MD Seminar
Pulmonary manifestation of cardiac diseases.

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Pulmonary circulation.

- Low pressure & high flow system

	Range	mean
Pulm art pressure (mmhg)	25/10	15
P.Capillary pressure	6-9	7
P. Venous pressure	1-4	2
P. vascular resistance (PVR)	1-4	3
P. Blood flow (L/min)	4-6	5

Causes

- Main way by which heart influence lung is by increasing pulmonary venous pressures.
- Main causes:
  1. Mitral valve diseases
  2. CHF
     - -IHD
     - -cardiomyopathy
     - -myocarditis
     - -pericardial restriction.

Starling principle and interstitial fluid dynamics

micro-vascular filtration and lymph flow. α Capillary pressure .

Pulmonary venous hypertension

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Fluid in interstitial space

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Alveolar septae

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Peri-bronchial and peri-vascular spaces (safety valve)

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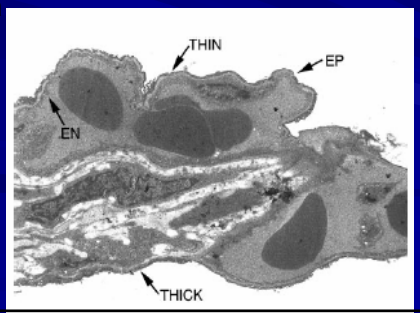
Via lymphatics to systemic veins

- in heart failure, systemic venous pressures are elevated
- This impair with proper lymphatic drainage.

Safety factors

- Extremely low permeability of alveolar epithelium.
- Active Na+ transport by alveolar epithelial cell.
- Perimicrovascular and peribronchial interstitial compliance
- Pleural spaces (25% of pulm edema fluid in experimental animals) (Broaddus VC, J appl Physiol.)
- Resorption into blood vessels
- expectoration



Histo-pathologic change in PVH

- PVH→ excess interstitial edema →organized and form hyaline material.
- Medial hypertrophy of muscular pulmonary arteries.
- Muscularisation of smaller branches → narrowing.

MECHANISM

- ❖ Sustained hypertension---- stimulates growth factor release
- ❖ e.g. fibronectin ,tenascin – C + release of endogenous serine elstase.
- ❖ Serine elastase breaks internal elastic lamina.
- ❖ Now growth factors directly stimulate muscular layer of B.vessels.

Pulmonary capillary stress failure
changes in blood gas barrier

Pulmonary venous hypertension

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Disruption of endothelial and alveolar epithelial layer

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Thickening of basement membrane

(pars dense)

↓

Alveolar fibrosis.

MECHANISM:

Changes in gene expression for extra-cellular matrix protein.

Pulmonary hemodynamics

- Poiseuille equation

pressure = flow x resistance

OR

Resistance = pressure / flow.

- Pulmonary venous resistance (PVR)
- Measured in “wood units”
- Which is “mmhg/ l/min”
- In normal adults PVR = 1 wood unit.

Pulmonary hemodynamics

- Butler etal (J Am coll Card,1999)

	Controls	CHF
O2 uptake (ml/min/kg)	35	13.7
PA pressure (mmhg)		
At Rest	12	24
At exersice	30	38
PVR (woods unit)	**Resistance = pressure / flow.**	
At rest	1.0	1.9
At max exersice.	0.6	2.0

- Fall in PVR was not seen in patient of CHF.
- **Inference :** in CHF recruitment of reserve cap beds is not possible because of elevated resistance in pulm circulation.

Sustained PHT : effect

- Gibbs etal (J Am Col Card,1990)
- Studied level of pulm pressure in day to day activities.

Peak tredmill pressure	100%
Climbing stairs	90 %
Walking at level ground	73%
Lying supine	63%

- **Inference :** these episodic rises in pressures; causes ongoing pulmonary vascular changes in otherwise compensated heart failure.

Natural history of PVH

- **Prototype :** severe Mitral stenosis.

Early in course	recurrent pulmonary edema
Over next few years	Less severe episodes. Ongoing remodeling
Old cases	Infrequent pul.edema Severe PAH Cor pulmonale

Pulmonary complication of PVH

Complications	Description
Pulmonary function abnormalities	Decreased lung volume Airflow obstruction, especially in acute pulmonary edema Air-trapping Decreased lung compliance Arterial hypoxemia Decreased diffusing capacity (may be irreversible in long-standing CHF)
Sleep-disordered breathing	CSR-CSA OSA
Myopathy of peripheral and respiratory muscles	
Unusual manifestations	Hemoptysis, pulmonary hemorrhage Hemosiderosis Ossific nodules Mediastinal lymphadenopathy[138]

Pulmonary function abnormality
Changes in lung volume.

