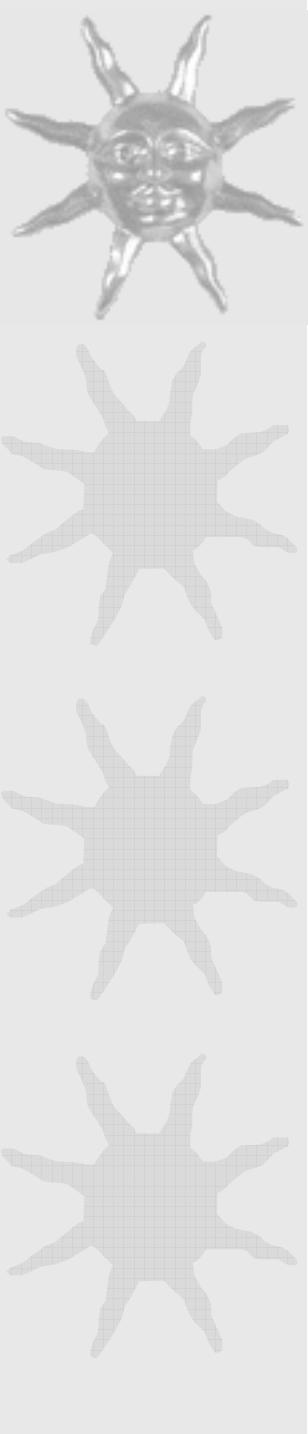


# *Parenchymal Lung Diseases*

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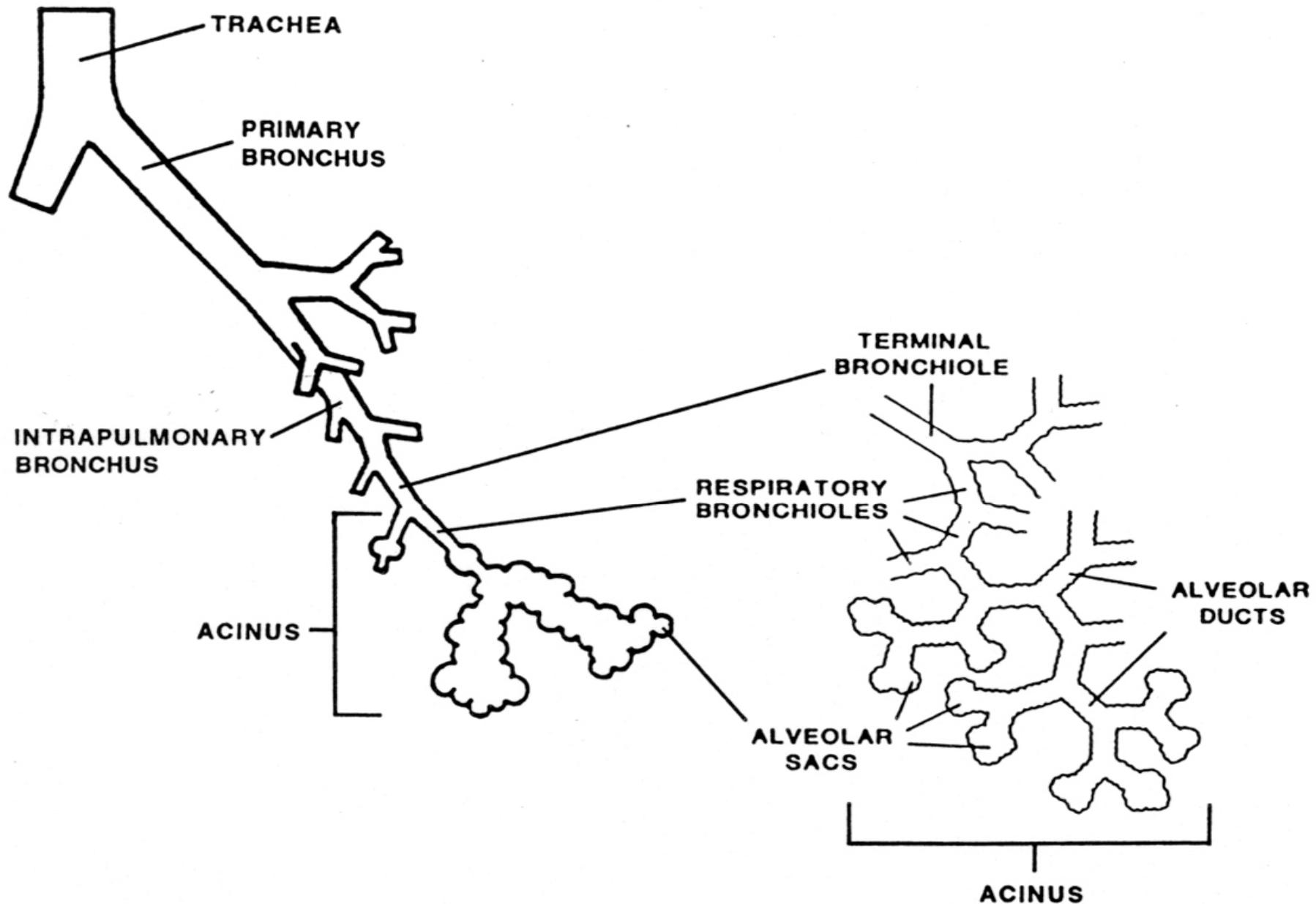
SS Visser  
Lung Unit  
UP and PAH



