Conference on Dyspnoea 2025

Pocket Booklet on Dyspnoea

Department of Respiratory Medicine
Sher-E-Bangla Medical College, Barishal, Bangladesh

MRC breathlessness scale	
Grade	**Degree of dyspnea**
1	no dyspnea except with strenuous exercise
2	dyspnea when walking up an incline or hurrying on the level
3	walks slower than most on the level, or stops after 15 minutes of walking on the level
4	stops after a few minutes of walking on the level
5	with minimal activity such as getting dressed, too dyspneic to leave the house

"Pocket Booklet on Dyspnoea"- Dyspnoea Conference 2025

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INTRODUCTION

Prof. Dr. H. N. Sarker
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The members of the department of respiratory medicine of Sher E Bangla Medical College, Barishal feel need to share knowledge and experience about dyspnea among doctors of southern part of Bangladesh to improve the overall management of dyspnea patients. That's why this conference is arranged and this pocket booklet is published to subserve the purpose.

Dyspnea is a common symptom with which patient may present to doctor of any discipline of medical science.

About 3.5% of people present to the emergency department in the United States with dyspnea. Among them, approximately 51% are admitted to the hospital and 13% die within a year[1]. Some studies have suggested that up to 27% of hospitalized patients develop dyspnea[2], while 75% will experience dyspnea at their departure[3]. Acute dyspnea is the most common reason for palliative care patients to visit an emergency department[4]. Up to 70% of adults with advanced cancer also experience dyspnea[5].

Dyspnea (in American English) or **Dyspnoea** (in British English) means shortness of breath (SOB). The American Thoracic Society defines dyspnea as a subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity[6].

Other definitions of dyspnoea include:-

- awareness of respiratory distress
- an uncomfortable sensation of breathing
- difficult, laboured, uncomfortable breathing
- the sensation of feeling breathlessness or experiencing air hunger.

Dyspnea is a normal symptom of heavy physical exertion that is physiological and disappears after rest, but becomes pathological if it occurs in unexpected situations, when resting or during light exertion[7].

DiagnosisPro, an online medical expert system, listed 497 distinct causes of dyspnea in October 2010[8]. Cardiorespiratory causes such as asthma, chronic obstructive pulmonary disease, pneumonia, COVID-19, interstitial lung disease, cardiac ischemia, and congestive heart failure accounts for 85% cases of dyspnea[7,9]. The rest is due to other causes like psychogenic causes such as panic disorder and anxiety, obesity, pregnancy etc.[10].

Its presentatation varies from acute, acute on chronic and chronic dyspnea. Approach also varies depending on presentation, causes and severity. The primary treatment of shortness of breath is directed at its underlying cause[11]. Extra supplemental oxygen is effective in those with hypoxia; however, this has no effect in those with normal blood oxygen saturations[9,12].

Subsequent discussions will help the readers to understand dyspnea more easily.

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Approach to Dyspnea

How to manage common causes of dyspnoea

প্রশান্তি ভরা শ্বাস, আমাদের প্রয়াস।

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Dyspnea is defined as the subjective awareness of breathing which results from the diseases of the lungs, heart or many other conditions. The diseases causing dyspnea are shown in **Box 1**.

Box 1 The diseases causing dyspnea	
Respiratory Disease and Disease of chest Wall	Asthma, COPD, ILD, Pneumothorax, Pleural effusion, Myasthenia gravis
Cardiovascular Disease	Heart Failure (Acute, Chronic), Pulmonary embolism
Metabolic Causes	Hypercapnia, Acidosis
Other Causes	Anemia, obesity, Physical deconditioning

Dyspnea can also be classified as acute or chronic dyspnea depending on duration and onset which is shown in **Box 2**.

Box 2 Causes of acute and chronic dyspnea	
Acute dyspnea	**Chronic exertional dyspnea**
Acute Asthma	COPD
Acute left ventricular failure	Chronic persistent asthma
Pneumonia	Chronic heart failure
Acute exacerbation of COPD	DPLD
ARDS	Chronic pulmonary thromboembolism
Pneumothorax	Large pleural effusion
Anaphylaxis	
Foreign body inhalation in children	
Psychogenic hyperventilation	
Other causes:	
Neuromuscular disorders affecting respiratory muscles	Myasthenia gravis, GBS, Muscular dystrophies, MND
Elevation of the diaphragm	Pregnancy, Diaphragmatic palsy, Abdominal surgery or trauma
Physical deconditioning	

- **Evaluation of dyspnea:**
  Evaluation is done by history taking, examination and investigation. In most cases, it becomes possible to reach diagnosis from history and examination; investigations are needed to help or confirm diagnosis.

  - **History:** Important points in the history are speed of onset (Minutes, Minutes to hours, hours to days), duration (short- hours, days to weeks, Long- months to years) and associated features (Fever, chest pain, cough, sputum production, conscious level, aggravating factors and relieving factors).
    History can narrow the differential diagnoses which are illustrated in **Box 3**.

