Sri Lakshmi Narayana Institute of Medical Sciences

Date: 01.06.2019

From

Dr.G.Somasundram
Principal of Allied Health Sciences,
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

To

The Dean,
Sri Lakshmi Narayana Institute of Medical College
Bharath Institute of Higher Education and Research,
Chennai.

Sub: Permission to conduct value-added course: Congestive Heart Failure and Pulmonary Edema

Dear Sir,

With reference to the subject mentioned above, the department proposes to conduct a value-added course titled: **Congestive Heart Failure and Pulmonary Edema** from July to September 2019. We solicit your kind permission for the same.

Kind Regards

Dr.G.Somasundram

FOR THE USE OF DEANS OFFICE

Names of Committee members for evaluating the course:

The Dean: **Dr. Jayalakshmi**

The HOD: **Dr. Somasundram. G**

The Expert: **Dr. Sangeetha**

The committee has discussed about the course and is approved.

Dean	Subject Expert	HOD
(Sign & Seal)	(Sign & Seal)	(Sign & Seal)

Dr. G. JAYALAKSHMI, BSC., MBBS., DTCD., M.D.,
DEAN
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OFFICE OF THE DEAN

Sri Lakshmi Narayana Institute of Medical Sciences

OSUDU, AGARAM VILLAGE, VILLIANUR COMMUNE, KUDAPAKKAM POST,
PUDUCHERRY - 605 502.
[Recognised by Medical Council of India, Ministry of Health letter No. U/12012/249/2005-ME (P -II) dt. 11/07/2011]
[Affliated to Bharath University, Chennai - TN]

Circular

30.06.2019

Sub: Organizing Value-added Course: "Congestive Heart Failure and Pulmonary Edema".reg

With reference to the above mentioned subject, it is to bring to your notice that Sri Lakshmi Narayana Institute of Medical Sciences, **Bharath Institute of Higher Education and Research** is organizing **"Congestive Heart Failure and Pulmonary Edema"**. The course content and registration form is enclosed below."

The application must reach the institution along with all the necessary documents as mentioned. The hard copy of the application should be sent to the institution by registered/ speed post only so as to reach on or before July to September 2019. Applications received after the mentioned date shall not be entertained under any circumstances.

Dean

Dr. G. JAYALAKSHMI, BSC., MBBS., DTCD., M.D.,
DEAN
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Agaram, Kudapakkam Post,
Villianur Commune, Puducherry - 605502.

Encl: Copy of Course content

VALUE ADDED COURSE

1. Name of the programme & Code

"Congestive Heart Failure and Pulmonary Edema" & VAC01/AHS/2019-15/07

2. Duration & Period

30 hrs. & July to September 2019

3. Information Brochure and Course Content of Value Added Courses

Enclosed as Annexure- I

4. List of students enrolled

Enclosed as Annexure- II

5. Assessment procedures:

Assessment - *Enclosed as Annexure- III*

6. Certificate model

Enclosed as Annexure- IV

7. No. of times offered during the same year:

1 time July to September 2019

8. Year of discontinuation:

9. Summary report of each program year-wise

Value Added Course- July to September 2019					
Sl. No	**Course Code**	**Course Name**	**Resource Persons**	**Target Students**	**Strength & Year**
1	VAC01/AHS/2019-15/07	Congestive Heart Failure And Pulmonary Edema	Dr. Sangeetha	AHS	30 AHS students July to September 2019

10. Course Feed Back

Enclosed as Annexure- V

RESOURCE PERSON

COORDINATOR
Dr.G.Somasundram

PRINCIPAL
Allied Health Sciences
Sri Lakshmi Narayana Institute of Allied Health Sciences
Osudu, Agaram Post, Puducherry - 605 502.

Course Proposal

Course Title: "**Pulmonary Edema with Left Ventricular Failure"**

Course Objective:

1. To enhance the performance skill in Pulmonary Edema with Left Ventricular Failure.
2. To assess the reaction of target allied Health students in Pulmonary Edema with Left Ventricular Failure getting their feedback.

Course Outcome: Improvement in the "Pulmonary Edema with Left Ventricular Failure"
Course Audience: Students of AHS Batch
Course Coordinator: Dr.G.Somasundram
Course Faculties with Qualification and Designation:
1. Dr.Sangeetha
Course Curriculum/Topics with schedule (Min of 30 hours)

SlNo	Date	Topic	Time	Hours
1.	15.07.2019	Introduction to Pulmonary Edema with Left Ventricular Failure	4-6p.m	2
2.	17.07.2019	What is Pulmonary Edema	2-4p.m	2
3.	20.07.2019	What is Left Ventricular	4-6p.m	2
4.	22.07.2019	Cardiogenic and Non-Cardiogenic Pulmonary Edema	4-6p.m	2
5.	23.07.2019	Drugs used in Pulmonary Edema	4-6p.m	2
6.	25.07.2019	Complications of Pulmonary Edema	4-6p.m	2
7.	27.07.2019	Drugs used in Left Ventricular Failure	4-6P.M	2
8.	29.07.2019	Complications of Left Ventricular Failure	4-6p.m	2
9.	03.08.2019	Contraindication for drugs used in Left Ventricular Failure with Pulmonary Edema	4-6p.m	2
10.	05.08.2019	COPD	4-6p.m	2
11.	07.08.2019	Chronic Bronchitis	4-6p.m	2
12.	09.08.2019	Emphysema	4-6p.m	2
13.	10.08.2019	Pre course and Post Course evaluation, Feedback analysis	2-4p.m	2
14.	12.08.2019	Steps model explanation and various performance assessment	4-6p.m	2 m
15.	17.08.2019	Orientation of the students about the training program and assessment	2-4p.m	2
		Total		30 hrs

REFERENCE BOOKS:

1. Dorland's illustrated medical dictionary (32nd Ed.). Saunders/Elsevier. p. 593. ISBN 9781416062578.

2. Ware LB, Mathai MA (December 2005). "Clinical practice. Acute pulmonary edema". N. Engl. J. Med. 353 (26): 2788–96. Doi: 10.1056/NEJMcp052699. PMID 16382065.

3. Οἴδημα, οἰδέω. Liddell, Henry George; Scott, Robert; a Greek–English Lexicon at the Perseus Project.

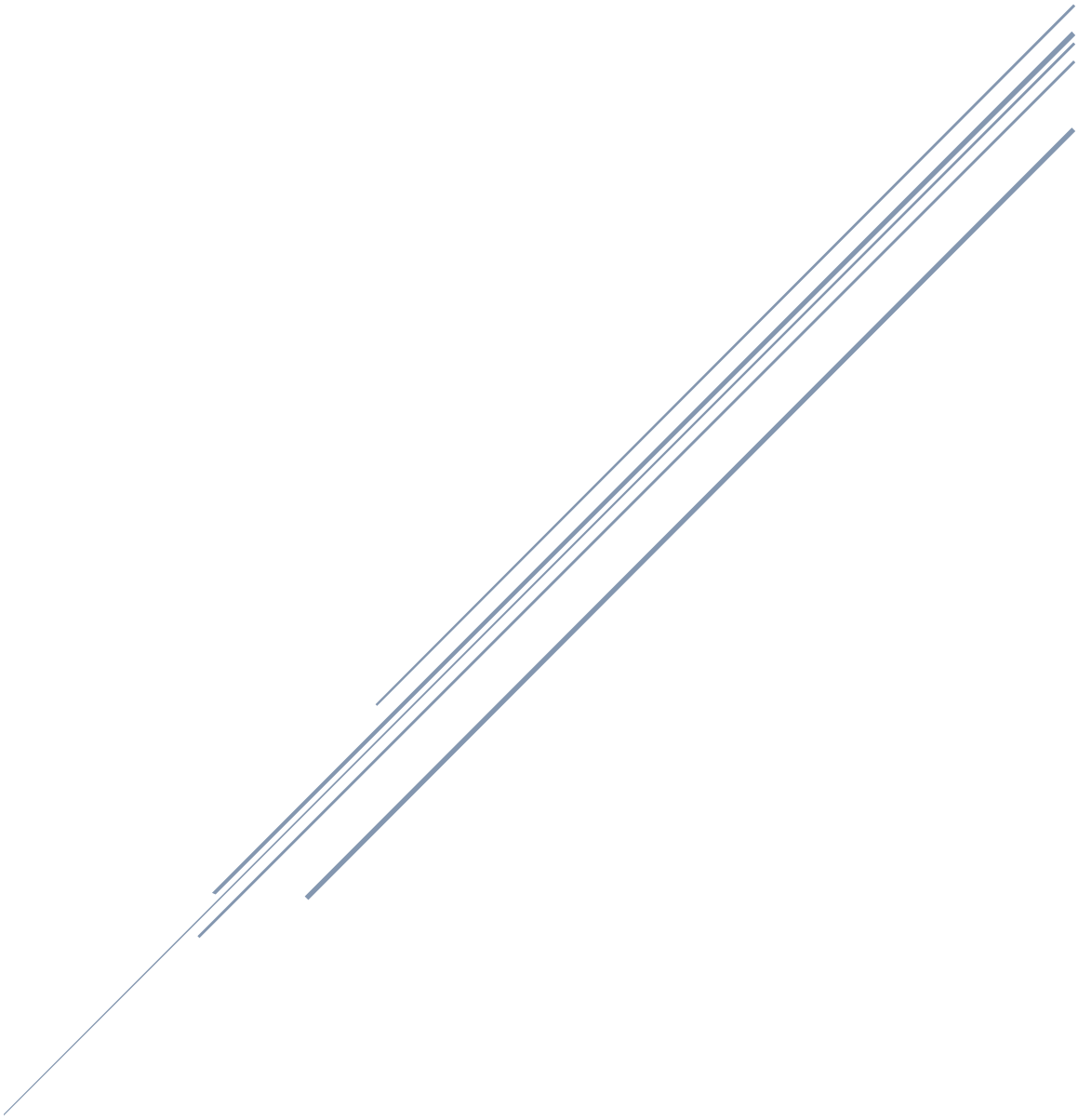
4. What Is Pulmonary Hypertension? From Diseases and Conditions Index (DCI). National Heart, Lung, and Blood Institute. Last updated September 2008. Retrieved on 6 April 2009.