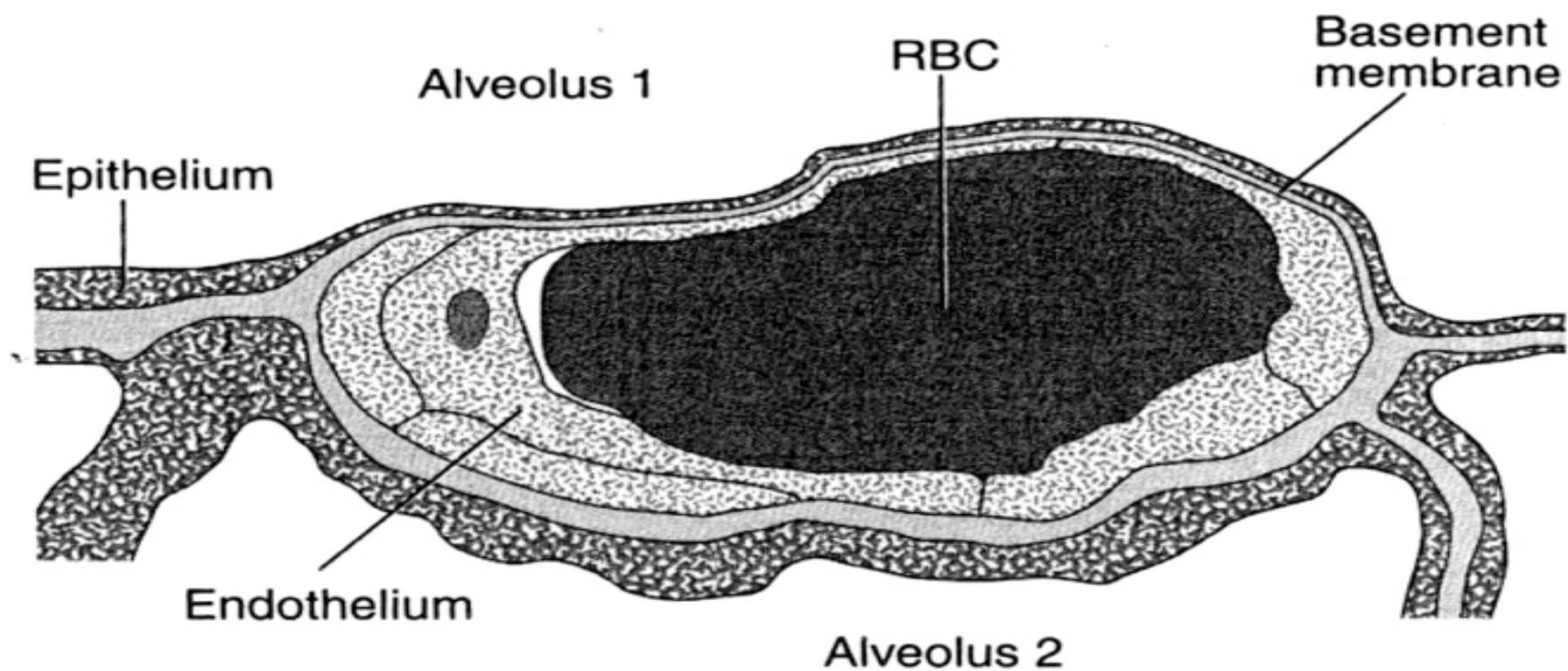
# *Contents*

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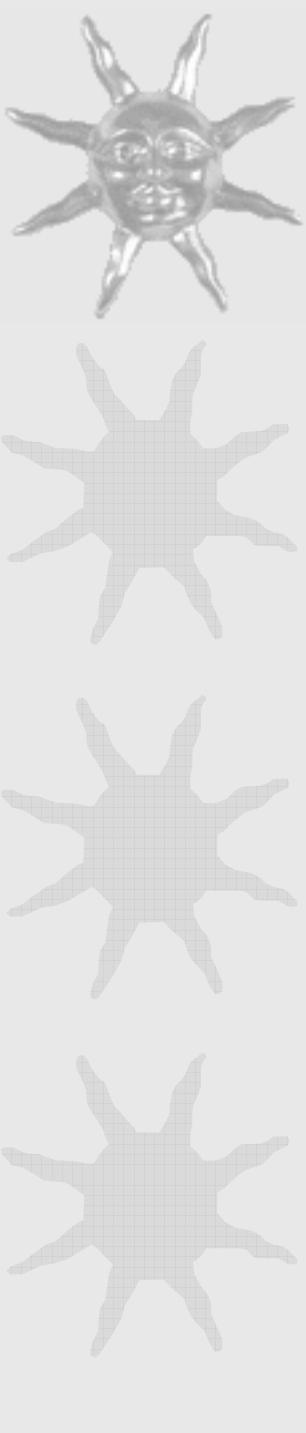
- ★ Structure of the lung parenchyma
- ★ Etiology of parenchymal diseases
- ★ Pathogenesis
- ★ Physical signs
- ★ Diagnosis
- ★ Complications
- ★ Management



The tracheobronchial airway tree: the trachea, primary bronchi, intrapulmonary bronchi, terminal bronchioles, and air passages of the pulmonary acinus.



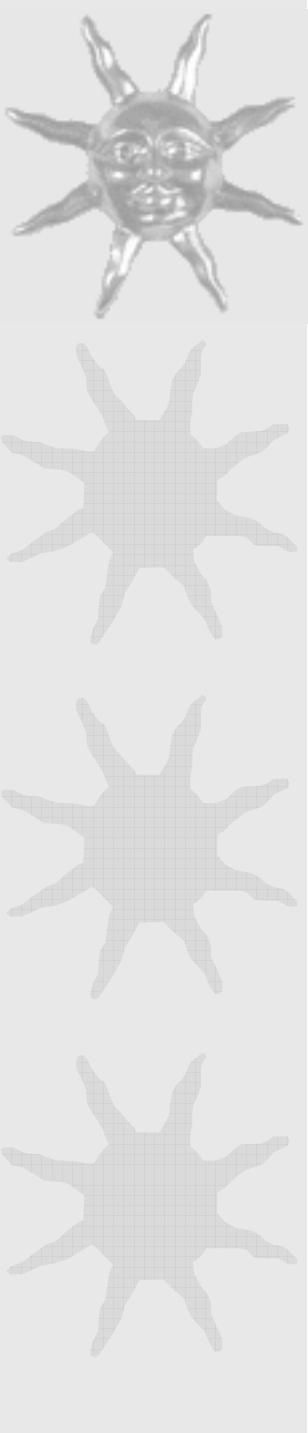
**The alveolar–capillary membrane.** This diagram of an electron micrograph shows the way the alveolar and capillary cells on one side of the alveolar septum fuse to form an ultrathin layer which offers little barrier to diffusion. The other side of the septum is thicker and provides physical support. RBC, red blood cell.