Box 3 Differential diagnoses of dyspnea from history			
Short duration (days/weeks)		**Long duration (months/years)**	
With fever	**Without fever**	**Predominant cough**	**Predominant respiratory distress**
Viral respiratory infection	Acute LVF Acute asthma attack	Asthma	Chronic Heart failure
Pneumonia	Pneumothorax	COPD	DPLD
ARDS	Pulmonary embolism		Chronic pulmonary embolism
	Psychogenic hyperventilation		Pulmonary HTN

- **Bedside examinations:** Physical examination further narrows down the differential diagnoses and leads to clinical diagnosis; Following signs are sought during bedside examination-
  - Respiratory Rate
  - Anemia
  - Cyanosis
  - Blood pressure
  - Temperature
  - JVP
  - Pedal edema
  - Pulse oximetry (SpO2)
  - Examination of the chest.

During examination, we also assess signs of severe respiratory distress and red flag signs which are shown in **Box 4**.

Box 4 The red flag signs
• **Signs of severe respiratory distress** ▪ Respiratory rate> 24 breaths per minute ▪ Prominence of respiratory muscles ▪ Patient unable to complete a sentence in one breath.)
• **Impairment of consciousness**
• **Cyanosis**
• **Spo2<90%**
• **Respiratory arrest**

Clinical features of short and long duration dyspnea are summarized in **Box 5.**

Box 5 Summarization of Clinical features of short and long duration dyspnea	
Short duration dyspnea (Days to Weeks)	
Clinical features	**Clinical diagnosis**
With fever, cough, chest pain+/- features of Consolidation	PNEUMONIA
With wheeze, chest tightness and features of Allergy	ACUTE ASTHMA
Very sudden onset (minutes), diminished breath sounds, resonant percussion note	PNEUMOTHORAX
Sudden onset of breathlessness (hours to days), tachycardia, low volume pulse, bi-basal crepitations with frothy sputum	ACUTE LEFT VENTRICULAR FAILURE
With or without fever, features of respiratory failure, SpO2<90, Bilateral coarse crepitations.	ARDS
Within minutes, peripheral chest pain, decreasedSpO2 with minimum chest findings	PULMONARY EMBOLISM

Inability to take deep breath, frequent erratic ventilation at rest, short breath holding time despite absence of severe respiratory disease	PSYCHOGENIC HYPERVENTILATION
Suggestive history, especially in children	FOREIGN BODY INHALATION
History of exposure to allergens, wheeze, urticarial, hypotension	ANAPHYLAXIS
Long duration (Months to Years)	
Cough prominent	
Smoker, age of onset at forty years, Gradual onset and prolonged duration of dyspnea, cough with sputum, barrel shaped chest	COPD
Chest tightness, wheeze, history of allergy	Chronic Asthma
Respiratory distress prominent	
Dyspnea at rest, PND like features, raised JVP, peripheral edema, shifted apex beat, Bi basal Crackles	CHRONICHEARTFAILURE
Dry distressing cough and progressive dyspnea, Bilateral Crepitations, fall of spo2 on exertion	DPLD

Non-cardiorespiratory diseases	
BMI $\geq$ 30 kg / m^2	OBESITY
Pale looking, H/O Blood loss	ANEMIA
H/O immobilizations	PHYSICAL DECONDITIONING

Physicians sometimes confuse to differentiate dyspnea of respiratory origin from cardiac origin, because they frequently share same features. Despite that clinicians should differentiate for optimum management. Some differentiating points are given in **Box 6**.

Box 6 Compare and Contrast between Dyspnea of Respiratory & Cardiac origin	
Dyspnea of Respiratory diseases	**Dyspnea of Cardiac diseases**
Dyspnea is associated with cough and sputum production	Dyspnea is associated with central chest pain, palpitation and PND like features.
Aggravates with dust, fume, cold weather, sometimes with allergens.	Aggravates with exertion.
Pulse–tachycardia	Pulse–low volume and tachycardia, there may be pulsus alternans.
Wide spread Wheeze	Bi-basal crepitations
Respiratory failure is more common	Respiratory failure is less common

- **Initial investigations:**
  - Chest X-ray
  - ECG
  - CBC
  - RBS
  - D-dimer (suspected pulmonary embolism)
  - Bed side Echocardiography (if available).

- **Initial management of respiratory dyspnea:** This is given in **Box 7**.

Box 7 Initial management of respiratory dyspnea

- Evaluation and management should go side by side in case of acute or severe respiratory distress or presence of red flag signs.

Common initial treatment:

- Nebulization with salbutamol & Normal saline (1:1), minimum 4-6ml.
- Consider O2 inhalation if SpO2 <92%.
- Consider Furosemide injection if suspected heart failure
- Empirical antibiotic if signs of infection (fever & sputum) are present.

