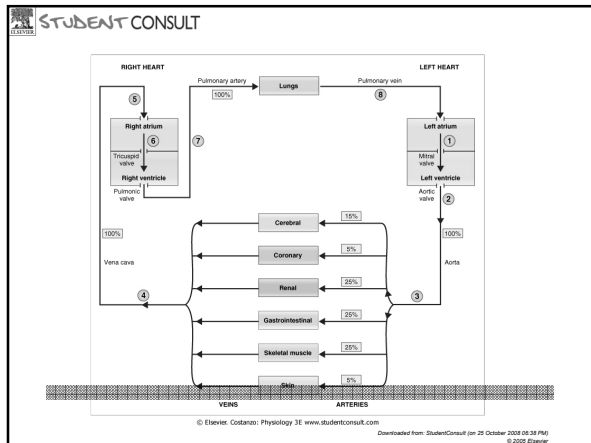


Pulmonary Vascular Changes in Heart Disease

Objectives

- To review the normal physiology of the pulmonary circulation
- To define pulmonary hypertension, its causes especially related to heart disease, and consequences
- To understand the mechanisms responsible for the clinical and radiological manifestations of a congested lung seen in all types of cardiac disease



Pressure, Flow, Resistance

Perfusion pressure: pressure gradient across a vascular bed

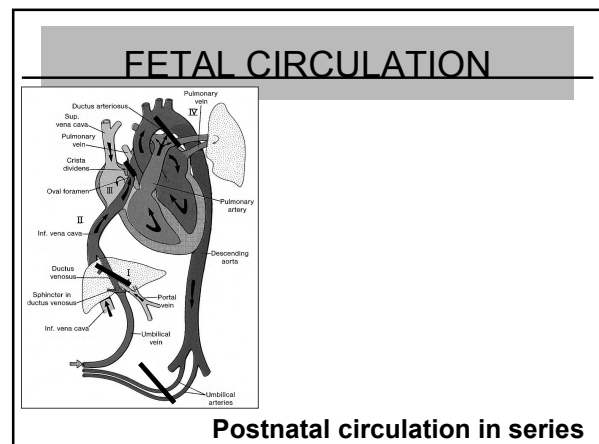
Flow: volume of blood that travels across the vascular bed

Resistance: Opposition to flow
vessel diameter
vessel structure and organization
physical characteristics of blood

Pressure, Flow, Resistance

Pulmonary blood flow is directly proportional to the pressure gradient between the pulmonary artery and the left atrium and is inversely proportional to the resistance of the pulmonary vasculature

$$Q = P \text{ gradient}/R$$

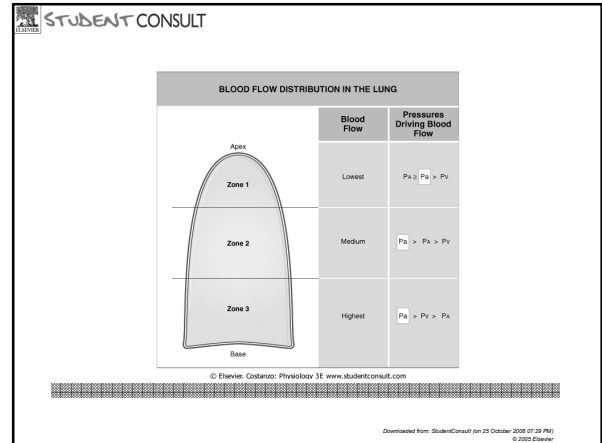
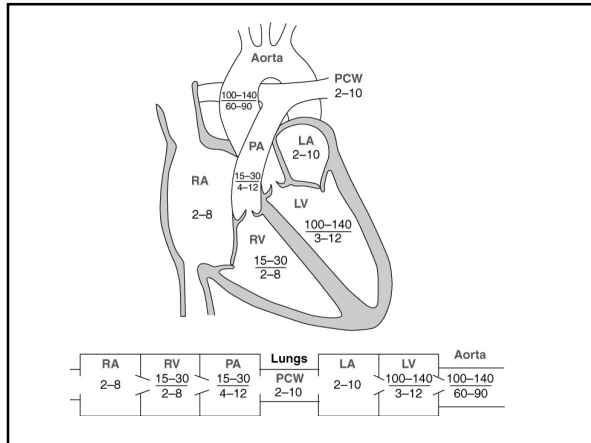
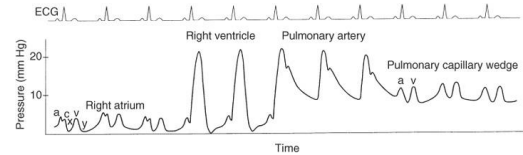


