Review of Contemporary Philosophy
ISSN: 1841-5261, e-ISSN: 2471-089X
Vol 23 (02), 2024
pp. 7518 - 7534

Respiratory Failure In Adults: An Updated Review Article for Healthcare Professionals

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Abstract:

Background: Respiratory failure is a critical condition characterized by the inability of the respiratory system to maintain adequate oxygenation (Type 1) or carbon dioxide elimination (Type 2). It can be acute, chronic, or acute-on-chronic, with varying etiologies including pulmonary, cardiac, neuromuscular, and systemic disorders. Prompt recognition and intervention are essential to prevent life-threatening complications.

Aim: This review provides an updated clinical approach to diagnosing and managing respiratory failure in adults, emphasizing early identification, pathophysiology, and evidence-based treatment strategies.

Methods: A comprehensive evaluation of respiratory failure involves history, physical examination, arterial blood gas (ABG) analysis, imaging (chest X-ray, CT), capnometry, and point-of-care ultrasound. Laboratory tests, electrocardiography, and specialized diagnostics (e.g., bronchoscopy, polysomnography) are used as needed.

Results: Type 1 respiratory failure (hypoxemic) arises from impaired oxygen diffusion, ventilation-perfusion (V/Q) mismatch, or shunting, while Type 2 (hypercapnic) results from alveolar hypoventilation due to central, neuromuscular, or mechanical causes. Treatment includes oxygen therapy, noninvasive ventilation (NIV), or invasive mechanical ventilation, depending on severity. Mortality varies by etiology, with higher rates in ARDS (44.3%) and pneumonia (48.4%) requiring intubation.

Conclusion: Early diagnosis and tailored interventions improve outcomes. Multidisciplinary care involving pulmonologists, intensivists, nurses, and respiratory therapists is crucial. Patient education on medication adherence, vaccination, and smoking cessation reduces exacerbation risks.

Keywords: Respiratory failure, hypoxemia, hypercapnia, mechanical ventilation, ARDS, COPD, oxygenation, ABG analysis.

Received: 04 October 2024 **Revised:** 23 November 2024 **Accepted:** 10 December 2024

Introduction:

The respiratory system plays a central role in supporting aerobic metabolism by enabling gas exchange between the external environment and the human body. This system is responsible for two essential functions: the delivery of oxygen to tissues and the elimination of carbon dioxide, a byproduct of cellular respiration. When the respiratory system is unable to maintain either of these functions, the result is respiratory failure, a condition that requires immediate medical attention. Respiratory failure is classified into two primary types. Type 1 respiratory failure, also referred to as hypoxemic respiratory failure, arises when there is an inadequate supply of oxygen, which leads to a drop in arterial oxygen levels (hypoxemia). In contrast, Type 2 respiratory failure, or hypercapnic respiratory failure, occurs when the body is unable to expel carbon dioxide efficiently, causing elevated levels of carbon dioxide in the blood (hypercapnia). In addition to this functional classification, respiratory failure can be categorized by its temporal profile as acute, chronic, or acute on chronic. Acute respiratory failure develops rapidly and may be reversible if treated early, while chronic respiratory failure progresses over time and is often associated with underlying chronic pulmonary conditions. Acute chronic respiratory failure represents an acute worsening of pre-existing chronic respiratory failure. Recognizing the type and temporal pattern of respiratory failure is essential for determining the appropriate clinical response. Failure to promptly identify and manage respiratory failure can lead to rapid deterioration, including respiratory arrest, coma, and death. This article addresses the clinical approach to adult patients suspected of having respiratory failure, including both hypoxemic and hypercapnic forms. It also outlines current practices in the diagnosis and treatment of both acute and chronic respiratory failure, emphasizing the importance of early recognition and intervention to prevent fatal outcomes.

Etiology:

Respiratory failure can develop due to abnormalities in any part of the respiratory system. The respiratory system includes the upper and lower respiratory tracts, the central and peripheral nervous systems, the chest wall, and the muscles involved in breathing [1]. Disruption in any of these components can impair gas exchange and lead to either hypoxemia, hypercapnia, or both. For example, obstruction, infection, or trauma in the upper respiratory tract may block airflow, while lower respiratory tract conditions such as pneumonia or chronic obstructive pulmonary disease can interfere with alveolar oxygenation. Neurological control is also critical. Damage to the central nervous system—such as brainstem injury or drug-induced respiratory depression—can suppress respiratory drive. Similarly, disorders of the peripheral nervous system, including Guillain-Barré syndrome or myasthenia gravis, may weaken the signal transmission to respiratory muscles, impairing ventilation. Muscle weakness or fatigue, whether from neuromuscular disease or critical illness, reduces the capacity for effective breathing. Structural issues in the chest wall, such as kyphoscoliosis or rib fractures, can limit chest expansion and compromise lung volumes. Each of these disruptions affects the respiratory system's ability to perform oxygen delivery and carbon dioxide clearance. Identifying which component is impaired is essential for diagnosing the cause of respiratory failure. The pathophysiology section of this article will review specific etiologies of respiratory failure [1], offering a clearer understanding of how distinct conditions contribute to the failure of respiratory function. This forms the basis for accurate diagnosis and tailored treatment.

Epidemiology:

Respiratory failure (RF) is not a single disease but a clinical syndrome resulting from various underlying conditions. This complexity makes it difficult to determine precise epidemiological patterns. Despite these challenges, data from the United States in 2017 indicated that the incidence of respiratory failure was approximately 1,275 cases per 100,000 adults, based on diagnostic codes that included respiratory failure as part of the diagnosis [2]. This figure illustrates the significant burden of RF in the adult population, but it must be interpreted within the context of the diverse etiologies and varying clinical definitions used across healthcare settings. The epidemiology of respiratory failure largely reflects the frequency of its root causes. For example, respiratory failure associated with acute myocardial infarction (AMI-RF) accounted for 439,436 hospital admissions between 2000 and 2014. Of these cases, 57% were diagnosed with respiratory failure, and 43% required mechanical ventilation [3]. These figures emphasize the respiratory complications that can arise from cardiac events and the substantial proportion of patients needing advanced respiratory support. Acute respiratory distress syndrome (ARDS) represents another major cause of acute respiratory failure. Its incidence varies widely—from 10 to 80 cases per 100,000 per year—depending on geographic location and clinical practices. This variation is influenced by differences in diagnostic criteria, thresholds for intubation, and availability of critical care resources. One report suggests that ARDS accounts for 10% of all intensive care unit (ICU) admissions and 23% of mechanically ventilated patients [4]. The COVID-19 pandemic significantly altered the landscape of respiratory failure epidemiology. Early in the pandemic, up to 79% of hospitalized COVID-19 patients developed respiratory failure severe enough to require invasive mechanical ventilation [5]. This surge in cases strained healthcare systems and highlighted the importance of preparedness for respiratory pandemics. Another frequent cause of respiratory failure is acute exacerbation of chronic obstructive pulmonary disease (AECOPD). It ranks as the third most common cause among hospitalized patients with acute respiratory failure [6]. This trend underscores the ongoing impact of chronic respiratory diseases and their acute complications on healthcare systems worldwide.

