Continuing Medical Education - News & Information

April 2010 - Volume 16, Issue 4 **Multi-Agency Edition**

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Inside this issue:
(bold = new content)

Journal CME Newsletter

Published monthly

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From the Editor

New Mandatory REMAC Credentialing Fee

A new $25 fee has been instituted by NYC REMAC for all new or recertifying paramedic credentials. On successfully completing a REMAC exam, candidates will receive a temporary letter verifying certification. They will soon after be mailed a memo directly from NYC REMSCO requiring a completed application, proof of NY State paramedic certification, and credentialing fee by money order only. On receipt, a permanent NYC REMAC certification card7 will be issued.

Please direct inquires on this process to NYC REMSCO at 212-870-2301

Important Change to Protocol Updates

A new protocol update schedule has been adopted for both the field and the certification process. Rollouts now take place only once per year. The final version will be published January 1, beginning a three month training period. The new protocols are then implemented for all agencies on April 1.

During January, February and March, only the *prior* version is in effect, not the new April protocol changes. Only on April 1 will the new version be available for use in the field and on certification exams.

Exceptions make take place when it is urgent that a specific life-saving treatment be available right away. In such a case, the change would be implemented on a selected date for both the field and REMAC exams.

Always see **nycremsco.org** for the current approved protocols.

REMEMBER: the protocols on the street are the protocols on the exam!

Effective April 1, 2010, NYC REMAC protocol revisions are to be implemented by paramedics updated by their Medical Director.

Per REMAC, ambulance services in NYC are responsible to provide copies of the protocols to their personnel. REMAC Advisories and Protocols are available to all at www.nycremsco.org

After April 1, only the April 2010 protocols may be used in the field and on NYC REMAC exams.

Questions may be referred to the REMAC Liaison at swansoc@fdny.nyc.gov or 718-999-2671.

Outline of April 2010 NYC REMAC protocol changes

see REMAC Advisory 2010-01 at nycremsco.org:

General Operating Procedures

- Oxygen Admin: removes respiratory rate as criterion for ventilation; removes mouth-to-mouth & mouth-to-nose ventilation
- Prehospital sedation: adds etomidate for cardioversion and pacing
- Communication with Medical Control: removes 20 minute on-scene time limit

BLS Protocols

- 401 Resp Distress: removes respiratory rate as criterion for ventilation; removes mouth-to-mouth & mouth-to-nose ventilation
- 407 Wheezing: adds epinephrine under Standing Orders with repeat Medical Control Option
- 410 Anaphylaxis: changes initial epinephrine dose to Standing Orders
- 421 Head & Spine Injuries: clarifies criteria for immobilization
- 423 Chest Injuries: removes bulky dressings for flail segments
- 425 Bone & Joint Injuries: note to request ALS for pain management; clarifies traction splint for closed injuries
- 428 Burns: note to request ALS for pain management; clarifies bandaging by BSA
- 430 EDP: note to request ALS for sedation
- 431 Heat-related Emergencies: removes saline PO

ALS Protocols

- 500-A Smoke Inhalation & 500-B Cyanide Exposure: clarifies sodium thiosulfate preparation
- 502 Obstructed Airway: removes needle cricothyroidotomy; adds procedure for right-mainstem bronchus displacement
- 503 Non-traumatic Arrest: removes reference to paddles
- 503-A V-fib/V-tach: changes joule setting
- 503-B PEA/Asystole: adds dextrose administration
- 504 Suspected MI: adds prompt OLMC contact; changes transport prior to IV admin
- 505-A, B & C Dysrhythmias: removes biphasic
- 505-D Brady Dysrhythmias: removes epi drip
- 506 APE: changes furosemide to Medical Control Option
- 510 Anaphylaxis: removes epi drip
- 521 Head Injuries: clarifies use of hyperventilation
- 540 Severe Pre-Eclampsia/Eclampsia: renames protocol; removes treatment for post-partum hemorrhage
- 551 Peds Obstructed Airway: removes needle cricothyroidotomy; adds procedure for right-mainstem bronchus displacement
- 554 Peds Asthma: clarifies ipratropium use
- 555 Peds Anaphylaxis: removes epi drip

Appendices

Appendix N Needle Cricothyroidotomy: deleted

REMAC Exam Study Tips

REMAC candidates have difficulty with:
* Epinephrine use for peds patients
* 12-lead EKG interpretation
* ventilation rates for peds & neonates

REMAC Written exams are approximately:

15% Protocol GOP	40% Adult Med. Emerg.
10% BLS	10% Adult Trauma
10% Adult Arrest	15% Pediatrics

Certification & CME Information

- *Of the 36 hours of Physician Directed Call Review CME required for REMAC Refresher recertification, at least 18 hours must be ACR/PCR Review (which may include QA/QI Review). The remaining 18 hours may include ED Teaching Rounds and OLMC Rotation.*
- **Failure to maintain a valid NYS EMT-P card will invalidate your REMAC certification.**
- **By the day of their refresher exam all candidates must present a letter from their Medical Director verifying fulfillment of CME requirements. Failure to do so will prevent recertification.**
- **FDNY paramedics, see your ALS coordinator or Division Medical Director for CME letters.**
- **CME letters must indicate the proper number of hours, per REMAC Advisory # 2000-03:**
  - 36 hours - Physician Directed Call Review
    - *ACR Review, QA/I Session* (**minimum 18 hours of ACR/QA review**)
    - *Emergency Department Teaching Rounds, OLMC Rotation*
  - 36 hours - Alternative Source CME - **Maximum of 12 hours per venue**
    - *Online CME*
    - *Lectures / Symposiums / Conferences*
    - *Journal CME*
    - *Clinical rotations*
    - *Associated Certifications: BCLS / ACLS / PALS / NALS / PHTLS*

REMAC Refresher Written examinations are held monthly, and may be attended up to 6 months before your expiration date. See the exam calendar at the end of this Journal. To register, call the Registration Hotline @ 718-999-7074 by the last day of the month prior to your exam.