# *Structure of lung parenchyma: Respiratory compartment*

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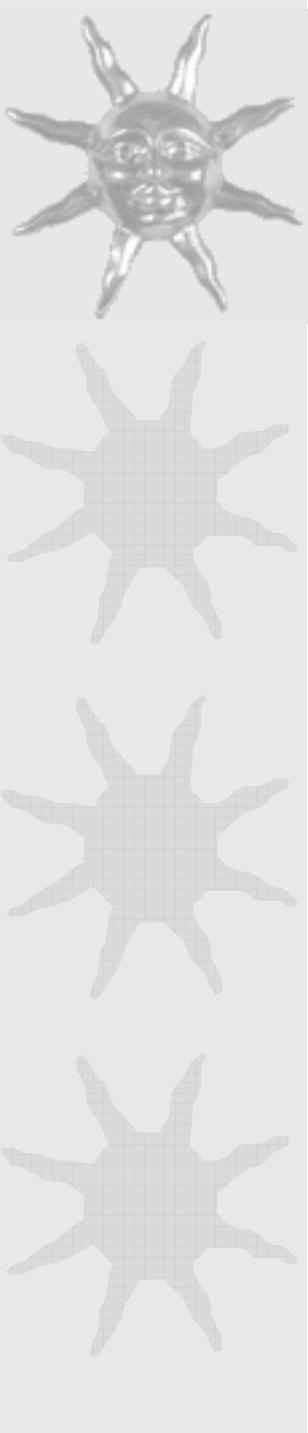
- ★ Contact area between air and blood = surface area of a tennis court, 30 000 acini and 300 million alveoli
- ★ Tissue barrier between air and blood is extremely thin to allow saturation of hemoglobin in erythrocytes within 1 sec.
- ★ Tissue barrier contains capillaries lined with complete endothelium
- ★ Alveoli are densely packed and lined by epithelium.
- ★ Interstitium contains few cells – fibroblasts with contractile filaments



## *Cells of the lung parenchyma*

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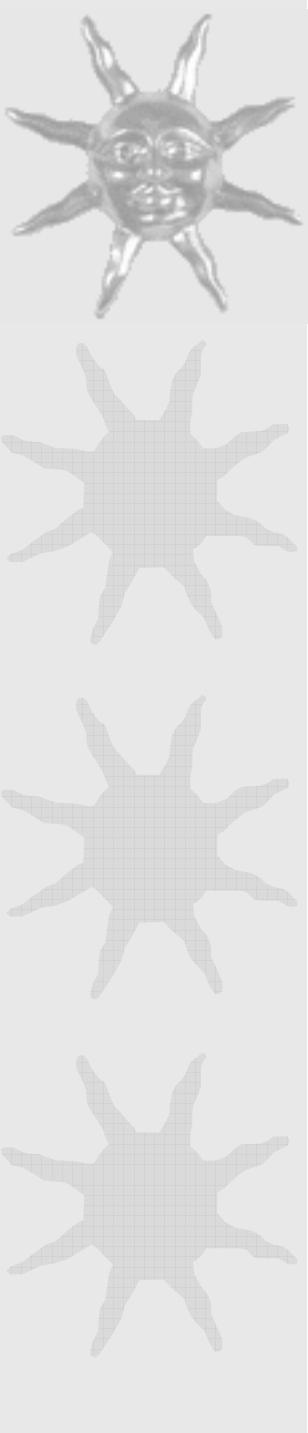
- ★ 75% of all lung cells are contained in the parenchyma.
- ★ The air:blood barrier consists of 25% epithelial cells, 25% endothelial cells, 35% interstitial cells and 15% collagen fibres.
- ★ Alveolar eithelium consists of lining cells (type 1), secretory cells ( type 2 pneumocytes) and brush cells



## *Function of parenchymal cells*

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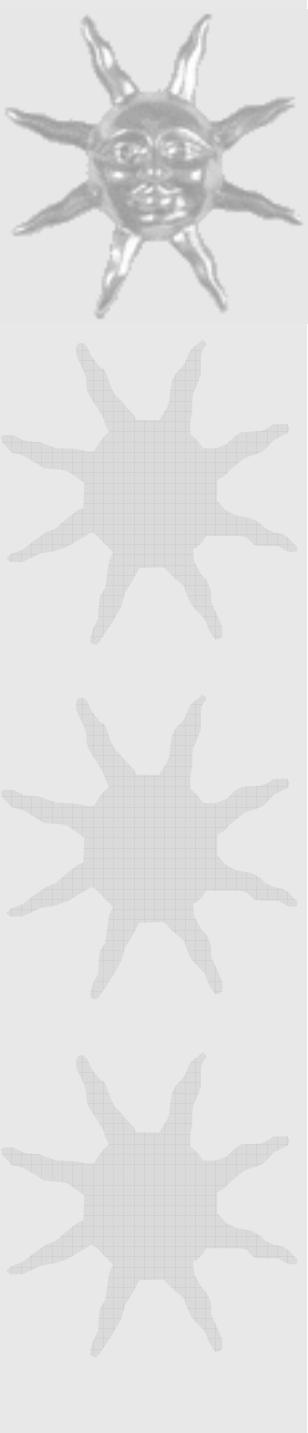
- ★ Type 1: tight junctions, cannot multiply (mitosis)
- ★ Endothelial cells: leaky junctions with exchange of  $H_2O$ , solutes and molecules between capillaries and interstitium.
- ★ Type 2: synthesis, storage and secretion of surfactant
- ★ Injury to type 1 cells: Type 2 cells undergo transformation into type 1 cells ( 2-5 days).
- ★ Catastrophic injury: repair mechanism too slow and respiratory failure occurs which require ICU and mechanical ventilation and supplemental  $O_2$ .



## *Parenchymal Defense system*

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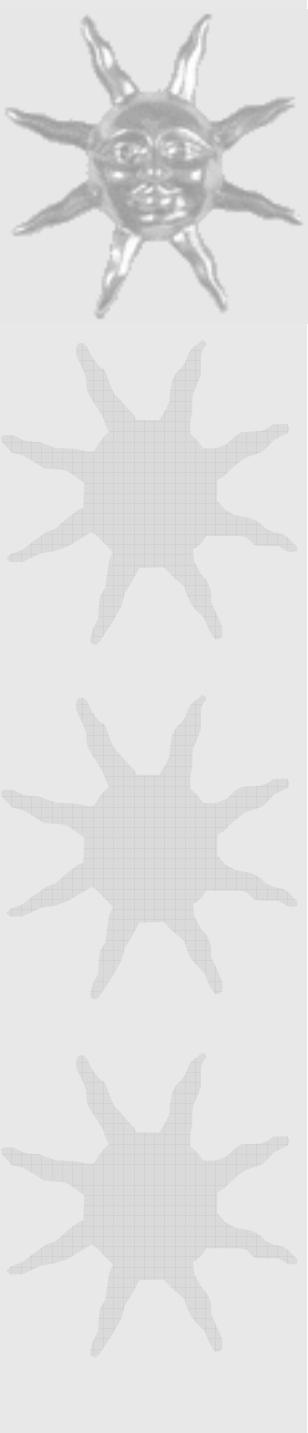
- ★ 1<sup>st</sup> defense: alveolar macrophages
- ★ 2<sup>nd</sup> defense: histiocytes, plasma cells, lymphocytes, leucocytes (rare in healthy lungs), mast cells.
- ★ 3<sup>rd</sup> defense: Lymph nodes