CHF / MS (Am J Respir Crit Care,2000)

as disease progressed NYHA I → IV

- TLC↓
- ERV↓
- RV↑
- FRC→

Acute pulmonary edema.

- no studies.
- Only one case report: reduction in all volumes

(Br Heart J,1951)

Pulmonary function abnormality
change in lung compliance

alveolar destruction and fibrosis produced by PVH.

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Reduction in lung compliance.

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Results in low TLC and VC

- NOTE: Despite fibrosis; lung recoil pressures do not increase (unlike ILD).
- REASON: simultaneously existing resp muscle dysfunction in CHF.

Pulmonary function abnormality respiratory muscle weakness.

- Meyer etal (circulation ,2001)
- Max insp pressure (MIP)—diaphragm— alt test SNIFF
- Max. exp.pressure (MEP)—Truncal muscle— COUGH
- 2yr prospective study of CHF pt.

Non surviver (n=57)	MIP 60% control
Survivor (n=187)	MIP 77%control

Pulmonary function abnormality respiratory muscle weakness.

	McPortland	Hughes	Hammond
VC % pred	93	86	60
MEP %control	100	80	49
MIP % control	81	76	40

- MIP is sensitive indicator of 1 year survival.
- PATHOGENESIS : structural , biochemical and functional changes in muscle cells.

e.g. atrophy of type 1 muscle fibers.

Pulmonary function abnormality
diffusion capacity

DLCO = overall alveolar capillary conductance

PULMONARY RESISTANCE TO GAS TANSFER:

1 / DLC0 = 1 / DM + 1 / VC

DM= membrane diffusive conductance.

VC= red cell conductance (pulmonary capillary volume)

Pulmonary function abnormality
diffusion capacity

	NYHA II	NYHA III	NYHA IV
DLCO (%CONTROL)	80	72	71
DM (%CONTROL)	68	35	61
VC (%CONTROL)	77	129	87

Role of angiotensis system

ACE -converts angiotensin
-inactivate bradykinin.

2 weeks of enalapril therapy (Guazzi etal circulation,1997)		
	CHF patient	Only sys HTN, no CHF
DLCO Change	84%→96%	No change

- blocked by asprin
- no effect from ARB

MECHANISM : ACE gene polymorphism (DD,ID,II)

DD genotype -- higher ACE levels, low DLCO, FEV1, VO2.

PULMONARY SYMPTOMS OF HEART DISEASE: Orthopnea

- Elevated diaphragm.
- ↑ respiratory resistance (upto 80%) on lying supine, which was reversed on sitting up.

	Normal		CHF	
	SITTING	SUPINE	SITTING	SUPINE
Breathlessness (VAS)	0	0	2.2	3.9
VC (L)	4.5	4.4	2.6	2.4
FRC (L)	3.7	3.05	3.3	3.1
FEV1(%pred)	103	97	76	66

PULMONARY SYMPTOMS OF HEART DISEASE : wheeze

ACUTE LVF: -geometric narrowing of airways
-increased vagal tone -- bronchoconstriction

- Light &George (arch int med)
- FEV1 / FVC ratio studied.
- Rise from 66% to 71% after treatment of acute LVF.

CHF:

overall pattern is restrictive
(1)FEV1/FVC = 80%
(2)TLC reduced.

- Increased methacholine responsiveness
- blocked by methoxamine (alpha agonist)

inference: mucosal vessel dilation , generalized mucosal swelling.

PULMONARY SYMPTOMS OF HEART DISEASES: poor exercise tolerance.

AT REST : -low O2 (Increasing with clinical severity of CHF)
-no change in PaCO2.
-rise in lactate levels (low pH)

AT EXERCISE: - low PaO2 (unrelated to clinical severity)
- low PCO2 levels
- earlier and higher rise in lactate levels.

Cause : (1) no recruitment of blood flow at apex during exercise
(2) regional VA/ Q mismatch.

(Wasserman etal, circulation,1997)

Interesting observation

REGIONAL VARIATION IN VENTILATION

- SERIES OF 180 pts of CHF with cardiomegaly.
  (Alexander etal, BMJ,1989)
- Ventilation checked by Krypton 81m gas
- OBSERVATION 1 : reduction in left lower lobe ventilation in supine posture which improved on turning prone.
- OBSERVATION 2 : CHF pt with large LV avoid sleeping on left side.
  (Wiener etal Am Rev Respir Dis,1990)

Sleep disorders in CHF

- **Cheyne stokes resp-central sleep apnea (CSR-CSA)**
  - frequently observed in severe CHF.
  - more in CHF with large LV & high PCWP.
  - PATHOGENESIS: respiratory control instability that is caused by fluctuation of CO_2 levels.