REMAC Quarterly Written and Oral examinations are held every January, April, July & October. Registration is limited to the first 50 applicants. See the exam calendar at the end of this journal.

REMAC CME and Protocol information is available, and suggestions or questions about the newsletter are welcome. Call 718-999-2671 or email swansoc@fdny.nyc.gov

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REMSCO: www.NYCREMSCO.org
NYS/DOH: www.Health.State.NY.US

Online CME: www.EMS-CE.com www.MedicEd.com www.EMCert.com www.WebCME.com www.EMINET.com

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  Andrea Katsanakos
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  Matthew Rightmeyer & Gary Simmonds
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  Mike Romps
- **Division 5 718-979-7175**
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  Director of Prehospital Research
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  Associate Medical Director of Training
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Acosta, Juan	**80286**	**Huie, Frederick**	**80300**
Alexandrou, Nikolaos	**80282**	**Isaacs, Doug**	**80299**
Asaeda, Glenn	**80276**	**Jacobowitz, Susan**	**80297**
Barbara, Paul	**80306**	**Kaufman, Bradley**	**80289**
Ben-Eli, David	**80298**	**Lombardi, Gary**	**80225**
Cordi, Heidi	**80279**	**McIntosh, Barbara**	**80246**
Cox, Lincoln	**80305**	**Munjal, Kevin**	**80308**
Freese, John	**80293**	**Pascual, Jay**	**80287**
Giordano, Lorraine	**80243**	**Safford, Mark**	**80307**
Gonzalez, Dario	**80256**	**Schenker, Josef**	**80296**
Hansard, Paul	**80226**	**Schoenwetter, David**	**80304**
Hegde, Hradaya	**80262**	**Schneitzer, Leila**	**80241**
Hew, Phillip	**80267**	**Silverman, Lewis**	**80249**
		Soloff, Lewis	**80302**

Respiratory Emergencies

The ability to breathe is essential to life. The lack of it is one of the leading reasons for calling 9-1-1. Respiratory call-types account for 15% of NYC 9-1-1 responses, approximately 160,000 calls per year. The Center for Disease Control (CDC) lists chronic lower respiratory disease as the fourth leading cause of death in the United States.

Much of EMS education is focused on airway management because a blocked or unstable airway is life threatening and needs to be addressed immediately. However, the majority of cases of dyspnea are caused by problems with the respiratory system itself. It is for that reason that this month, we have chosen to focus the journal article on respiratory emergencies.

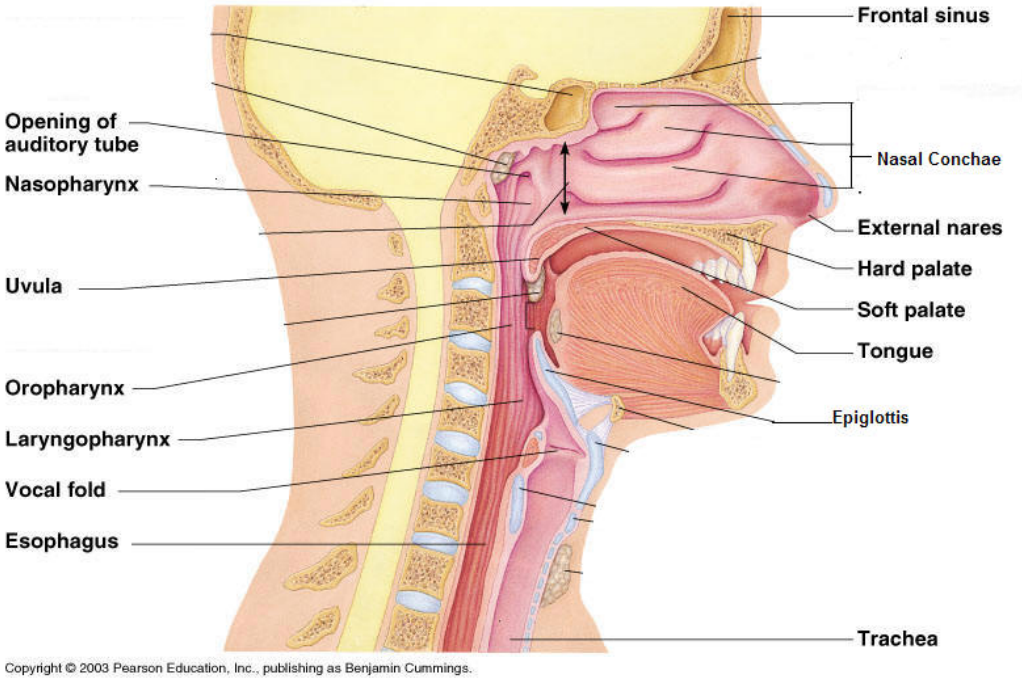
Clearly there is an important overlap between respiratory and cardiac causes of shortness of breath, most notably the commonplace disease known as Congestive Heart Failure. In this journal, we will try to avoid in depth discussion of the cardiogenic causes of dyspnea to allow for emphasis on respiratory system anatomy, pathophysiology and prehospital management and treatment.