Pulmonary and Systemic Circulation

When compared with the systemic circulation, the pulmonary circulation is characterized by much lower pressures and resistances, although the blood flow is the same.

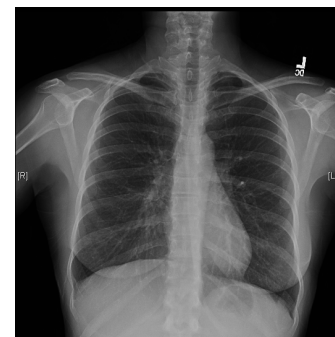
The reason that pulmonary blood flow can be equal to systemic blood flow is that pulmonary pressures and resistances are proportionately lower than systemic pressures and resistances.

Right Heart Catheterization



The distribution of blood flow within the lungs is uneven and the distribution can be explained by the effects of gravity.

In the upright position, Zone 1 has the lowest blood flow and zone 3 has the greatest blood flow. In the supine position, blood flow is uniform.



Normal frontal view of the chest



Normal lateral view of the chest

Pulmonary Blood Flow

The normal distribution of blood flow becomes disturbed in disease states affecting the left side of the heart. These changes include increased vascular markings, redistribution of blood flow, pulmonary edema, and pleural effusions and will be reviewed later in the lecture.

When Dr. Williams discusses congenital heart disease, blood flow in the lung can also be disturbed and she will discuss these changes with you.

Pulmonary Circulation

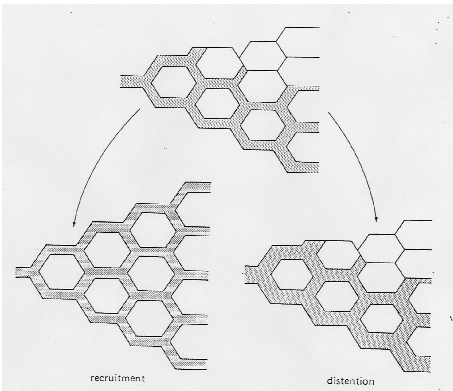
Low resistance, high compliance vascular bed
Changes in cardiac output as well as pleural/alveolar pressure affect pulmonary blood flow

The pulmonary circulation reacts differently to stimuli such as hypoxia than the systemic circulation

The pulmonary circulation is normally in a state of mild vasodilatation

Exercise

With exercise, cardiac output will increase
Pulmonary blood flow can increase up to 4-5x baseline levels
Increased blood flow is accommodated by both recruitment and vasodilatation
Net effect is a decrease in pulmonary vascular resistance



Pulmonary Hemodynamics

	Sea level Rest	Sea level Exercise	High Alt Rest
PA pressure (mean)mmHg	20/10 (15)	30/13 (20)	38/14 (26)
CO, L/min	5.0	9.0	5.0
PVR	1.7	0.9	3.3

Pulmonary Hemodynamics

What would happen when you try to exercise at high altitude?

	High altitude rest	High altitude exercise
PA pressure	38/14	??
CO, L/min	5	9
PVR	3.3	??

Hypoxia

High altitude with decreased pO₂ is a good example of the potent vasoconstrictive effect of hypoxia on the pulmonary bed.