5. Adair, Olivia Vynn (2001). Cardiology secrets (2nd Ed.). Elsevier Health Sciences. Chapter 41, page 210. ISBN 1-56053-420-6.

6. Papaioannou V, Terzi I, Dragoumanis C, Pneumatikos I (2009). "Negative-pressure acute tracheobronchial hemorrhage and pulmonary edema". J Anesth. 23 (3): 417–20. Doi: 10.1007/s00540-009-0757-0. PMID 19685125. S2CID 9616605.

7. Hines, Roberta L. and Marschall, Katherine. Stoelting's Anesthesia and Co-Existing Disease. 6th edition. 2012. Pages 178 and 179.

PULMONARY EDEMA WITH HEART FAILURE



CONTENT

Congestive Heart Failure and Pulmonary Edema

LV FAILURE

Introduction

The heart is comprised of the pericardium, myocardium, and endocardium. Pathology in any of those structures can lead to heart failure. Left ventricular failure occurs when there is dysfunction of the left ventricle causing insufficient delivery of blood to vital body organs. Left ventricular failure can further subdivide into heart failure with preserved ejection fraction (HFpEF with EF over 50%), heart failure with reduced ejection fraction (HFrEF with EF less than 40%), or heart failure with mid-range ejection fraction (EF between 41 and 49 percent).

Etiology

The most common etiologies of left heart failure are coronary artery disease and hypertension. The latter can cause left heart failure through left ventricular hypertrophy (leading to HFpEF), and also serves as a risk factor for coronary artery disease (which can lead to HFrEF). Diabetes, smoking, obesity, male gender, and a sedentary lifestyle are also considered risk factors. Many of these causes are preventable, and as such risk factor control remains of extreme importance in preventing heart failure.

Epidemiology

Heart failure is more prevalent and has a higher incidence in the elderly population. Approximately 5.7 million people in the United States have diagnosed heart failure. The incidence is around 10 per 1000 in people over 65. Approximately 50% of all patients with heart failure are considered to have HFrEF; this diagnosis is becoming more prevalent with time. HFpEF, as opposed to HFrEF, is more common in women (79% versus 49%) and also tends to affect an older population.

Pathophysiology

Multiple mechanisms can lead to left heart failure. Chronic or poorly controlled hypertension causes increased afterload and therefore increased cardiac workload, which can lead to hypertrophy of the left ventricle. Initially, this hypertrophy serves as a compensatory mechanism and can help maintain cardiac output, but long-term can inhibit relaxation of the myocardium leading to impaired cardiac filling and decrease left ventricular output. Coronary arterial disease causes direct ischemic damage to the myocardium, leading to remodeling and scar formation, which decreases contractility and cardiac output. Arrhythmias can cause remodeling, but in general, decrease cardiac output by impaired ventricular filling and decreased ventricular

relaxation. Cardiomyopathies encompass a diverse pathologic spectrum and have variable mechanisms causing cardiac dysfunction.

History and Physical

Patients with left heart failure may present with complaints of shortness of breath (often on exertion, a sensitivity of 89%), orthopnea (a specificity of 89%), paroxysmal nocturnal dyspnea and/or symptoms of volume overload (e.g., leg swelling, weight gain, increased abdominal girth, or right upper quadrant pain due to liver congestion). Interestingly, some patients with advanced disease might experience weight loss, referred to as "cardiac cachexia."

On physical exam, the most common signs encountered are:

- Rales on lung auscultation indicative of pulmonary edema
- Decreased breath sounds on lung auscultation suggestive of pleural effusion
- S3 gallop on heart auscultation indicative of elevated left ventricular end-diastolic pressure
- Point of maximal impulse displaced laterally on palpation characteristic of increased heart size
- Jugular venous distention (jugular venous pressure over 8 cm of water) indicative of elevated right atrial pressure
- Positive hepatojugular reflux (exerting manual pressure on the congested liver causing increased jugular venous pressures)
- Increased abdominal girth due to ascites
- Swelling of the scrotum
- Low blood pressure and rapid heart rate can occur in severely decompensated failure due to decreased cardiac output

Evaluation

The diagnosis of heart failure is clinical. However, several tests are available for further evaluation:

- Laboratory tests: brain natriuretic peptide (BNP) or NT-proBNP may be the most helpful as it can differentiate acute heart failure from other causes of shortness of breath. However, this test lacks specificity, and a high level of this hormone is not diagnostic of acute heart failure. Other laboratory tests include troponin T (to detect myocardial infarction, although the levels may be high due to heart failure itself), complete blood count, and basic metabolic panel (low sodium, in particular, indicates advanced disease) and liver function tests (to detect liver injury due to volume overload).

- Electrocardiography can show nonspecific findings, like ischemic changes, left ventricular hypertrophy, or arrhythmias.
- Echocardiography can help distinguish HFrEF from HFpEF by determining the ejection fraction, and diastolic left ventricular function can evaluate associated regional wall motion abnormalities that may be suggestive of an ischemic component, as well as valvular and pericardial pathologies.
- Coronary angiography is indicated in patients with anginal symptoms and may also be indicated in patients with worsening heart failure symptoms.

Treatment / Management

Patients should receive education on the importance of lifestyle modification for improving the outcome of their disease. This includes reasonable salt consumption and avoidance of alcohol, nicotine, and recreational drugs.

Treating the underlying cause is of extreme importance as some heart failure conditions may be reversible when the precipitating factors are addressed, like cardiomyopathies induced by alcohol, tachycardia or ischemia. Tight control of blood pressure will also help prevent further deterioration. Besides loop diuretics for volume overload, the pharmacologic treatment differs between HFrEF and HFpEF: -For HFrEF, the mainstay of treatment is the combination of an angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs) with a beta blocker (carvedilol, metoprolol or bisoprolol). If the patient remains symptomatic on a maximal dose of ACE inhibitor or ARB, an angiotensin receptor-neprilysin inhibitor may be substituted. Other medications include hydralazine, nitrates, and mineralocorticoid receptor antagonists such as spironolactone, ivabradine, and digoxin (as a last resort). Nitrates in combination with hydralazine may be especially efficacious in African American patients. Digoxin, ivabradine and the diuretics have not been shown to offer any mortality benefit.

- For HFpEF, the treatment focuses on the underlying cause or contributing factors: control of blood pressure, revascularization if ischemic cardiomyopathy, and management of arrhythmias. A mineralocorticoid receptor antagonist may be beneficial in these patients.

Severely symptomatic patients with an ejection fraction less than 35% should obtain a referral for implantable cardioverter-defibrillator (ICD) or cardiac resynchronization therapy (CRT) (depending on QRS width and the type of intraventricular conduction delay) after medical optimization. In advanced cases, mechanical circulatory assist devices such as an LVAD, continuous infusion of inotropic medications such as dobutamine or milrinone (which is still possible in the ambulatory setting), and cardiac transplantation are options.

Differential Diagnosis

- When approaching a patient with shortness of breath on exertion, a broad differential diagnosis exists. A patient with established left heart failure might have a concomitant disease that might

contribute to the patient's presentation, and additional workup for other conditions is warranted in case of atypical presentations. Those conditions include other cardiovascular causes (like primary pulmonary hypertension), pulmonary causes (like chronic obstructive pulmonary disease and interstitial lung disease) and extra-cardiopulmonary causes (like anemia).

- Isolated lower limb edema is less likely heart failure, and other causes must be ruled out first, like venous insufficiency, cirrhosis, nephrotic syndrome, lymphedema and thrombosis of the veins.

Staging

The "2009 focused update incorporated into the ACC/AHA 2005 guidelines for the diagnosis and management of heart failure in adults" recommends a staging for heart failure. Patients with stages A and B are those who are asymptomatic but are prone to develop heart failure in the future, for example, diabetic and hypertensive patients. Stage B differs from stage A by the presence of a structural cardiac abnormality, like ventricular hypertrophy or systolic dysfunction, but again with no symptoms. Stage C and D patients include those who have ever experienced symptoms of heart failure. Stage D patients are those with severe heart failure who require advanced treatment like mechanical support, around-the-clock inotropic agents and heart transplantation.Stage C and D patients can be further classified based on the New York Heart Association (NYHA) Functional Classification:

- Class I: symptoms not restricting daily activities.
- Class II: symptoms occurring at moderate effort slightly restricting daily activities.
- Class III: symptoms occurring at minimal effort with significant restriction of daily activities.
- Class IV: debilitating symptoms are occurring at rest.

Prognosis

The mortality rate in heart failure in 2008 was 18.2 per 100,000 for males and 15.8 per 100,000 for females based on the "Morbidity and Mortality: 2012 Chart Book on Cardiovascular, Lung and Blood Diseases". Patients with HFpEF have been known to have a lower mortality rate than those with HFrEF.

Multiple variables have associations with worse outcome in heart failure: male gender, advancing age, low ejection fraction, high NYHA functional class, low hematocrit, and sodium levels, high brain natriuretic peptide, low peak exercise oxygen uptake, wide QRS, renal failure, low blood pressure, elevated heart rate, and volume overload refractory to medical treatment.

