From the Editor

To ensure the highest-possible quality of patient care in NYC, REMAC has raised CME and exam requirements for all re-certification and new candidates.

**All candidates must now meet CME requirements**

- All REMAC paramedics and candidates should review Certification & CME Information on page 3 journal and plan accordingly.
- All upcoming exam candidates, see registration instructions at the bottom of the last page of this journal.
- Candidates who will not have a CME letter at the time of their REMAC exam must email Christopher.Swanson@fdny.nyc.gov ASAP.

**The exam format has changed for all candidates**

- Early testing is strongly encouraged, there is no loss of certification time.
- Study Tips – to pass the exam, candidates MUST:
  - memorize the REMAC GOP, BLS and ALS protocols, and appendices
  - interpret 3 and 12-lead ECGs
  - calculate drug doses based on patient weight
- 120 question multiple-choice exam with a 3-hour time limit
  - 20 Scenario questions: two new intensive patient-care scenarios
    - one adult and one pediatric, 10 questions each
    - similar to past REMAC Orals and Scenario exams
    - testing the candidate’s ability to integrate history, physical exam, ECG interpretation, diagnosis, treatment using the NYC REMAC protocols
  - 100 General questions: the same format and content as past REMAC exams, on protocol content and patient care
- Passing score is 80%. Exam failure permits a retest the same month.
**On August 1, 2015 REMAC Protocol revisions take effect for the field and exams**

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

Always see [nycremsco.org](http://www.nycremsco.org) for the current approved protocols

For updates, see REMAC Advisory 2015-03, 04, 05 & 07 at nycremsco.org

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**General Operating Procedures**

- Spinal Precautions
  - Removes rapid-takedown
  - New policy language

- Pediatric Patients
  - Changes age parameters

- Prehospital Sedation
  - Removes etomidate administration rate for intubation
  - Increases etomidate maximum dose for cardioversion

- IO Administration
  - Limits number of attempts

- Pre-existing Central Venous Catheter
  - New GOP section

**BLS Protocols**

- 407 – Wheezing & 410 – Anaphylaxis
  - Changes note to not delay transport
  - Changes OLMC contact requirements

- 411 – Altered Mental Status
  - Adds pediatric dosing for naloxone
  - Removes contraindications for pediatrics and therapeutic opiate use
  - Initiate transport prior to repeating treatment
  - Adds QA component

- 421 – Head and Spine Injuries
  - Removes immobilization
  - Adds spinal precautions
  - Removes hyperventilation

- 430 – Excited Delirium
  - Changes name of protocol
  - Language changes to criteria & procedures

**ALS Protocols**

- 500-A – Smoke Inhalation & 500-B – Cyanide
  - Changes blood drawing to “if available”
  - Changes age requirement
  - Changes bottle use of hydroxocobalamin
  - Deletes Table 2

- 530 – Excited Delirium
  - Changes name of protocol
  - Standing Orders:
    - adds midazolam IM/IN
    - add normal saline rapid infusion
  - Medical Control Options:
    - reorganized as a table
    - adds ketamine IM/IN
    - removes IV lorazepam & midazolam
    - reduces IN lorazepam & midazolam
  - Adds QA component

**Appendices**

- Appendix P – CPAP
  - Removes pregnancy as contraindication
REMAC Exam Study Tips

REMAC candidates have difficulty with:  
* Epinephrine use for peds patients 15%  
* 12-lead EKG interpretation 10%  
* ventilation rates for peds & neonates 10%

REMAC Written exams are approximately:  
10% BLS  15% Adult Trauma  
10% Adult Arrest  15% Pediatrics

Certification & CME Information

- By the day of their exam, all REMAC paramedics and candidates must present a letter from their Medical Director verifying fulfillment of CME requirements.

- Upcoming candidates without a CME letter ASAP must email Christopher.Swanson@fdny.nyc.gov

- 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 # 2007-11:
  - 36 hours - Physician Directed Call Review  
    - ACR Review  
    - QA/I Session  
    - Emergency Department Teaching Rounds - Maximum of 18 hours  
  - 36 hours - Alternative Source CME - Maximum of 12 hours per venue  
    - Online CME (see examples below) - Clinical rotations  
    - Lectures / Symposiums / Conferences - Associated Certifications – 4 hours each: BCLS / ACLS / PALS / NALS / PHTLS

- Failure to maintain a valid NYS EMT-P card will suspend your NYC REMAC certification until NYS is recertified.

