRS
- FEV1 = 27% predicted
- **In 91%, mean PAP > 20 mmHg**
- In 5%, PAP > 35 mmHg
- Poor correlation between PFT and PAP
- PAP correlated with occlusion pressure (air trapping vs LV dysfunction)

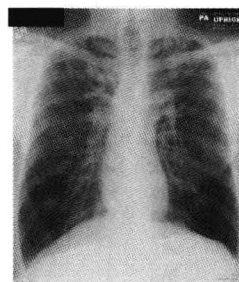
Scharf SM et al, AJRCCM 166:314-22, 2002

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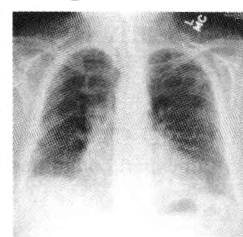
Why should a mild PHTN cause RHF in COPD?

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PHTN from other lung diseases



Cystic Fibrosis



Idiopathic Pulmonary Fibrosis

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