Complications

Left heart failure can be complicated with severe volume overload leading to respiratory distress and anasarca as well as arrhythmias (tachyarrhythmias or bradyarrhythmias), cardiogenic shock due to pump failure and death. It was also reported that pulmonary embolism, acute coronary syndrome, cerebrovascular accidents and rupture of the myocardium are common causes of sudden death in patients with heart failure. In addition to implantable cardioverter-defibrillators, several medications (stated above) reduce mortality in left heart failure and initiation of therapy should be as soon as indicated.

Deterrence and Patient Education

Compliance with lifestyle modification and pharmacologic treatment and control of precipitating factors (like hypertension and arrhythmias) are crucial to prevent hospitalizations for heart failure exacerbation and improve the quality of life. Many of those precipitating factors are potentially under patient control. That is why patient education is an integral part of a multidisciplinary approach to decrease mortality and morbidity due to heart failure.

Enhancing Healthcare Team Outcomes

An interprofessional approach is crucial in patients with advanced heart failure with an increased risk of readmissions. In a systematic review of 29 randomized controlled trials, an interprofessional approach decreased heart-failure related hospital admissions by 27% and all-cause mortality by around 25%. This approach was also cost-effective. Well-trained nurses are an integral part of this approach since heart failure clinics led by nurses and in coordination with the patients' providers were very successful in enhancing patient outcome. In addition to the availability of heart failure clinics, training personnel in heart failure management and patient education are cornerstones to a successful an interprofessional approach. Telephone follow up reduced hospital readmissions due to heart failure but not mortality.

Risk Factors and Etiology of Heart Failure

There are many causes of heart failure and the most common of which is coronary artery disease in the United States. The importance of identifying the risk factors for heart failure is that heart failure is preventable. In recognition of the preventable nature of the condition, the American College of Cardiology and the American Heart Association have modified their classification schemes so that patients currently without any structural abnormalities are identified early and treated appropriately. Treatment of systolic and diastolic hypertension concurrently in alignment with contemporary guidelines reduces the risk of heart failure by approximately 50%.

Risk Factors for Heart Failure

- Coronary artery disease (CAD)
- Connective tissue disorders (i.e., rheumatoid arthritis, scleroderma, systemic lupus erythematous)

- Endocrine disorders (i.e., diabetes mellitus, thyroid function disorders, growth hormone deficiency)
- Hypertension
- High-output conditions (i.e.: anemia, Paget disease)
- Valvular heart disease
- Metabolic causes (i.e., obesity)
- Myocarditis (i.e., secondary to HIV/AIDS, medications, or viruses)
- Infiltrative disorders (i.e., amyloidosis, sarcoidosis)
- Peripartum cardiomyopathy
- Stress cardiomyopathy (Takotsubo)
- Valvular heart disease
- Medications (i.e., amphetamines, anabolic steroids)
- Tachycardia induced cardiomyopathy
- Toxins (i.e., cocaine, alcohol)
- Nutritional deficiency (i.e., L-carnitine deficiency, thiamine)

Risk Factors and Etiology Acute Heart Failure (ADHF) / Flash Pulmonary Edema

Acute heart failure is the worsening of heart failure symptoms to the point that the patient requires intensification of therapy and intravenous treatment. Acute heart failure can be dramatic and rapid in onset, such as flash pulmonary edema or more gradual with the worsening of symptoms over time until a critical point of decompensation is reached. For those with a history of pre-existing heart failure, there is often a clear trigger for decompensation.

Potential Triggers of Acute Decompensated Heart Failure / Flash Pulmonary Edema

- Arrhythmia
- Acute coronary syndrome (CAD)
- Infection
- Worsening hypertension (i.e., hypertensive crisis)
- Medication non-adherence
- Acute renal artery stenosis
- Left ventricular diastolic dysfunction
- Obstructive sleep apnea
- Stress (Takotsubo) cardiomyopathy

Epidemiology

Heart Failure is a major public health problem and is now the most common cause of hospitalization in the US among patients 65 years and older, and approximately 915000 new cases of heart failure are diagnosed each year in the United States. The increasing prevalence of heart failure is most likely secondary to the aging of the population, increased risk factors, better outcomes for acute coronary syndrome survivors, and a reduction in mortality secondary to improved management of chronic conditions. Incidence rates for heart failure increase with age for both sexes.

The lifetime risk of developing heart failure for those over age 40 years residing in the U.S. is 20%. The risk and incidence of heart failure continue to increase from 20 per 1000 people age 60 to 65 years to over 80 per 1000 people over age 80. There are also differences in risk for heart failure based on the population, with African Americans having the highest risk and greater five-year mortality for heart failure than the white population in the U.S. The European Society of Cardiology states that the prevalence of heart failure is 1 to 2% and rises to greater than 10% in the over 70 population.

Heart Failure Statistics

- Heart Failure survival has improved over time, yet the absolute 5-year mortality rates from diagnosis for HF have remained at 50 percent.
- Heart failure is the number one diagnosis among all hospitalizations, and the cost of annual heart failure care exceeds $30 billion every year.
- Most of the cost spent on heart failure patients is for hospitalizations and re-admissions.

Acute Heart Failure

While consensus guidelines tend to use the term heart failure to refer to those with established chronic disease, acute heart failure is defined as a more rapid onset of signs and symptoms or the gradual worsening of chronic symptoms that necessitate intravenous treatment. Acute heart failure exacerbation that requires hospitalization tends to occur in the more elderly population mean age of 79 years old with a slightly higher preponderance of women affected than men. Data from the UK National Heart Failure Audit shows mortality rates of approximately 10% during the index admission, 30-day post-discharge mortality of 6.5% and 1-year mortality of 30%.

Pathophysiology

Different cardiovascular or metabolic abnormalities can cause heart failure, but in most patients, the clinical symptoms are secondary to left ventricular dysfunction. In cases caused by left ventricular dysfunction, the ejection fraction may be preserved or compromised. The ejection fraction is important because most clinical trials select patients based on the percentage of ejection fraction and use the data on ejection fraction to help guide therapy. In contrast,

pulmonary edema associated with acute decompensated heart failure is secondary to dysregulation of pulmonary fluid homeostasis and the forces that balance fluid movement into the alveolar space

History and Physical

History

Heart failure is mainly a clinical diagnosis. It is essential to consider the following during the history and physical.

1. The presentation of heart failure may vary based on each patient. If the patient has a history of heart failure in the past, ask them if this is the same presentation as when they had previous episodes of heart failure or an acute decompensation.

2. Consider non-cardiac and other causes for the patient's symptoms. It is important to ensure that there is a broad differential diagnosis and to avoid anchoring bias, premature closer, and diagnostic inertia.

3. Heart failure symptoms:

- Increasing dyspnea (on exertion, on lying flat or at rest, exercise intolerance)
- Increasing leg swelling, ascites, edema
- Increased body weight -ask patients if they have been tracking their weight at home and if it has increased since their symptoms have become worse.
- What are the patient's baseline symptoms, and are the current symptoms worse or similar to when they had previous heart failure exacerbations?
- Palpitations, automatic implantable cardioverter-defibrillator (*AICD*) shocks (associated with worse prognosis)
- Chest pain, fatigue
- Duration of Illness, recent or frequent hospitalizations for heart failure
- Medications or diet changes
- Anorexia, cachexia, or early satiety (associated with worse prognosis)
- Symptoms of transient ischemic attack (TIA) or thromboembolism (indicate a possible need for anticoagulation)
- Social history and family history (to assess for possible familial cardiomyopathy, alcohol or other cause
- Travel history (exposure risk some tropical diseases)

Physical Examination

The physical examination should include the following:

- Vital signs - assess blood pressure, heart rate, temperature, oxygen saturation, and respiratory rate. Vital signs are important in helping develop and refine the differential diagnosis and help the healthcare provider to better tailor the physical examination.
- Check patient weight – The patient's weight and BMI should be checked during each office visit. The information can be used to track response to treatment and the potential progression of heart failure or ADHF. Losing weight can also be a warning sign of worsening HF.
- Assessment of jugulovenous distension – Jugulovenous distension can be a marker for fluid overload. The patient should be lying at a 45-degree angle in bed to get an accurate assessment.
- Pulse - Assess the regularity and strength of the pulse
- Cardiac Examination
    - Extra heart sounds (i.e., S3 is associated with a worse prognosis), murmurs
    - Size and location of maximal cardiac impulse (can suggest ventricular enlargement if displaced)
    - Presence of right ventricular heave
- Pulmonary Examination - Respiratory rate, rhonchi, rales (note: pleural effusions can mask reduce breath sounds, and rhonchi or rales may be less prominent)
- Abdominal examination - check for ascites, hepatomegaly, hepatojugular reflux
- Lower extremity examination - assess for peripheral edema, the temperature of skin (cool lower extremities may be suggestive of worse cardiac output)

Evaluation

Classification of Heart Failure

Classification is one of the key determinates of how to evaluate and treat heart failure. When a patient is in acute or decompensated heart failure, our focus is on expeditious identification and treatment of life threats. When evaluating chronic heart failure, different classification schemes are available. The classification scheme used to categorize the type and degree of heart failure is based on the presentation and will affect the treatment and prognosis of the condition. Heart failure classification schemes are generally based on one of the following:

1. Anatomic findings (i.e., heart failure with different degrees of ejection fraction)
2. The chamber of the heart involved (including functional)
3. Symptoms of the patient

All heart failure patients should also be classified based on the ACCF/American Heart Association stages of heart failure, a New York Heart Association functional classification.[1]

ACCF/AHA Stages of Heart Failure

The ACC/AHA stages of heart failure are defined by the risk of heart failure, the presence of active heart failure, and whether structural heart disease is present. In general, the higher the classification, the greater the treatment and interventions that the patient may require.