REMARC certification exams are held monthly for new and expired candidates, and for currently certified paramedics who may attend up to 6 months before their expiration date.

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

REMSCO: www.NYCREMSCO.org  

Online CME: www.EMS-CE.com  
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March – April 2016 – Journal CME Newsletter
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HEART FAILURE AND CPAP

Introduction

As a result of the aging of the U.S. population, heart failure has become a large and growing problem. It is the most common diagnosis in Medicare patients. With more than five million Americans diagnosed with heart failure and a five-year mortality approaching 50%, it is the most common cause of hospitalization in patients older than 65 years and is the single most expensive diagnosis in the U.S. healthcare system. The good news is that with recent improvements in treatment, and a drop in the incidence of coronary artery disease, the last reported 10-year trend showed a decline in ED admissions from this diagnosis. The rate of one-year all-cause mortality, however, remained high at approximately 30% for patients hospitalized due to heart failure. For those patients who present with frank pulmonary edema, the corresponding rates of morbidity and mortality are approximately double. Patients who present with acute myocardial infarction and cardiogenic shock have a mortality rate of 70% or more despite aggressive therapy.

Pathophysiology

The heart must be able to adequately pump blood to and from the lungs and the body organs and tissues to maintain perfusion. If the heart, as a pump, fails, perfusion will stop. If the pump is weakened but has not failed, compensation may be possible. If oxygen consumption is conserved, as with rest, symptoms may be minimal. However, with exertion, oxygen consumption and oxygen demand will increase. If the heart is unable to meet this demand, the patient will become symptomatic.

Heart failure results when the heart's ability to pump fails. Most causes of pump failure have a gradual onset and therefore produce a more gradual onset of symptoms. If the pump failure is sudden, symptom onset may be dramatic because of the limited ability of the body to compensate. Shock as a result of pump failure is referred to as cardiogenic shock.

One of the more dramatic causes of pump failure is a heart attack, or myocardial infarction (MI). When blood flow to an area of heart muscle is blocked, that area of heart muscle will become ischemic and die unless the blood flow is restored quickly. If a large enough area of muscle is involved, the wall motion of the ventricular contraction will be compromised and pump failure will occur. Which part of the heart is affected and the extent of the damage will determine the patient's ability to recover. If the left ventricle is involved, more severe symptoms of heart failure will result.

Attempts to limit the damage to the heart by opening the blocked vessel has been behind the FDNY initiative to transport patients having an ST elevation MI (STEMI) to a hospital with cardiac catheterization capability. When oxygen is restored to the heart muscle quickly, it can save heart muscle, and prevent the development of cardiogenic shock.
shock. In the long term, it can mean the difference between someone returning to work, struggling with the symptoms of heart failure, or waiting for a heart transplant.

**Severity of Heart Failure**

The severity of failure can be described in many ways, which may include a measure of how chronic failure affects the **quality of life** or more acute clinical parameters. Table 1 below describes the new American Heart Association classification, and Table 2 describes the commonly used New York Heart Association (NYHA) classification.

**Table 1. American Heart Association Classification of Heart Failure:**

<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage A</td>
<td>Patients are at high risk for heart failure but have not developed structural heart disease and have no symptoms.</td>
</tr>
<tr>
<td>Stage B</td>
<td>Patients have developed structural heart disease but have not (yet) developed symptoms.</td>
</tr>
<tr>
<td>Stage C</td>
<td>Patients with past or current heart failure symptoms in association with structural damage to the heart.</td>
</tr>
<tr>
<td>Stage D</td>
<td>Patients with end-stage, or terminal, heart failure requiring specialized treatment strategies (circulatory support, such as LVAD).</td>
</tr>
</tbody>
</table>

**Table 2. New York Heart Association - (NYHA) Classification of Heart Failure**

<table>
<thead>
<tr>
<th>Class</th>
<th>Functional state</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No limitation</td>
<td>Asymptomatic during usual daily activities</td>
</tr>
<tr>
<td>II</td>
<td>Slight limitation</td>
<td>Mild symptoms (dyspnea, fatigue, or chest pain) with ordinary daily activities</td>
</tr>
<tr>
<td>III</td>
<td>Moderate limitation</td>
<td>Symptoms noted with minimal activity</td>
</tr>
<tr>
<td>IV</td>
<td>Severe limitation</td>
<td>Symptoms at rest</td>
</tr>
</tbody>
</table>

Ventricular failure occurs when the amount of blood entering the ventricle is more than the ventricle can pump out, or when the ventricle is damaged to the degree that it cannot deliver enough blood to meet the body's metabolic needs. Right or left ventricular failure refers to which ventricle has failed. **Right heart failure** results in the impaired return of blood to the heart from the central venous circulation. It is most commonly a consequence of severe or long-standing **left heart failure** but can also occur in isolation (as in the case of cor pulmonale) or result from right ventricular infarction.