ANATOMY

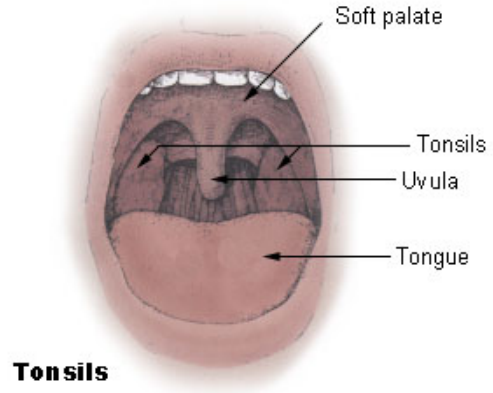
The anatomy of the respiratory system can be divided into two major segments: the upper airway and the lower airway. The upper airway consists of the oral and nasal cavities, the pharynx, and the larynx. The lower airway consists of the trachea, the bronchial tree, and the alveoli. We shall review the anatomy sequentially as we track the flow of air.

The Upper Airway

Air enters the respiratory system primarily via the two nares (nostrils) which are lined with tiny hairs meant to catch particulate matter before it enters and damages the lung. As it flows through the nasal passage (nasal cavity), it is humidified and warmed by the highly vascular mucosa.

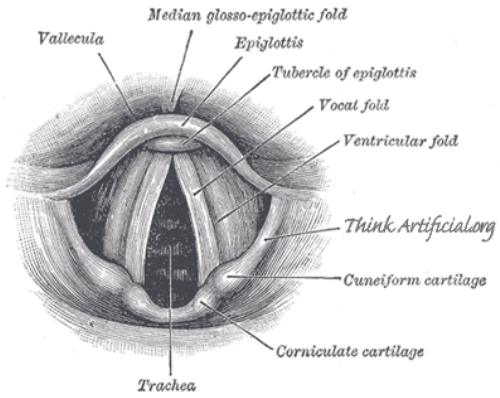


Air may also enter via the mouth (oral cavity) where it is similarly warmed and humidified but it does not get filtered as well. Upon direct visual inspection of the oral cavity, we notice that it is separated from the nasal cavity by the hard palate (bone) anteriorly and the soft palate (cartilage) posteriorly. Hanging down from the posterior edge of the soft palate in the midline is a piece of connective tissue known as the uvula. On both sides, we can visualize the palatine tonsils, and posterior to the tonsils and uvula, we can visualize a portion of the pharynx.



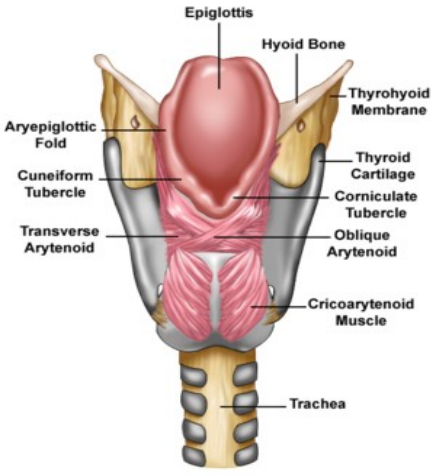
Tonsils

The pharynx is a muscular membranous that connects the nasal airway passages and the airway passages. It is this area that is stimulated during an intact gag reflex. Air continues to move inferiorly where it enters the larynx, or voicebox. The larynx is protected by the thyroid and cricoid cartilage externally and the epiglottis internally. Posteriorly, it is marked by the presence of the aretynoid cartilages, often the first landmark seen during laryngoscopy. In the space of the larynx lie the vocal cords, and it is here that the movement of air is manipulated to produce sound. The opening to the trachea located within the larynx and between the vocal cords is also known as the glottis.

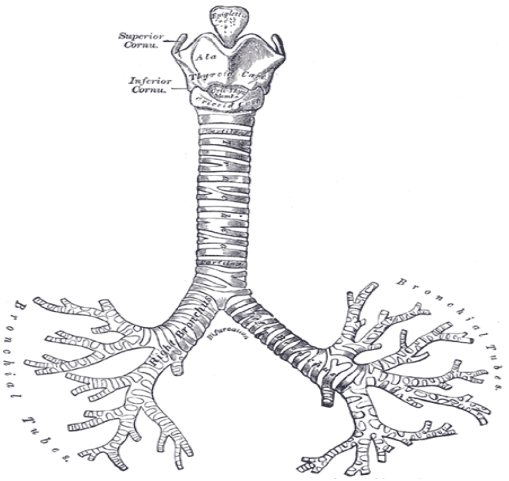


The Lower Airway

Air enters the lower airway through the glottis and into the trachea, a cylindrical tube about 10 cm long in an adult, which is kept patent by the presence of incomplete cartilaginous rings on the anterior 2/3 of the tube. At its inferior-most point known as the carina, the trachea divides into the left and right mainstem bronchi. In most patients, the right side branches off at a less acute angle which explains why endotracheal tubes that are passed too far often end up in the right mainstem bronchus.



Each bronchus further subdivides into lobar bronchi, then segmental bronchi, subsegmental bronchi and bronchioles. Collectively, this system of air tubes is known as the tracheobronchial tree and each division of the tree yields a smaller and thinner walled cartilaginous passageway for air. Beginning in the bronchi, the airway passages are lined with goblet cells that produce mucus that lines the airway, and tiny hair-like structures known as cilia which beat rhythmically to move particulate matter, fluid or debris up and out of the airway. These cilia are often damaged in smokers making it harder to clear particulate matter and more prone to infection such as pneumonia.