Pathophysiology

Type 1 Respiratory Failure

Type 1 respiratory failure is defined by a reduction in the partial pressure of oxygen in arterial blood (PaO_2) to below 60 mmHg, while the partial pressure of carbon dioxide ($PaCO_2$) remains normal or reduced. This condition primarily reflects a failure of oxygen exchange across the alveolar-capillary membrane. A key diagnostic tool in evaluating this condition is the alveolar-arterial (A-a) oxygen gradient, calculated by subtracting the PaO_2 from the alveolar partial pressure of oxygen (PAO_2). The A-a gradient helps determine whether hypoxemia results from alveolar hypoventilation or other mechanisms such as diffusion defects or ventilation-perfusion mismatch. The alveolar gas equation, expressed as $PAO_2 = FiO_2\ (PB - Pwater) - PaCO_2/0.8$, estimates the alveolar oxygen pressure. In this equation, FiO_2 is the fraction of inspired oxygen, PB is the barometric pressure, Pwater is the water vapor pressure at body temperature (47 mmHg), and $PaCO_2$ represents the arterial carbon dioxide pressure [7]. This equation is critical in identifying whether a high A-a gradient is present and in interpreting the physiological basis of the observed hypoxemia. Some etiologies of Type 1 respiratory failure present with a normal A-a gradient. In alveolar hypoventilation, $PaCO_2$ increases due to reduced alveolar ventilation. According to the alveolar gas equation, this increase in $PaCO_2$ leads to a decrease in PAO_2. Since both PAO_2 and PaO_2 fall proportionally, the A-a gradient remains within normal limits. If untreated, hypoventilation can progress into Type 2 respiratory failure due to carbon dioxide retention [8]. Another cause of Type 1 respiratory failure with a normal A-a gradient is reduced inspired oxygen or atmospheric pressure. In high-altitude environments, atmospheric pressure (Patm) decreases, leading to a lower PAO_2. Similarly, a decrease in inspired oxygen (FiO_2) also reduces PAO_2. In response, hyperventilation occurs, lowering $PaCO_2$. Despite this compensatory mechanism, arterial oxygen remains low. The A-a gradient, however, remains normal, as both alveolar and arterial oxygen levels decline simultaneously [9].

When the A-a gradient is elevated, the cause of hypoxemia typically involves impaired oxygen transfer despite normal ventilation. One such mechanism is a diffusion defect, where structural changes at the alveolar-capillary interface hinder oxygen movement into the bloodstream. This may result from reduced surface area or thickening of the alveolar membrane. A shortened pulmonary capillary transit time, as seen in high-output states, may also contribute to inadequate oxygen transfer. However, carbon dioxide diffusion usually remains intact because it crosses the alveolar-capillary membrane more efficiently than oxygen [10]. Common causes of diffusion defects include emphysema and interstitial lung disease. Ventilation-perfusion (V/Q) mismatch is another major cause of Type 1 respiratory failure with an elevated A-a gradient. Normally, the V/Q ratio across the lungs is about 0.8, reflecting the balance between alveolar ventilation and perfusion. When this balance is disrupted, gas exchange becomes inefficient. If ventilation exceeds perfusion, the V/Q ratio is high, which leads to increased dead space ventilation. Conversely, when perfusion exceeds ventilation, the V/Q ratio is low, resulting in shunting of blood that bypasses oxygenation. This mismatch is the most frequent cause of hypoxemia in clinical practice [11]. Diseases commonly associated with V/Q mismatch include acute respiratory distress syndrome (ARDS), chronic obstructive pulmonary disease (COPD), congestive heart failure (CHF), and pulmonary embolism.

A true shunt occurs when blood flows through the lungs without participating in gas exchange, resulting in a V/Q ratio of zero. This is comparable to intracardiac right-to-left shunting, such as that seen in atrial septal defect or patent foramen ovale. Unlike V/Q mismatch, hypoxemia due to shunt is unresponsive to supplemental oxygen, since no oxygen enters the affected alveoli for exchange [12]. Pulmonary causes of right-to-left shunting include arteriovenous malformations, areas of complete atelectasis, severe pneumonia, and massive pulmonary edema. In summary, Type 1 respiratory failure involves hypoxemia with a normal or decreased $PaCO_2$ and can result from diverse pathophysiological processes. A normal A-a gradient suggests problems related to ventilation or atmospheric conditions, while an increased A-a gradient indicates structural or functional disruptions in oxygen exchange. The most common underlying mechanisms include diffusion impairment, V/Q mismatch, and true shunting. Accurate identification of the specific cause is essential for guiding therapy and improving clinical outcomes. Understanding these mechanisms also informs the use of oxygen therapy, mechanical ventilation, and other interventions tailored to the underlying defect in gas exchange.

Type 2 Respiratory Failure

Type 2 respiratory failure, also referred to as hypercapnic respiratory failure, occurs when the arterial partial pressure of carbon dioxide ($PaCO_2$) rises above 45 mmHg, accompanied by a decrease in blood pH to below 7.35. This reflects inadequate alveolar ventilation relative to the body's metabolic CO_2 production. The imbalance can result from either insufficient CO_2 elimination or, more rarely, increased CO_2 production. The alveolar ventilation equation explains this relationship: $PaCO_2$ is directly proportional to the rate of carbon dioxide production (VCO_2) and inversely related to alveolar ventilation (VA), expressed as $PaCO_2 = VCO_2 / VA$. Any condition reducing VA or increasing VCO_2 can result in hypercapnia [13]. VA itself depends on minute ventilation (VE) and the ratio of dead space (VD) to tidal volume (Vt). The equation $VA = VE \times (1 - VD/Vt)$ highlights that reductions in effective alveolar ventilation may occur even with normal or increased minute ventilation if a large portion of that ventilation is wasted as dead space. Although rare, increased CO_2 production can contribute to hypercapnia, especially if the respiratory system cannot compensate with increased ventilation. In most cases, however, decreased VA is the underlying cause. The two main mechanisms underlying this condition can be categorized as "won't breathe," referring to impaired central respiratory drive, or "can't breathe," reflecting structural, mechanical, or neuromuscular limitations. Both result in alveolar hypoventilation and CO_2 retention. Whether the PaO_2 remains normal or drops depends on the balance between hypoventilation, dead space ventilation, and underlying pulmonary disease. The respiratory pump comprises the central nervous system, peripheral nerves, respiratory muscles, chest wall, pleura, and lung parenchyma. Failure in any of these components can cause reduced ventilation and lead to hypercapnia.