Pulmonary Hypertension

Table 1. General Classification of Pulmonary Hypertension

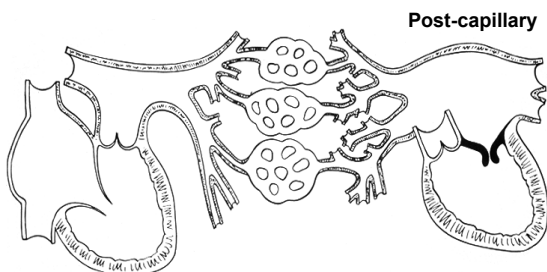
1. PAH
2. Pulmonary hypertension with left-sided heart disease
3. Pulmonary hypertension associated with lung diseases and/or hypoxemia
4. Pulmonary hypertension due to chronic thrombotic and/or embolic disease
5. Miscellaneous

(Circulation, 2008;118:1195-1201.)

Pulmonary Venous Hypertension

For most of this course, we will be talking about disease states that affect the left ventricle, the left atrial pressure, and thus the pulmonary venous pressure. You will learn about the other causes of pulmonary hypertension next month in your pulmonary section.

Localizing the problem



Pulmonary Venous Hypertension

Post-capillary PH or pulmonary venous hypertension

PAP mean of 25mmHg or greater at rest or 30mmHg or greater with exercise

AND

PCWP or LVED > 15mmHg

Pulmonary Arterial Hypertension

Pre-capillary PH or pulmonary arterial hypertension

PAP mean 25mmHg or greater at rest or 30mmHg or greater with exercise

AND

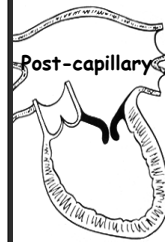
PCWP or LVEDP 15mmHg or less

PVRI 3units/m2 or greater

No left-sided heart disease

Post-capillary PH: Pulmonary Venous Hypertension Localizing the Problem

- Left Heart Etiologies
 - Valvular heart disease
 - Cardiomyopathies
 - Ischemic Heart Disease
 - Pericardial Disease
 - Tumors (myxoma)
 - Congenital (cor triatriatum, coarctation)



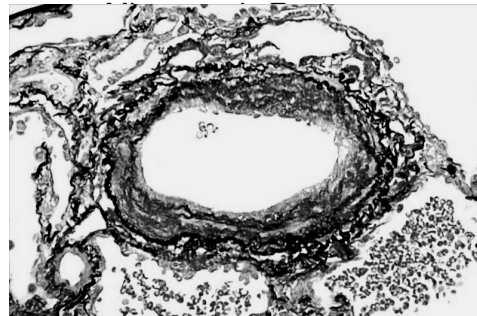
Chronic Pulmonary Venous Hypertension

Table 1. Histologic Changes Associated With Chronic Pulmonary Venous Hypertension

Pulmonary arterial medial hypertrophy
Muscularization of pulmonary arterioles
Arterialization of pulmonary veins
Intimal and adventitial fibrosis
Capillary dilatation
Venous dilatation
Focal alveolar hemosiderosis
Lymphatic dilatation

Progress in Cardiovascular Diseases, Vol XXXVII, No 6 (May/June), 1995: pp 347-370

Pulmonary Venous Hypertension

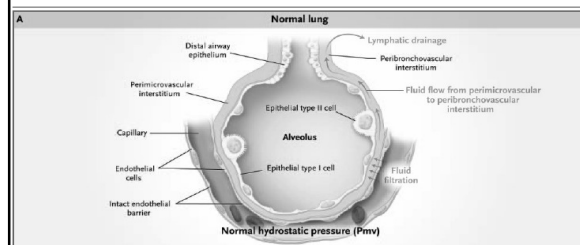


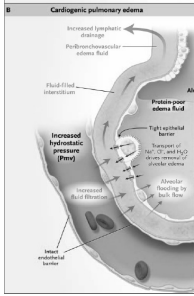
Thickened Pulmonary Vein (VVG Stain)

Physiology of Microvascular Fluid Exchange in the Lung

In the normal lung, fluid moves continuously outward from the vascular to the interstitial space.