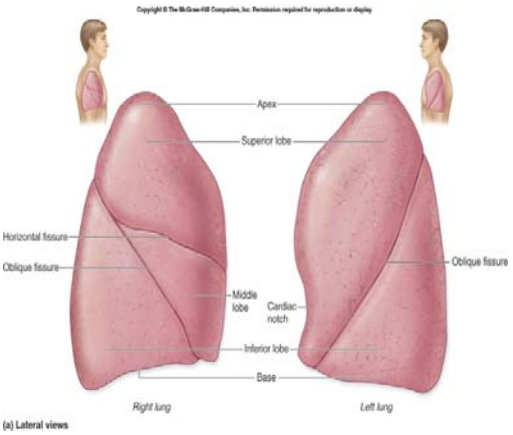


At its distal most point, the bronchial tree provides air into microscopic functional units of air exchange known as alveoli (some air can be exchanged in the terminal bronchioles). Each alveolus is lined with surfactant, a thin mucus like substance that serves to reduce surface tension thereby keeping the alveoli open. Though small, these functional units of air exchange make up the majority of the lung mass and give the lung its macroscopic spongy appearance.

The lung has fissures which divide the lung tissue into distinct lobes, each of which is supplied by a branch of the right or left mainstem bronchus. Notice in the diagram that the lungs are asymmetrical; the right has three lobes, and the left just two. The lung is covered by a serous membrane known as the visceral pleura which slides along the parietal pleura (the inner lining of the chest wall). The potential space between these two linings is the pleural space (or pleural cavity) and the presence of air, fluid, or blood represents a pneumothorax, hydrothorax, or hemothorax respectively.

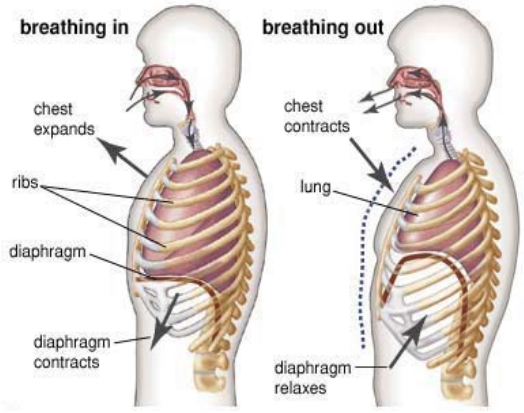
PHYSIOLOGY

Ventilation



We breathe by first creating negative intrathoracic pressure which like a vacuum, sucks air into the chest through the upper airways and down into the lung tissue (inspiration), and then creating a positive intrathoracic pressure which forces air out (expiration). The changes in pressure are made possible by downward movement of the diaphragm and expansion of the chest wall during inspiration (active process), and upward movement of the diaphram and contraction of the chest wall during expiration (passive process). The amount of air in each breath multiplied by the number of breaths per minute is our minute ventilation.

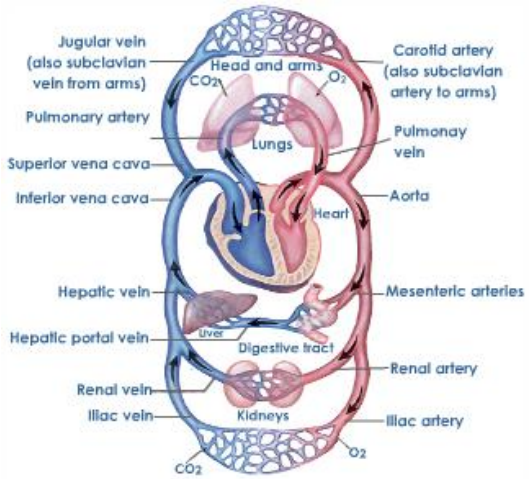
During a respiratory emergency, a patient may use additional (accessory) muscles to create even greater changes in intrathoracic pressure to move more air in and out and at faster rates to allow for greater air exchange. The rate, depth and rhythm of our ventilation is regulated by the brain which is why head trauma can cause changes to our breathing patterns.



Oxygenation

Oxygen diffuses across the membrane of the alveoli into the nearby cluster of tiny blood vessels known as capillaries while carbon dioxide diffuses across the membranes in the opposite direction. The oxygenated blood is transported by the circulatory system to all of our organs and tissues. Low levels of oxygen in our blood is known as hypoxia.

Respiration



This occurs in each of our cells. It is the process of oxygen being taken up into our cells along with glucose to produce energy allowing the cell to perform its function. The waste product of the cell's activity is carbon dioxide which then must be removed from our cells, diffuse into the blood stream and finally be exchanged for new oxygen in the lungs.

PATHOLOGY

The respiratory system has numerous interactions with other systems in our body. Thus, a respiratory complaint or abnormal breathing can be the result of an abnormal condition in the neurological, cardiovascular, musculoskeletal, or renal systems. Conversely, a respiratory system disturbance could manifest itself in one of these systems.

Anything that disturbs the function of the brain, such as head trauma, stroke, or medication could depress the part of our brain responsible for stimulating or controlling breathing. Any disruption to the spinal cord or certain peripheral nerves could result in paralysis of the diaphragm or chest wall muscles used in ventilation. And dysfunction of the respiratory system could lead to hypoxia which may manifest as confusion, loss of consciousness, or seizure.

A heart attack or failure of the heart to pump blood effectively can lead to a back up of blood causing the pulmonary circulatory system to fill with oxygenated blood. This congestion in the lung tissues could lead to leaking of fluid into the lung tissues. The shortness of breath that ensues is known as congestive heart failure. A lack of oxygen to the myocardium, the muscle of the heart could lead to chest pain secondary to ischemia.

Musculoskeletal injuries or weakness can result in an inability to generate sufficient negative intrathoracic pressures to ventilate. Lack of oxygenated blood to muscles can result in lactic acid build up, followed by weakness and cramping.