Dyspnea of Cardiac origin

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Dyspnea is a term used to characterize a subjective experience of breathing discomfort that is comprised of qualitatively distinct sensations that vary in intensity. The experience derives from interactions among multiple physiological, psychological, social, and environmental factors, and may induce secondary physiological and behavioral responses."

We can classify dyspnea in various ways, such as dyspnea of cardiac origin, dyspnea of respiratory origin; acute dyspnea and chronic dyspnea.

In this write-up we are discussing about dyspnea of cardiac origin. Common etiologies of dyspnea of cardiac origin are-

- Heart Failure;
- Pulmonary hypertension;
- Anaemia;
- Physical deconditioning.

In heart failure, dyspnea develops due to either reduction of cardiac output or increased pulmonary or systemic hypertension; hypoxia and anemia may aggravate dyspnea of any origin.

- **Definition of heart failure**: Heart failure is defined as the clinical syndrome that develops when the heart cannot maintain adequate output, or can do so only at the expense of elevated ventricular filling pressure.
- **Pathophysiology of cardiac dyspnea in HF:** It is shown in **Box.1**.

Box. 1 Pathophysiology of cardiac dyspnoea in HF

Two major classes:

- Those due to a reduction in cardiac output (low cardiac output), oxygen delivery to the tissues is reduced, stimulation of metabo- or ergoreceptor;
- Increased pulmonary or systemic venous pressure- Hypoxemia stimulating pulmonary vascular and/or interstitial receptors.

- **Types of heart failure:**

  - Acute and chronic heart failure
  - Left, right and biventricular heart failure
    - Left-sided heart failure.
    - Right-sided heart failure.
    - Biventricular heart failure (CCF).
  - High-output failure and low output failure
  - Diastolic and systolic dysfunction

  ❖ Heart Failure usually refers to Left ventricular systolic dysfunction unless otherwise specified. For clinical use, HF is categorized as Acute & Chronic Heart Failure.

Some factors may precipitate and aggravate heart failure which are shown in **Box 2**.

Box 2 Precipitating factors

- Myocardial ischaemia or infarction
- Intercurrent illness, e.g. infection
- Arrhythmia, e.g. atrial fibrillation
- Inappropriate reduction of therapy
- Administration of a drug with negative inotropic (β-blocker) or fluid-retaining properties (NSAIDs, corticosteroids)
- Pulmonary embolism
- Conditions associated with increased metabolic demand, e.g. pregnancy, thyrotoxicosis, anaemia
- IV fluid overload, e.g. post-operative IV infusion

Clinical evaluation of heart failure:

History:

Symptoms of Acute heart failure

- Acute respiratory distress
- Orthopnoea
- Prostration
- Agitation, pale and clammy
- Peripheries are cold to touch

Symptoms of Chronic heart failure

- Fatigue
- Exertional breathlessness, cough or wheeze
- Paroxysmal Nocturnal Dyspnea
- Orthopnea
- Cheyne stokes respiration

Other symptoms:

- Anorexia
- Nausea
- Early satiety
- Abdominal pain
- Abdominal fullness
- Confusion
- Disorientation
- Sleep disturbance
- Nocturia
- Cardiac cachexia

Examination:

General examination

- Laboured breathing
- Cool peripheries
- Cyanosis (peripheral first, central in severe HF)
- Anemia
- Pulse- Sinus tachycardia due to increased sympathetic activity
- Blood pressure- may be normal or high in early HF; may decrease consequently and is usually low in severe HF.
- Low pulse

Systemic examination

Cardiovascular system

- Apical impulse may shift inferiolaterally.
- Sustained apical impulse is felt in severe LVH.
- Third Heart Sound (S3/S4 gallop)
- S4 gallop is usually present in diastolic dysfunction.
- Pansystolic murmur of MR or TR may be present additionally.

Respiratory system

- Bilateral basal rales/crepitations
- Expiratory wheeze (cardiac asthma)
- Pleural effusion may/may not be present (Common in CCF).

pressure (reduced stroke volume)
- Depression

Abdomen
- Hepatomegaly (tender / pulsatile)
- Pulsatile liver indicates tricuspid regurgitation
- Ascites
- Jaundice and raised liver enzymes

- **Features of Right and left heart failure:** Features of Right and left heart failure are shown in **Figure 1.**