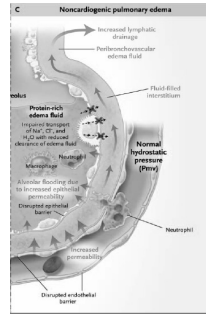
Depends on the net difference between hydrostatic and protein oncotic pressure and the permeability of the capillary membrane





When left atrial pressure increases, hydrostatic pressure increases in the microcirculation and the rate of transvascular fluid filtration rises.

When lung interstitial pressure exceeds pleural pressure, fluid moves across the visceral pleura, creating pleural effusions.

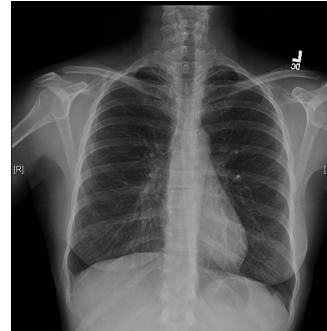


Non-cardiogenic pulmonary edema can occur in critically ill patients when there is injury to the microvascular membrane resulting in a marked increase in the amount of fluid and protein leaving the vascular space (adult respiratory distress syndrome)

Normal Chest X-ray

Normal (PCW 8-12mmHg)

Blood flow is greater to the lower lobes than to the upper lobes. The lower lobe vessels are 2-3x larger in diameter than the upper lobe vessels (gravity and alveolar pressure differences)



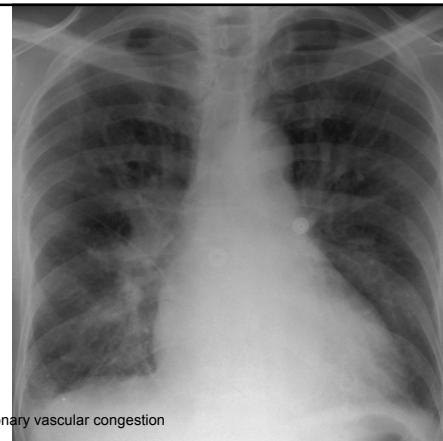
Normal frontal view of the chest

Pulmonary Vascular Redistribution

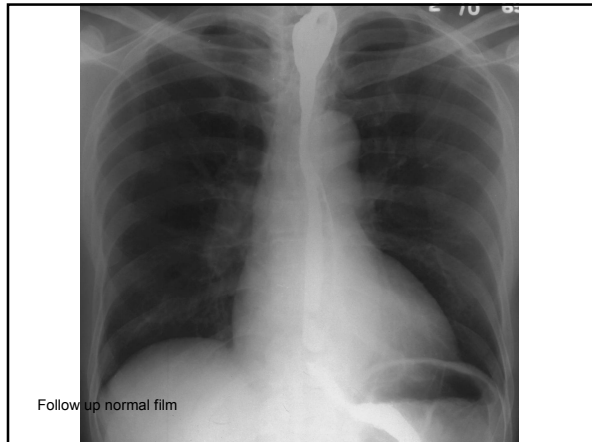
PCW 12-18mmHg

Pulmonary vascular redistribution – pulmonary blood flow is redirected into the upper lobes