The renal system controls our fluid volume status which relates to blood pressure, our acid-base balance, and the cleanliness of our blood. Patients with renal disease may become acidemic which might stimulate tachypnea that is meant to blow off CO_2 and maintain a pH of 7.4. Patients may have difficulty removing fluid from the body thereby making them both hypertensive and prone to congestive heart failure, which may present with a respiratory complaint.

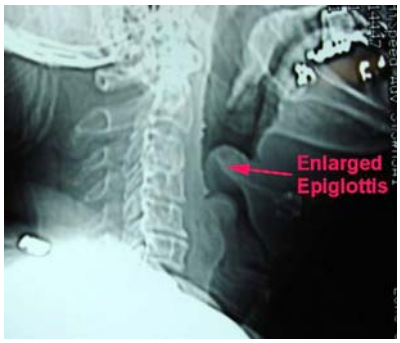
Infection

A variety of infections can result in respiratory emergencies. Beginning with the upper airway, some infections can result in a narrowing or complete occlusion resulting in respiratory distress.

Croup is a viral infection that causes inflammation and thickening of the mucosa near the glottis. A small amount of inflammation in an adult results in little more than a hoarse voice and cold symptoms. However, that same narrowing in a child, who already has a narrow airway to begin with, creates a potentially dangerous clinical condition. A child typically has a seal-like barking cough that typically occurs at night. It commonly improves with cool or warm humidified air. Avoid manipulating the airway unless absolutely necessary as this may cause further swelling and worsen the condition.

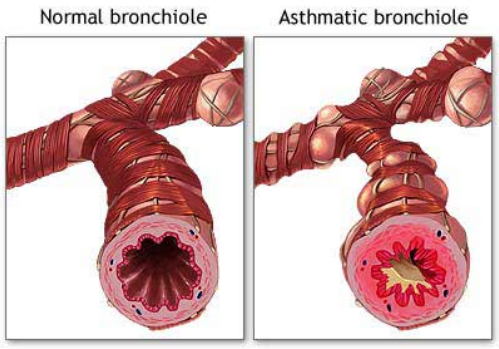
Epiglottitis is usually caused by a bacteria (*Haemophilus Influenza* type b) which has become rare due to routine vaccination. Unlike croup, it can occur at any age. Patients are usually febrile and have difficulty swallowing their own saliva due to the swelling of the epiglottis. The danger is that the increased weight of the epiglottis sometimes causes the entire structure to "flop down" and obstruct the airway resulting in stridor or respiratory arrest. If you suspect this condition, do not manipulate the airway.

Other upper airway infections such as tonsillitis, peritonsillar abscess, and retropharyngeal abscess primarily cause pain or difficulty with swallowing, but rarely can result in airway obstruction.



The most common lower airway infection is pneumonia. Pneumonia is any bacterial or viral infection of the lung tissue and can result in fever, cough, hypoxia, dyspnea, and dehydration. Upon auscultation, rales or rhonchi may be heard in one or more lung fields. Infection causes the blood vessels in the lungs to become leaky, allowing a pleural effusion to accumulate in the pleural space, the area between the lung and the chest wall. You might hear absent or decreased breath sounds with an effusion. This effusion can become infected creating an abscess in the chest. In the hospital, a patient with pneumonia usually requires antibiotics and intravenous fluids to avoid going into septic shock. An infected pleural effusion may need to be drained similarly to an abscess on the skin.

Inflammatory Disease



Asthma is a chronic inflammatory disease of the bronchial tree with acute manifestations. Usually in response to an environmenal trigger, the bronchioles become edematous, they constrict or spasm, and produce excess mucus. This can lead to mucus plugging further decreasing their ability to exchange air. Patients present with audible expiratory wheezing, tachypnea, and can proceed to hypoxia or even respiratory arrest.

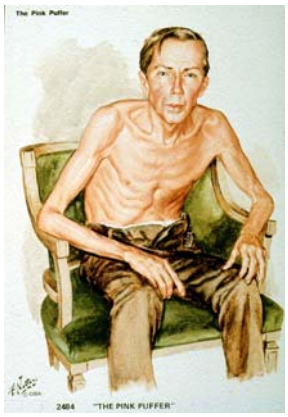
Management of an acute asthma exacerbation is focused on reversing the bronchospasm that is occuring through the use of inhaled beta agonists (albuterol) and anticholinergics (ipratropium bromide). The effect is to relax the smooth muscle cells of the bronchioles. In addition, supplemental oxygen allows for greater oxygenation of blood despite less air exchange and steroids are used to suppress the inflammatory response. In severe asthmatics, upper airways can be so constricted that the albuterol medication cannot reach the lower airway where it is most needed. In these cases, epinephrine is used because it can be delivered to the lungs via the bloodstream to achieve a similar level of smooth muscle relaxation

Bronchitis is an inflammation of the bronchi or bronchioles that results in excessive mucus production by the goblet cells which presents with a cough and occasionally compromised air exchange. Though this generally occurs over a period of weeks to months, patients with bronchitis may present with an acute shortness of breath if it acutely worsens or a mucus plug develops.

Chronic Obstructive Pulmonary Disease (COPD)

COPD is a chronic condition in which the bronchial tree is irreversibly narrowed. This can occur with years of exposure to toxic substances, polluted air, smoking or from a history of lung infections. There are two main types of COPD, chronic bronchitis and emphysema, and they often co-exist.

Chronic bronchitis results from damage and inflammation of the bronchi and bronchioles. It is similar to acute bronchitis in that the airways have excessive mucus production and are inflamed. It is characterized by a cough with productive sputum, dyspnea with expiratory wheezing and cyanosis. These patients are usually overweight and are subject to recurrent respiratory infections. These patients are classically described as "blue bloaters."