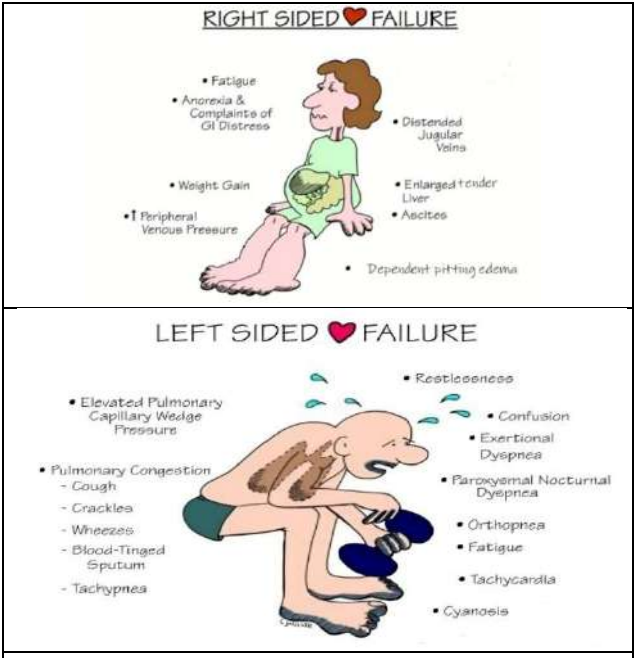

Figure 1. Features of Right and left heart failure

- **NYHA Functional Classification**:

Heart failure (HF) is classified by the New York Heart Association (NYHA) into four functional classes based on the

severity of symptoms and how the heart is functioning, which is shown in **Box 3**.

Box 3 NYHA Functional Classification
Class I: no limitation of ordinary physical activity **Class II:** slight limitation of ordinary physical activity **Class III:** marked limitation of ordinary physical activity, but comfortable at rest **Class IV:** unable to carry out physical activity, symptomatic at rest

- **Stages of Heart Failure:**

In collaboration with the American College of Cardiology, the AHA has identified four stages of heart failure. They are described in **Box 4**.

Box 4 ACC/AHA Staging of Heart Failure
Stage A: At risk for heart failure • At high risk of HF but without structural hear disease. **Stage B: Pre-heart failure** • Structural heart disease without signs or symptoms of HF. **Stage C**: **Symptomatic heart failure** • Structural heart disease with prior HF or current HF. **Stage D**: **Advanced heart failure** • Heart failure symptoms interfere with daily life functions or lead to repeated hospitalizations.

Investigations:

- NT-Pro BNP/Brain natriuretic peptide (BNP)
- ECG
- X-ray chest PA view
- Echocardiography
- Serum urea and electrolytes
- S.creatinine
- CBC with Hemoglobin,
- Thyroid function

Management of Heart Failure

♥ **Management of Acute Heart Failure:**

- Sit the patient up to reduce pulmonary congestion.
- Give oxygen (high-flow, high-concentration).
- Non-invasive positive pressure ventilation (continuous positive airways pressure (CPAP) of 5–10 mmHg).
- Administer a loop diuretic such as furosemide (40–80 mg IV).
- Administer nitrates such as IV glyceryl trinitrate until clinical improvement occurs or systolic BP falls to less than 110 mmHg.
- Intravenous opiates (Morphine)
- Inotropic agents may be required to augment cardiac output, particularly in hypotensive patients- Noradrenalin, Dobutamin, Dopamin, Levosimendan.

♥ **Management of Chronic heart failure:**

♣ **Heart failure with reduced ejection fraction:**

Systolic dysfunction of heart-

♦ **General measures:** General measures necessary for heart failure patients are shown in **Box 5**.

Box 5 General measures for the management of heart failure
Education
▪ Explanation of nature of disease, treatment and self-help strategies
Diet
▪ Good general nutrition and weight reduction for the obese ▪ Avoidance of high-salt foods and added salt, especially for patients with severe congestive heart failure
Alcohol
▪ **Moderation or elimination of alcohol consumption; alcohol-induced cardiomyopathy requires abstinence**
Smoking
▪ Complete and permanent cessation
Exercise
▪ Regular moderate aerobic exercise within limits of symptoms
Vaccination
▪ Consideration of influenza and pneumococcal vaccination

- **Specific measures**
  - **Volume overload management-** Use of loop diuretics considering following factors
    - BP, Electrolytes, PH, edema
    - Dosage of furosemide
    - Bolus vs Syringe pump
    - ceiling effects
    - When to withdraw loop diuretics
    - Resistance: sequential blocking of nephron
  - **Counteracting Neurohumeral activation-**
    - **ARB, ACEi**
    - **ARNI** (angiotensin receptor +neprilysin inhibitor)
    - **Beta blocker**
  - **Addressing precipating cause – see Box no 2**
  - **Prevention of cardiac remodeling and fibrosis-**
    - Spironolactone
    - SGLT2 inhibitor
  - **Lifestyle modification**
  - **Rehabilitation**
  - **Interventions and surgical considerations**

Childhood Dyspnea

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Respiratory Distress has been discussed here in three categories- respiratory distress in newborn, dyspnea in infants and childhood dyspnea.