# *Etiology of parenchymal diseases*

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- ★ Inhalation Route:
  1. bacteria, viruses, fungi, protozoa
  2. Organic materials: bird prot, wood dust
  3. Anorganic substances: asbestos, silica
  4. Gastric contents
  5. Water, blood, toxic gases
  6. Idiopathic diseases ? inhalation

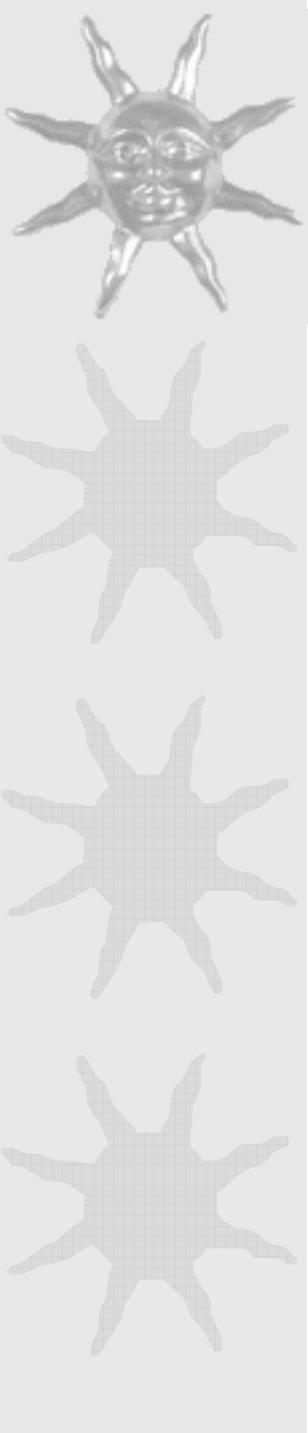


## *Etiology of parenchymal diseases*

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### ★ Vascular route:

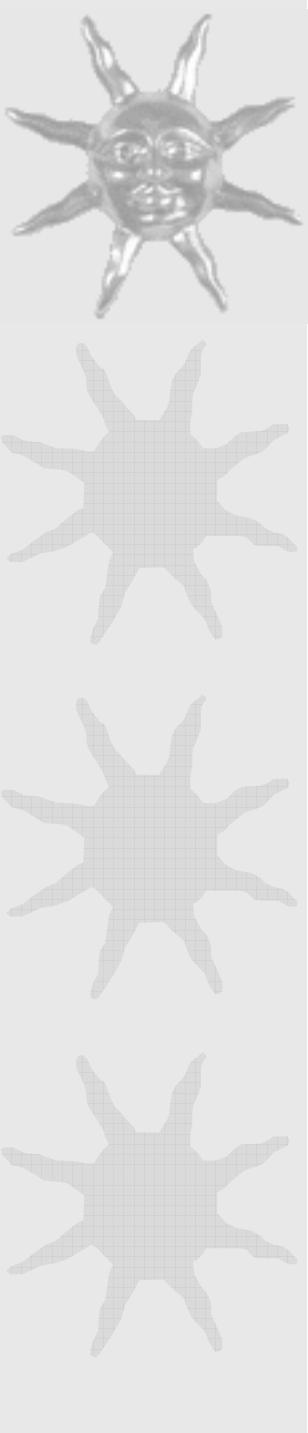
1. Vasculitis: Wegeners, SLE, Churg Strauss
2. Increased vascular permeability: ARDS
3. Increased Starling forces: pulm oedema
4. Pulmonary embolism
5. Septicaemia: multiple lung abscesses
6. Primary Pulmonary Hypertension



## *Pathogenesis*

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- ★ Alveolitis with desquamation of epithelial cells, inflammatory cell infiltrates, hialine membranes, necrosis.
- ★ Involvement of interstitium with fibroblast proliferation and granuloma formation, entrapment or occlusion of lymphatics
- ★ Obliteration/occlusion of capillary bed and necrosis
- ★ Honey combing