Central drive impairment is a common contributor to Type 2 respiratory failure. Depressants such as alcohol, opioids, and benzodiazepines blunt the brain's respiratory response to CO_2. Neurological diseases including stroke, encephalitis, tumors, and spinal cord injuries (SCI) can disrupt respiratory centers or neural pathways, also reducing ventilatory effort [14]. Neuromuscular disorders that affect signal transmission between the brain and the respiratory muscles are another key group of causes. Conditions like amyotrophic lateral sclerosis, myasthenia gravis, Guillain-Barré syndrome, botulism, and transverse myelitis reduce muscle strength or coordination. These impair the mechanics of breathing and compromise

CO_2 clearance [14]. Mechanical issues affecting the chest wall or pleura also reduce the ability to generate adequate tidal volumes. Kyphoscoliosis, flail chest, thoracoplasty, large pleural effusions, and severe obesity can impose restrictive loads on the lungs. These conditions reduce lung compliance and increase the breathing work, leading to ventilatory failure.

Muscular abnormalities further contribute to respiratory pump failure. Diaphragmatic paralysis, muscular dystrophy, and respiratory muscle atrophy decrease respiratory force generation. These conditions commonly appear in long-term neuromuscular disease and can worsen during periods of increased ventilatory demand. Dead space ventilation plays a pivotal role in hypercapnia. When large portions of ventilation do not participate in gas exchange, as seen in emphysema, pulmonary embolism, or ARDS, the effective alveolar ventilation drops. Hypoventilation can occur when this dead space exceeds 50% of total ventilation. Tachypnea can exacerbate this imbalance by increasing VD/Vt, thereby reducing the proportion of air reaching functioning alveoli. In COPD, elevated dead space and ventilation-perfusion mismatch are central mechanisms in the development of chronic hypercapnia [15]. Increased CO_2 production is a less common cause of Type 2 respiratory failure but can occur under specific conditions. Fever, sepsis, thyrotoxicosis, intense physical exertion, and overfeeding (hyperalimentation) accelerate metabolic activity, thereby increasing CO_2 output. When this increase exceeds the compensatory capacity of the lungs to ventilate, $PaCO_2$ rises. Although the lungs are usually capable of adapting through higher VE, failure to do so results in respiratory acidosis [16].

Alveolar hypoventilation serves as the final common pathway for the majority of Type 2 respiratory failure causes. Whether due to neuromuscular dysfunction, impaired central drive, or mechanical restriction, insufficient VA leads to CO_2 retention. If left untreated, this can progress to severe acidosis, altered mental status, and respiratory arrest. PaO_2 may remain stable early on, but hypoxemia becomes more likely as the condition advances [8]. Understanding the diverse etiologies of Type 2 respiratory failure is critical for accurate diagnosis and treatment. Clinical assessment must determine whether the issue lies in respiratory drive, neuromuscular transmission, pulmonary mechanics, or metabolic demands. Treatment strategies depend on addressing the root cause. This may involve reversing CNS depression, supporting ventilation with noninvasive or mechanical ventilation, treating infections, or optimizing neuromuscular function. Identifying whether CO_2 retention is acute or chronic also influences management decisions, especially regarding the use of oxygen therapy in patients with chronic hypercapnia who rely on hypoxic drive. Effective intervention requires a systems-level view of ventilation, incorporating mechanics, control, and metabolic factors. Without timely correction, persistent hypercapnia leads to worsening acidosis, organ dysfunction, and death.

History:

A comprehensive history and physical examination are vital in assessing patients with respiratory failure, given the broad range of potential underlying etiologies. Respiratory failure is not a disease in itself but a clinical syndrome that arises from diverse causes, including pulmonary, cardiac, neuromuscular, and systemic conditions. Accurate diagnosis depends on a detailed understanding of the patient's clinical background, current symptoms, and risk factors. Patients commonly present with a range of respiratory symptoms such as dyspnea, wheezing, cough, hemoptysis, and sputum production. However, the diagnostic process should not be limited to these manifestations. Symptoms from other organ systems can provide essential diagnostic clues. For example, chest pain may indicate a pulmonary embolism or cardiac ischemia, while fever and chills may suggest an infectious cause. Constitutional symptoms like weight loss, decreased appetite, and fatigue may point toward malignancy or chronic systemic illness. Upper respiratory symptoms such as loss of smell, along with exposure to sick contacts, particularly individuals infected with COVID-19, must be assessed to evaluate the likelihood of viral pneumonia and subsequent respiratory failure. This is particularly relevant in high-risk populations, including elderly patients, males, and individuals with obesity [17]. Immunosuppression is another critical factor to investigate. Patients with known immunocompromised states, such as those on immunosuppressive therapy or with conditions like HIV/AIDS, are at higher risk for opportunistic infections leading to respiratory compromise. Early identification of these risk factors allows for more targeted diagnostic evaluation and treatment.

In patients with pre-existing airway diseases like asthma or chronic obstructive pulmonary disease (COPD), it is necessary to assess adherence to inhaler therapy and proper usage techniques. Questions should also cover recent use of systemic steroids and exposure to known environmental triggers. For patients experiencing chronic cough and hypertension, clinicians should consider whether the patient is using angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, both of which are known to induce cough. A thorough social history enhances the assessment of risk. Alcohol use and history of sexually transmitted infections may suggest immunosuppression. Sedentary behavior can increase the risk of thromboembolic events. Lifestyle habits and hobbies may offer additional clues; for example, bird ownership may be relevant in hypersensitivity pneumonitis, and activities like diving or flying might relate to barotrauma or decompression-related issues. A detailed smoking history is also essential, including use of cigarettes, marijuana, e-cigarettes, and exposure to secondhand smoke or vaping, as all have established links with respiratory pathology [18]. Occupational history plays a pivotal role, especially when considering work-related lung diseases such as hypersensitivity pneumonitis or pneumoconiosis. Inhalation exposures in industrial, agricultural, or construction settings may contribute to the pathogenesis of respiratory symptoms. Additionally, a family history may provide clues to inherited or genetic disorders like cystic fibrosis or alpha-1-antitrypsin deficiency. It may also help assess susceptibility to infectious diseases such as tuberculosis [19]. The physical examination should be systematic and thorough, as findings can emerge from multiple regions of the body. General inspection might reveal signs such as respiratory distress, altered mental status, cachexia, or obesity. The patient's use of accessory muscles and presence of diaphoresis can indicate increased respiratory effort. Cyanosis, either central or peripheral, and clubbing of the fingers are signs that may point toward chronic hypoxemia.

Examination of the head and neck may uncover features such as pale conjunctiva, suggestive of anemia, or Horner's syndrome, potentially indicating a Pancoast tumor. Jugular venous distension or tracheal deviation may suggest intrathoracic pathology. Chest auscultation and percussion remain critical, as they can reveal asymmetrical breath sounds, dullness or hyperresonance, crackles, wheezes, rhonchi, or pleural rubs. Tactile fremitus and vocal resonance may help localize areas of consolidation or effusion. In the abdomen, hepatomegaly may be present in cases of right heart failure secondary to chronic pulmonary hypertension. Upper extremity findings such as asterixis or tremor may indicate carbon dioxide retention or metabolic encephalopathy, while digital clubbing may reflect chronic pulmonary disease. Lower extremity examination should assess for edema and signs of deep venous thrombosis, both of which may suggest cor pulmonale or pulmonary embolism [20]. Each element of the history and physical examination contributes to narrowing the differential diagnosis and guiding further investigations. Timely and accurate assessment is essential for initiating effective treatment and improving outcomes in patients presenting with respiratory failure.