Respiratory Distress in Newborn

Respiratory Distress in Newborn is a crucial condition for the attending physicians to combat. The causes of such condition depend on the gestational age, which are enumerated in **Box 1**.

Box 1 Most common and clinically important causes of respiratory distress according to gestational age

Preterm Newborns	**Term/Near term Newborns**	**Irrespective of gestational age**
• Respiratory distress syndrome (RDS) • Delayed transition • Hypo/ hyperthermia	• Transient tachypnea of the newborn (TTN) • Perinatal Asphyxia (PNA) • Meconium aspiration syndrome	• Pneumonia, Sepsis • Pneumothorax • Congenital heart diseases (CHD) • Hypoglycemia • Acidosis, Inborn metabolic error

	• Congenital malformations • Persistent pulmonary hypertension of the newborn (PPHN)	• Meningitis, Seizure • Anemia, Polycythemia. • Maternal sedation, narcotic withdrawal

- **IMCI classify cough or difficult breathing as shown in Box 2.**

Box 2 Classification of Cough or Difficult Breathing according to IMCI	
Sign	**Classified as**
Any General Danger Signs or Stridor in Calm Child or Oxygen Saturation <90%	♣ **Severe Pneumonia** Or ♣ **Very Severe Disease**
❖ Chest Indrawing Or ❖ Fast Breathing*	♣ **PNEUMONIA**
♣ No sign of Pneumonia or Very Severe Disease	♣ **Cough or Cold**

* Landmark of fast breathing is sketched in **Box 3.**

Box 3 Landmark of fast breathing	
Age	**Respiratory Rate /minute**
• 0 - < 2 months	60 or More
• Completed 2 months- < 12 Months	50 or More
• Completed 12 months– < 5 Years	40 or More

Causes of dyspnea in children differ from those in infants and neonates, which are enlisted in **Box 4**.

Box 4 Common causes of Childhood Dyspnea	
With Fever	
• Bronchiolitis • Pneumonia • Acute Epiglottitis • Croup • Pulmonary TB • Pertussis	• Asthma • Foreign Body Inhalation • Metabolic e.g. DKA • Heart Failure

- **Evaluation of dyspnea in children**
  - **History:** Points for detecting the causes are:
    - Onset (Acute or chronic);
    - Fever (Infectious)
    - Associated symptoms (Cyanosis),

- Barking cough: croup;
- Family history: Asthma, CHD, CF;
- Drug history: anticonvulsants;
- Surgery: Cardiac cause.

♣ **Clinical examination:** Points should be noted during clinical examinations are:

- Vital signs: Tachypnea or Tachycardia;
- Color: Pallor or Cyanosis;
- Dysmorphic features e.g. Down syndrome;
- Signs of Respiratory Distress: Working alae nasi and retractions;
- Signs of Heart Failure: Tachycardia and cyanosis;
- Growth parameters: Failure to thrive;
- General feature: Edema or clubbing or hepatomegaly
- Chest exam: Ronchi or basal rales.

♣ **Signs of Dyspnea in Infant:** Respiratory distress can be discovered by noticing carefully some signs e.g.

- Head nodding;
- Nasal flaring;
- Tachypnea;
- Suprasternal and intercostal recession;
- Chest indrawing;
- Irritability;
- Cyanosis;
- Inability to feed.

- **Investigations:** Plan of Investigations depends on the provisional diagnosis.
    - CBC: Leukocytosis or Leucopenia may be found in infective causes.
    - Inflammatory markers like C-Reacive Protein (CRP) and ESR etc. are usually raised also in infective causes.
    - Blood for C/S though done on selective cases can discover the exact offending organism.
    - When Pulmonary Tuberculosis is in mind of the attending Physician he may go for
        - Mantaux Test (MT);
        - Sputum for Gene Xpert;
        - Stool Xpert.
    - For Asthma patient-
        - Serum Ig-E level;
        - Spirometry;
        - Skin Prick test.
    - Arterial Blood Gas Analysis (ABG) should be monitored regularly in critically ill patients.
    - Sometimes Direct Larygoscopy or Brochoscopy needed immediately in Foreign Body Inhalation.
    - Uncommon tests like PCR for Viral Serology, Sweat Chloride test for Cystic Fibrosis etc. are done in few cases.
    - Imaging studies:
        - Chest X-Ray- Chest X-Ray is mandatory near about in all cases. Radiological evidence of Consolidation, Effusion or Pneumothorax etc. may be found depending on the diagnosis.
        - Color Dopler Echocardiography-

sometimes needed to find out Congenital Cardiac Defects.

- Gastrographin Study- done when there is suspicion of Tracheo-Esophageal Fistula.
- CT scan of Chest sometimes gives more precise diagnosis than conventional Chest X-Ray.

- **Differentiation among Bronchiolitis, Pneumonia and Asthma**

Sometimes it is very much confusing to distinguish among these three illnesses, but some features help to differentiate shown in **Box 5**.