When the right ventricle fails, the neck veins become distended (JVD); blood backs up in the liver, resulting in chronic passive congestion of the liver; and extremity edema occurs, because the lower extremity is so gravity-dependent. **Cor pulmonale** refers to the impaired function of the right ventricle that results from pulmonary hypertension, and is seen in patients with advanced lung disease, such as chronic obstructive pulmonary disease (COPD). As COPD advances past the early stages of airway inflammation and narrowing (remodeling), there is an eventual loss of lung capillary bed surface area. Hypoxemia now becomes almost continuous. These changes cause a marked increase in pulmonary artery pressure, making it harder for the right ventricle to pump blood. If not treated, pulmonary hypertension will cause the right ventricle to fail.
The term **congestive heart failure** refers to failure of the left ventricle. When the left ventricle fails, blood “congests” in the lungs (pulmonary edema). If allowed to continue, this congestion will eventually cause the right ventricle to fail, the liver to become congested, and peripheral edema to appear. There are many causes of left ventricular failure, with coronary artery disease being the most common. Abnormal heart rhythms, failing heart valves, toxic damage to the ventricular muscles (alcohol and certain antitumor drugs) and viral infections are other causes of heart muscle damage leading to ventricular failure.

When the left ventricle fails to pump sufficient blood to the body, needed nutrients such as oxygen, glucose, fatty acids and proteins are in short supply to the body's cells. All organs of the body need these nutrients in order to be healthy enough to perform their specific tasks. Equally important, the waste products of metabolism (CO₂ and urea to name two) are no longer eliminated efficiently. The symptoms of left ventricular failure include shortness of breath, heart palpitations, fatigue, increased urination at night (nocturia), and the need to sleep sitting up (orthopnea).

Physical findings in patients with left ventricular failure include rales in the lungs on auscultation, peripheral edema, distended neck veins, and abnormal rhythms and heart sounds (third heart sound-S3). These findings are a result of blood backing up in the chamber of the left ventricle, the lungs, and subsequently the right ventricle, causing congestion in every part of the vascular system carrying blood to the heart.
Other methods of diagnosing heart failure include the chest ultrasound, chest x-ray, electrocardiogram and a blood test called the brain natriuretic peptide (BNP). BNP is a peptide that naturally occurs in heart muscle (myocardium) and is excreted into blood in increased amounts when myocardium is stretched. The more the myocardium is stretched, as in congestive heart failure, the higher the level of BNP. This test helps doctors differentiate between respiratory failure due to CHF or other causes such as COPD.

The etiologies of heart failure are numerous and diverse. In the United States, the vast majority of heart failure arises as a consequence of coronary artery disease and/or long-standing hypertension. Acute heart failure can present for the first time in the out-of-hospital setting; for example, in acute myocardial infarction (MI) or with acute valvular insufficiency. Much more commonly, patients have chronic heart failure that has decompensated as the result of one or more precipitating factors shown on the next page.

### Etiologies of Heart Failure.

- Coronary artery disease
- Hypertension
- Valvular disease
- Cardiomyopathy
  - Idiopathic cardiomyopathy
  - Alcoholic cardiomyopathy
  - Toxin-related cardiomyopathy (e.g. adriamycin)
  - Postpartum cardiomyopathy
  - Hypertrophic obstructive cardiomyopathy (HOCM)
  - Tachyarrhythmia-induced cardiomyopathy
- Infiltrative disorders (e.g. amyloid)
- Congenital heart disease
- Pericardial disease
- Hyperkinetic states
  - Anemia
  - Arteriovenous fistula
  - Thyroid disease
  - Beriberi

By definition, heart failure is the inability of the heart to produce adequate cardiac output to meet the perfusion and oxygenation requirements of the body’s tissues. It can arise from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood, resulting in decreased cardiac output. There are two mechanisms by which heart failure can occur:

- Systolic dysfunction, the result of impaired cardiac **contractile** function; or
- Diastolic dysfunction, the result of abnormal cardiac **relaxation**, **stiffness** or **filling**.
Systolic Heart Failure

In systolic heart failure (SHF), the heart has impaired **contractile function**, resulting in a decreased stroke volume (SV) and cardiac output (CO) and subsequent low blood pressure. Patients with SHF also have a decreased ejection fraction (EF). The EF is the percentage of blood pumped out of the ventricle with each heartbeat. A healthy adult would be expected to have an ejection fraction between 50%–75%. A patient with an EF less than or equal to 40% is said to have heart failure.