Patient may be asymptomatic



Pulmonary vascular congestion

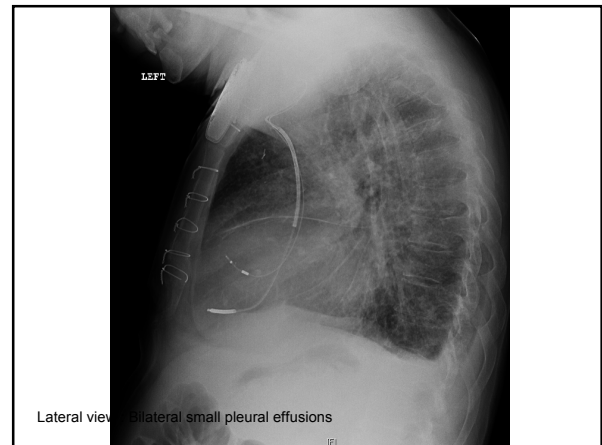
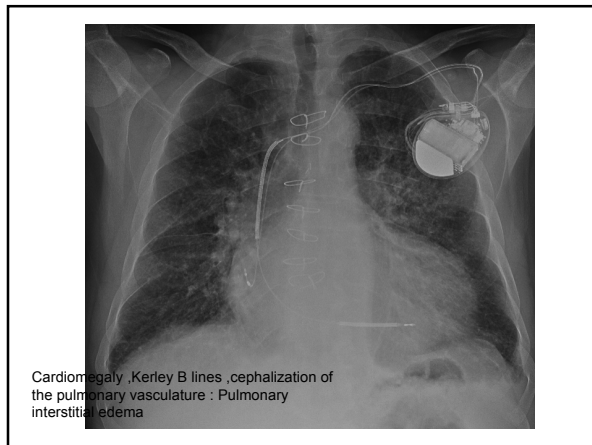


Pulmonary Interstitial Edema

PCW >18mmHg

Get pulmonary interstitial edema, causing haziness of the vessels and Kerley B lines (linear markings at the periphery of the lower lung fields indication interlobular edema)

Patient will be short of breath

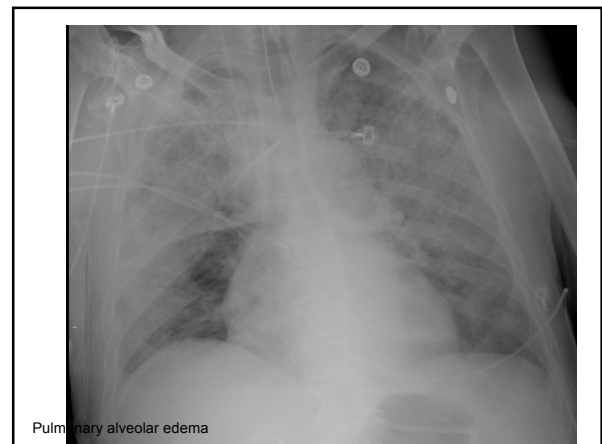


Pulmonary Edema

PCW > 25mmHg

Get alveolar edema, patient in marked distress, with opacification of the air spaces, a butterfly pattern around the hila, and pleural effusions

The patient is cyanotic (blue), rales and wheezing, frothy pink sputum



Acute versus Chronic Changes

If elevation of pulmonary venous pressure is slowly progressive and chronic, higher pulmonary capillary wedge pressures can be accommodated with fewer clinical and radiological signs due to enhanced lymphatic drainage and the chronic changes to the vasculature previously described.

Case Examples

A 30 year old woman is undergoing an elective surgical procedure. She has no heart disease. Her blood pressure drops unexpectedly from the anesthesia. She is given some IV fluid to bring up her blood pressure but she gets too much. Her left atrial pressure will rise suddenly and she develops pulmonary congestion. If a Swan-Ganz catheter were placed, her PCW would be 18mmHg instead of 10mmHg.

Case Examples

The second patient is a 30 year woman from India with a history of mitral stenosis. Chronically her left atrial pressure is 24mmHg (rheumatic heart disease is a slowly progressive disease). Suddenly she develops atrial fibrillation which makes her heart go rapidly and her PCW rises to 30mmHg. She develops pulmonary congestion but at a much higher pressure because of the chronic changes that have occurred in her lung vasculature and increased lymphatic drainage.

Symptoms Associated with Venous Congestion

<u>Pulmonary congestion</u>	<u>Systemic congestion</u>
Dyspnea	Edema
Orthopnea	Ascites
Paroxysmal nocturnal dyspnea	RUQ pain (liver congestion)
Hemoptysis	Central and peripheral cyanosis
Cough	
Fatigue	

Dyspnea

One of the principle symptoms of cardiac and pulmonary disease

Described as an abnormally uncomfortable awareness of breathing

Cardiac dyspnea is most commonly associated with and caused by pulmonary congestion. The interstitial and alveolar edema stiffens the lung and stimulates respiration by activating "J" receptors in the lung

Cardiac dyspnea can also occur in the setting of a reduced cardiac output.