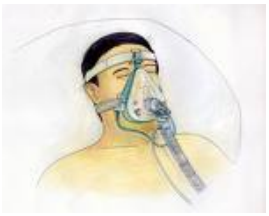
Emphysema results from damage and inflammation to the alveoli that makes it difficult to exhale and results in air trapping. It is characterized by progressively worsening shortness of breath. Instead of cyanosis, these patients get flushed cheeks due to the increased effort it takes to breathe. They also experience tachypnea, prolonged expiration, and over time, develop barrel chests. They are classically described as "pink puffers."

In both cases, decreased ability to exchange air leads to a chronic mild hypoxia and hypercarbia (elevated levels of CO_2). Patients often benefit from home oxygen therapy which allows them to extract more oxygen from the air they breathe with less work. This improves their exercise tolerance but they usually still feel short of breath. Patients are often on similar preventative medication as asthmatics to manage their chronic inflammation.

Environmental or infectious triggers can result in an acute worsening of a patient's COPD. Patients become acutely short of breath, develop severe productive coughs, and may develop profound hypoxia or hypercarbia. The hypercarbia can cause a change in mental status and eventually cause a patient to lose consciousness. During such episodes, patients not already having a respiratory infection are prone to catching one. Management includes supplemental oxygen, the use of steroids, and for severe cases, positive pressure ventilation.

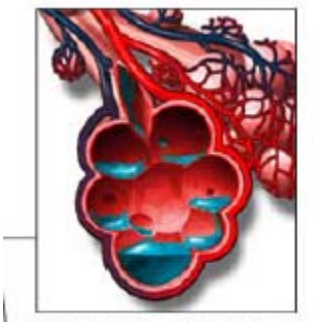
Acute Pulmonary Edema (APE)

Pulmonary edema is simply the presence of fluid in the lung tissues. It usually occurs when either the blood in the pulmonary circulation is under high pressure, the capillaries become leaky, or the ability to drain fluid is compromised. Some examples in which high pressure develops include sudden cardiac pump failure in which fluid backs up into the pulmonary veins, and pulmonary embolus in which a backup occurs in the pulmonary arteries. The capillaries can become leaky during a pneumonia, or in response to allergic or immunologic reactions that release substances like histamine. Impaired drainage might occur from trauma resulting in a pulmonary contusion. A variety of other causes include inhalation of toxins, renal failure resulting in uremia, narcotic overdoses, and eclampsia.



Acute pulmonary edema is a common and very dangerous medical emergency. Patients are profoundly short of breath, hypoxic and have rales heard in all lung fields. Management

depends upon rapid diagnosis and treatment of the underlying cause. All patients with hypoxia should receive supplemental oxygen, and these patients are no exception. In many cases, the patient will be hypertensive. The mainstay of treatment for these patients will be nitroglycerin which dilates arteries and veins throughout the body including those in the heart and lung. This will allow the pressure in those vessels to decrease thereby preventing the further pushing of fluid into the lung tissue, and in fact may reverse the flow of fluid back into the vessels. Morphine has been used in the past but it is now known that little or no blood vessel dilation occurs at normal doses. Its main benefit was likely from the reduction of anxiety, but this can be accomplished more effectively with benzodiazepines like midazolam available as medical control options.


Accumulation of fluid in the air sacs (alveoli) in the lungs

Furosemide (Lasix) was commonly used in the past to help remove fluid from the body, but its onset is relatively slow and it is possibly harmful because it can acutally cause patients to become hypovolemic. For these reasons, it has been moved from a standing order to a medical control option. Although not available currently to FDNY medics, Non-invasive Positive Pressure Ventilation (NIPPV), a general term that refers to CPAP or BIPAP, has been shown to be effective and useful in the management of both severe CHF and APE.

ASSESSMENT

Due to a patient's respiratory distress, obtaining a history of present illness or past medical history can be difficult. Initially, it is important to determine the chief complaint and stick to yes or no questions whenever possible. Try to obtain the key elements such as onset, duration, presence of chest pain, history of similar episodes, history of intubations, and any respiratory diagnoses. After initial management and stabilization, you can come back and ask for additional information.

Onset is very important with respiratory complaints. An acute onset of dyspnea is indicative of bronchospasm that occurs with asthma or anaphylaxis, or a sudden disturbance to available lung tissue for gas exchange such as a pneumothorax, pulmonary embolism, or flooding of the alveoli that occurs in APE. A gradual onset is more typical of an infectious cause or chronic disease state.

Other helpful diagnostic clues include position of comfort and exacerbating or alleviating factors. Patients with orthopnea (more difficulty breathing while laying flat) probably have an element of congestive heart failure. Patients able to lay down and speak in full sentences are likely of less severity than someone who is sitting up and only able to give one or two word answers.

Physical Exam

Begin with visual inspection. How distressed is the patient? Are they diaphoretic, cyanotic, tachypneic? Are they using accessory muscles? Inspection of the neck may reveal distention of the jugular veins (JVD) which could represent high intrathoracic pressure, possibly due to a pneumothorax or pericardial tamponade, or cardiac etiology of the dyspnea, such as CHF. It can also occur in COPD or asthma. Inspection of the extremities may reveal digital clubbing, a sign of chronic hypoxia, or pitting edema, often secondary to CHF but also found in renal disease.