Evaluation:

Respiratory failure results from various underlying conditions, which makes its assessment complex and dependent on clinical context. There is no universal diagnostic algorithm for respiratory failure, and evaluation must be tailored based on history, physical examination, and suspected etiology. Several diagnostic modalities are used in combination to determine the type, severity, and cause of respiratory failure. These include laboratory studies, imaging, electrocardiography, arterial blood gas analysis, capnometry, and point-of-care ultrasound. A thorough laboratory evaluation often starts with a complete blood count with differential, which helps identify infection, anemia, or leukemoid reactions. A comprehensive metabolic panel provides information on renal function, electrolytes, and bicarbonate levels. Abnormalities in magnesium or phosphorus may indicate metabolic imbalances affecting respiratory muscle function. Cardiac biomarkers such as troponin are useful in identifying myocardial injury as a contributing factor. Procalcitonin levels may help distinguish bacterial infections from non-infectious causes. In cases of suspected endocrine contributions, thyroid-stimulating hormone testing can uncover hypothyroidism-related hypoventilation. If infection is suspected, clinicians may perform blood cultures, sputum cultures, respiratory pathogen panels, and urinary antigen testing for organisms like Streptococcus pneumoniae or Legionella. Electrocardiography is commonly used to rule out cardiac causes of respiratory symptoms, such as acute myocardial infarction or arrhythmias that can contribute to hypoxia or decompensation. Chest imaging, including chest X-ray or CT scan, helps identify structural abnormalities such as pneumonia, pleural effusion, pneumothorax, or pulmonary embolism. Arterial blood gas (ABG) remains the gold standard for confirming respiratory failure. It measures arterial pH, PaO2, PaCO2, and serum bicarbonate. Hypoxemia is defined by a PaO2 below 60 mmHg, while hypercapnia is defined by a PaCO2 above 45 mmHg. The ABG also allows clinicians to assess acid-base status and categorize respiratory failure into acute or chronic forms. This differentiation is based on bicarbonate compensation. In acute respiratory acidosis, the renal response is minimal, resulting in only a slight increase in HCO3. In chronic respiratory acidosis, the kidneys have had time to compensate, and HCO3 levels are significantly elevated. However, since bicarbonate on ABG is calculated, confirmation with the value from a basic metabolic panel, where bicarbonate is directly measured, is more reliable [21].

Capnometry is another useful diagnostic tool, particularly in emergency settings. It measures carbon dioxide in exhaled gas. Colorimetric capnometry provides a qualitative assessment, indicating the presence or absence of CO2 using a color-changing device. Quantitative capnometry, often using infrared sensors, gives precise readings of end-tidal carbon dioxide (PETCO2). Under normal physiology, PETCO2 is only slightly lower than PaCO2, typically by 2 to 3 mmHg. When this gap widens, it signals impaired gas exchange or increased dead space ventilation, which occurs in conditions like pulmonary embolism or severe COPD [22][23]. Quantitative capnometry data can be visualized as waveform capnography. The capnogram reflects each phase of the respiratory cycle and provides valuable insight into ventilation patterns. Abnormal waveforms may indicate conditions such as bronchospasm, hypoventilation, or apnea. For instance, a sloped or prolonged expiratory upstroke can signal obstructive disease, while a flat capnogram may suggest apnea or significant hypoventilation [24]. Pulse oximetry, though commonly used, has limitations. It measures oxygen saturation but cannot detect hypercapnia or changes in ventilation. It can also give falsely reassuring results in patients receiving supplemental oxygen despite worsening ventilation. Therefore, pulse oximetry must be interpreted alongside ABG and clinical findings. Point-of-care ultrasound can support rapid bedside evaluation. Lung ultrasound may detect pneumothorax, pleural effusion, pulmonary edema, or consolidation. Cardiac ultrasound helps evaluate for right heart strain, pericardial effusion, or ventricular dysfunction, which may contribute to or result from respiratory failure. Together, these tools offer a comprehensive framework for evaluating respiratory failure. Each test provides distinct but complementary information that contributes to diagnosis, guides treatment, and allows for monitoring disease progression. Accurate and early evaluation is essential to prevent further deterioration and tailor interventions to the underlying cause.

Radiography:

Radiographic evaluation plays a central role in the diagnostic workup of respiratory failure. Several imaging modalities are employed depending on the clinical scenario and suspected underlying pathology. These include chest radiography, computed tomography (CT), magnetic resonance imaging (MRI), nuclear medicine scans, angiography, and ultrasonography [25]. Chest radiography, or plain film X-ray, is often the first imaging study performed. It is widely available, quick, and inexpensive. It can identify causes of respiratory failure such as pneumonia, pulmonary edema, pleural effusion, pneumothorax, and atelectasis. In the intensive care setting, portable chest X-rays are frequently used to monitor disease progression and detect complications such as misplaced tubes or evolving infiltrates. However, its sensitivity is limited, especially in early or subtle disease processes. CT imaging provides more detailed visualization of lung parenchyma, vasculature, and mediastinal structures. It is especially valuable in identifying interstitial lung disease, pulmonary embolism, small nodules, or masses not visible on chest radiography. High-resolution CT (HRCT) is preferred when evaluating diffuse parenchymal lung disease. In the setting of acute respiratory failure, CT pulmonary angiography is commonly used to confirm or exclude pulmonary embolism. The use of intravenous contrast improves vascular assessment but may be contraindicated in patients with renal dysfunction.

MRI is less commonly used for lung imaging due to limited spatial resolution and longer acquisition times. However, it may be helpful in evaluating vascular or mediastinal abnormalities and is preferred in specific populations such as pregnant patients to avoid radiation exposure. Nuclear medicine techniques such as ventilation-perfusion (V/Q) scans are used in patients with suspected pulmonary embolism who cannot undergo CT angiography. These scans compare areas of the lung that are ventilated with those that are perfused, and mismatched defects are suggestive of embolic disease. Angiography, once the standard for diagnosing pulmonary embolism, is now rarely used due to the availability of CT pulmonary angiography. It remains an option when less invasive imaging is inconclusive or not feasible. Ultrasonography has become increasingly important, particularly in acute and critical care settings. Lung ultrasound provides real-time, bedside information without radiation. It is helpful in detecting pneumothorax, pleural effusions, pulmonary edema, and consolidations. As discussed further below, the BLUE protocol is a systematic approach to lung ultrasound specifically designed to identify causes of acute respiratory failure.

