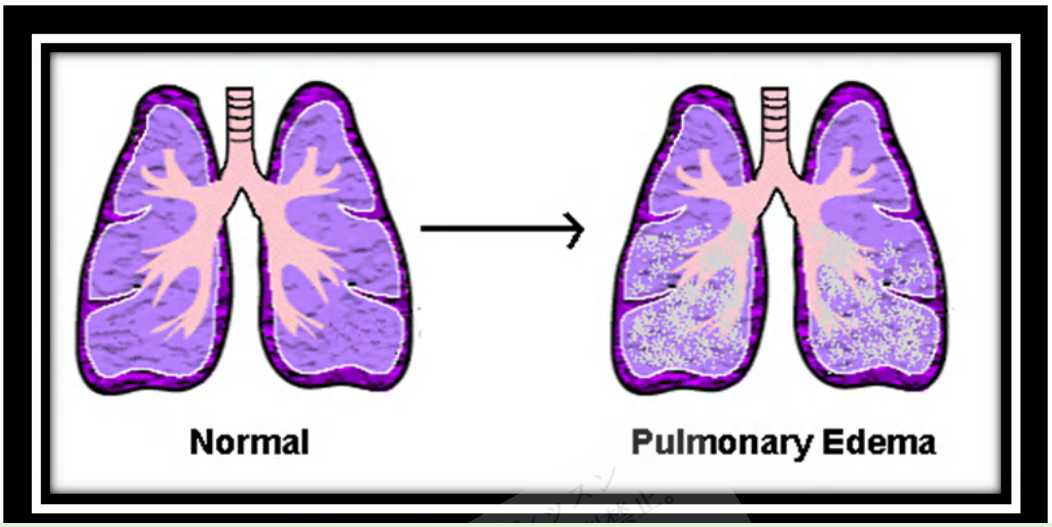
Pulmonary Edema



edema. Although precipitating causes vary by age and country, about one half of cases result from acute coronary ischemia; some from **decompensation** of significant underlying heart failure (HF), including diastolic dysfunction HF due to hypertension; and the rest from arrhythmia, an acute valvular disorder, or acute volume overload often due to IV fluids. Drug or dietary non-adherence is often involved.

Symptoms and Signs

Patients present with extreme dyspnea, restlessness, and anxiety with a sense of suffocation. Cough producing blood-tinged sputum, **pallor**, **cyanosis**, and marked **diaphoresis** are common; some patients froth at the mouth. **Frank hemoptysis** is uncommon. The pulse is rapid and low volume, and BP is variable. Marked hypertension indicates significant cardiac reserve; hypotension with systolic BP < 100 mg Hg is ominous. Inspiratory fine crackles are widely **dispersed** anteriorly and posteriorly over both lung fields. Marked wheezing (cardiac asthma) may occur. Noisy respiratory efforts often make cardiac auscultation difficult; a summation **gallop**—

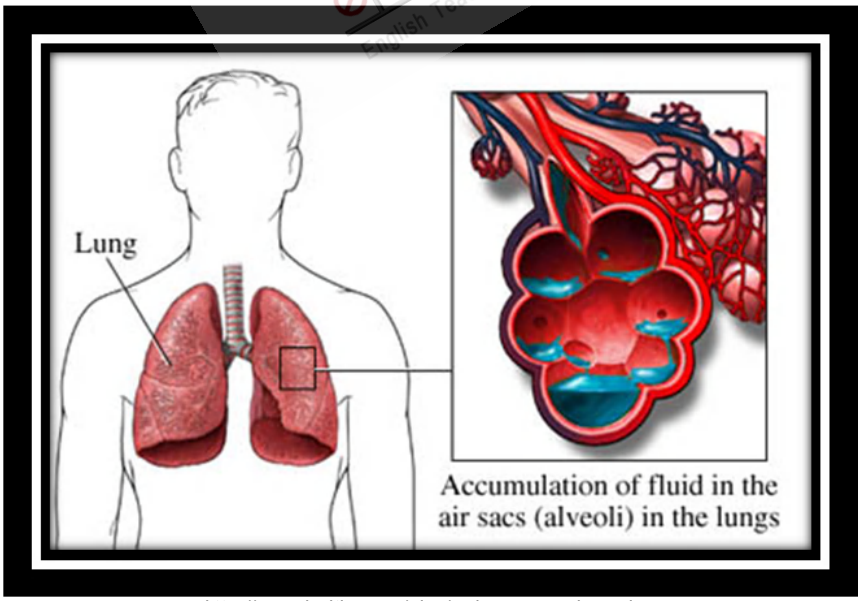
merger of 3rd (S_3) and 4th (S_4) heart sounds—may be present. Signs of right ventricular (RV) failure (eg, neck vein distention, peripheral edema) may be present.