Dyspnea

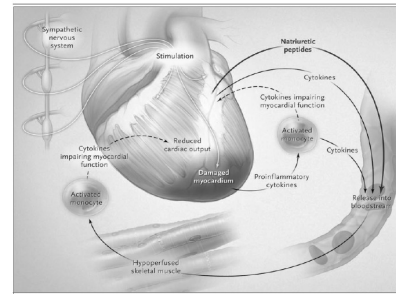
Sudden onset: pulmonary edema
pulmonary embolism
pneumothorax
asthma

Dyspnea on exertion: how much exertion??
heart failure
pregnancy (normal)
pleural effusion from cancer

Dyspnea

Differential Diagnosis:

Pulmonary disease
Anemia
Obesity
Deconditioning
Psychogenic/anxiety attack



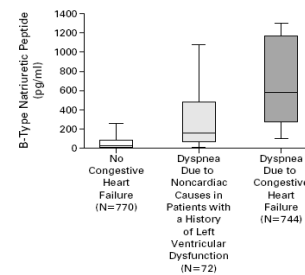
The stressed myocardium releases natriuretic peptides, denoted in red; their release improves the circulation. N Engl J Med 358:20 www.n engl j med May 15, 2008

BNP

A vasoactive peptide that is released by myocardial stress. The actions of BNP oppose the physiologic abnormalities of heart failure.

A useful test in the emergency room for patients presenting with dyspnea. BNP normal in patients with lung disease.

“BREATHING NOT PROPERLY” Study



N Engl J Med, Vol. 347, No. 3

BNP

Levels of BNP are correlated with severity of congestive heart failure and predict prognosis/mortality.

Dyspnea in Heart Failure

Table 4. Mechanisms of Dyspnea in Heart Failure

- Decreased pulmonary function
 - Decreased compliance
 - Increased airway resistance
- Increased ventilatory drive
 - Hypoxemia: \uparrow PCW
 - V/Q mismatching: \uparrow PCW, \downarrow CO
 - \uparrow CO₂ production: \downarrow CO-lactic acidosis
- Respiratory muscle dysfunction
 - Decreased strength
 - Decreased endurance
 - Ischemia

Abbreviations: PCW, mean pulmonary capillary wedge pressure; V/Q, ventilation perfusion mismatch; CO, cardiac output; CO₂, carbon dioxide production.

Progress in Cardiovascular Diseases, Vol XXXVII, No 6 (May/June), 1995: pp 347-370

Orthopnea

Dyspnea that develops in the recumbent position that is relieved by elevation of the head by pillows

Mechanism: in the recumbent position, there is reduced pooling of blood in the lower extremities and abdomen and blood is displaced from the extrathoracic to the thoracic compartment (increased venous return). There is a further rise in baseline pulmonary venous and capillary pressures

In advanced heart failure, patients often sleep sitting up in a chair.

Differential diagnosis: lung disease, ascites (any condition in which the vital capacity is low)

Paroxysmal Nocturnal Dyspnea

Usually occurs at night

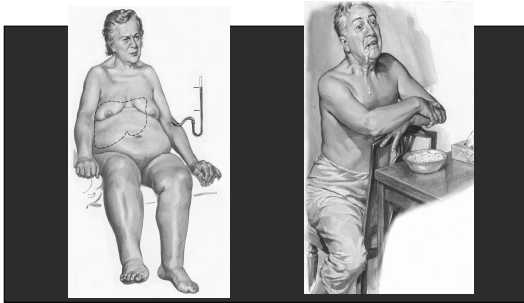
Patient awakens suddenly, with a feeling of severe anxiety and suffocation, sits bolt upright, and gasps for breath

May be associated with wheezing (cardiac asthma)

Distress may persist for 30 minutes even when patient sits up; patient may be afraid to go back to sleep

Mechanisms: redistribution of blood flow from dependent portion of the body, reduced adrenergic support of left ventricular function during sleep, normal nocturnal depression of the respiratory center