Pulse Oximetry

Pulse oximetry is a non-invasive method for monitoring oxygen saturation (SpO2). It is based on spectrophotometry, measuring the absorption of light at two different wavelengths through pulsatile arterial blood. Deoxygenated hemoglobin absorbs light at 660 nm, while oxygenated hemoglobin absorbs light at 940 nm. These values are processed by proprietary algorithms to calculate the percentage of hemoglobin saturated with oxygen. Pulse oximetry is a valuable tool for continuous monitoring in both inpatient and outpatient settings [26]. Although it provides rapid feedback on oxygenation status, pulse oximetry has several limitations. It does not assess ventilation or detect hypercapnia, and readings can be falsely elevated in patients receiving supplemental oxygen despite inadequate ventilation. In conditions like carbon monoxide poisoning or methemoglobinemia, pulse oximetry may provide misleading results due to the inability to differentiate abnormal hemoglobin derivatives.

Ultrasonography

Ultrasound of the lungs is a rapid, bedside diagnostic tool increasingly used in emergency and critical care settings. The BLUE protocol is a validated method for evaluating acute respiratory failure using thoracic ultrasound. It involves examining six standardized thoracic points on the anterior and lateral chest walls. Each of these "BLUE points" corresponds to specific lung zones and is examined for characteristic sonographic findings [27]. The protocol identifies ten ultrasonographic signs that correspond with particular clinical conditions. Normal lung is indicated by the presence of the bat sign, lung sliding, and horizontal A-lines. Lung rockets, or B-lines, are associated with interstitial syndromes such as pulmonary edema or fibrosis. Consolidation appears as tissue-like echotexture and irregular borders, known as the fractal and tissue-like signs. Pleural effusions are detected using the quad and sinusoid signs, indicating fluid accumulation. Pneumothorax is diagnosed by absence of lung sliding (stratosphere sign) and detection of the lung point, which marks the boundary between normal and collapsed lung. Lung ultrasound offers distinct advantages, especially in unstable patients, by avoiding radiation and enabling repeat assessments. Its diagnostic accuracy is high when performed by trained providers. However, it is operator-dependent, and interpretation requires familiarity with lung anatomy and pathology. Other diagnostic tools such as bronchoscopy, echocardiography, nocturnal polysomnography, and pulmonary function tests are employed selectively based on the suspected etiology. Bronchoscopy may be needed for airway visualization, biopsy, or secretion clearance. Echocardiography helps assess cardiac function and pulmonary pressures. Pulmonary function tests are more relevant in the chronic setting but can guide diagnosis and prognosis. Polysomnography is used when sleep-related breathing disorders are suspected. In complex cases requiring advanced diagnostics, pulmonary consultation is essential for comprehensive evaluation and management.

Table 1: Diagnostic Techniques.

Technique	Purpose	Key Details
Arterial Blood Gas (ABG)	Assess oxygenation, ventilation, acid-base balance	Gold standard; measures pH, PaO_2, $PaCO_2$, calculated HCO_3; PaO_2 < 60 mmHg = hypoxemia; $PaCO_2$ > 45 mmHg = hypercapnia
Capnometry/Capnography	Measure exhaled CO_2	$PETCO_2$ approximates $PaCO_2$ in healthy lungs; quantitative method preferred; detects ventilation abnormalities
Pulse Oximetry	Estimate oxygen saturation (SpO_2) noninvasively	Uses 660/940 nm wavelengths; target SpO_2 90–94%; easy bedside monitoring
Radiography (CXR/CT/MRI)	Identify structural abnormalities	Chest X-ray for initial assessment; CT/MRI for detailed views; identify causes like pneumonia or effusion
Ultrasonography (BLUE)	Rapid bedside diagnosis	Uses 6 points and 10 profiles; detects pneumothorax, effusions, consolidation, interstitial syndrome
Infectious Workup	Identify infectious causes	Includes blood/sputum cultures, respiratory panel, urinary antigens
ECG	Evaluate cardiac contribution	Rule out myocardial infarction or arrhythmias

Treatment / Management

The management of respiratory failure hinges on two fundamental principles: addressing the underlying cause and providing immediate support to maintain adequate oxygenation and ventilation. The initial approach must always begin with a structured assessment of airway, breathing, and circulation (ABC). This ensures stabilization of vital functions while diagnostic efforts are underway. Without a patent airway and adequate breathing, corrective measures for blood gas abnormalities and tissue oxygenation are ineffective. The correction of hypoxemia is a priority in all cases. The therapeutic goal is to maintain arterial oxygen tension (PaO2) of at least 60 mmHg or arterial oxygen saturation (SaO2) around 90%. Achieving this ensures sufficient tissue oxygen delivery without inducing oxygen toxicity. Administering excessive oxygen, especially in patients with chronic hypercapnia such as those with COPD, can lead to oxygen-induced hypoventilation and CO2 narcosis. Therefore, oxygen supplementation must be titrated to maintain target saturations, typically between 90% and 94%. Various oxygen delivery devices are used depending on the severity of hypoxemia and the patient's clinical condition. These range from nasal cannulae and simple face masks to non-rebreather masks and high-flow nasal cannula systems. In severe or refractory cases, where conventional oxygen delivery methods fail, extracorporeal membrane oxygenation (ECMO) may be necessary to support gas exchange [28].

Correction of hypercapnia and respiratory acidosis follows the principle of either reversing the cause of alveolar hypoventilation or initiating ventilatory support [29]. In some patients, treating infections, clearing airway obstructions, or managing exacerbations of underlying diseases such as asthma or COPD may resolve the issue. However, when these approaches fail or when respiratory acidosis worsens, mechanical ventilation becomes essential. Mechanical ventilatory support is indicated when a patient shows signs of respiratory muscle fatigue, altered mental status, or hemodynamic instability, or fails to maintain adequate oxygenation or ventilation despite supplemental oxygen. Specific clinical thresholds include apnea, a respiratory rate above 30 breaths per minute, PaO2 under 60 mmHg despite oxygen, or arterial pH less than 7.25 due to hypercapnia [30]. Ventilation serves to correct gas exchange abnormalities, reduce the work of breathing, and allow respiratory muscles to recover. The decision between invasive and noninvasive ventilation depends on several factors. These include the acuity and severity of the condition, the underlying cause of respiratory failure, the patient's level of consciousness, airway protective reflexes, and hemodynamic stability [31]. Noninvasive ventilation (NIV), using masks or nasal interfaces, is increasingly used and often preferred in select conditions. Evidence supports its use in acute exacerbations of COPD, cardiogenic pulmonary edema, and obesity hypoventilation syndrome [32][33][34][35][36]. NIV reduces the need for intubation, shortens hospital stay, and improves survival in these conditions. Invasive mechanical ventilation is reserved for patients with more severe forms of respiratory failure or when NIV fails. It involves endotracheal intubation and precise control over ventilation settings, including tidal volume, respiratory rate, and oxygen concentration. This allows for tailored management but carries risks such as ventilator-associated pneumonia, barotrauma, and prolonged weaning challenges. Ultimately, successful management of respiratory failure requires a multidisciplinary approach that addresses both immediate physiological needs and the root pathology. Frequent reassessment, appropriate escalation of care, and transition planning are critical in ensuring both survival and recovery.