Diagnosis

- Clinical evaluation showing severe dyspnea and pulmonary crackles
- Chest x-ray
- Sometimes serum brain natriuretic peptide (BNP) or N-terminal-pro BNP (NT-pro-BNP)
- ECG, cardiac markers, and other tests for etiology as needed

A COPD **exacerbation** can mimic pulmonary edema due to LV failure or even that due to **biventricular failure** if cor pulmonale is present. Pulmonary edema may be the presenting symptom in patients without a history of cardiac disorders, but COPD patients with such severe symptoms usually have a history of COPD, although they may be too **dyspneic** to relate it.

A chest x-ray, done immediately, is usually diagnostic, showing marked interstitial edema. Bedside measurement of serum BNP levels (elevated in pulmonary edema; normal in COPD exacerbation) is helpful if the diagnosis is in doubt. ECG, pulse oximetry, and blood tests (cardiac markers, electrolytes, BUN, creatinine, and, for severely ill patients, ABGs) are done. An echocardiogram may be helpful to determine the cause of the pulmonary edema (eg, MI, valvular dysfunction, hypertensive heart disease, dilated cardiomyopathy) and may influence the choice of therapies. Hypoxemia can be severe. CO_2 retention is a late, ominous sign of secondary hypoventilation.



http://www.beltina.org/pics/pulmonary_edema.jpg

Treatment

- O_2
- Furosemide
- Nitrates
- IV morphine
- Ventilatory assistance as needed
- Treatment of cause

Initial treatment includes 100% O_2 by nonrebreather mask; upright position; furosemide 0.5 to 1.0 mg/kg IV; nitroglycerin 0.4 mg sublingually q 5 min, followed by an IV drip at 10 to 20µg/min, titrated upward at 10 µg/min q 5 min as needed to a maximum 300 µg/min if systolic BP is > 100 mm Hg; and morphine 1 to 5 mg IV once or twice. If hypoxia is significant, noninvasive ventilatory assistance with bilevel positive airway pressure (BiPAP) is helpful, but if CO_2 retention is present or the patient is obtunded, tracheal **intubation** and assisted ventilation are required.

Specific additional treatment depends on etiology:

- For acute MI or another acute coronary syndrome, **thrombolysis** or direct percutaneous coronary angioplasty with or without a stent
- For severe hypertension, an IV vasodilator
- For supraventricular or ventricular tachycardia, direct-current cardioversion
- For **rapid atrial fibrillation**, to slow the ventricular rate, an IV β-blocker, IV digoxin, or cautious use of an IV Ca channel blocker (cardioversion is preferred)

Other treatments, such as IV BNP (nesiritide) and new **inotropic drugs** (levosimendan), remain under study to **elucidate** safety profiles and efficacy. Because fluid status before onset of pulmonary edema is usually normal in patients with acute MI, diuretics are less useful than in patients with chronic HF and may precipitate hypotension. If systolic BP falls < 100 mm Hg or shock develops, IV dobutamine and an intra-aortic balloon pump (counterpulsation) may be required.

Once patients are stabilized, long-term HF treatment is as described above.