# *Acute Respiratory Failure*

***2.0 Contact Hours***

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# Acute Respiratory Failure

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## Objectives:

At the completion of this course, the learner will be able to:

1. Identify structures that make up the respiratory components.
2. Recognize conditions that cause dysfunction of the respiratory components.
3. Identify signs and symptoms of ARF.
4. Recognize proper ventilatory care of the patient with ARF.

## The Grand Picture

Acute Respiratory Failure (ARF) is a common diagnosis in the CCU with a mortality rate of 50%. ARF occurs when one or both of the prime functions of the respiratory system fail suddenly in the absence of long-term pulmonary disease. A sufficient amount of oxygen fails to diffuse into the blood (hypoxia), carbon dioxide builds up instead of being exchanged and vented into the atmosphere (hypercarbia), perfusion of tissues fails to provide enough oxygen to the brain and vital organs, and death occurs. V/Q mismatch, shunting, and hypoventilation from trauma or other conditions cause ARF. ARF occurs rapidly due to dysfunction in any one of five critical areas of the respiratory components. Let's look at the acute problems in these areas that can result in ARF.

## Respiratory Components:

### The Nervous System

The medulla oblongata is a mass of neurons on the lower end of the brainstem and at the top of the spinal cord. It lies above the foramen magnum and ends at the lower end of the pons. It has both white and gray matter. The white matter consists of tracts that conduct both ascending and descending signals. Gray matter in the medulla contains areas where several neurons join together to receive impulses from one another called nuclei groups. One of these nuclei groups is the cardiovascular center that regulates the heart beat. Another is the medullary rhythmicity area which controls the rhythm of respirations. Other nuclei groups control coughing and sneezing, as well as swallowing, hiccupping, and vomiting. The inspiratory part of the medullary rhythmicity area sends signals for 2 seconds to the external intercostal muscles and diaphragm to contract. When they do, inspiration occurs. Then the signal ends for 3 seconds, the muscles relax and exhalation occurs. If natural exhalation does not suffice, the expiratory area sends signal to the internal intercostal and abdominal muscles to contract, forcing out a larger amount of air. The medulla receives other signals from its own chemoreceptors and those

in the aortic and carotid areas that notify it when $O_2$, $CO_2$ and $H^+$ are abnormal so that the respiration rate can be altered to compensate.

The pons lies just above the medulla oblongata and also contains white matter with tracts and gray matter with nuclei groups. Two of these nuclei groups, the pneumotaxic area and the apneustic areas, work together with the medulla to control respirations. The pneumotaxic area sends impulses to the medulla to stop inhalation before the lungs fill with too much air. The apneustic area signals the medulla to prolong the signals so that deeper breaths occur.

The Hypothalamus and limbic system of the brain can cause changes in respiration during emotional moments. The cerebral cortex offers voluntary control of respirations so that we can hold our breath or hyperventilate at will. When unconsciousness occurs, the medulla takes over and breathing returns to normal.

Trauma to the back of the head or the upper neck can damage the medulla and pons resulting in ARF. Increased intracranial pressure can also result in pressure forcing these areas downwards until ischemia occurs and breathing ceases. Brain trauma, CVA, hypothermia after surgery, and depression of the CNS from an overdose in drugs or poisoning also causes ARF. When ARF occurs as a result of **controller dysfunction**, it is called central apnea. Patients will exhibit respiratory rates of less than 12 breaths per minute.

**The Bones and Muscles of the Chest and Abdomen**

Integrity of the thorax is necessary for normal respiration. The clavicles, ribs, sternum, and spine offer the framework that keeps the chest from collapsing and causing dyspnea. Trauma to bones can cause lung punctures and a pneumothorax to occur. While fractures of these bony areas do not result in ARF per se, respiratory muscle exhaustion can occur from the increased work of breathing.