Table 2: Treatment and Supporting Measures.

Intervention	**Indication**	**Details**
Oxygen Therapy	Hypoxemia	Nasal cannula, simple mask, non-rebreather, high-flow nasal cannula; avoid oxygen toxicity
Mechanical Ventilation (Invasive)	Severe respiratory failure, apnea, coma, pH < 7.25	Intubation; correct acidosis and hypoxemia; rest ventilatory muscles
Noninvasive Ventilation (NIV)	COPD exacerbation, cardiogenic pulmonary edema	BiPAP or CPAP; avoids intubation; effective in selected chronic conditions
Extracorporeal Membrane Oxygenation	Refractory hypoxemia	Requires specialized centers; used when ventilation and oxygenation fail
Treat Underlying Cause	All cases	E.g., antibiotics for pneumonia, steroids for asthma, anticoagulation for embolism
Pharmacological Therapy	Disease-specific	Includes bronchodilators, corticosteroids, diuretics, antibiotics, vasopressors
Nursing Care	All hospitalized patients	Suctioning, repositioning, feeding, infection control, patient monitoring
Respiratory Therapy	All ventilated or oxygen-requiring patients	Administers O_2, performs chest physiotherapy, supports ventilation
Pharmacist Involvement	Polypharmacy, chronic conditions	Ensures safe medication use, adjusts doses, prevents interactions, patient counseling
Patient Education	Discharge planning, chronic disease	Focus on inhaler technique, risk factor modification, vaccination, and symptom awareness

Differential Diagnosis

Respiratory failure is a clinical syndrome with diverse causes that impact either oxygenation, ventilation, or both. Establishing an accurate diagnosis requires systematic evaluation of possible etiologies across various organ systems. The differential diagnosis encompasses pulmonary, cardiovascular, neuromuscular, and systemic conditions. Acute respiratory distress syndrome (ARDS) is a leading cause of hypoxemic respiratory failure. It often follows sepsis, trauma, or pneumonia and presents with diffuse bilateral infiltrates and refractory hypoxemia. Aspiration pneumonia and aspiration pneumonitis are also common, particularly in patients with impaired consciousness or swallowing dysfunction. These two conditions differ in etiology—pneumonia is infectious, while pneumonitis is chemical—but both can lead to acute respiratory compromise. Obstructive lung diseases like asthma and emphysema (a form of chronic obstructive pulmonary disease) are frequent causes of hypercapnic respiratory failure. Asthma presents bronchospasm and reversible airway obstruction, while emphysema involves alveolar destruction and reduced elastic recoil. Acute bronchitis, although less severe, can contribute to respiratory distress, especially in vulnerable patients. Infections such as bacterial and viral pneumonia can cause severe inflammation and alveolar filling, impairing oxygen diffusion and leading to respiratory failure.

Cardiac conditions also feature prominently. Cardiogenic pulmonary edema, often secondary to myocardial infarction or cardiogenic shock, presents with dyspnea, orthopnea, and pulmonary congestion. Neurogenic and distributive shock can also result in impaired oxygen delivery and secondary respiratory failure. Pulmonary embolism, a life-threatening condition, causes sudden obstruction of the pulmonary artery, leading to ventilation-perfusion mismatch. Likewise, fat embolism, seen in long bone fractures, can produce similar effects. Pulmonary fibrosis, pneumoconiosis, and granulomatous lung diseases restrict lung expansion, leading to progressive respiratory insufficiency. Pleural effusion and pneumothorax can compromise lung volumes and shift mediastinal structures, causing acute decompensation. Neuromuscular disorders such as diaphragmatic paralysis, cervical cord injury, primary muscle disorders, and myxedema can impair respiratory muscle function and reduce ventilatory capacity. Obesity hypoventilation syndrome and obstructive sleep apnea also contribute to chronic hypoventilation and may present acutely in decompensated states. Other notable conditions include idiopathic pulmonary arterial hypertension, restrictive lung diseases like kyphoscoliosis, and central sleep apnea, often seen in neurologic disorders. Accurate differentiation among these requires integration of clinical presentation, imaging, and laboratory studies to guide appropriate management.

Prognosis

The prognosis of respiratory failure varies widely due to the broad range of underlying causes and individual patient factors. Outcomes depend on the etiology, severity of the disease, comorbid conditions, timeliness of intervention, and the patient’s overall physiological reserve. Respiratory failure can present as either hypoxemic, hypercapnic, or a combination of both, and may be acute, chronic, or acute-on-chronic in nature. Each of these classifications influences prognosis. In the United States in 2017, the in-hospital mortality rate for respiratory failure was 12%, based on a broad case definition that included all diagnosis codes for respiratory failure [2]. This general statistic reflects the variability in the severity and etiology of cases. However, when the data is stratified by specific diagnoses requiring mechanical ventilation, the outcomes differ significantly. Patients intubated for asthma exacerbation had an in-hospital mortality rate of 9.8%, which is relatively low given the generally reversible nature of asthma with proper treatment [37]. In contrast, those requiring intubation for an acute exacerbation of chronic obstructive pulmonary disease (COPD) had a much higher mortality rate of 38.3% [38]. This reflects the chronic and progressive nature of COPD, as well as the increased risk of complications in patients with longstanding respiratory compromise. Patients with pneumonia who required intubation faced an even more serious prognosis, with an in-hospital mortality rate of 48.4% [39]. This high rate is often due to severe hypoxemia, systemic infection, or sepsis, which frequently accompanies severe pneumonia. The highest mortality was observed in patients with acute respiratory distress syndrome (ARDS), who had a reported in-hospital mortality rate of 44.3% [40]. ARDS often results from severe infections, trauma, or systemic inflammation, and is associated with refractory hypoxemia and multiorgan failure. These statistics underline the importance of early recognition, appropriate treatment, and intensive monitoring. Mortality rates remain high in patients with severe respiratory failure, especially when mechanical ventilation is required. Chronic conditions, advanced age, and comorbidities further worsen outcomes.

Complications

Respiratory failure, particularly in its acute form, often leads to a wide range of complications that can worsen clinical outcomes and prolong recovery. These complications may arise directly from the underlying respiratory condition, from interventions such as mechanical ventilation, or as secondary consequences of prolonged critical illness. Pulmonary complications are common. Bronchopleural fistula, an abnormal connection between the bronchial tree and pleural space, can occur following prolonged ventilation or barotrauma and is associated with significant morbidity. Nosocomial pneumonia, especially ventilator-associated pneumonia, frequently develops in patients requiring intubation. This type of pneumonia is typically caused by multidrug-resistant organisms, complicating treatment and increasing the risk of mortality. Pneumothorax, the presence of air in the pleural space, may occur spontaneously in patients with underlying lung disease or as a complication of positive-pressure ventilation. Pulmonary embolism is another potentially fatal complication that arises from thrombus migration to the lungs, often due to prolonged immobilization and hypercoagulability. In some cases, especially with chronic or unresolved inflammation, respiratory failure may progress to pulmonary fibrosis, leading to long-term impairment in gas exchange and reduced lung compliance.