Box 5 Differentiation among Bronchiolitis, Pneumonia and Asthma

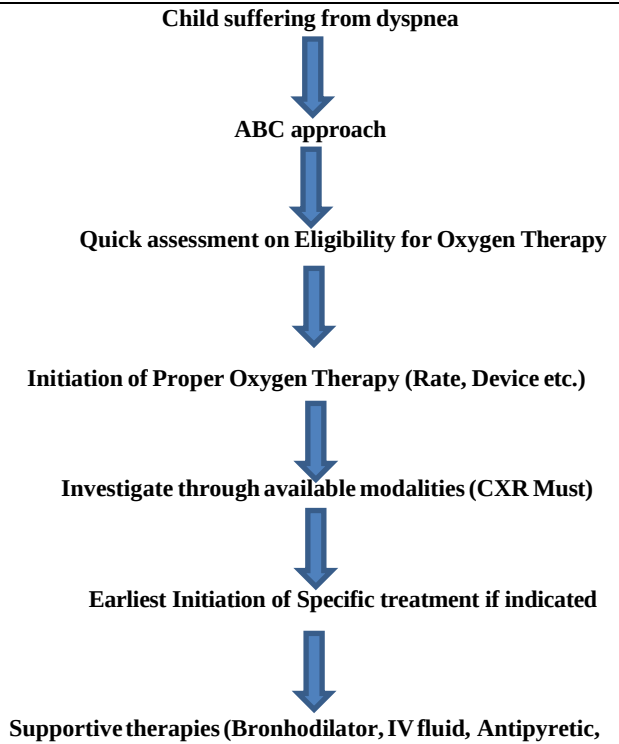
Feature	Bronchiolitis	Pneumonia	Asthma
Age	<2 years	Any age	>2 years
Symptoms	• Cough • Runny nose • Low grade fever • Wheeze	• High grade fever • Respiratory distress • Toxic look	• Recurrent cough • Wheeze • Concomitant allergic manifestations
Sign	Rhonchi>Rales	Rales > Rhonchi	Rhonchi>Rales

WBC count	Usually normal	Neutrophilic leukocytosis	Eosinophilia
Chest X-ray	Hypertranslucency	Consolidation	Hypertranslucency

- **Management outline:**
  - **Emergency situation:** Management of ABC-
    - Airway clearance
    - Maintenance of Breathing
    - Maintenance of effective Circulation.
  - **Specific Treatment-** depends on the underlying diagnosis
    - **Pneumonia and Epiglottitis-** Appropriate antibiotic
    - **Bronchiolitis:** Supportive Treatment
      3% NaCl Nebulization
      Chest physiotherapy
    - **Asthma:** Step Care Management
    - **FB aspiration:** Bronchoscopic removal
    - **Croup:** Steroids
    - **CHD:** Device Closure/Surgery
    - **Heart Failure:** Diuretics, Digoxin
    - **Anemia:** Blood Transfusion
    - **DKA:** Management of DKA and metabolic correction.
  - **Treatment of complications:** Acidosis, Respiratory Failure etc.

- **Practical Algorithm:** Practical Algorithm for management of dyspnea in children is shown in **Box 6**.

Box 6 Practical Algorithm for management of dyspnea in children



Steroid etc.) with justification

Regular Close Follow Up

Prompt Identification of Complications(Heart Failure, Respiratory Failure etc.) & Handle accordingly

Complete the institutional treatment as needed

Give proper drug & lifestyle advice during discharge & give knowledge regarding when to return immediately

Encourage EBF, Vaccination, Vit-A Supplementation etc. for Infection Prevention

Management of Dyspnea in Critical Care Unit

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Dyspnea is a common presentation at critical care unit; patients transferring from different wards, especially from Medicine, Surgery and Gynae & Obs ward. Most of them have Respiratory failure either Type I or Type II Respiratory failure. Some patients have CNS causes while others have metabolic causes.

Type I respiratory failure means arterial hypoxia with normo- or hypocapnia (Hypoxia (PaO2 <8.0 kPa (60 mmHg)); normal or low PaCO2 (≤ 6 kPa (45 mmHg)).

Type II respiratory failure means arterial hypoxia with hypercapnia (Hypoxia PaO2 <8.0 kPa (60 mmHg); raised PaCO2 >6 kPa (45 mmHg)).

Respiratory failure may be acute or chronic depending on underlying cause and mechanism; Causes are shown in **Box 1**.