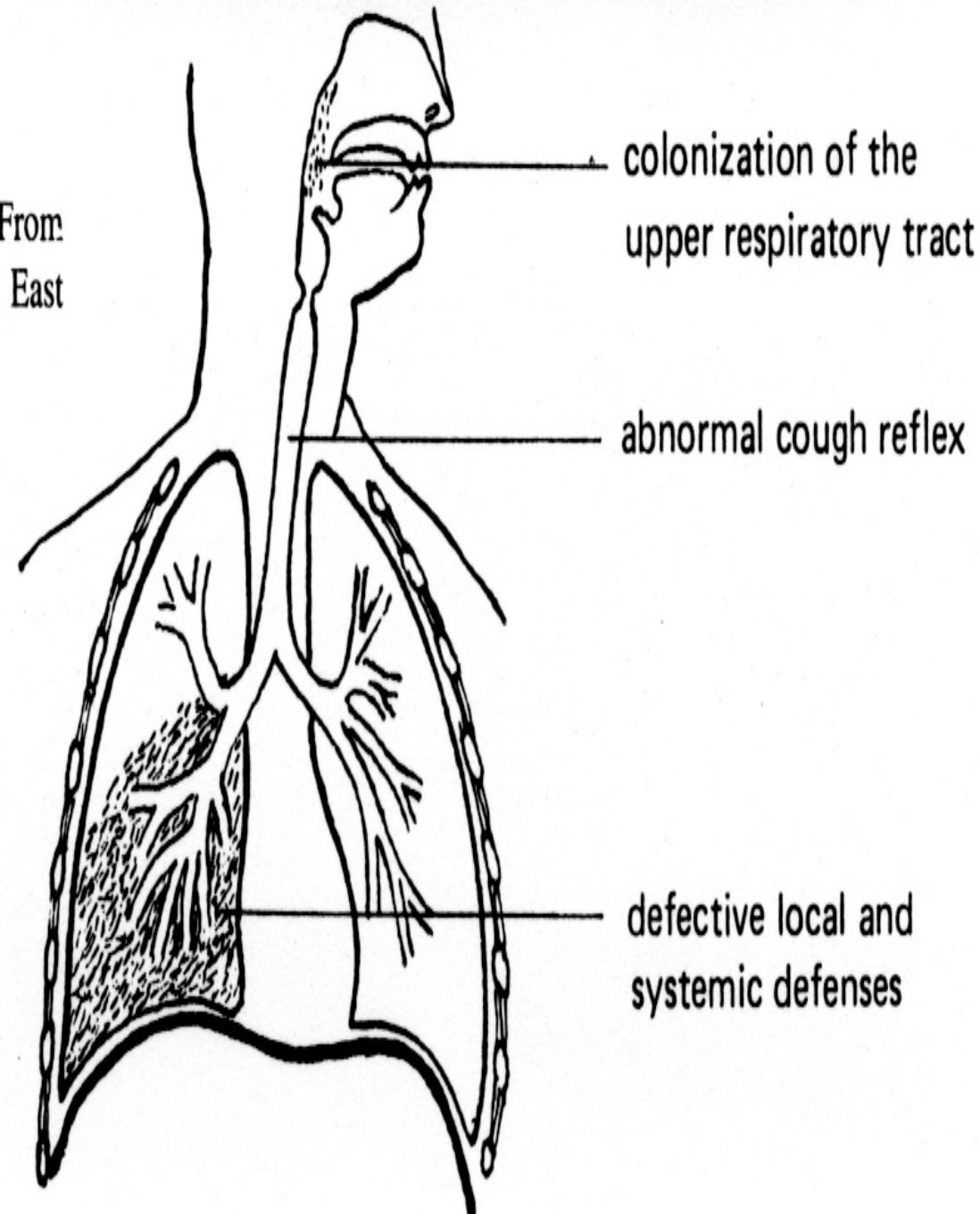
## *Alveolar diseases*

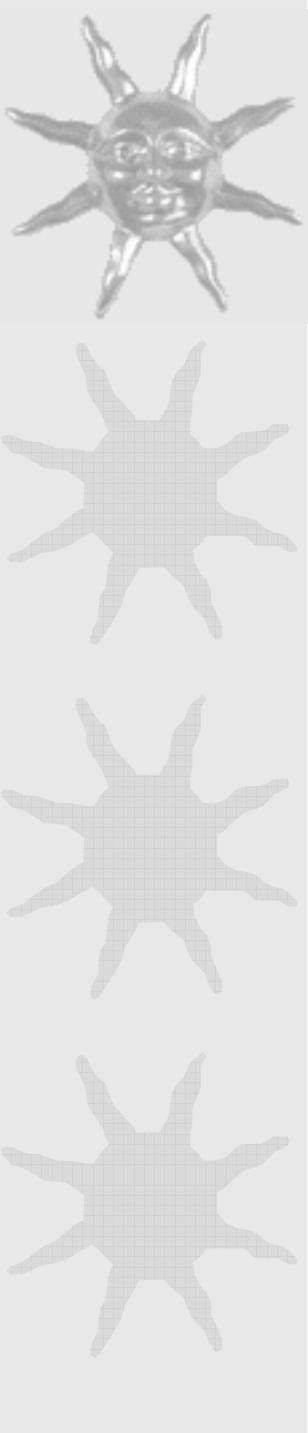
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★ Alveoli filled by:

- 1.Pus: pneumonia, lung abscess
- 2.Water:pulm oedema, ARDS
- 3.Blood:necrotizing infection, ITP, bleeding tendency.
- 4.Cells:Malignant, T4 lymphocytes (sarcoidosis), neutrophils (IPF), eosinophils ( E pneumonia)
- 5.Phospholipoprot: surfactant (Pulm alveolar proteinosis)

Pathogenesis of gram-negative pneumonia. (From concise handbook of respiratory diseases, ed 2, Easton, 1985, Appleton & Lange.)

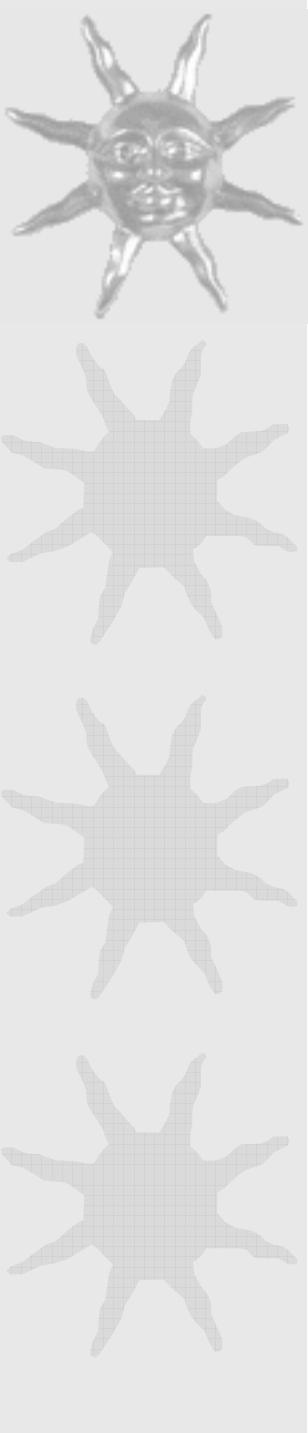




# *Interstitial diseases*

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- ★ Unknown etiology: Idiopathic pulmonary fibrosis, sarcoidosis, collagen vascular diseases
- ★ Known etiology: asbestosis, silicosis, hypersensitivity pneumonitis

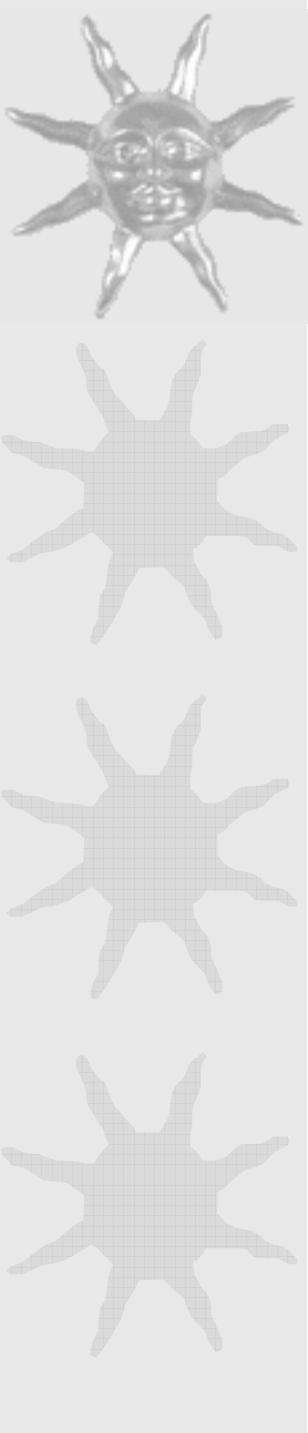


## *Physical signs of alveolar disease*

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### ★Consolidation:

- 1.dullness on percussion,
- 2.increased vocal sounds on palpation and auscultation
- 3.Bronchial breathing
- 4.Aegophony and E to A (auscultation)
- 5.Whispering pectoriloquy