A number of factors can lead to impaired myocardial contractile function. Acute myocardial infarction (AMI) can acutely lead to impaired contractility, as infarcted myocardium can be significantly weaker than healthy myocardium. After an MI, the scarred, remodeled ventricular myocardium will have less contractile force than healthy myocardium. Dilated cardiomyopathy can result from many etiologies, including chronic hypertension.

Chronically increased blood pressure stretches and dilates the ventricular tissue, making it weaker. Valvular disease and ineffective heart valves can allow the backflow of blood during systole, resulting in decreased stroke volume. In addition, faulty heart valves (such as the aortic and pulmonary semilunar valves) can impede the forward movement of blood, resulting in decreased stroke volume as well as increased intraventricular pressures, which can cause further problems such as cardiomyopathies.

Diastolic Heart Failure

In diastolic heart failure (DHF), the ventricle wall cannot adequately relax, resulting in **inadequate ventricular filling** during diastole and a subsequent decrease in stroke volume and cardiac output. The inadequate ventricular filling occurs as a result of a stiffening of the ventricular wall that prevents the normal ventricular relaxation that occurs during diastole. Numerous etiologies can lead to ventricular wall stiffening. **Chronic hypertension** can result in ventricular hypertrophy as the ventricle is chronically pushing against an elevated systemic blood pressure and increased afterload. In amyloidosis, protein is deposited in the ventricular wall, causing it to stiffen. Patients with DHF do not suffer a marked decrease in their EF unless there is coexistent systolic failure.

The differences between systolic and diastolic mechanisms are typically not apparent to the prehospital provider managing a patient in the field. From an EMS perspective, it makes more practical sense to approach heart failure from the standpoint of right-sided heart failure versus left-sided heart failure, which are clinically distinctive and apparent to EMS providers.

As was mentioned earlier, when the right ventricle fails, blood and pressure back up into the central venous circulation, starting with the vena cava. Increased pressures in the superior vena cava carry over to the jugular veins, leading to jugular venous distension (JVD). Increased pressures traveling down the inferior vena cava lead to physical findings such as abdominal ascites and peripheral (pedal) edema.

With left ventricular failure, blood and pressure back up into the pulmonary circulation, leading to increased capillary pressure, capillary dilation and congestion. Increased capillary pressures result in the leaking of fluid from the capillaries into the surrounding tissues, resulting in fluid buildup (edema). Left untreated, this will lead to pulmonary edema and congestive heart failure. Rales, or crackles, are the hallmark finding on lung examination. Left unchecked, left heart failure will lead to right heart failure, as pressure backs up through the pulmonary vasculature and into the right ventricle.

History

Most patients presenting with decompensated heart failure will have a prior cardiac history. Patients can generally tell you if they have had “fluid on the lungs” in the past. More sophisticated patients may be able to provide details of previous echocardiograms or cardiac catheterizations. Most patients commonly complain of dyspnea or trouble sleeping. It is important to determine the degree of dyspnea and its onset. Does it occur at rest? Does the patient...
have paroxysmal nocturnal dyspnea (PND)? Crews working tour one are used to the 0400 call for the elderly “Difficulty Breathing” patient who was fine at bedtime, only to awake in severe distress. Ask the patient how he or she has been sleeping and on how many pillows. Spending the night sleeping while sitting on a couch or chair is an indication of worsening heart failure.

The rapidity of symptom onset may also suggest an etiology for the decompensation. An abrupt deterioration raises concern for arrhythmia, acute coronary syndrome, or valvular rupture. Prior episodes of a similar nature can provide important clues.

Associated symptoms are also important. Determine whether the patient has had any chest pain or other anginal equivalent such as shoulder, neck, arm or epigastric discomfort. The combination of syncope and heart failure is worrisome and is associated with a high one-year mortality rate. Ask the patient and family about recent weight gain, leg swelling, urinary output, exercise tolerance, fatigue, and compliance with diet and medications. New prescriptions or changes in dosages could provide an important clue.