The critical muscle of respiration is the diaphragm. The diaphragm lies in the inferior aspect of the thorax and is shaped like a dome when at rest. It receives impulses from the medulla via the phrenic nerves. Contraction of the diaphragm causes the dome to collapse downwards, raising the lower ribs upwards. This increases volume in the thorax, lowering pressure within the lungs below that of atmospheric pressure, causing air to rush in. When the diaphragm relaxes and rises, pressure within is increased and air rushes out of the lungs during expiration. About 500 ml of air is inhaled with each inspiration. Pressure from clothing, fat, or a gravid uterus can impinge upon the diaphragm and decrease the depth of inspiration.

The external intercostal muscles work in concert with the diaphragm to increase the size of the thorax. They are 25% responsible for the decrease in pressure within the lungs; the diaphragm decreases pressure 75%. During contraction of the external intercostal muscles, the ribs move upwards and the sternum moves forward. Relaxation reverses this action, contributing to passive exhalation. The sternocleidomastoid and scalene muscles can be used during dyspnea to assist during inspiration to help raise the ribs.

Exhalation is normally passive due to recoil of the lungs, but when directed by the expiratory area of the medulla the internal intercostal muscles and the abdominal muscles contract to lower the ribs to force more air out. The external oblique, internal oblique, transversus abdominis, and rectus abdominis are the abdominal muscles that assist forced exhalation in the abdomen by contracting and forcing the diaphragm upwards and the ribs downwards.

The phrenic nerves exit the spinal cord at C3, 4, and 5. Spinal cord injury above this level will result in paralysis of the muscles of respiration, resulting in ARF. The intercostal nerves exit between vertebrae T1 to T11 and are affected by spinal cord injury in that area. Electrolyte problems, toxins, poliomyelitis and drugs can affect the muscles and nerves. Other neurological and muscular injuries can also cause ARF. ARF that is caused by failure of the muscles of respiration is said to be caused by **pump dysfunction**. The patient will exhibit paradoxical respiratory motions or no respirations at all if paralysis is present.

**The Airways**

The airways include the nose and the pharynx, larynx, trachea, bronchi, and bronchioles. The nasal airway is kept open by its cartilage and bone composition. The larynx has open cartilage rings, muscles and ligaments and is the smallest part of the adult airway. The Cricoid cartilage is the only closed cartilage ring and is the smallest part of a child's airway. The trachea below these structures has open cartilage rings and branches off into the right and left bronchus. The bronchioles do not have cartilage; they consist of smooth muscles instead.

Any portion of the airway can become blocked. Foreign bodies or mucous can lodge in any of the airways. Swelling of tissues of the nose, septal deviation, and a fractured nose can close this portion. Swelling of the tongue or larynx during an anaphylactic reaction or trauma to the trachea can block the airway at this level. Bronchospasm from noxious fumes or an acute asthmatic attack can close the bronchioles. Blockage of the airways causes ARF via **airway system dysfunction.** The patient's lung sounds will exhibit wheezing, stridor, or rhonchi and resistance to ventilatory efforts.

**The Alveoli**

Type I and Type II epithelial cells are found in the walls of the alveoli. Type II cells exude fluid containing surfactant which keeps alveoli from collapsing with each exhalation, enclose inhaled particles, and detoxify toxic gases. Type I cells are responsible for allowing the diffusion of oxygen into the blood stream and carbon dioxide into the lungs through the epithelial and capillary basement membranes. Blockage of these respiratory membranes by vomitus, blood, pneumonia, pulmonary edema, or particulate matter will prevent proper exchange of gases and cause an intrapulmonary shunt. Adult Respiratory Distress Syndrome (ARDS) occurs as fluid fills alveoli secondary to receipt of multiple transfusions, sepsis, trauma, pancreatitis, or drug overdose. Inhalation of toxic gas can damage the Type I cells restricting diffusion of $O_2$ and $CO_2$. ARF resulting from these problems is due to **alveolar compartment dysfunction**. The

patient will exhibit abnormal blood gases, chest x-ray consolidation, dullness to percussion over the involved area, and decreased lung sounds. Resistance to ventilation is also present.