Box 1 Causes of respiratory failure			
Type I respiratory failure		**Type II respiratory failure**	
Acute	**Chronic**	**Acute**	**Chronic**
• Acute asthma • Pneumonia • Pulmonary oedema • Pneumothorax • Lobar collapse • Pulmonary embolus • ARDS	• COPD (Emphysema) • Lung fibrosis • Lymphangitic carcinomatosis • Right-to-left shunts	• Acute severe asthma • Acute exacerbation of COPD • Upper airway obstruction • Acute neuropathies /paralysis • Flail chest injury • Primary alveolar hypoventilation • Narcotic drugs	• COPD • Sleep apnoea • Kyphoscoliosis • Myopathies/ muscular dystrophy • Ankylosing spondylitis

Acute decompensation of chronic respiratory failure i.e. acute on chronic respiratory failure may develop due to some precipitating factors like

- ❖ Respiratory infection
- ❖ Drugs (Sedatives, Narcotics)
- ❖ Heart failure
- ❖ Exacerbation of underlying Neuromuscular diseases (e.g. Myasthenia Gravis)

- **Presentation of Hypoxia:** Hypoxic patient may present with
  - ♣ Tachypnea
  - ♣ Tachycardia
  - ♣ Anxiety
  - ♣ Diaphoresis
  - ♣ Altered mental status
  - ♣ Confusion
  - ♣ Cyanosis
  - ♣ Bradycardia
  - ♣ Seizure
  - ♣ Coma

- **Presentation of Hypercapnia:** Hypercapnic patient may present with
  - ♣ Somnolence
  - ♣ Lethargy
  - ♣ Restlessness
  - ♣ Tremor
  - ♣ Slurred Speech

- Headache
- Asterixis
- Papilledema
- Coma

Management

- **GOAL of management:** Whatever may be the cause, our GOAL is:
  - Improvement of Hypoxia
  - Improvement of Acidosis
  - Maintenance of Cardiac output
  - Treatment of underlying diseases process
  - Avoidance of predictable complications

- **Improvement of Hypoxia:** Hypoxia can be corrected by
  - Supplemental oxygenation
  - Positive pressure ventilation
    - NIPPV(Non Invesive Positive Pressure Ventilation)
    - IPPV (Invasive Positive Pressure Ventilation, Mechanical Ventilation)
  - Prone positioning
  - Extracorporial respiratory support

- **Supplemental oxygenation:**
  - **Aims-** PaO2 >60mmhg or SpO_2 95%
  - **Means of O2 Supplementations:**
    - Nasal Prongs (1-6L/min , 24%-44% FiO2)
    - Simple Face Mask (5-8L/min, 40-60%FiO2)
    - Partial Rebreather (7-15 L /min, 65-80%FiO2)
    - Non-Rebreather (Set to prevent collapse of the bag, 85-100%FiO2).
  - **Indications of High Flow Oxygen:** High Flow Oxygen is given in
    - Spontaneously Breathing patient
    - Need Higher concentration of Oxygen
    - Oxygen is humidified
    - 10-60 L/min flow rate
    - Low PEEP
    - Modest increase in CO2 elimination.

- **Positive pressure ventilation or Mechanical Ventilation**:

Those who are not responsive to Oxygen inhalation by any above procedure

or there is progressive hypercapnia, needs Positive Pressure Ventilation

(PPV)

- **Two types of positive pressure ventilation (Mechanical Ventilation)-**
    - **NIPPV**(Non Invesive Positive Pressure Ventilation)
    - **IPPV** (Invasive Positive Pressure Ventilation)

NIPPV- NIPPV refers to positive pressure ventilation through a non-invasive interface. NIPPV is the delivery of positive pressure ventilation via a tight-fitting mask that covers the nose or both the nose and mouth. Proper fitting of the mask is a **KEY** to successful NIPPV.

- **Benefit of NIPPV:**
    - Noninvasiveness
    - Avoid complications related to endotrachial intubation (trauma, infection)
    - Allows intermittent application
    - Reduces need for sedation
    - Preserves speech, cough and swallowing
    - Decreases hospital stay, rate of intubation, mortality and morbidity
    - Keeps the airway patent and relieves obstruction
    - Prevents expiratory collapse of airway and alveoli

- Recruits more and more alveoli
- Increases tidal volume
- Decreases preload of the heart
- Decreases work of breathing.

♣ **Indications of NIPPV**:

- Acute exaggeration of COPD with hypercapnic acidosis (pH<7.3, $PaCO_2$>45mmHg) despite maximal medical treatment who do not need emergent intubation;
- Deterioration of Asthma patient with confusion, drowsiness and ABG with falling PaO_2(<60 mmHg) +/-rising $PaCO_2$(45 mmHg);
- Respiratory failure due to acute cardiogenic pulmonary edema;
- Decompensation of OSA;
- Hypercapnic respiratory failure due to neuromuscular and chest wall diseases;
- To prevent postextubation respiratory failure;
- Hypoxemic respiratory failure (e.g. Pneumonia, ARDS, Acute Severe Asthma);

- Postoperative hypoxemia;
- Intubation of refusal.