**Physical Examination**

The patient’s degree of distress should be determined early in the encounter; severe pulmonary edema can be a “doorway diagnosis.” In severe cases, audible rales (crackles) can be heard without a stethoscope. Confusion, cyanosis, diaphoresis, inability to speak, and extreme weakness may indicate impending respiratory failure. Patients may also have a cough with pink frothy sputum. Keep in mind that rales and wheezing, sometimes occurs with pneumonia and COPD. Wheezing in heart failure has been referred to as “cardiac asthma,” since it is not uncommon in acute heart failure patients.

Vital signs not only suggest the severity of illness but can also indicate etiologic factors. High blood pressure, and especially high diastolic pressure, is common. While hypotension can be the baseline for patients with end-stage cardiomyopathy, in the symptomatic patient it should raise concern for sepsis, massive pulmonary embolism, or cardiogenic shock. In the absence of heart rate-controlling medications, tachycardia is nearly universal in decompensated heart failure. Bradycardia should raise concern for high-degree AV block, hyperkalemia, digoxin (or other drug) toxicity, or severe hypoxia.

**EKG**

The 12-lead ECG is useful for detecting ischemia, arrhythmias, or electrolyte disturbances, and should be obtained by paramedics. The ECG is likely to be abnormal in patients with heart failure. In one study, left ventricular hypertrophy on resting ECG was noted in 42% and electrocardiographic evidence of cardiac ischemia or a prior MI was present in approximately 70%. Comparison with prior ECGs may be helpful, especially with patients who have subtle changes or “baseline abnormal” tracings.

**Treatment**

For patients in respiratory distress, application of high-flow oxygen via non-rebreather mask is indicated. Although there have been reports that excessive oxygen can adversely affect left ventricular function, hypoxia is by far the greater concern. Sitting the patient upright (full Fowler’s position) will improve airflow. Studies in patients with chronic heart failure show a large rise in airflow resistance after lying supine for five minutes, a condition that is reversed by sitting erect. Pulse oximetry, blood pressure, and continuous cardiac monitoring are necessary to provide early signs of deterioration or response to treatment.
Medications

**Nitrates** are recommended as first-line therapy for acutely decompensated heart failure of both ischemic and non-ischemic origin. The beneficial hemodynamic effects of nitrates in the setting of heart failure have long been appreciated. *No other group of agents improves the symptoms of congestion as rapidly as nitrates.* Treatment with sublingual nitroglycerin (tablets or spray) results in noticeable hemodynamic and clinical improvement within five minutes. If chest pain is present, BLS providers may assist the patient with self-administration of the patient’s own previously prescribed nitroglycerin provided that the patient’s systolic BP is at least 120mm Hg (REMAC BLS Protocol 404). Single doses of 0.4 mg can be given repeatedly every five (5) minutes by paramedics provided the patient’s systolic blood pressure is greater than 100mm Hg (REMAC ALS Protocol 504-A).

Hypotension with standard nitrate therapy is generally mild and transient. Nitrates are short acting and sublingual nitrates last for about 4-5 minutes. Severe or persistent hypotension should raise suspicion for hypovolemia, cardiac tamponade, right ventricular infarction, or recent use of sildenafil (Viagra) or tadalafil (Cialis). If these conditions are known or suspected, online medical control should be consulted.

**Diuretics** represent the mainstay of therapy for patients with volume overload. On the other hand, it is important to recognize that *patients who present with acutely decompensated heart failure are not necessarily volume overloaded.* The indiscriminate use of diuretics carries the risk of over-diuresis, particularly among elderly patients or patients who are volume depleted despite their pulmonary edema. For this reason, diuretics may be harmful for some patients with pulmonary edema and furosemide is a medical control option. In preparing your Telemetry presentation, the patient who has been noncompliant with prescribed diuretics and/or has had a recent high sodium meal, would be a possible candidate for furosemide.

**Morphine** was once the mainstay of treatment in acute pulmonary edema, but has fallen out of favor as research has shown that patients treated with morphine show higher mortality. REMAC Protocol 506 Acute Pulmonary Edema allows for morphine as a medical control option. Treatment with benzodiazepines (On-Line Medical Control Options: Lorazepam and Midazolam) are available as an option for decreasing catecholamine release with fewer side effects.

**Dialysis Patients**

Heart failure is present in about one-third of patients who begin dialysis and will develop over time in an additional 25%. Among hemodialysis patients who no longer have urinary output, heart failure is the most common cause of ED visits. In these patients, acutely decompensated failure is most often due to volume overload between dialysis treatments or due to missed dialysis sessions. Although hemodialysis is the treatment of choice for these patients, it may not be immediately available. Treatment is directed at stabilizing these patients until hemodialysis can be performed.