**Pulmonary Blood Flow**

Blood supply to the lungs occurs through the pulmonary and bronchial arteries. The pulmonary arteries and capillaries bring deoxygenated blood to the alveoli for the exchange of carbon dioxide for oxygen, and then return to the heart via the pulmonary veins. The bronchial arteries supply the bronchi and bronchioles with oxygen. About 60 to 140 ml of blood is normally in contact with the alveoli at any given time. Pulmonary arteries will constrict in areas of the lungs that are damaged, diverting the blood to undamaged portions of the lungs (ventilation-perfusion coupling). When there is a lot of damaged lung tissue there will be major vasoconstriction throughout the pulmonary vasculature resulting in pulmonary hypertension. ARF occurs quickly in those with cardiogenic, septic, or hypovolemic shock. A pulmonary embolus, fat embolus, or intracardiac shunt can also cause ARF from **pulmonary vascular dysfunction**. The patient may exhibit sudden hypoxemia, with or without activity. Blood gases will be abnormal.

## Signs and Symptoms

ARF can be distinguished from chronic respiratory failure (CRF) by its sudden occurrence within a few short minutes or hours rather then gradually over a period of days. Look to the patient history for any of the conditions that can interfere with the respiratory components listed above. Blood gases show a pH of < 7.3 in ARF, whereas renal compensation will cause the blood pH in CRF to be decreased a very small amount.

Possible signs and symptoms depend on the cause and can include:

- pH< 7.3
- $PaO_2$ < 60 mm Hg
- $PaCO_2$ >50 mm Hg
- Apnea or dyspnea that occurs in minutes to within a few hours
- Flaring of nostrils
- Pursed-lip breathing
- Use of accessory muscles of respiration
- Cyanosis
- Abnormal respiration rate, depth, rhythm
- Increased pulmonary secretions
- Anxiety
- Seizures and myoclonus with severe hypoxia
- Coma
- Increased length of expiration
- Wheezing, stridor, or rhonchi
- Decreased breath sounds with dullness to percussion over areas of pulmonary edema

- Egophony (the patient says the letter "e" which sounds like "a" during auscultation of the chest)
- Hyperresonance if pneumothorax is present
- Signs of hypercapnea and acidosis: headache, vertigo, restlessness, irritability problems with concentration, confusion, sleepiness, tachycardia, heart arrhythmias
- Recent chest pain indicative of MI
- Abdominal pain

## Diagnostic Studies and Procedures

Often, the rapid progression of ARF will necessitate treatment prior to the diagnostic workup. Once oxygenation has improved, the cause of the ARF will be determined as quickly as possible. Diagnostic studies and procedures can include:

- Arterial blood gases and oximetry
- TSH, CBC, electrolytes
- C & S of pulmonary secretions
- Lung and heart auscultation
- Chest x-rays
- CT of the chest and head with contrast
- MRI of head
- Toxicology screen
- Ventilation-perfusion scanning
- Shunt studies
- Core body temperature measurement
- Bronchoscopy
- Electrocardiogram
- Echocardiogram with bubble study
- Measurement of vital capacity
- Measurement of inspiratory force
- Transdiaphragmatic pressure measurements
- Electromyography
- Nerve conduction studies
- Airway resistance measurement
- Measurement of lung and chest wall stiffness
- Jugular venous pressure measurement
- Right heart catheterization
- Diaphragmatic ultrasound or fluoroscopy

## Medical Treatment

Medical treatment will depend on the cause of the ARF. The airway will first be secured. A basic head tilt-chin lift can open the airway and a bag-and-mask used to provide ventilation. If the airway remains occluded, a Heimlich maneuver is attempted. If this fails to dislodge a foreign body, laryngoscopy with suctioning and removal of a foreign body with forceps is attempted. A tracheostomy or cricothyrotomy is performed as needed. A bronchoscopy may be needed if a foreign body is deep in the lungs.

When the airway is patent, an endotracheal tube is usually inserted and mechanical ventilation is begun. Analgesia and sedation may be required. Ventilation may also be given via non-invasive methods such as BiPAP or CPAP.

Central and peripheral line placements, vascular monitoring, urinary catheterization, chest tube placement, nasogastric, percutaneous endoscopic gastrostomy (PEG), or small-bore feeding tubes may be inserted as needed.