## *Physical signs 2*

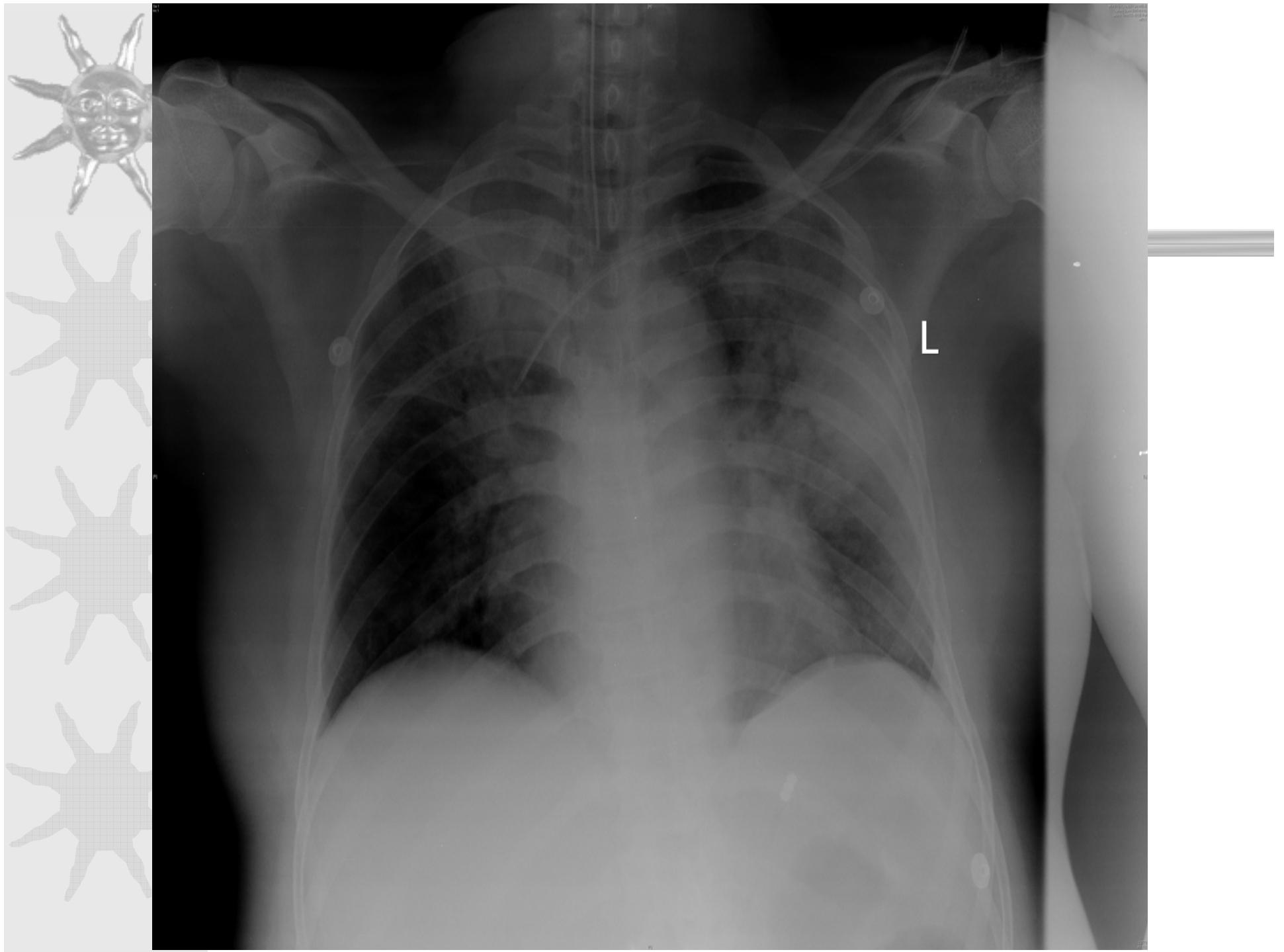
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### ★Crepitations:

- 1.Alveolar disease: fine, early inspiration.
- 2.Interstitial disease: coarse, high tone, late inspiration – velcro type

### ★Cough:

- 1.Productive:pneumonia
- 2.Non-productive:interstitial disease



Se:2  
Im:24

[A]

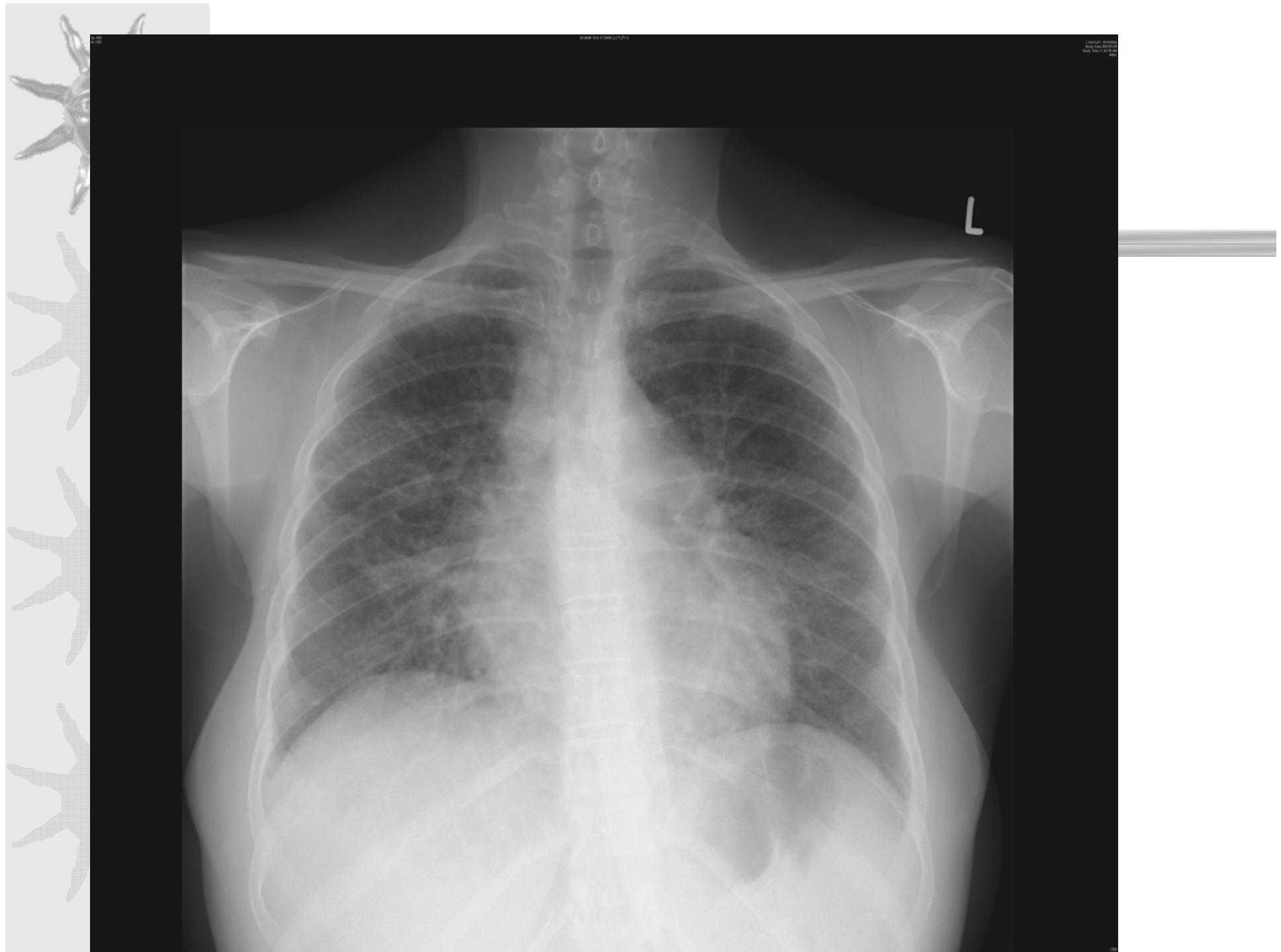
M.SELINAH  
Study Date:2007/01/10  
Study Time:08:42:44 AM  
MRN:

[R]

[L]

[P]

C-600  
W1200









Se:3  
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[A]

M.SHEILA, ANN  
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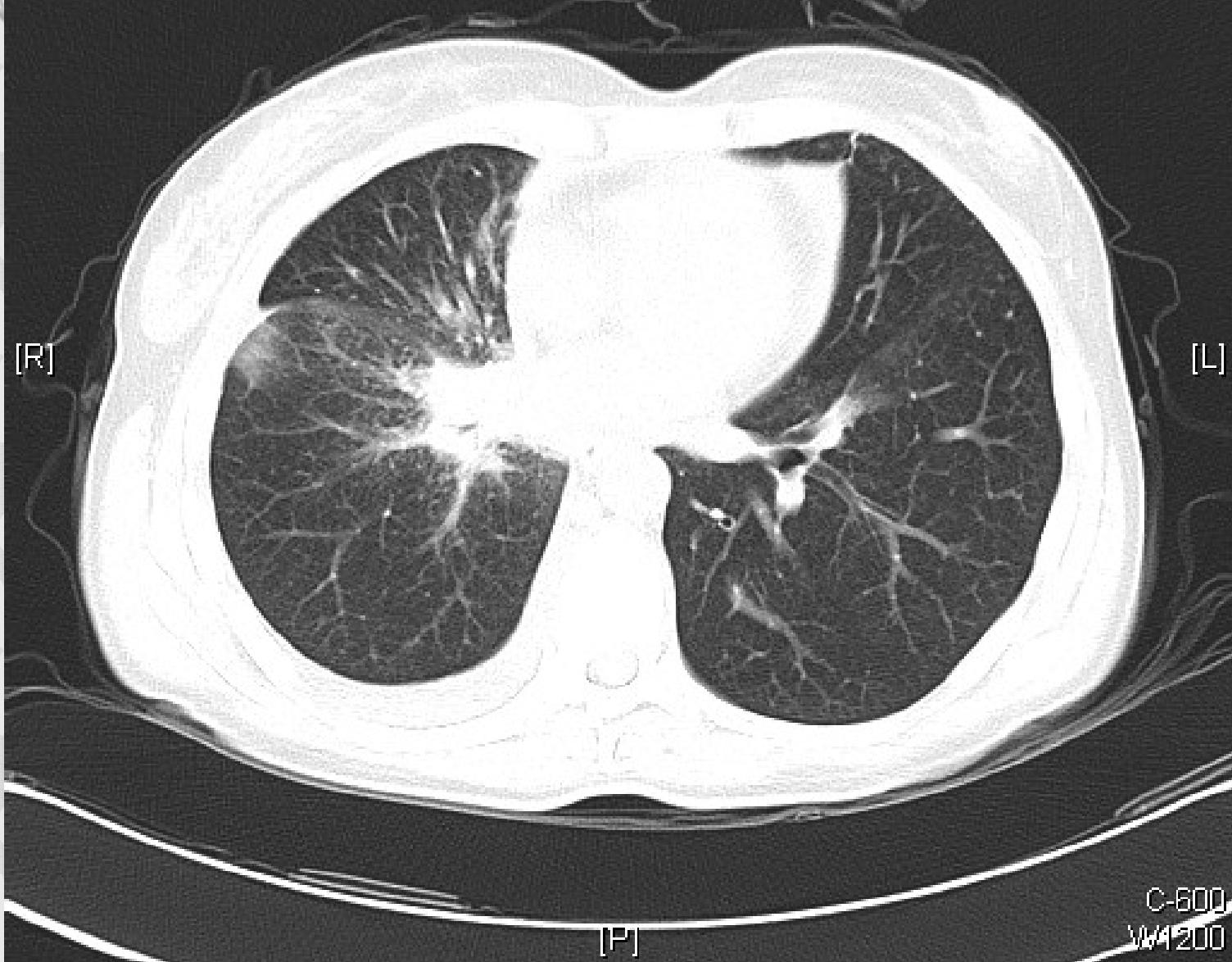
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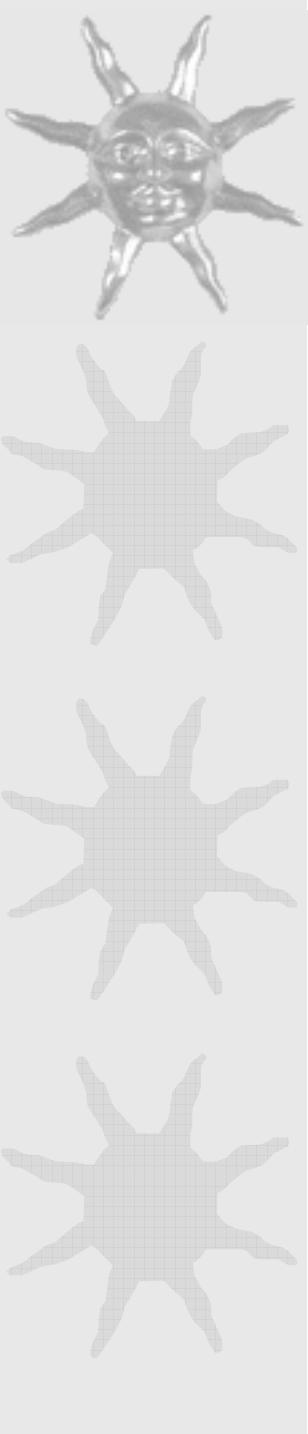
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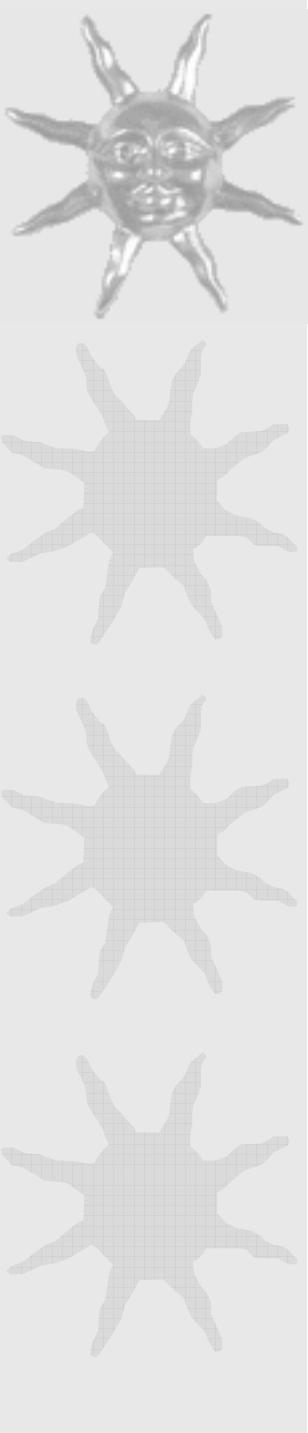