**Continuous Positive Airway Pressure Ventilation (CPAP)**

Noninvasive positive-pressure ventilation (NIPPV) has become increasingly important in the management of respiratory insufficiency and respiratory failure. There are two modes, biphasic positive airway pressure (BiPAP) and continuous positive airway pressure (CPAP). Positive airway pressure devices have been shown to rapidly improve the patient’s condition by:

- Increasing oxygen diffusion,
- Decreasing work of breathing,
- Decreasing pre-load and after-load,
- Decreasing dyspnea,
- Decreasing the need for endotracheal intubation.
It has been shown to be effective in patients who suffer shortness of breath from asthma, COPD, pulmonary edema, CHF, and pneumonia. CPAP has been shown to be safe and effective for shortness of breath even where the cause has not been determined. In patients with CHF, fluid in the alveolar space creates a barrier to gas exchange and makes lung less compliant.

Extrinsic pressure from CPAP forces fluid out of the alveolar space. In addition, CPAP improves hemodynamics by increasing intrathoracic pressure, which reduces preload and afterload. CPAP improves pulmonary mechanics by recruiting atelectatic (collapsed and airless) alveoli and improving the ability of oxygen to diffuse from the alveoli onto red blood cells. **Pulmonary recruitment** is a phenomenon that occurs in the lung when pressure is applied to the airways in such a way as to open up unused or collapsed alveolar segments not participating in gas exchange. The increased velocity of oxygen delivered by CPAP allows for better diffusion of oxygen into the bloodstream.

CPAP is frequently described as being able to **stent** or stretch the airways open. This allows for deeper penetration into the alveolar region of the lung by each breath, and this stenting holds the airways open longer during exhalation. There is a subsequent decrease in work of breathing, due to an increase in lung compliance. Distribution of ventilation improves, as does secretion removal. Both ventilation (CO₂ removal) and oxygenation are improved as a result.

The majority of patients with respiratory distress respond to supplemental oxygen and standard pharmacologic therapy, but patients not showing improvement, in severe distress, with persistent hypoxemia, or progressive fatigue may benefit from CPAP.

The success of noninvasive respiratory support depends on appropriate patient selection. For patients with **compromised upper airway function** or significantly **altered level of consciousness**, intubation and bag-valve-mask ventilation remain the appropriate therapy. The following patients are candidates for use of CPAP:

- Age 18+ years old
- Has shortness of breath (for reasons other than trauma)
- Is awake and able to follow commands
- Has a systolic blood pressure above 100 mmHg (CPAP may raise intrathoracic pressures, reducing preload, resulting in a drop in blood pressure)
- Presents with signs and symptoms consistent with asthma, bronchitis, COPD, acute cardiogenic pulmonary edema, or congestive heart failure

In the setting of severe myocardial ischemia or infarction, full ventilatory support may be preferable in order to decrease the myocardial oxygen demand associated with respiratory effort. Contact with On-line Medical Control is required for a discretionary decision to use CPAP for these patients. Do not apply CPAP for the following conditions (Contraindications):

- Altered mental status (lethargy, agitation, combativeness)
- Respiratory failure, or other need for endotracheal intubation
- Hemodynamically unstable (SBP< 100)
- Patients who are unable to control their airway (i.e. copious secretions or active bleeding)
- Trauma, facial burns or impending cardiac or respiratory arrest
• Gastric Distention
• Aspiration risk:
  o Persistent Nausea/Vomiting
  o Active upper GI bleeding
• Known active unstable angina or acute MI
• Known pneumonia, pneumothorax, pulmonary embolism, or anaphylaxis

Although the decision to initiate noninvasive respiratory support is dependent on a variety of factors, the presumption is that the earlier therapy is instituted, the greater the likelihood of averting intubation. Recent studies suggest that the use of noninvasive ventilatory support in the prehospital setting is feasible and potentially beneficial for patients with presumed cardiogenic pulmonary edema. If there is progressive respiratory distress, in spite of aggressive therapy, the patient requires intubation and mechanical ventilation. CPAP should be discontinued immediately if advanced airway control is required, the patient becomes hemodynamically unstable, the patient cannot tolerate the mask due to pain or discomfort, or the patient experiences nausea or vomits. Patients using continuous positive airway pressure ventilation (CPAP) must be able to breathe spontaneously.