- A - No structural heart disease present, high risk for heart failure, and asymptomatic
- B - Structural heart disease present, asymptomatic
- C - Structural heart disease present and current or previous symptoms of heart failure
- D - Heart failure that is refractory and requiring specialized interventions

NYHA Functional Classification of Heart Failure

The NYHA classification is a functional classification of heart failure and based upon how much the patients' symptoms limit their physical activity and to what degree physical activity can cause the person to become symptomatic. The grading scale is from me up to the most severe of grade IV where the patient is unable to carry on physical activity and has symptoms at rest.

Diagnostic Tests

Testing for heart failure patients should be focused on the patient's symptoms, clinical suspicion, and the current and any pre-existing or current stage of heart failure. The ordering of multiple routine tests should be avoided in all heart failure patients. Basic tests that should merit consideration for all patients evaluated for heart failure are the following:

- Serum electrolytes and kidney function
- Complete blood count
- Lipid level
- Liver function tests
- Troponin level - if there is concern that myocardial injury as the cause for symptoms
- Thyroid-stimulating hormone
- An electrocardiogram should also be performed in all heart failure patients

Other tests that may be considered based on severity and classification of patient condition are:

- Chest x-ray to assess for signs of pulmonary congestion or edema in acute decompensated heart failure
- Biomarkers - used to assess patients with more complex symptoms than acute heart failure (i.e., B-type natriuretic peptide and N-terminal pro-B-type natriuretic peptide)

For further information on the recommendations for evaluating heart failure, please see the American Heart Association and New York Heart Association classification based heart failure guidelines.

Treatment / Management

Treatment of Acute Decompensated Heart Failure and Pulmonary Edema

The focus of treatment for patients in heart failure is dependent on the severity of the symptoms and the stage of heart failure. When patients are in acute decompensated heart failure or flash pulmonary edema, the most important focus for therapeutic interventions is the enhancement of hemodynamic status through reduction of vascular congestion and improving preload, afterload, and myocardial contractility.

In flash pulmonary edema, where there is a rapid onset of heart failure, the initial management, and treatment goals are very similar to acute decompensated heart failure. Treatment options for acute decompensated heart failure and flash pulmonary edema are as follows:

1. ABC's - As with all patients, it is important to assess airway, breathing, and circulation in the initial evaluation and initiate appropriate management based on patient status. In decompensated heart failure, patients should be hooked up to cardiorespiratory monitoring, have IV access and oxygen administered if hypoxic or tachypnea, ECG performed, and have labs drawn based on clinical suspicion and patient condition.
2. Diuretics - most patients presenting in heart failure have a form of volume overload. AHA and ACC guidelines recommend intravenous (IV) loop diuretic administration to treat fluid overload. IV administration is preferred over oral for diuretics to maximize the bioavailability of the medication and clinical effect. Furosemide is a common treatment for acute decompensated heart failure and flash-pulmonary edema because of its anti-vasoconstrictor and diuretic effects.
3. Vasodilators - Patients with acute decompensated heart failure with HTN and acute pulmonary edema can benefit from treatment with vasodilators. Most vasodilators promote smooth muscle relaxation and vasodilatation to reduce preload and afterload through the cyclic guanine monophosphate pathway. Vasodilation relieves pulmonary vascular congestion and improves left ventricular preload and afterload. The common vasodilator medications used are Nitroglycerin and Sodium Nitroprusside.

    4.

    - Nitroglycerin can be given transdermal, sublingual, or intravenously depending on patient condition. In acute decompensation or flash pulmonary edema, nitroglycerin is best given sublingually or intravenously to allow for titration of effect.
    - Sodium Nitroprusside is only given intravenously. Patients with renal dysfunction treated with sodium Nitroprusside may need to have their cyanide levels monitored. Check with your pharmacist about guidelines for monitoring cyanide levels

5. Ionotrophic Medications - Additional treatment options for patients in cardiogenic shock or who have signs of end-organ dysfunction secondary to hypo perfusion. Inotropes should only be used as a treatment adjunct in acute decompensated

heart failure since data from the ADHERE registry suggest increased mortality with use. Dobutamine and MIlrinone are two inotropes that are more commonly used. Dobutamine is preferable for beta-blocker naive patients, while Milrinone is preferred for patients previously taking oral beta blockers who experience an acute decompensation.

6. CPAP/BIPAP - Continuous Positive Airway Pressure (CPAP) and Bilevel Positive Airway Pressure (BIPAP) are noninvasive methods of respiratory support to treat respiratory insufficiency secondary to pulmonary vascular congestion and pulmonary edema. The use of CPAP and BIBAP has reduced the need for intubation and mechanical ventilation in heart failure patients with acute respiratory decompensation. In situations where CPAP and BIPAP are ineffective in improving the patient's respiratory status, consider early intubation and mechanical ventilation to help prevent further decompensation and progression of symptoms.
7. Coronary revascularization - When performed in appropriately selected patients, revascularization can reduce mortality and morbidity by improving diastolic and systolic dysfunction. According to the American Heart Association 2013 CHF guidelines, coronary artery revascularization may be indicated as an intervention for heart failure patients with angina, LV dysfunction, and CAD. Interventional cardiology should be consulted early for patients according to AHA recommendations.
8. Admission to inpatient service for further treatment and evaluation. Patients being treated for flash pulmonary edema should be admitted to the hospital with the level of monitoring and care appropriate for each case. For select patients with acute decompensated heart failure, it may be possible to treat them at home depending on the severity of symptoms

The Medical Management of Heart Failure – Risk Factor Modification and Prevention of Acute Decompensation

While acute decompensated heart failure and flash pulmonary edema can be dramatic and require intensive care and aggressive therapy, the main focus of heart failure management is on helping prevent the progression of the disease and mitigate episodes of acute exacerbation. The American Heart Association and the New York Heart Association stages of heart failure are what medical practitioners often use to guide the evidence-based treatment of heart failure. Treatment of early stages of chronic heart failure usually focuses on risk factor modification, and as the disease process progresses, it starts to include more aggressive interventions.

- Risk factor modification
    1. Dietary and lifestyle changes, such as decreased salt intake, reducing obesity and smoking cessation
    2. Tighter control and management of hypertension, diabetes and dyslipidemia and other chronic diseases that can exacerbate CHF
- More aggressive intervention for those with a higher degree of CHF

1. Echocardiography for patients with a higher risk of left ventricular ejection fraction reduction
2. Implantable cardiac defibrillator placement when indicated in patients with ischemic cardiomyopathy at high risk of sudden death

For further information on the medical management of chronic heart failure, please refer to the American Heart Association and New York Heart Association guidelines.

Advanced Treatment Strategies for End-Stage Congestive Heart Failure

For select patients with end-stage heart failure, which are refractory to other treatment strategies, the option of mechanical circulatory support and cardiac transplantation should be considered. Mechanical circulatory support, as for example, a left ventricular assist device, is often used as a bridge therapy until a heart transplant is available. In certain situations, mechanical circulatory support is utilized as destination therapy.

For patients who are not candidates for mechanical circulatory support or cardiac transplantation, palliative care, and continuous inotropic support should be a consideration and discussed with the patient.

Differential Diagnosis

When patients present in acute decompensated heart failure or flash pulmonary edema, there are many different diagnoses to consider, based on the risk factors for heart failure alone. It is also important to consider other potentially life-threatening causes of heart failure.

1. Sepsis - Septic patients are at risk of multiorgan system failure. Approximately 1 out of 3 patients with sepsis present with reversible left ventricular systolic dysfunction, reduced ejection fracture, and 1 out of 2 patients with sepsis have left ventricular or right ventricular diastolic dysfunction. The cardiac dysfunction associated with sepsis can result in significantly increased mortality. Left ventricular diastolic dysfunction is associated with an increased mortality risk of 80%, and right ventricular diastolic dysfunction is associated with a 60% increased mortality
2. Acute respiratory distress syndrome (ARDS) is characterized by acute respiratory failure, and diffuse pulmonary infiltrates, which could potentially mimic flash pulmonary edema.
3. Neurological causes - Hemispheric and hippocampal brain infarcts are associated with heart failure and sudden cardiac death. Infarct of certain areas of the brain tissue can result in a sympathetic storm and loss of vasomotor homeostasis precipitated neurogenic pulmonary edema.
4. Pulmonary embolism - While a massive pulmonary embolism can cause acute cardiac dysfunction secondary to obstruction of blood flow, post pulmonary embolism syndrome may also present with heart failure type symptoms. Post pulmonary embolism syndrome, reduced exercise tolerance has been associated with reduced left ventricular ejection fraction, arrhythmia, valvular dysfunction, n and left ventricular diastolic dysfunction.

5. Acute coronary syndrome - Acute coronary syndrome or myocardial infarction is a common cause of acute decompensated heart failure.

Prognosis

The diagnosis of heart failure alone can be associated with a mortality rate greater than many cancers. Despite advances made in heart failure treatments, the prognosis of the condition worsens over time, resulting in frequent hospital admissions and premature death. One recent study showed that patients recently diagnosed with new-onset heart failure had a mortality rate of 20.2% at one year and 52.6% at five years. The one and five-year mortality rates also increase significantly based on patient age. Another study showed that the one and five-year mortality for patients at 60-year-old is 7.4%, and 24.4% and for patients at 80-year-olds is 19.5% and 54.4%. The mortality rates were similar when evaluated across different cardiac ejection fractions.

The prognosis is worse for heart failure patients who are hospitalized. Heart failure patients commonly require repeat hospitalizations and develop an intolerance for standard treatments as the disease progresses. Data from U.S. Medicare beneficiaries hospitalized during 2006 showed 30-day and 1-year mortality rates post admission of 10.8% and 30.7, % respectively. Mortality outcomes at one year also demonstrate a clear relationship with age and increase from 22% at 65 years old to 42.7% for patient's age 85 years and older.