Incidence in CHF: of CSA - 40 %
of OSA- 11%

Effect of hypoxia on heart.

- Increased afterload
- increased sympathetic stimulation.

Unusual manifestations.

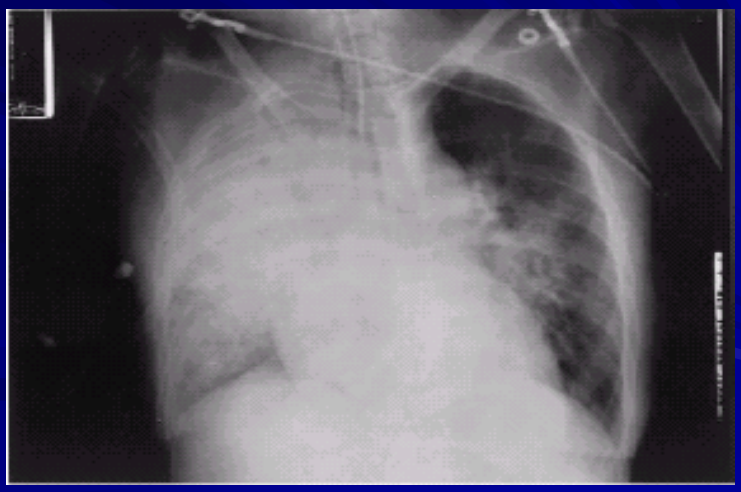
- Hemosidrosis from microvascular hemorrhage. (may be visible as small nodules on CxR.)
- OSSIFIC NODULES : consist of lamellated bone that forms with in alveoli.....(irregular shapes)
- increased interstitial markings---- pulmonary fibrosis.

Lung disease or heart disease.

- Not uncommon diagnostic dilemma in EMOPD.
- Over all accuracy of clinical evaluation=70%. (Mulrow CD etal, J Gen Int Med).
- on CxR extra shadows of coexisting lung disease may obscure pulmonary edema.
- Atypical pulmonary edema.

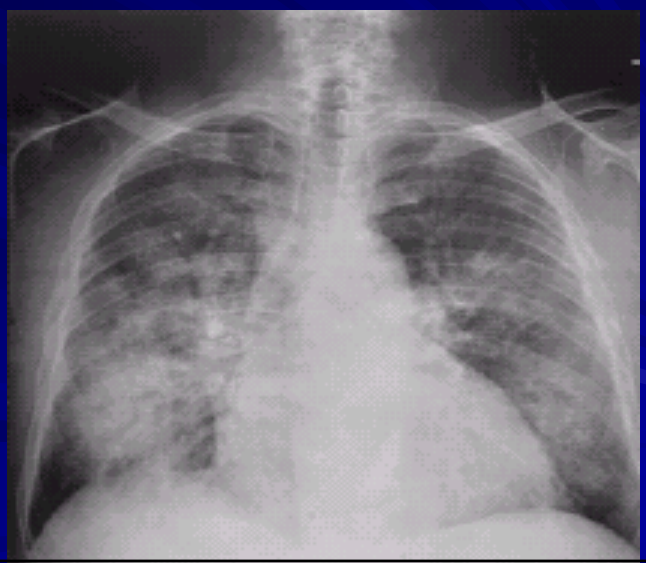
Atypical pumonary edema.

59/F, hypertensive, MR, resp failure.



Atypical pumonary edema.

60/M, CAD , progressive dyspnea



Lung disease or heart disease.

- CT scan may help:

  characteristic finding in CHF.

  – Septal thickening
  – ground glass opacities.
  – Peri-bronchovascular interstitial thickening.
  – Pleural effusion.
  – cardiomegaly

Lung disease or heart disease.

Pleural effusion: etio ?? CHF or lung dis.

- Indication for thoraco-centesis only if:
  – fever
  – unilateral effusion
  – B/L but sig. discrepancy in two side.

Lung disease or heart disease

Role of B-type natriuretic peptide (BNP)

- in emergency departments.
  - 80 to 90 % diagnostic accuracy
  - 96% negative predictive value at level <50 pg/ml.
  - Predictor of adverse outcome in CHF pts.

(Maisel etal, NEJM,2002)

Thank you