# *Diagnosis*

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## ★ Chest Xray:

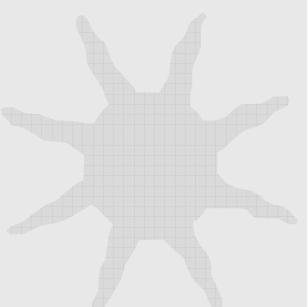
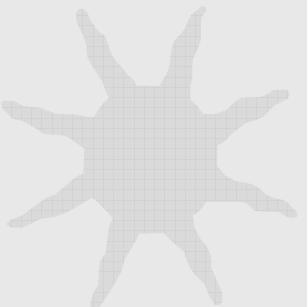
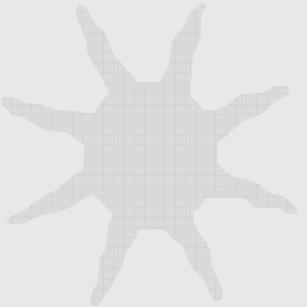
1. lobar veiling with air bronchograms –pneumonia
2. Diffuse alveolar veiling –pulm oedema-ARDS, aspiration
3. Interstitial veiling: linear:IPF,asbestosis  
nodular:miliary TB, sarcoidosis, silicosis
4. Focal decreased vascularity: pulm embolus, vasculitis with occlusion
5. Breakdown: cavitation: lung abscess



## *Laboratory investigations*

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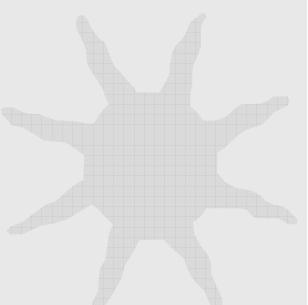
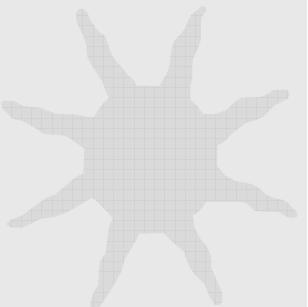
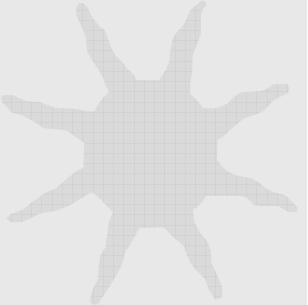
- ★ Sputum MCS, ZN+ culture
- ★ Blood culture
- ★ FBC
- ★ Serology: ANF, RF, ACE, chlamydia, mycoplasma
- ★ LFT+E
- ★ Renal function



## *Special Investigations*

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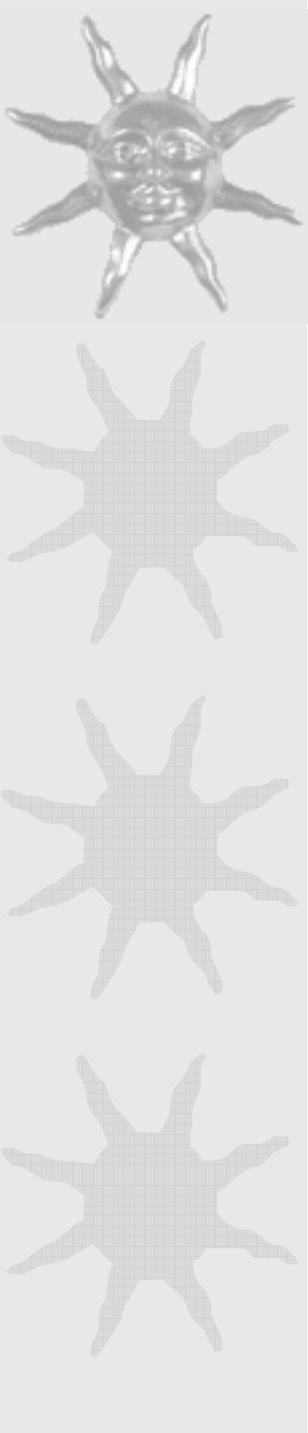
- ★ CT Scan
- ★ Bronchoscopy
- ★ Open lung biopsy



## *Complications*

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- ★ Respiratory failure
- ★ Cor pulmonale
- ★ Extra pulmonary manifestations: SIADH, meningism, arthralgia, septicaemia and DIC



# *Management*

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★ Depending on etiology:

Infection: Abs

ARDS: Mechanical ventilation and O<sub>2</sub>

IPF, sarcoidosis: Csteroids

Cor pulmonale

Respiratory failure: Type 1(hypoxaemic)-O<sub>2</sub>

Type 2 (Hypercarbic +hypoxaemia)-mechanical ventilation + O<sub>2</sub>.