Heart Failure



Cough

One of the most frequent of cardiorespiratory symptoms

For cardiovascular disorders:

- pulmonary venous hypertension
- pulmonary edema
- compression of the tracheobronchial tree by an aortic aneurysm

Cough is dry, irritating, spasmodic, and nocturnal

Pulmonary edema may be associated with frothy, pink-tinged sputum

Differential diagnosis: lung disease, allergic disease, infectious diseases, drug-reaction (ace-inhibitor)

Hemoptysis

Expectoration of blood

Due to escape of red cells into the alveoli from congested vessels

In cardiac disease, usually seen with chronic valve disease such as mitral stenosis

Differential diagnosis: lung disease, cancer, tuberculosis, pulmonary embolism, pulmonary AV fistula

Consequences of Pulmonary Hypertension

Whether the pulmonary hypertension is post-capillary or pre-capillary, the right ventricle which is used to working under low pressure is unable to work under higher pressures and the right ventricle will fail.

You are going to hear a lot about **heart failure** and its multiple causes and mechanisms.

Simplistically, heart failure means the heart is unable to do its job of pumping blood at a sufficient rate to meet the demands of the body.

The Right Ventricle

- You are going to hear a lot about how the heart adapts to pressure and volume overloads.
- The right ventricle demonstrates a heightened sensitivity to afterload change
- Failure of the right heart causes systemic venous congestion

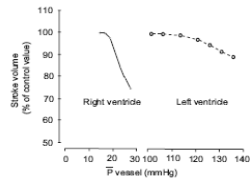


Figure 4. The response of the RV and LV to experimental increase in afterload. Reproduced from MacNeil²⁴ with permission from the publisher. Copyright © 1994, the American Thoracic Society.

Symptoms Associated with venous congestion

Pulmonary congestion Systemic congestion

Dyspnea	Edema
Orthopnea	RUQ discomfort (liver congestion)
Paroxysmal nocturnal dyspnea	Ascites
Hemoptysis	Central and peripheral cyanosis
Cough	
Fatigue	

Edema

Edema – multiple causes

- Cardiac
- Hepatic
- Renal
- Deep vein thrombosis/cellulitis
- Obstruction of the SVC (lung carcinoma)

Edema

Cardiac edema

associated with dyspnea
generally symmetrical
lower extremities to the abdominal wall
anasarca (total body edema)

Ascites

Ascites in cardiac disease usually reflects long-standing systemic venous hypertension.

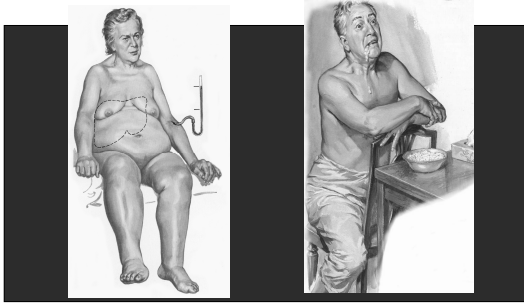
Cyanosis

Cyanosis is a bluish discoloration of the skin and mucous membranes due to an increased quantity of reduced hemoglobin

Central cyanosis: due to decreased arterial oxygen saturation (right-to-left shunting or impaired pulmonary function)

Peripheral cyanosis: cutaneous vasoconstriction due to low cardiac output or exposure to cold air

Heart Failure



The Right Ventricle

Although we will be concentrating predominantly on dysfunction of the left ventricle over the next three weeks, the right ventricle is also important.

Right ventricular dysfunction is an important predictor of survival and exercise capacity in cardiopulmonary disease.

The right ventricle is also important when we discuss congenital heart disease.

Summary

The pulmonary vasculature is adaptive to the demands of the heart.

Abnormalities of the left side of the heart causing elevation of pulmonary capillary wedge pressure or pulmonary venous pressure are reflected by changes in blood flow and fluid in the lung.

Symptoms of cardiac disease and pulmonary disease often overlap.