Next, listen to the lung fields, comparing left and right in the anterior chest wall and in the upper and lower posterior chest. It is important to become familiar with normal lung sounds, so practice on yourself or family member. Normal lungs usually sound soft and breezy. In CHF or APE, the sound often becomes louder and with crackles (rales) representing either interstitial edema or fluid in the alveoli. A pneumonia often has a localized area with rales but may be accompanied by rhonchi (low-pitched rattle). Another common lung sound is the wheeze, often heard diffusely in asthma but could be one sided or localized with a foreign body aspiration. The most ominous breath sound is the absent sound. If localized, it may represent a pneumothorax or effusion. If diffuse, it could mean the patient is not moving enough air to ventilate and is likely to go apneic.

Of course, use your adjunct device, the pulse oximeter to help determine how well a patient is oxygenating and perfusing. Keep in mind that in certain conditions, the pulse oximeter could be misleading. For example, if a patient has been exposed to carbon monoxide (CO), the pulse oximeter is likely to mistake the CO bound to hemoglobin for an oxygen molecule bound to hemoglobin. Thus, the reading will be falsely high. Also beware that the pulse oximeter reading may lag behind your patient's clinical condition. When compared to end-tidal CO_2, we can see that a patient that is not adequately ventilating may not drop their oxygenation status until several minutes after their CO_2 began to rise.

MANAGEMENT

The approach to the patient with dyspnea is relatively straightforward. As with all patients, begin with evaluating and ensuring that the patient has a patent airway. If a patient is unconscious or unable to maintain an airway, perform BLS maneuvers to adjust the airway. Use an airway adjunct such as an oropharyngeal airway (OPA) or a nasopharyngeal airway (NPA) as needed. Use the BVM to ventilate. ALS providers can insert an advanced airway. The more challenging situation is when they are in severe distress but still have a patent airway. It is here that experience is key to determine which patients are at high risk for their airway being compromised and when to intervene. It takes more skill and determination to monitor these patients closely while treating their underlying disease and NOT INTUBATE!

Place the patient in a comfortable position and try to decrease their work of breathing. Remove restrictive clothing, have them sit up, and insist that the patient NOT WALK. Provide them with supplemental oxygen.

If the patient is wheezing, administer a bronchodilator (albuterol) and anticholinergic (ipratropium bromide). These medications rarely hurt patients so we use them aggressively in the field.

Try to determine if the patient is fluid hypervolemic (fluid overloaded), euvolemic, or hypovolemic (dehydrated). This may impact on your choice of ALS intervention such as nitroglycerin and Lasix for the hypervolemic CHF patient versus fluid bolus for the pneumonia patient.

Always reassess your patient at set intervals, every 5 minutes for a patient in distress, or with any change in the patient's complaints or condition, and before and after every intervention. Avoid tunnel vision or narrow-mindedness. When a patient changes or something doesn't add up, do not hold on to your earlier presumptive diagnosis. Reassess, adapt, and stabilize the patient.

Written By: Dr. Kevin Munjal
EMS / Disaster Preparedness Fellow
Office of Medical Affairs

APRIL 2010 JOURNAL CME QUIZ

1) During inspiration, which of the following correctly tracks the flow of air through the body?
   a. Larynx → Pharynx → Alveoli → Bronchioles → Trachea
   b. Pharynx → Larynx → Trachea → Bronchioles → Alveoli
   c. Alveoli → Trachea → Pharynx → Larynx → Bronchioles
   d. Vocal Cords → Trachea → Glottis → Bronchioles → Alveoli
   e. Bronchioles → Glottis → Larynx → Pharynx → Trachea

2) What is ventilation?
   a. The process of oxygen diffusing across the alveolar membrane into the blood stream.
   b. The process of oxygen being taken up into our cells to produce energy.
   c. The process of moving air in and out of the lungs.
   d. The potential space between the parietal and visceral pleura.
   e. The tactic of creating a draft with an opening above or opposite the entry point so that heat and smoke will be released

3) Which medical condition is associated with a "pink puffer?"
   a. Restrictive lung disease
   b. Chronic Bronchitis
   c. Asthma
   d. Emphysema
   e. Intoxication

4) Physical Examination of the respiratory system begins with:
   a. Inspection
   b. Auscultation
   c. Pulse Oximetry
   d. Electrocardiogram
   e. Bag-Valve Mask Ventilation

5) Which of the following is **not** true about Asthma?
   a. It is a reversible bronchospasm.
   b. It is cured by albuterol.
   c. It can be triggered by environmental factors.
   d. It presents with wheezing.
   e. It can proceed to respiratory arrest.

6) Which of the following terms is correctly matched to its definition?
   a. Carina – gland that produces mucus to protect the bronchial tree.
   b. Glottis – cartilaginous structure that flops down and can obstruct the airway when infected with *H. Influenza* type B.
   c. Surfactant – thin mucus that helps keep the alveoli open.
   d. Parenchyma – opening to the trachea located at the level of the larynx.
   e. Uvula – posterior boundary of the larynx

7) All of the following can result from hypoxia except:
   a. Altered mental status.
   b. Myocardial Infarction
   c. Metabolic Alkalosis
   d. High levels of CO_2
   e. Diffuse weakness

8) All of the following are common causes of Acute Pulmonary Edema except:
   a. Pulmonary hypertension
   b. Pulmonary contusion
   c. Renal Failure
   d. Allergic reaction
   e. Right ventricular myocardial infarction

9) Which of the following best describes how epinephrine helps in Asthma exacerbations?
   a. Stimulates the goblet cells to secrete mucus
   b. Acts on the brain to stimulate respiratory drive
   c. Inhibits blood flow to the bronchial tree
   d. Relaxes smooth muscles in the bronchioles
   e. Increases intra-alveolar pressure

10) Jugular Venous Distention (JVD) could indicate all of the following except:
   a. Pericardial Tamponade
   b. Hypovolemia
   c. Chronic Obstructive Pulmonary Disorder
   d. Congestive Heart Failure
   e. Pneumothorax

Journal CME Credit Answer Sheet

Based on the CME article, place your answers to the quiz on this answer sheet.
Respondents with a minimum grade of **80%** will receive **1 hour** of Online/Journal CME.