Complications

Potential Complications of Heart Failure

- Worsening clinical status despite aggressive therapy
- Associated renal impairment and organ dysfunction which can compromise heart failure treatment efforts
- Recurrent hospitalizations for heart failure or most commonly associated co-morbidities, with resultant financial and personal cost to patients and families
- The progressive loss of ability to carry out activities of daily life
- Increased morbidity and mortality from the date of patient initial diagnosis with heart failure

Deterrence and Patient Education

Effective treatment of comorbidities and risk factor reduction can decrease the chance of developing heart failure. Patient education should be focused on ensuring compliance with prescribed evidence-based treatments.

- Hypertension - effective treatment of systolic and diastolic hypertension can reduce the risk of heart failure by approximately 50%
- Diabetes - is directly associated with the development of heart failure, independent of other associated clinical conditions
- Alcohol - heavy alcohol use is associated with heart failure
- Metabolic syndromes - important to keep up treatment based on evidence-based guidelines to decrease the risk of heart failure (i.e., lipid disorders)
- Patient education regarding dietary salt restriction and fluid restriction is imperative

Enhancing Healthcare Team Outcomes

The treatment of heart failure and acute decompensated heart failure is challenging despite the use of maximal evidence-based therapy based on the stage of heart failure. Given the limited effect that current treatment strategies have on the progression of heart failure, it is important to identify ways to maximize patient outcomes and quality of care by the interprofessional team.

Patients at potential risk for heart failure based on comorbidities or other identified risk factors should receive appropriate evidence-based preventative counseling and treatments. When appropriate, the primary care providers who may be the most involved in the management of the patients' risk factors should consult other specialists, including cardiologists, endocrinologists, pharmacists, cardiology nurses, and nutritionists, to ensure that they are providing the best advice and treatment for their patients. Nurses monitor patients, provide education, and collaborate with the physicians and the rest of the team to improve outcomes. Pharmacists review medications, inform patients and their families about side effects and monitor compliance.

Given the propensity of heart failure patients to require re-current admissions, often because of non-heart failure related conditions, the collaboration between inpatient and outpatient services can be of benefit in the continuity of care and helping promote improved outcomes.

PULMONARY EDEMA

Pulmonary edema is a condition caused by excess fluid in the lungs. This fluid collects in the numerous air sacs in the lungs, making it difficult to breathe.

In most cases, heart problems cause pulmonary edema. But fluid can collect in the lungs for other reasons, including pneumonia, exposure to certain toxins and medications, trauma to the chest wall, and traveling to or exercising at high elevations.

Pulmonary edema that develops suddenly (acute pulmonary edema) is a medical emergency requiring immediate care. Pulmonary edema can sometimes cause death. The outlook improves if you get treated quickly. Treatment for pulmonary edema varies depending on the cause but generally includes supplemental oxygen and medications.

Symptoms

Pulmonary edema signs and symptoms may appear suddenly or develop over time. The signs and symptoms you have depends on the type of pulmonary edema.

Sudden (acute) pulmonary edema signs and symptoms

- Difficulty breathing (dyspnea) or extreme shortness of breath that worsens with activity or when lying down
- A feeling of suffocating or drowning that worsens when lying down
- A cough that produces frothy sputum that may be tinged with blood
- Wheezing or gasping for breath
- Cold, clammy skin
- Anxiety, restlessness or a sense of apprehension
- Bluish lips
- A rapid, irregular heartbeat (palpitations)

Long-term (chronic) pulmonary edema signs and symptoms

- Difficulty breathing with activity or when lying flat
- Awakening at night with a cough or breathless feeling that may be relieved by sitting up
- More shortness of breath than normal when you're physically active

- Wheezing
- Rapid weight gain
- Swelling in your lower extremities
- Fatigue
- New or worsening cough

High-altitude pulmonary edema (HAPE) signs and symptoms

HAPE can occur in adults and children who travel to or exercise at high altitudes. Signs and symptoms are similar to those that occur with acute pulmonary edema and can include:

- Headache, which may be the first symptom
- Shortness of breath with activity, which worsens to shortness of breath at rest
- Decreased ability to exercise as you once could
- Dry cough, at first
- Later, a cough that produces frothy, pink sputum
- A very fast heartbeat (tachycardia)
- Weakness
- Chest pain
- Low-grade fever

Signs and symptoms of high-altitude pulmonary edema (HAPE) tend to get worse at night.

When to see a doctor

Pulmonary edema that comes on suddenly (acute pulmonary edema) is life-threatening. Call 911 or emergency medical help if you have any of the following acute signs and symptoms:

- Shortness of breath, especially if it comes on suddenly

- Trouble breathing or a feeling of suffocating (dyspnea)
- A bubbly, wheezing or gasping sound when you breathe
- Pink, frothy sputum when you cough
- Breathing difficulty along with a lot of sweating
- A blue or gray color to your skin
- Confusion
- A significant drop in blood pressure resulting in lightheadedness, dizziness, weakness or sweating
- A sudden worsening of any of pulmonary edema symptoms

Don't attempt to drive yourself to the hospital. Instead, call 911 or emergency medical care and wait for help.

Request an Appointment at Mayo Clinic

Causes

The causes of pulmonary edema vary. Pulmonary edema is grouped into two categories, depending on where the problem started.

- If a heart problem causes the pulmonary edema, it's called cardiogenic pulmonary edema. Most often, the fluid buildup in the lungs is due to a heart condition.
- If pulmonary edema is not heart related, it's called noncardiogenic pulmonary edema.
- Sometimes, pulmonary edema can be caused by both a heart problem and a non-heart problem.

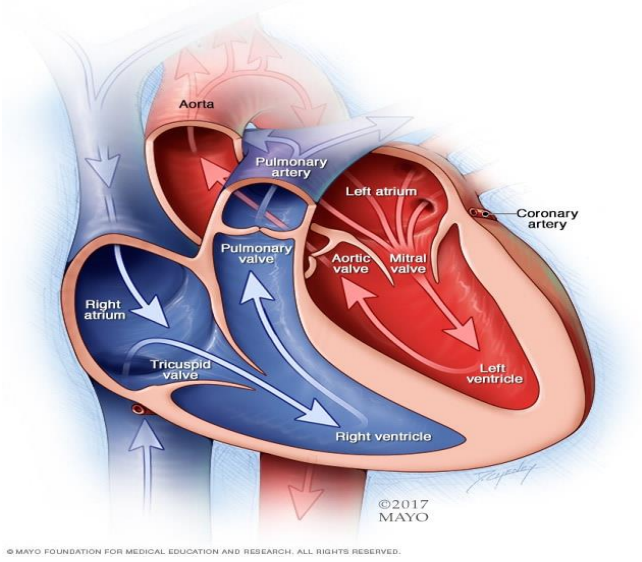
Understanding the relationship between your lungs and your heart can help explain why pulmonary edema may occur.

How your lungs work

Your lungs contain many small, elastic air sacs called alveoli. With each breath, these air sacs take in oxygen and release carbon dioxide. Normally, this exchange of gases occurs without problems.

But sometimes, the alveoli fill with fluid instead of air, preventing oxygen from being absorbed into your bloodstream.

How your heart works



Chambers and valves of the heart Open pop-up dialog box

Your heart is made of two upper and two lower chambers. The upper chambers (the right and left atria) receive incoming blood and pump it into the lower chambers (right and left ventricles). The lower chambers pump blood out of your heart.

Normally, deoxygenated blood from all over your body enters the right atrium then the right ventricle, where it's pumped through large blood vessels (pulmonary arteries) to your lungs. There, the blood releases carbon dioxide and picks up oxygen as it flows by the alveoli.

The oxygen-rich blood then returns to the left atrium through the pulmonary veins, flows through the mitral valve into the left ventricle and finally leaves your heart through the largest blood vessel in the body, called the aorta.

The heart valves keep blood flowing in the correct direction. The aortic valve keeps the blood from flowing backward into your heart. From the aorta, the blood travels to the rest of your body.

Heart-related (cardiogenic) pulmonary edema

Cardiogenic pulmonary edema is caused by increased pressures in the heart.

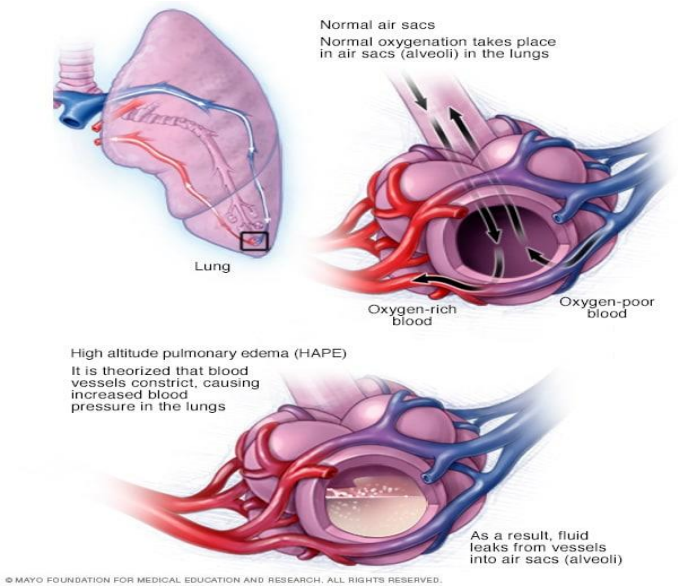
It's usually a result of heart failure. When a diseased or overworked left ventricle can't pump out enough of the blood it gets from your lungs, pressures in the heart go up. The increased pressure pushes fluid through the blood vessel walls into the air sacs.