Extrapulmonary complications are also significant. Acid-base disturbances frequently accompany respiratory failure, particularly respiratory acidosis from hypoventilation. These imbalances may further affect cardiovascular and neurological function. Decreased cardiac output may result from impaired oxygenation or from mechanical interactions with the heart during positive-pressure ventilation. Gastrointestinal hemorrhage, often stress-related, can occur in critically ill patients, particularly those not receiving prophylactic therapy. Hepatic dysfunction may result from hypoxia or sepsis, and ileus can develop due to altered gut perfusion or opioid use. Infection risk increases due to invasive devices and impaired immune function. Increased intracranial pressure, although less common, may occur in patients with severe hypercapnia. Malnutrition is another concern, especially in patients with prolonged hospitalization, and it impairs wound healing and immune response. Pneumoperitoneum, typically from barotrauma, can complicate ventilatory support. Renal failure, either from sepsis, hypoperfusion, or drug toxicity, further compounds respiratory failure management. Thrombocytopenia may occur due to sepsis, medications, or disseminated intravascular coagulation. Recognition and early management of these complications are essential. Prophylactic strategies such as thromboprophylaxis, stress ulcer prevention, early mobilization, and nutritional support are critical components of care. Tailored interventions and vigilant monitoring are required to reduce the incidence and impact of both pulmonary and systemic complications in patients with respiratory failure [41][42].

Consultations

Consultation with a pulmonary specialist is often necessary in cases of respiratory failure, particularly when the etiology is unclear, the clinical course is severe, or when advanced respiratory support is required. Pulmonologists bring expertise in interpreting complex diagnostic studies, guiding ventilatory strategies, and managing refractory hypoxemia or hypercapnia. In the acute setting, early involvement of a pulmonary consultant can assist in evaluating conditions such as acute respiratory distress syndrome, severe pneumonia, pulmonary embolism, and exacerbations of chronic obstructive pulmonary disease. These specialists help determine the need for invasive versus noninvasive ventilation, optimize oxygen delivery, and adjust ventilator parameters to minimize ventilator-induced lung injury. Chronic respiratory failure often requires long-term planning, including decisions about home oxygen therapy, noninvasive ventilation support, and follow-up for progressive diseases like interstitial lung disease or pulmonary hypertension. Pulmonologists play a key role in coordinating outpatient care, ensuring appropriate use of therapies, and monitoring disease progression. In cases involving complex comorbidities such as neuromuscular weakness, obesity hypoventilation syndrome, or overlap syndromes involving sleep-disordered breathing, consultation becomes crucial to guide appropriate interventions, including sleep studies, pulmonary rehabilitation, and the use of bilevel positive airway pressure devices. Patients with suspected occupational or environmental lung disease, unexplained hypoxemia, or recurrent hospital admissions for respiratory complaints also benefit from specialist input. Pulmonary consultation is equally important when considering advanced diagnostic interventions such as bronchoscopy, lung biopsy, or when initiating therapies like immunosuppressants for inflammatory lung disease. In summary, the decision to consult pulmonary specialists should be based on the complexity, severity, and persistence of respiratory failure. Timely referral enhances diagnostic accuracy, improves management, and contributes to better patient outcomes.

Patient Education

Patient education plays a critical role in the prevention, early recognition, and management of respiratory failure. Patients should understand the symptoms that may indicate respiratory deterioration, such as increased shortness of breath, rapid breathing, cyanosis, confusion, and fatigue. Early identification of these signs and prompt medical attention can significantly improve outcomes and reduce the likelihood of complications. Education should focus heavily on medication adherence. For patients with chronic respiratory diseases such as chronic obstructive pulmonary disease (COPD) or asthma, proper use of inhalers, compliance with prescribed therapy, and awareness of the correct administration technique are essential to maintain baseline respiratory function and prevent exacerbations. Patients must be instructed to follow dosing schedules strictly and avoid abrupt discontinuation of maintenance medications, especially corticosteroids or bronchodilators. Device use education is also essential. Those using home oxygen therapy or noninvasive ventilation must be trained in equipment setup, daily usage, hygiene protocols, and troubleshooting common device problems. Ensuring patients and caregivers understand when and how to use these devices reduces hospital readmissions and supports long-term respiratory management. Risk factor modification should be emphasized. Smoking cessation is one of the most effective interventions in slowing the progression of COPD and improving respiratory function. Referral to smoking cessation programs, behavioral counseling, and pharmacotherapy can enhance success rates. Avoiding secondhand smoke, reducing exposure to environmental pollutants, and minimizing contact with infectious individuals are also important.

Patients should be encouraged to receive routine vaccinations, especially annual influenza vaccines and pneumococcal vaccines, to reduce the risk of respiratory infections that can trigger acute respiratory failure. Participation in pulmonary rehabilitation programs helps improve exercise tolerance, reduce dyspnea, and enhance quality of life in those with chronic respiratory disease. Patients should also understand that while many causes of respiratory failure are preventable or manageable, some conditions are progressive or acute without warning. Therefore, individuals with risk factors or chronic respiratory diseases must be vigilant about changes in symptoms. Educating patients to seek timely evaluation when symptoms worsen supports earlier intervention and improved survival. Effective patient education requires a clear explanation of the disease process, individualized instruction, and reinforcement during follow-up visits. Empowering patients with knowledge and tools for self-management strengthens their role in maintaining respiratory health and preventing deterioration.

Enhancing Healthcare Team Outcomes

Managing respiratory failure requires coordinated care among a multidisciplinary healthcare team. The condition often stems from complex and overlapping pulmonary and extrapulmonary causes. Therefore, the involvement of various medical specialists is essential. Physicians from cardiology, neurology, infectious disease, and other relevant fields should be consulted when the clinical picture is unclear or when comorbid conditions may contribute to respiratory compromise. Radiologists also play a role in evaluating imaging studies, offering critical input that may influence diagnosis and management plans. Complications in respiratory failure can arise from factors beyond the disease itself. For example, improper patient positioning can lead to pressure ulcers, aspiration, or worsened pulmonary function. Nurses are central to preventing these complications. They monitor vital signs, manage feeding, provide suctioning, and ensure correct positioning, all while maintaining patient comfort. They also serve as a communication bridge between patients, families, and healthcare providers. By delivering timely updates and relaying changes in condition, nurses contribute directly to timely clinical interventions. Nurses also enforce infection control policies. Adherence to hand hygiene, use of personal protective equipment, and maintenance of sterile procedures in interventions such as suctioning or central line access all reduce hospital-acquired infections. Nurses are often responsible for educating patients and families on infection prevention practices at home, especially when discharge planning involves home care. Pharmacists ensure medication safety, particularly in patients with polypharmacy. They perform medication reconciliation during transitions of care, identify potential drug interactions, and advise on dosage adjustments, especially when organ dysfunction is present. Pharmacists also counsel patients on the proper use of inhalers, nebulizers, and other respiratory-related therapies. Their expertise improves medication adherence and minimizes adverse effects, which can be particularly dangerous in critically ill patients.