Please submit this page **only once**, by one of the following methods:

- FAX to 718-999-0119 or
- MAIL to FDNY OMA, 9 MetroTech Center 4th flr, Brooklyn, NY 11201

Contact the Journal CME Coordinator at 718-999-2790:

- three months before REMAC expiration for a report of your CME hours.
- for all other inquiries.

Monthly receipts are not issued. You are strongly advised to keep a copy for your records.

Note: if your information is illegible, incorrect or omitted you **will not** receive CME credit.

check one: ☐ EMT ☐ Paramedic ☐ _____
other

_____
Name

_____
NY State / REMAC # or "n/a" (not applicable)

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Work Location

_____
Phone number

_____
Email address

Submit answer sheet by the last day of this month.

April 2010 CME Quiz		
1.		**Required for BLS & ALS providers**
2.		
3.		
4.		
5.		
6.		**Required for ALS providers only**
7.		
8.		
9.		
10.		

Citywide CME – April 2010

Sessions are subject to change without notice. Please confirm through the listed contact.

Boro	Facility	Date	Time	Topic	Location	Host	Contact
BK	Brooklyn Hospital	1st Wed	0800-0900	Nov 1 Lecture	121 Dekalb Ave, Mazer Lecture Room near ED	Dr Lehrfeld	David Lehrfeld MD 503-961-5113
	Kingsbrook	4/15	1530	Telemetry Presentations	ED Conference Room	Dr Hew	Manny Delgado 718-363-6644
		5/20		Stroke Assessment			
		6/17		Environmental			
	Lutheran	4th Wed	1730-1930	Call Review **RSVP** →	Call for location →	Dr Chitnis	Dale Garcia 718-630-7230 dgarcia@lmcmc.com
MN	NY Presbyterian	TBA	TBA	TBA: call to inquire →	Weill Auditorium, enter at E 69 St and York Ave	Dr. Samuels	212-746-0885 x2
	NYU School of Medicine	TBA	TBA	TBA: call to inquire →	Schwartz Lecture Hall 401 E. 30th Street	TBA	Jessica Kovac 212-263-3293
QN	FDNY-BOT	4/21	1030-1430	Call Review or Lecture	Fort Totten Bldg 325	TBA	swansoc@fdny.nyc.org
		5/26					
		6/23					
	Flushing Hosp	3rd Wed	1330-1530	Call Review	Board Room	Dr Crupi	Mordechai Lax 718-240-5570
	NYH Queens	Mondays	1600-1800	Call Review/Trauma Rounds	East bldg, courtyard flr	Dept of Surgery	Lisa Galati 718-670-2501
	Mt Sinai Qns	last Tues	1800-2100	Lecture	25-10 30 Ave, conf room	Dr. Dean	Donna Smith-Jordan 718-267-4390
	Parkway Hosp	3rd Wed	1830-2130	Call Review	Board Room, 1st flr		pabruzzino@capitolhealthmgmt.com
	Queens Hosp	2nd Thurs	1615-1815	Call Review	Emergency Dept		718-883-3070
		4th Thurs					
SI	Richmond UMC	TBA	TBA	TBA: call to inquire →	MLB conference room	Dr. Ben-Eli	William Amaniera 718-818-1364

2010 NYC REMAC Examination Schedule

Month	REMAC Refresher Exam (Written only - CME letter required)		REMAC Quarterly Exam - $100 fee (Written & 3 Orals Scenarios)			NYS/DOH Written Exam
	Registration Deadline	Exam Date (on Wednesdays)	Registration Deadline	Written @18:00	Orals @09:00	
January	12/31/09	1/20/10	Thursday 1/7/10	Thursday 1/21/10	Wednesday 1/27/10	1/21/10
February	1/31/10	2/17/10				
March	2/28/10	3/24/10				3/18/10
April	3/31/10	4/21/10	Thursday 4/8/10	Thursday 4/22/10	Thursday 4/29/10	
May	4/30/10	5/26/10				5/20/10
June	5/31/10	6/23/10				6/17/10
July	6/30/10	7/21/10	Thursday 7/8/10	Thursday 7/22/10	Tuesday 7/27/10	
August	7/31/10	8/25/10				8/19/10
September	8/31/10	9/22/10				
October	9/30/10	10/20/10	Thursday 10/7/10	Thursday 10/21/10	Wednesday 10/27/10	
November	10/31/10	11/17/10				11/18/10
December	11/30/10	12/22/10				12/16/10

The **REMAC Refresher Written examination** is offered monthly for paramedics who meet CME requirements **and** whose REMAC certifications are either current or expired **less** than 30 days. To enroll, call **718-999-7074** before the register registration deadline above. Candidates may attend an exam no more than 6 months prior to expiration. Refresher exams are held at 07:00 or 18:00 hours at FDNY-EMS Bureau of Training, Fort Totten, Queens.

The **REMAC Quarterly Written & Orals examination** is for initial certification, **or** for inadequate CME, **or** for certifications expired **more** than 30 days. Registrations **must** be postmarked by the deadline above. Email swansoc@fdny.nyc.gov for instructions. You are encouraged to **register at least 30 days** prior to the exam - seating is limited. The exam fee as above is by **money order only**. The Quarterly is held at FDNY-EMS Bureau of Training, Fort Totten, Queens.