Medical conditions that can cause heart failure and lead to pulmonary edema include:

- **Coronary artery disease.** Over time, the arteries that supply blood to your heart muscle can become narrow from fatty deposits (plaques). A slow narrowing of the coronary arteries can make the left ventricle weak. Sometimes, a blood clot forms in one of these narrowed arteries, blocking blood flow and damaging part of your heart muscle, resulting in a heart attack. A damaged heart muscle can no longer pump as well as it should.
- **Cardiomyopathy.** This term means heart muscle damage. If you have cardiomyopathy, your heart has to pump harder, and pressures go up. The heart may be unable to respond to conditions that require it to work harder, such as exercise, infection or a rise in blood pressure. When the left ventricle can't keep up with the demands that are placed on it, fluid backs up into your lungs.
- **Heart valve problems.** Narrowing of the aortic or mitral heart valves (stenosis) or a valve that leaks or doesn't close properly affects blood flow into the heart. The heart has to work harder, and pressures go up. If valve leakage develops suddenly, you may develop sudden and severe pulmonary edema.
- **High blood pressure (hypertension).** Untreated or uncontrolled high blood pressure can enlarge the heart.

- **Other heart problems.** Inflammation of the heart muscle (myocarditis), congenital heart defects and abnormal heart rhythms (arrhythmias) also may cause pulmonary edema.
- **Kidney disease.** High blood pressure due to narrowed kidney arteries (renal artery stenosis) or fluid buildup due to kidney disease can cause pulmonary edema.
- **Chronic health conditions.** Thyroid disease and a buildup of iron (hemochromatosis) or protein (amyloidosis) also may contribute to heart failure and cause pulmonary edema.

Non-heart-related (noncardiogenic) pulmonary edema



High-altitude pulmonary edema

Open pop-up dialog box

Pulmonary edema that is not caused by increased pressures in your heart is called noncardiogenic pulmonary edema.

Causes of noncardiogenic pulmonary edema include:

- **Acute respiratory distress syndrome (ARDS).** This serious disorder occurs when your lungs suddenly fill with fluid and inflammatory white blood cells. Many

conditions can cause ARDS, including severe injury (trauma), widespread infection (sepsis), pneumonia and severe bleeding.

- **Adverse drug reaction or drug overdose.** Many drugs — ranging from aspirin to illegal drugs such as heroin and cocaine — are known to cause pulmonary edema.
- **Blood clot in the lungs (pulmonary embolism).** If a blood clot travels from the blood vessels in your legs to your lungs, you can develop pulmonary edema.
- **Exposure to certain toxins.** Inhaling toxins or breathing in some of your stomach contents when you vomit (aspiration) causes intense irritation of the small airways and alveoli, resulting in fluid buildup.
- **High altitudes.** Pulmonary edema has been seen in mountain climbers, skiers, hikers and other people who travel to high elevations, usually above 8,000 feet (about 2,400 meters). High-altitude pulmonary edema (HAPE) generally occurs in those who don't first become acclimated to the elevation (which can take from a few days to a week or so). But people who live at high altitudes can get HAPE with no elevation change if they have a respiratory infection.
- **Near drowning.** Inhaling water causes fluid buildup in the lungs that is reversible with immediate medical care.
- **Negative pressure pulmonary edema.** Pulmonary edema can develop after a blockage in the upper airway causes negative pressure in the lungs from intense efforts to breathe despite the blockage. With treatment, most people with this type of pulmonary edema recover in about 24 hours.
- **Nervous system conditions or procedures.** A type of pulmonary edema called neurogenic pulmonary edema can occur after a head injury, seizure or brain surgery.
- **Smoke inhalation.** Smoke from a fire contains chemicals that damage the membrane between the air sacs and the capillaries, allowing fluid to enter your lungs.
- **Transfusion-related lung injury.** Blood transfusions may cause fluid overload in the left ventricle, leading to pulmonary edema.
- **Viral infections.** Pulmonary edema can be caused by viruses such as the Hantavirus and dengue virus.

Risk factors

Heart failure and other heart conditions that raise pressure in the heart increase the risk of pulmonary edema. Risk factors for heart failure include:

- Abnormal heart rhythms (arrhythmias)
- Alcohol use
- Congenital heart disease
- Coronary artery disease
- Diabetes
- Heart valve disease
- High blood pressure
- Sleep apnea

However, some nervous system conditions and lung damage due to near drowning, drug use, smoke inhalation, viral infections and blood clots also raise your risk.

People who travel to high-altitude locations above 8,000 feet (about 2,400 meters) are more likely to develop high-altitude pulmonary edema (HAPE). It usually affects those who do not first become acclimated to the elevation (which can take from a few days to a week or so).

Children who have existing pulmonary hypertension and structural heart defects may be more likely to get HAPE.

Complications

Complications depend on the underlying cause.

In general, if pulmonary edema continues, the pressure in the pulmonary artery can go up (pulmonary hypertension). Eventually, the heart becomes weak and begins to fail, and pressures in the heart and lungs go up.

Complications can include:

- Breathing difficulty
- Swelling of the legs, feet and abdomen
- Buildup of fluid in the membranes that surround your lungs (pleural effusion)
- Congestion and swelling of the liver

Immediate treatment is necessary for acute pulmonary edema to prevent death.

Prevention

You may be able to prevent pulmonary edema by managing existing heart or lung conditions and following a healthy lifestyle.

For example, you can reduce your risk of many kinds of heart problems by taking steps to control your cholesterol and blood pressure. Follow these tips to keep your heart healthy:

- Eat a healthy diet rich in fresh fruits, vegetables, whole grains, fat-free or low-fat dairy, and a variety of proteins.
- Manage your weight.
- Get regular exercise.
- Don't smoke.
- Limit salt and alcohol.
- Manage stress.

Preventing high-altitude pulmonary edema (HAPE)

To prevent HAPE, gradually ascend to high elevations. Although recommendations vary, most experts advise increasing elevation no more than 1,000 to 1,200 feet (about 300 to 360 meters) a day once you reach 8,200 feet (about 2,500 meters).

Some climbers take prescription medications such as acetazolamide or nifedipine (Adalat CC, Procardia) to help prevent signs and symptoms of HAPE. To prevent HAPE, start taking the medication at least one day before ascent. Ask your

doctor how long you need to take the medication after you've arrived at your high-altitude destination.

Diagnosis

Breathing problems require immediate diagnosis and treatment. Your doctor can make a preliminary diagnosis of pulmonary edema based on your signs and symptoms and the results of a physical exam, electrocardiogram and chest X-ray.

Once your condition is more stable, your doctor will ask questions about your medical history, especially whether you have ever had cardiovascular or lung disease.

Tests that may be done to diagnose pulmonary edema or to determine why you developed fluid in your lungs include:

- **Chest X-ray.** A chest X-ray can confirm the diagnosis of pulmonary edema and exclude other possible causes of your shortness of breath. It's usually the first test done when someone has signs or symptoms of pulmonary edema.
- **Chest CT.** A computed tomography (CT) scan of the chest may not provide the cause for the pulmonary edema, but can give your doctor indirect clues to help make a diagnosis.
- **Pulse oximeters.** A sensor is attached to your finger or ear and uses light to determine how much oxygen is in your blood.
- **Arterial blood gas test.** Blood is taken, usually from an artery in your wrist, and checked for the amount of oxygen and carbon dioxide it contains (arterial blood gas concentrations).
- **B-type natriuretic peptide (BNP) blood test.** Increased levels of BNP may signal a heart condition.
- **Other blood tests.** Blood tests to diagnose pulmonary edema and its causes also usually include a complete blood count, metabolic panel to check kidney function and thyroid function test.
- **Electrocardiogram (ECG or EKG).** This painless test detects and records the timing and strength of your heart's signals using small sensors (electrodes) attached to the skin on your chest and legs. The signals are recorded in the form of

waves on graph paper or a monitor. An ECG can show signs of heart wall thickening or previous heart attack. A portable ECG machine such as a Holter monitor may be used to continuously monitor your heartbeat at home.

- **Echocardiogram.** An echocardiogram creates a moving picture of your heart using sound waves (ultrasound). It can identify areas of poor blood flow, abnormal heart valves and heart muscle that is not working normally. Your doctor can use this test to help diagnose fluid around the heart (pericardial effusion).
- **Cardiac catheterization and coronary angiogram.** This test may be done if an ECG, echocardiogram or other tests don't show the cause of pulmonary edema, or if you also have chest pain.

  During cardiac catheterization, a doctor inserts a long, thin tube (catheter) in an artery or vein in your groin, neck or arm. X-rays help guide the catheter through the blood vessel to your heart. During a coronary angiogram, dye flows through the catheter, allowing blood vessels to show up more clearly on the X-rays. A coronary angiogram can reveal any blockages and measure the pressure in your heart chambers.
- **Ultrasound of the lungs.** This painless test uses sound waves to measure blood flow through the lungs. It can quickly reveal signs of fluid buildup and plural effusions. Lung ultrasound has become an accurate tool for diagnosing pulmonary edema.

More Information

- Cardiac catheterization
- Chest X-rays
- Echocardiogram

Treatment

The first treatment for acute pulmonary edema is supplemental oxygen. You usually receive oxygen through a face mask or nasal cannula — a flexible plastic tube with two openings that deliver oxygen to each nostril. This should ease some of your symptoms.

Your doctor will monitor your oxygen level closely. Sometimes it may be necessary to assist your breathing with a machine such as a mechanical ventilator or one that provides positive airway pressure.

Depending on the severity of your condition and the reason for your pulmonary edema, you may also receive one or more of the following medications:

- **Diuretics.** Doctors commonly prescribe diuretics, such as furosemide (Lasix), to decrease the pressure caused by excess fluid in your heart and lungs.
- **Morphine (MS Contin, Oramorph, others).** This narcotic may be taken by mouth or given through an IV to relieve shortness of breath and anxiety. But some doctors believe that the risks of morphine may outweigh the benefits and are more likely to use other drugs.
- **Blood pressure drugs.** If you have high or low blood pressure when you develop pulmonary edema, you'll be given medications to help manage the condition. Your doctor may also prescribe medications that lower the pressure going into or out of your heart. Examples of such medicines are nitroglycerin (Nitromist, Nitrostat, others) and nitroprusside (Nitropress).
- **Inotropes.** This type of medication is given through an IV if you are in the hospital with severe heart failure. Inotropes improve heart pumping function and maintain blood pressure.

