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

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

\[ Q = \frac{P \text{ gradient}}{R} \]

Fetal Circulation

Postnatal circulation in series
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

<table>
<thead>
<tr>
<th>Sea level</th>
<th>Sea level</th>
<th>High Alt</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Exercise</td>
<td>Rest</td>
</tr>
<tr>
<td>PA pressure</td>
<td>20/10</td>
<td>30/13</td>
</tr>
<tr>
<td>(mean)mmHg (15)</td>
<td>(20)</td>
<td>(26)</td>
</tr>
<tr>
<td>CO, L/min</td>
<td>5.0</td>
<td>9.0</td>
</tr>
<tr>
<td>PVR</td>
<td>1.7</td>
<td>0.9</td>
</tr>
</tbody>
</table>
**Pulmonary Hemodynamics**

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

<table>
<thead>
<tr>
<th></th>
<th>High altitude rest</th>
<th>High altitude exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>PA pressure</td>
<td>38/14</td>
<td>??</td>
</tr>
<tr>
<td>CO, L/min</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td>PVR</td>
<td>3.3</td>
<td>??</td>
</tr>
</tbody>
</table>

**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.

**Hypoxia**

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

**Localizing the problem**

**Pulmonary 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)

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

Chronic Pulmonary Venous Hypertension

<table>
<thead>
<tr>
<th>Table 1: Histologic Changes Associated With Chronic Pulmonary Venous Hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary arteriolar medial hypertrophy</td>
</tr>
<tr>
<td>Alveolar septal thickening</td>
</tr>
<tr>
<td>Arterialization of pulmonary veins</td>
</tr>
<tr>
<td>Intimal and subintimal thickening</td>
</tr>
<tr>
<td>Capillary dilatation</td>
</tr>
<tr>
<td>Venous dilatation</td>
</tr>
<tr>
<td>Focal intimal hemodynamics</td>
</tr>
<tr>
<td>Lymphatic dilatation</td>
</tr>
</tbody>
</table>

Thickened Pulmonary Vein (VVG Stain)
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).

Pulmonary Vascular Redistribution

PCW 12-18mmHg
Pulmonary vascular redistribution – pulmonary blood flow is redirected into the upper lobes
Patient may be asymptomatic

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)
Follow up normal film

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

<table>
<thead>
<tr>
<th>Pulmonary congestion</th>
<th>Systemic congestion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnea</td>
<td>Edema</td>
</tr>
<tr>
<td>Orthopnea</td>
<td>Ascites</td>
</tr>
<tr>
<td>Paroxysmal nocturnal dyspnea</td>
<td>RUQ pain (liver congestion)</td>
</tr>
<tr>
<td>Hemoptyis</td>
<td>Central and peripheral cyanosis</td>
</tr>
<tr>
<td>Cough</td>
<td></td>
</tr>
<tr>
<td>Fatigue</td>
<td></td>
</tr>
</tbody>
</table>

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

“BREATHING NOT PROPERLY” Study

BNP

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

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.

Dyspnea in Heart Failure

Table 4. Mechanisms of Dyspnea In Heart Failure

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Decreased pulmonary compliance</td>
<td>Decreased compliance</td>
</tr>
<tr>
<td></td>
<td>Increased artery resistance</td>
</tr>
<tr>
<td>2. Increased sympathetic drive</td>
<td>Hypoxemia, PCW, C02, C02 ventilation, pH, wedge, C02 production</td>
</tr>
<tr>
<td>3. Respiratory muscle dysfunction</td>
<td>Hypoxemia, increased work</td>
</tr>
</tbody>
</table>

Abnormalities: PCW, mean pulmonary capillary wedge (mmHg); V/Q ventilation-perfusion mismatch, C02, arterial acidosis; C02, carbon dioxide production.

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)

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)

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

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

Heart Failure

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.

Edema

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

Symptoms Associated with venous congestion

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Ascites

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

Edema

Edema – multiple causes
Cardiac
Hepatic
Renal
Deep vein thrombosis cellulitis
Obstruction of the SVC (lung carcinoma)

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.