Studies show that CPAP is therapeutic when applied to patients in CHF. CPAP has also been shown to reduce the need for heart failure patients to be intubated and placed on a ventilator. Thus, EMS plays a critical role in the management of these patients, because the sooner proper treatment is initiated, the higher the chances that the patient will avoid an ICU admission. This is a “win-win” for both the patient and the entire healthcare system.

Coaching the CPAP Patient

Crucial to the success of CPAP is active patient involvement. The first step to actively engage the patient in achieving success is to identify likely barriers, then work to overcome these. For first time CPAP patient, the most likely barrier is the perception that the mask will suffocate them. We can address this concern by verbalizing it at the outset, then letting the patient hold the mask and gradually get comfortable:

  Explain that once they try it, they will see that it will improve their breathing. Demonstrate the quick release clips. ("It’s going to feel like being in a car with all the windows down. It may take a minute to get comfortable, but you will be getting more oxygen and you will find you will start feeling better. You know you can remove the mask easily any time you want.") Once the patient has become comfortable with the mask against their face, pull the harness over the head and adjust the Velcro straps. Since comfort is important to continued compliance, make sure the forehead pads are properly adjusted to take pressure off the nose. Slide up the tab on the bridge of the nose to relieve pressure on the nose.

For your female patients, the hair and earrings may be tangled in the head straps. To address this, ask the patient to remove the earrings before applying the mask. Move the hair away from the face before securing the head straps.

Nebulized Medication

Nebulizer solutions may be added by detaching the nebulizer container from the CPAP system. After medication is placed in the nebulizer, turn switch to “ON” position. The flow rate may need to be increased to maintain the correct pressure while the nebulizer is in use. Nebulized medications may not “mist” due to the increased size of the chamber and increased flow rate of nebulized medication but they are still being delivered to the patient.
Adjust oxygen liter flow to achieve 5 to 10 cm H₂O on the manometer. Using a “D” oxygen cylinder, you will expect to have about 44 minutes of oxygen delivery at a flow rate of 8 liters per minute. The chart below shows approximate time intervals for various settings.

<table>
<thead>
<tr>
<th>O₂ Flow (LPM)</th>
<th>CPAP/PEEP (cmH₂O)</th>
<th>Minutes</th>
</tr>
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<tbody>
<tr>
<td>8-9</td>
<td>5</td>
<td>44</td>
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<tr>
<td>10-12</td>
<td>7.5</td>
<td>30-35</td>
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<td>13-14</td>
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<td>24-30</td>
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</table>

**Conclusion**

Heart failure is a serious medical condition that is on the increase. The sooner a patient is diagnosed and treatment started, the better the patient's chance for survival, especially when congestive heart failure has occurred acutely. Continuous positive airway pressure ventilation (CPAP) is a critical prehospital therapy that will lead to improved patient outcomes. EMS providers have been successfully trained to initiate it safely so as to improve survival of the patients committed to their care. If CPAP is applied to these patients, there is evidence that these patients will avoid endotracheal intubation, and their hospital stays will be shorter.

**Submitted by:**  
Lt. Joan Hillgardner, FDNY Office of Medical Affairs

**Edited by:**  
Dr. Nathan Reisman, FDNY EMS Fellow  
Dr. Bradley Kaufman, FDNY First Deputy Medical Director

**References**


All 10 questions for ALS and BLS Providers

1. The rate of one-year mortality for patients hospitalized for heart failure is:
   a. 10 percent
   b. 30 percent
   c. 50 percent.
   d. 70 percent.

2. Heart failure is the most common cause of hospitalization in patients older than 65 years of age.
   a. True
   b. False

3. A blocked coronary artery may cause severe damage to the heart muscle (myocardium) leading to cardiogenic shock. Intervention for the treatment of S-T Elevation MI (STEMI) is designed to:
   a. prevent further damage to the myocardium
   b. restore coronary artery blood flow
   c. promote a better functional outcome
   d. all of the above

4. Which of the following statements regarding heart failure is true?
   a. chronic pulmonary diseases causes left heart failure
   b. sleeping flat improves symptoms
   c. heart failure can be sudden or progressive
   d. cardiac asthma is related to right heart failure

5. Severe respiratory failure in the prehospital setting should be addressed with:
   a. prone positioning.
   b. continuous high-dose furosemide infusion.
   c. CPAP with ambient air.
   d. ventilatory support by BVM or endotracheal intubation