It is important to diagnosis and treat, if possible, any nervous system problems or causes of heart failure.

Treating high-altitude pulmonary edema (HAPE)

As with other forms of pulmonary edema, oxygen is the usually the first treatment. If supplemental oxygen isn't available, you may use portable hyperbaric chambers, which imitate a descent for several hours until you are able to move to a lower elevation.

Treatments for high-altitude pulmonary edema (HAPE) also include:

- **Immediately descending to a lower elevation.** If you're climbing or traveling at high altitudes and have mild symptoms of HAPE, descend 1,000 to 3,000 feet (about 300 to 1,000 meters) as quickly as you can, within reason. Depending on

the severity of your condition, you may need rescue assistance to get off the mountain.

- **Stop exercising and stay warm.** Physical activity and cold can make pulmonary edema worse.
- **Medication.** Some climbers take prescription medications such as acetazolamide or nifedipine (Adalat CC, Procardia) to help treat or prevent symptoms of HAPE. To prevent HAPE, medication is started at least one day before ascent.

Clinical trials

Explore Mayo Clinic studies testing new treatments, interventions and tests as a means to prevent, detect, treat or manage this condition.

Lifestyle and home remedies

Lifestyle changes are an important part of heart health and can help you manage some forms of pulmonary edema.

- **Keep blood pressure under control.** If you have high blood pressure, take your medications as prescribed and check your blood pressure regularly. Record the results. Ask your doctor for your target blood pressure.
- **Manage other medical conditions.** Address any underlying medical conditions, such as controlling your glucose levels if you have diabetes.
- **Avoid the cause of your condition.** If pulmonary edema results from drug use or high altitudes, for example, you'll want to avoid these things to prevent further lung damage.
- **Don't smoke.** It's always a healthy idea to stop smoking. If you need help quitting, talk to your doctor. He or she can provide tips and, sometimes, medications to help you quit smoking.

- **Eat less salt.** Salt helps your body retain fluid. In some people with severely damaged left ventricular function, getting too much salt may be enough to trigger congestive heart failure. Your doctor may recommend a low-salt diet. If you need help, a dietitian can show you how to determine the salt content in foods and create a nutritious, good-tasting diet. In general, most people should consume less than 2,300 milligrams a day of salt (sodium). Ask your doctor what level is safe for you.
- **Choose a healthy diet.** You'll want to eat a plenty of fruits, vegetables and whole grains. Limit saturated fats and Trans fats, added sugars, and sodium.
- **Manage your weight.** Being even slightly overweight increases your risk of cardiovascular disease. On the other hand, even losing small amounts of weight can lower your blood pressure and cholesterol and reduce your risk of diabetes.
- **Get regular exercise.** Healthy adults should get at least 150 minutes of moderate aerobic activity or 75 minutes of vigorous aerobic activity a week, or a combination of the two. If you're not used to exercise, start out slowly and build up gradually. Be sure to get your doctor's OK before starting an exercise program.

SRI LAKSHMI NARAYANA INSTITUTE OF ALLIED HEALTH SCIENCE

MEDICAL EDUCATIONAL PROJECT

Pulmonary edema and Left Ventricular Failure - VAC01/AHS/2019-15/07

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5	**ARYA A.G**	**UAH1802154**	
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10	**GOKUL PRASAD**	**UAH1802159**	
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14	**KRITHIKSHA.S**	**UAH1802164**	
15	**MOHAMMED AFSAL.N**	**UAH1802166**	
16	**MOHAMMED AMEER.K**	**UAH1802167**	
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29	**ADARSH P G**	**UAH1902201**	Adar
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SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Annexure - III

Assessment Form

Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry

Course code: VAC01/AHS/2019-15/07

Multiple Choice Question 10x2=20

1) A 73-year-old male presents with acute pulmonaryoedema. His blood pressure is 180/110 mm Hg And heart rate 120 beats min−1 (sinus rhythm, QRSduration <100 ms), and he has cool peripheries. His serum lactate is 5 (normal 0.6–1.3) mmol litre−1 and there is left ventricular hypertrophy (LVH) on his ECG but no ischaemic changes. Thecardiac silhouette is not enlarged on chest X-ray. The following statements regarding cardiac function are correct:

(a). His cardiac output is likely to be limited byimpaired left ventricle (LV) filling.
(b). The presence of pulmonary oedema and elevated serum lactate concentrations indicates a reduced ejection fraction.
(c). Myocardial oxygen demand is likely to beincreased during this episode.
(d). The patient's blood pressure suggestsadequate tissue perfusion.

2) Regarding the pharmacological management ofacute decompensated heart failure (ADHF):
(a). Dobutamine is likely to be useful in the supportive treatment of ADHF caused by mitral incompetence.
(b). Dopamine has a positive inotropic effect.
(c). Levosimendan is likely to be usefullycombined with a α-1 adrenergic agonist.
(d). Opioid toxicity is associated with increased right ventricular (RV) afterload.
(e). Continued use of drugs with negative inotropic effects should generally be avoided in decompensated cardiac failure

3) An elderly, normotensive female patient is recovering from an episode of acute decompensated heart failure during which she hada good response to nitrates and diuretics, and a short spell of non-invasive ventilation. She is now in a 'post-stabilization' phase of her management. Her echocardiogram shows a mildly dilated left ventricle with an ejection fraction (EF) of 40%. She has a resting heart rate of 90 beats min−1 (sinus rhythm), blood pressure of 110/65 mm Hg and oxygen saturation of 95% on room air, but shortness of breath when moving from bed to chair. Her creatinine clearance is normal. The following therapies may be advocated as demonstrating survival benefit for her heart failure:
(A). Calcium antagonists.
(b). β-Blockers.
(c). Aldosterone antagonists.
(d). Furosemide infusion.
(e). Low-dose epinephrine infusion to relieve pulmonary congestion and improve tissueperfusion.

4) A 76-year-old male who has had a previous myocardial infarction presents with acute decompensated heart failure (ADHF). His transthoracic echocardiogram confirms an ejectionfraction (EF) of <35% and the 12-lead ECG is reported as 68 beats min−1 with second-degree heart block and left bundle branch block. The QRS duration is 160 Ms. He is haemodynamically stable, in no

respiratory distress and comfortable, but requires dobutamine and noradrenaline to maintain adequate cardiac output and perfusion pressure. From the information, available, the following therapies should be considered:

(a). B-Block.
(b). Angiotensin II receptor blockers.
(c). Implantable cardioverter defibrillator (ICD).
(d). Morphine.
(e). Cardiac resynchronization therapy

5) In acute respiratory distress syndrome (ARDS):(a). The diagnosis can be made on the ratio of Arterial oxygen partial pressure to fractional inspired oxygen (PaO2/FiO2 ratio), regardless of pressures used to ventilate the lungs.
(a). The diagnosis requires direct measurement ofintracardiac pressures.
(b). The mild form has a mortality rate of 27%.
(c). The onset is slow and insidious.
(d). Patients develop reduced pulmonary compliance and increased ventilation perfusion mismatch.

6) The pathophysiology of acute respiratory distresssyndrome (ARDS) includes:

(a). Production of inflammatory mediators, usually reduced by non-steroidal anti-inflammatory drugs (NSAIDs) as management of the disease.
(b). Fibroproliferation and microvascular thrombusformation in the first few hours of a known insult.
(c). An acute phase characterized by hypoxaemiaand flooding of the lungs with low-protein fluid.
(d). An inevitable progression to widespreadfibrosis and lung remodelling.
(e). Radiological imaging demonstratinghomogeneous lung fields.

7) Regarding management strategies for acuterespiratory distress syndrome (ARDS):

(a). Aiming for normal gas exchange can worsenthe disease process.
(b). An optimal level of positive end-expiratory pressure (PEEP) at 5 cm H2O has been identified and recommended by the ARDSnetgroup.
(c). Driving ventilator pressure (ΔP), when reduced,is strongly associated with survival.
(d). High-frequency oscillation ventilation (HFOV) is associated with higher mortality than conventional ventilation.
(e). Prone positioning is a simple, low-risk procedure that should be performed in anypatient with a diagnosis of ARDS.

8) Appropriate statements concerning acute respiratory distress syndrome (ARDS) include the following:

(a). There is level-1 evidence supporting the use ofsteroids in early ARDS.
(b). Initial ventilator settings for a newly diagnosedARDS patient include a tidal volume of 6 ml kg^{-1}.
(c). There is clear evidence of a mortality benefit for a conservative fluid management strategyin ARDS.
(d). Inhaled nitric oxide improves oxygenation at24 h but does not improve the likelihood of survival.
(e). On follow-up, most survivors of ARDS returnto normal function by 5 years after the illness

9) With reference to modern defibrillators: (a). They are most likely to use a triphasic Waveform.
(b). The duration of the biphasic truncated exponential (BTE) waveform is limited to 10Ms.
(c). The rectilinear biphasic (RB) waveform uses an inductor to control current flow during the initial phase of the biphasic waveform.
(d). Defibrillators using either the RB or the BTEwaveform have similar success rates for cardioversion of atrial fibrillation.
(e). Biphasic defibrillators can be used for direct defibrillation or direct current (DC) cardioversion of the heart during cardiac surgery.