Medications are given depending on the cause of the ARF:
Antibiotics
Anti-inflammatory or immunosuppressive drugs
Antiallergy medications
Bronchodilators
Anticoagulants or thrombolytics
Diuretics
Adrenocorticosteroids

## Nursing Care

The patient is usually on a ventilator with an endotracheal tube, but may have noninvasive BiPAP or CPAP. Hospital policies and procedures will guide frequency of patient monitoring. Interventions will include:

- An endotracheal tube will need to be secured according to your hospital's procedure. Record the level of the tube. Check for breath sounds on each side to ensure it is placed properly. Check security of the tube frequently as you give care to be sure no dislodgement occurs. Tubing should be adequately supported.
- Check endotracheal tube cuff pressure and keep as low as possible to prevent tracheal erosion.
- Be sure the tube is centered down through the nares or oropharynx. If the tube is off to one side of the mouth, change sides if ordered every 8 to 12 hours to prevent damage to vocal cords. Otherwise avoid manipulating the tube to prevent damage to nares, oral mucosa, larynx, and trachea.
- Check skin and mucosa frequently for pressure or tissue breakdown from the tube or tape.

- Provide humidification and change according to policy to prevent infection. Empty water in the ventilation tubing as needed. Work with Respiratory Therapists to maintain settings and sterility of the system.
- Record vital signs, most recent blood gases, and ventilator settings hourly and as needed. Assess respirations to see if there is synchrony with the ventilator.
- Give excellent and frequent mouth and dental care to prevent ventilator-associated pneumonia.
- Suction as needed using strict sterile technique. Hyperventilate prior to suctioning with 100% oxygen for 3-4 breathes. Limit suctioning to 15 seconds or less. Watch for arrhythmias during and after suctioning. Examine sputum for color, consistency, and amount and record.
- Turn the patient every 1 to 2 hours. Perform chest physiotherapy and postural drainage as needed to maximize ventilation/perfusion. Position the patient with head of the bed up 30-45 degrees to prevent aspiration.
- Watch for ventilation complications such as pneumothorax, pneumomediastinum, and subcutaneous emphysema from ruptured alveoli.
- Monitor for signs of barotrauma: increased airway peak pressure, decreased respirations on the affected side, cyanosis, and restlessness.
- Monitor for atelectasis: decreased lung sounds, crackles, or rales.
- Assess for deep vein thrombosis. Maintain compression stockings. Give anticoagulants and thrombolytics as ordered.
- Monitor for decreased cardiac output: pulse rate changes, decreased output, and decreased blood pressure.
- Foster rest and sleep patterns.

**Fluid balance:**

- Maintain strict I & O.
- Monitor daily weight.
- Administer IV fluids as ordered and maintain sites.
- Monitor urine output hourly via catheter.
- Assess for dehydration in skin turgor, mucous membranes, and sputum viscosity.
- Monitor hemoglobin and hematocrit.

**Nutrition:**

- The patient may have a nasogastric tube in place. Record gastric secretion color and amount. Monitor for sign of bleeding from stomach ulcers that can occur with ventilation therapy. Give antacids, sucralfate, or histamine-2 receptor antagonists as ordered. Monitor stools for occult blood as well.
- If there is no NG tube, monitor for nausea and vomiting.
- Administer TPN via central line or enteral feedings via nasogastric tube or percutaneous gastrostomy (PEG) tube as ordered. Maintain PEG site according to procedure.

**Medications:**

- Use a reliable scale to measure anesthesia or sedation. Administer benzodiazepines, paralytics, or anesthetics as ordered.
- Monitor for pain and medicate with narcotics as needed
- Give other medications as ordered.

**Psychosocial:**

- Provide a communication board for the patient to communicate needs.
- Give assurance, encouragement, empathy, and support as needed.
- Allow family to ask questions; teach as needed. Offer empathy and an outlet for emotions. Refer to social services as necessary for support, especially for end-of-life decisions.

The patient with ARF will need ventilatory support, proper treatment for the particular respiratory component that is causing the ARF and intensive care by nursing to survive.

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