♣ **Avoidance of NIPPV:**

- Cardiac or Respiratory arrest
- Severe encephalopathy
- Upper GI bleeding
- Upper airway obstruction or unable to protect airway (Decreased cough reflex)
- High risk of aspiration
- Hemodynamically unstable cardiac arrhythmia

♣ **Modes of NIPPV:**

1. Continuous positive airway pressure (CPAP)
2. Bilevel positive airway pressure (BiPAP)

*In **CPAP**, constant pressure is maintained throughout the respiratory cycle with no additional inspiratory support. With **BiPAP**, the physician sets both the expiratory positive airway pressure (EPAP) and the inspiratory positive airway pressure (IPAP), with respirations triggered by the patient.*

BiPAP:

- Delivers positive pressure at different level during Inspiration and Expiration;
- Pressure delivers during inspiration is IPAP;
- Pressure delivers during expiration is EPAP;
- IPAP-EPAP =PS;
- Tidal volumes depends on PS;
- IPAP augments inspirations and improves ventilation;
- Reduces work of breathing;
- EPAP prevents airway and alveolar collapse, recruits more alveoli and improves oxygenation.

♣ If **CO_2** remains **high**, following measures are taken-

- Adjust FIo2 (SPO2 85-95%);
- Check for excessive leakage;
- Consider increase in Respiratory rate;
- Increase inspiratory time;
- Increase IPAP.

♣ If **PO_2** remains **low**; do

- Increase FIO2;
- Increase EPAP

IPPV:

- Protect the airways against aspiration of gastric content
- To deliver an increase FiO2
- Facilitate Mechanical Ventilation

➢ **Prone Position Ventilation**:

- Improves Ventilation-Perfusion mismatch by recruiting atelectatic alveoli;
- Improves Drainage of secretions;
- Reduces 20% of FiO2.

➢ **Extra corporial respiratory support:**

- If patient has a reversible cause of respiratory failure and facilities are available, then extracorporeal respiratory support should be considered.
- Types-
  - Venous-venous extracorporeal Oxygenation
  - Extracorporeal CO_2 removal

❖ **Supportive Measures:** Every patient of Acute Respiratory Failure is at risk of developing :

- DVT
- Pulmonary Thromboembolism
- Gastric Stress Ulceration

So, measures should be taken to prevent them.

Journey of dyspnea patient from entry to critical care unit to extubation is shown in **Figure 1**.

Figure 1. SO JOURNEY OF DYSPNEA TO EXTUBATION

Dyspnea → O2 by **rebreather →Neck extension →Suction→** Checking any **FB/secretion** in respiratory tract & **removal** by suction → **Umbooing**, **Circulation** maintain by **fluid/ ionotrops/ chronotropes → NIPPV** (BiPAP: IPAP &EPAP/ CPAP) → **IPPV** (Intubation) & MV(Mandatory Ventilation) →**CMV** mode (Continuous Mandatory Ventilation) (Tidal volume, FiO2, RR, PEEP, altering IE ratio, suction, Bronchodilator) →**Improvement→SIMV** (synchronized intermittent mandatory ventilation)→**PS mode** (pressure-supported spontaneous breaths) →**T-Piece Trial→Extubation**.

Brief summary of management of dyspnea due to different causes are shown in tabulated form in **Box 2**.

Box 2. Brief Summary of Management of Dyspnea due to Different Causes	
Disease causing dyspnea	**Management**
Acute exaggeration of COPD	Maximal Medical Treatment (MMT) + controlled O2 (PaO_2 60% or SpO_2 88-92%) by Nasal prongs (NP) / Facemask (FM) → If patient still dyspneic and $PaCO_2$ >45mmhg+pH <7.35 → NIPPV(BiPAP) → Still dyspnec or rising $PaCO_2$→IPPV.
Acute Severe Asthma	Maximal Medical Treatment + High conc. O_2 (NP-FM-Partial rebreather-Rebreather) → **S**till dyspnec → NIPPV→ Refractory Hypoxia (PaO_2 <60mmhg and falling), $PaCO_2$ >45 mmHg and rising, low and falling pH, confusion, somnolence, coma, respiratory or cardiac arrest → IPPV.

Pneumonia(Bacteria/Viral)	Maximal Medical Treatment + High conc. O_2 (NP-FM-Partial Rebreather-Rebreather) → CURB 4-5, persistent hypoxia, progressive hypercapnia, severe acidosis, circulatory shock, reduced conscious level → NIPPV → IPPV.
Septic shock/Hypovolemic shock	Maximal Medical Treatment + Fluid Challenge (25 ml/kg) + Inotropes + O_2 administration (NP-FM-Rebreather) → NIPPV → IPPV.
Cardiogenic Cause (Acute LVF, Vulvular heart diseases, etc.)	Maximal Medical Treatment + Diuretics+ O_2 (NP-FM-Rebreather) → ionotropes/Chronotropes → CPAP → IPPV.

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