10) Appropriate indications for direct current (DC)cardioversion include the following:

(a). Atrial fibrillation in a patient with Wolff–Parkinson–White (WPW) syndrome.
(b). A patient with digoxin-induced ventriculartachycardia (VT).
(c). A multifocal atrial tachycardia (MAT) in a critical care patient with pneumonia receiving norepinephrine and dobutamine infusions.
(d). A narrow complex tachyarrhythmia in a patientwith associated haemodynamic instability.
(e). A patient with atrial fibrillation in whom a rate control strategy has proved unsuccessful, with poor symptomatic control.

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MOHAMED RIJAS. K
UAH18021 65

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Annexure - III

Assessment Form

Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry

Course code: VAC01/AHS/2019-15/07

Multiple Choice Question 10x2=20

1) A 73-year-old male presents with acute pulmonaryoedema. His blood pressure is 180/110 mm Hg And heart rate 120 beats min−1 (sinus rhythm, QRSduration <100 ms), and he has cool peripheries. His serum lactate is 5 (normal 0.6–1.3) mmol litre−1 and there is left ventricular hypertrophy (LVH) on his ECG but no ischaemic changes. Thecardiac silhouette is not enlarged on chest X-ray. The following statements regarding cardiac function are correct:

(a). His cardiac output is likely to be limited byimpaired left ventricle (LV) filling.
(b). The presence of pulmonary oedema and elevated serum lactate concentrations indicates a reduced ejection fraction.
(c). Myocardial oxygen demand is likely to beincreased during this episode.
(d). The patient's blood pressure suggestsadequate tissue perfusion.

2) Regarding the pharmacological management ofacute decompensated heart failure (ADHF):
(a). Dobutamine is likely to be useful in the supportive treatment of ADHF caused by mitral incompetence.
(b). Dopamine has a positive inotropic effect.
(c). Levosimendan is likely to be usefullycombined with a α-1 adrenergic agonist.
(d). Opioid toxicity is associated with increased right ventricular (RV) afterload.
(e). Continued use of drugs with negative inotropic effects should generally be avoided in decompensated cardiac failure

3) An elderly, normotensive female patient is recovering from an episode of acute decompensated heart failure during which she hada good response to nitrates and diuretics, and a short spell of non-invasive ventilation. She is now in a 'post-stabilization' phase of her management. Her echocardiogram shows a mildly dilated left ventricle with an ejection fraction (EF) of 40%. She has a resting heart rate of 90 beats min−1 (sinus rhythm), blood pressure of 110/65 mm Hg and oxygen saturation of 95% on room air, but shortness of breath when moving from bed to chair. Her creatinine clearance is normal. The following therapies may be advocated as demonstrating survival benefit for her heart failure:
(A). Calcium antagonists.
(b). β-Blockers.
(c). Aldosterone antagonists.
(d). Furosemide infusion.
(e). Low-dose epinephrine infusion to relieve pulmonary congestion and improve tissueperfusion.

4) A 76-year-old male who has had a previous myocardial infarction presents with acute decompensated heart failure (ADHF). His transthoracic echocardiogram confirms an ejectionfraction (EF) of <35% and the 12-lead ECG is reported as 68 beats min−1 with second-degree heart block and left bundle branch block. The QRS duration is 160 Ms. He is haemodynamically stable, in no

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respiratory distress and comfortable, but requires dobutamine and noradrenaline to maintain adequate cardiac output and perfusion pressure. From the information, available, the following therapies should be considered:

(a). B-Block.
(b). Angiotensin II receptor blockers.
(c). Implantable cardioverter defibrillator (ICD).
(d). Morphine.
(e). Cardiac resynchronization therapy

5) In acute respiratory distress syndrome (ARDS):(a). The diagnosis can be made on the ratio of Arterial oxygen partial pressure to fractional inspired oxygen (PaO2/FiO2 ratio), regardless of pressures used to ventilate the lungs.
(a). The diagnosis requires direct measurement ofintracardiac pressures.
(b). The mild form has a mortality rate of 27%.
(c). The onset is slow and insidious.
(d). Patients develop reduced pulmonary compliance and increased ventilation perfusion mismatch.

6) The pathophysiology of acute respiratory distresssyndrome (ARDS) includes:

(a). Production of inflammatory mediators, usually reduced by non-steroidal anti-inflammatory drugs (NSAIDs) as management of the disease.
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(d). High-frequency oscillation ventilation (HFOV) is associated with higher mortality than conventional ventilation.
(e). Prone positioning is a simple, low-risk procedure that should be performed in anypatient with a diagnosis of ARDS.

8) Appropriate statements concerning acute respiratory distress syndrome (ARDS) include the following:

(a). There is level-1 evidence supporting the use ofsteroids in early ARDS.
(b). Initial ventilator settings for a newly diagnosedARDS patient include a tidal volume of 6 ml kg^{-1}.
(c). There is clear evidence of a mortality benefit for a conservative fluid management strategyin ARDS.
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9) With reference to modern defibrillators: (a). They are most likely to use a triphasic Waveform.
(b). The duration of the biphasic truncated exponential (BTE) waveform is limited to 10Ms.
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(e). A patient with atrial fibrillation in whom a rate control strategy has proved unsuccessful, with poor symptomatic control.

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ARULMOZIHI. A
UAH1802153.

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Annexure - III

Assessment Form

Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry

Course code: VAC01/AHS/2019-15/07

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(e). A patient with atrial fibrillation in whom a rate control strategy has proved unsuccessful, with poor symptomatic control.

Sri Lakshmi Narayana Institute of Medical Sciences

Affiliated to Bharath Institute of Higher Education & Research

(Deemed to be University under section 3 of the UGC Act 1956)

CERTIFICATE OF MERIT

This is to certify that **GOPIKA.K (UAH1802160)** has actively participated in the Value Added Course on **Congestive Heart Failure And Pulmonary Edema(VAC01/AHS/2019-15/07)** held during month Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

RESOURCE PERSON

Dr. G.Somasundram

COORDINATOR

Sri Lakshmi Narayana Institute of Medical Sciences

Affiliated to Bharath Institute of Higher Education & Research

(Deemed to be University under section 3 of the UGC Act 1956)

CERTIFICATE OF MERIT

This is to certify that **ARAVINTH.S(UAH1802151)** has actively participated in the Value Added Course on **Congestive Heart Failure And Pulmonary Edema(VAC01/AHS/2019-15/07)** held during month Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

RESOURCE PERSON

Dr. G.Somasundram

COORDINATOR

Annexure- V

Student Feedback Form

Course Name: Congestive Heart Failure and Pulmonary Edema

Subject Code: VAC01/AHS/2019-15/07

Name of Student: _____ Roll No.: _____

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

Feedback Form

	Strongly agree	Agree	Neutral	Disagree	Strongly disagree
1. The course met my expectations.	○	○	○	○	○
2. I will be able to apply the knowledge learned.	○	○	○	○	○
3. The course objectives for each topic were identified and followed.	○	○	○	○	○
4. The content was organised and easy to follow.	○	○	○	○	○
5. The quality of instruction was good.	○	○	○	○	○
6. Class participation and interaction were encouraged.	○	○	○	○	○
7. Adequate time was provided for questions and discussion.	○	○	○	○	○

8. How do you rate the course overall?
   - ○ Excellent
   - ○ Good
   - ○ Average
   - ○ Poor
   - ○ Very poor

9. The aspects of the course could be improved?

10. Other comments?

Signature of the Student:
Date:

Annexure- V

Student Feedback Form

Course Name: Congestive Heart Failure and Pulmonary Edema

Subject Code: VAC01/AHS/2019-15/07

Name of Student: ABIJITH.C.C Roll No.: 0AH1802150

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

Feedback Form

	Strongly agree	Agree	Neutral	Disagree	Strongly disagree
1. The course met my expectations.	✓				
2. I will be able to apply the knowledge learned.		✓			
3. The course objectives for each topic were identified and followed.			✓		
4. The content was organised and easy to follow.	✓				
5. The quality of instruction was good.		✓			
6. Class participation and interaction were encouraged.		✓			
7. Adequate time was provided for questions and discussion.	✓				

8. How do you rate the course overall?
   - Excellent
   - ✓ Good
   - Average
   - Poor
   - Very poor

9. The aspects of the course could be improved?

   NO.

10. Other comments?

    Class was

Signature of the Student: Abijith.
Date: 14/8/19

Annexure- V

Student Feedback Form

Course Name: Congestive Heart Failure and Pulmonary Edema

Subject Code: VAC01/AHS/2019-15/07

Name of Student: ARUL. B Roll No.: UAH1802152

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

Feedback Form

	Strongly agree	Agree	Neutral	Disagree	Strongly disagree
1. The course met my expectations.	✓				
2. I will be able to apply the knowledge learned.		✓			
3. The course objectives for each topic were identified and followed.	✓				
4. The content was organised and easy to follow.		✓			
5. The quality of instruction was good.			✓		
6. Class participation and interaction were encouraged.	✓				
7. Adequate time was provided for questions and discussion.		✓			

8. How do you rate the course overall?
   - ✓ Excellent
   - Good
   - Average
   - Poor
   - Very poor

9. The aspects of the course could be improved?

   NO.

10. Other comments?

    Now I can able. understand CHF and PE

Signature of the Student: Arul.

Date: 14/08/19

Date: 20.08.2019

From

Dr.G.Somasundram
Department of Pharmacology,
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

Through Proper Channel

To

The Dean,
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

Sub: Completion of value-added course: Congestive Heart Failure and Pulmonary Edema

Dear Sir,

With reference to the subject mentioned above, the department has conducted the value-added course titled: "**Congestive Heart Failure and Pulmonary Edema"** July to September 2019 for 30 AHS Students. We solicit your kind action to send certificates for the participants that is attached with this letter. Also, I am attaching the photographs captured during the conduct of the course.

Kind Regards,

Dr.G.Somasundram

Encl: Certificates

Photographs