Respiratory therapists are key in managing ventilator settings, administering bronchodilators, performing chest physiotherapy, and educating patients on breathing exercises. Their assessments of respiratory patterns and airway clearance contribute to ventilator weaning protocols and help prevent ventilator-associated complications. Communication among all team members must be continuous and transparent. Shared access to patient records ensures that all disciplines operate with up-to-date information. Interprofessional rounding and documentation improve coordination and reduce the likelihood of miscommunication or errors. Any change in patient status should prompt immediate communication with the appropriate team member to initiate corrective action. A collaborative approach improves diagnostic accuracy, reduces complications, enhances patient safety, and shortens hospital stays. Each discipline brings unique skills, and when these are integrated effectively, the team can provide higher-quality, patient-centered care.

Conclusion:

Respiratory failure remains a life-threatening syndrome requiring prompt recognition and intervention to prevent fatal outcomes. This review highlights the critical distinctions between Type 1 (hypoxemic) and Type 2 (hypercapnic) respiratory failure, emphasizing their underlying pathophysiology, diagnostic approaches, and management strategies. Type 1 respiratory failure, marked by PaO_2 <60 mmHg, commonly results from V/Q mismatch, diffusion impairment, or shunting, as seen in ARDS, pneumonia, or pulmonary embolism. Diagnostic tools such as ABG analysis, imaging, and capnometry help differentiate these mechanisms. Treatment focuses on correcting hypoxemia through supplemental oxygen, high-flow nasal cannula, or mechanical ventilation while addressing the primary cause. In contrast, Type 2 respiratory failure ($PaCO_2$ >45 mmHg) stems from alveolar hypoventilation due to reduced respiratory drive (e.g., opioid toxicity), neuromuscular weakness (e.g., Guillain-Barré syndrome), or mechanical limitations (e.g., COPD exacerbation). Management includes NIV or invasive ventilation to restore ventilation and reverse acidosis. The prognosis of respiratory failure varies significantly based on etiology. While asthma-related respiratory failure has a lower mortality (~9.8%), COPD exacerbations and pneumonia carry higher mortality rates (38.3% and 48.4%, respectively). ARDS, often complicating sepsis or trauma, remains particularly lethal (44.3%). Early intervention, including lung-protective ventilation and prone positioning in ARDS, improves survival. A multidisciplinary approach is essential, integrating pulmonologists, intensivists, nurses, and respiratory therapists to optimize care. Complications such as ventilator-associated pneumonia, barotrauma, and thromboembolism necessitate vigilant monitoring and preventive strategies. Pharmacists play a key role in medication safety, while rehabilitation specialists aid recovery in chronic cases. Patient education is vital for long-term management, particularly in chronic conditions like COPD. Emphasis on smoking cessation, vaccination, and adherence to inhaler therapy reduces exacerbations. Home oxygen and NIV protocols should be clearly communicated to patients and caregivers to prevent readmissions. In summary, respiratory failure demands a systematic, evidence-based approach tailored to its type and etiology. Advances in diagnostic tools and ventilatory strategies have improved outcomes, but early detection and interdisciplinary collaboration remain paramount. Future research should focus on personalized therapies and innovations in noninvasive support to further reduce morbidity and mortality.

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فشل الجهاز التنفسي لدى البالغين: مراجعة محدثة موجهة لأخصائي الرعاية الصحية

الملخص

الخلفية: يُعد فشل الجهاز التنفسي حالة حرجة تتمثل في عجز الجهاز التنفسي عن الحفاظ على الأكسجة الكافية (النوع الأول) أو التخلص من ثاني أكسيد الكربون (النوع الثاني). وقد يكون حاداً أو مزمناً أو حاداً على مزمن، وتختلف أسبابه وتشمل اضطرابات رئوية، قلبية، عصبية عضلية، أو جهازية. الكشف المبكر والتدخل السريع ضروريان لتفادي مضاعفات مهددة للحياة.

الهدف: تقدم هذه المراجعة نهجاً سريرياً محدثاً لتشخيص وعلاج فشل الجهاز التنفسي لدى البالغين، مع التركيز على التعرف المبكر، الفسيولوجيا المرضية، واستراتيجيات العلاج المبنية على الأدلة.

الطرق: يتطلب التقييم الشامل لفشل الجهاز التنفسي أخذ التاريخ المرضي والفحص السريري وتحليل غازات الدم الشرياني (ABG) والتصوير (أشعة الصدر، الأشعة المقطعية)، وقياس ثاني أكسيد الكربون(Capnometry) ، والتصوير بالموجات فوق الصوتية في نقطة الرعاية. وتُستخدم الفحوصات المخبرية وتخطيط القلب الكهربائي والفحوصات المتخصصة (مثل تنظير القصبات ودراسة النوم) حسب الحاجة.

النتائج: ينتج فشل الجهاز التنفسي من النوع الأول (نقص الأكسجة) عن ضعف في انتشار الأكسجين أو اضطراب التهوية/التروية أو التحويل(Shunt) ، بينما ينجم النوع الثاني (فرط ثاني أكسيد الكربون) عن انخفاض التهوية السنخية بسبب أسباب مركزية أو عصبية عضلية أو ميكانيكية. يشمل العلاج إعطاء الأكسجين، التهوية غير الغازية(NIV) ، أو التهوية الميكانيكية الغازية حسب شدة الحالة. تختلف معدلات الوفاة حسب السبب، إذ تصل إلى 44.3٪ في متلازمة الضائقة التنفسية الحادة (ARDS)و48.4٪ في حالات الالتهاب الرئوي التي تتطلب تنبيباً.

الاستنتاج: التشخيص المبكر والتدخل العلاجي المخصص يُحسنان النتائج. ويُعد التعاون متعدد التخصصات بمشاركة أطباء الرئة، وأطباء العناية المركزة، والممرضين، وأخصائي العلاج التنفسي ضرورياً. كما يُسهم تثقيف المرضى حول الالتزام الدوائي، والتطعيم، والإقلاع عن التدخين في تقليل مخاطر التفاقم.

الكلمات المفتاحية: فشل الجهاز التنفسي، نقص الأكسجة، فرط ثاني أكسيد الكربون، التهوية الميكانيكية، متلازمة الضائقة التنفسية الحادة، مرض الانسداد الرئوي المزمن، الأكسجة، تحليل غازات الدم الشرياني.