6. Diastolic failure primarily involves impairment of:
   a. myocardial contractility
   b. ventricular filling
   c. atrioventricular conduction
   d. systemic vascular resistance
7. Which medication improves congestive symptoms of heart failure most rapidly?
   a. nitrates
   b. beta-blockers
   c. diuretics
   d. amiodarone

8. An ideal drug (or drug combination) for the treatment of acutely decompensated heart failure would:
   a. reduce preload
   b. enhance left ventricular function
   c. address volume overload
   d. all of the above

9. Which of the following excludes a patient from a trial of CPAP?
   a. diaphoresis
   b. renal failure
   c. left ventricular hypertrophy on ECG
   d. severe agitation

10. Patients using CPAP for the first time may:
    a. feel like the mask is making it more difficult to breathe
    b. be unable to support the weight of the mask
    c. develop carbon monoxide poisoning
    d. develop a fever
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)

Work Location

Phone number

Email address

Submit answer sheet by the last day of April 2016

March – April 2016 CME Quiz

1. Questions 1-10 for all providers
2.
3.
4.
5.
6.
7.
8.
9.
10.
Regional CME – Sessions are subject to change. Please confirm through the listed contact.

See other opportunities at [www.nycemsco.org](http://www.nycemsco.org) under News & Announcements

**Note**: A potential source of Call Review is E.D. Teaching Rounds (maximum of 18 hours)
See any hospital E.D. Administrator for availability (especially HHC hospitals)

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<tr>
<th>Boro</th>
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<td>ED Conference Room</td>
<td>Aaron Scharf 718-363-6644</td>
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<tr>
<td></td>
<td>Lutheran</td>
<td>contact to inquire →</td>
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<td>Dale Garcia 718-630-7230 <a href="mailto:dgarcia@lmcme.com">dgarcia@lmcme.com</a></td>
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<td>MN</td>
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<td>contact to inquire →</td>
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<td>Brian Lynch 512-589-9128 Lenox Hill Hospital EMS</td>
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<td>contact to inquire →</td>
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<td>Eunice Wright <a href="mailto:eunice.wright@mountsinai.org">eunice.wright@mountsinai.org</a></td>
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<td></td>
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<td>contact to inquire →</td>
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<td>Steven M. Samuels 212-746-0596</td>
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<tr>
<td></td>
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<td>contact to inquire →</td>
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<td><a href="mailto:danielle.milbauer@nyumc.org">danielle.milbauer@nyumc.org</a> <a href="http://cme.med.nyu.edu/course">http://cme.med.nyu.edu/course</a></td>
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<td>QN</td>
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<td>Call Review, Trauma Rounds</td>
<td>A1-22 Auditorium 3rd Wednesdays, 0830-0930</td>
<td>Anju Galer RN 718-334-5724 <a href="mailto:galera@nychhc.org">galera@nychhc.org</a></td>
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<td>Mt Sinai Qns</td>
<td>Call Review, Lecture</td>
<td>25-10 30 Ave, conf room last Tuesdays, 1800-2100</td>
<td>Donna Smith-Jordon 718-267-4390</td>
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<tr>
<td></td>
<td>NYH Queens</td>
<td>contact to inquire →</td>
<td>East bldg, courtyard flr</td>
<td>Mary Ellen Zimmermann RN 718-670-2929</td>
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<td>Queens Hosp</td>
<td>Call Review</td>
<td>Emergency Dept 2nd &amp; 4th Thurs 1615-1815</td>
<td>Maria Jones or Julia Fuzailov 718-883-3070</td>
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<td></td>
<td>St John’s University</td>
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<td>175-05 Horace Harding Expwy</td>
<td>718-990-8436 <a href="http://www.stjohns.edu/ems/cme">www.stjohns.edu/ems/cme</a></td>
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<td>Michelle Scarlett <a href="mailto:mscarlet@ehs.org">mscarlet@ehs.org</a></td>
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<tr>
<td>SI</td>
<td>RUMC</td>
<td>contact to inquire →</td>
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<td>Tony McKay NRP <a href="mailto:amckay@rumcsi.org">amckay@rumcsi.org</a></td>
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<td></td>
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<td>Holly Acierno RN <a href="mailto:hacierno@SIUH.edu">hacierno@SIUH.edu</a></td>
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# 2016 NYC REMAC Examination Schedule

<table>
<thead>
<tr>
<th>Month</th>
<th>Registration Deadline</th>
<th>Refresher exams(^1) – no fee for exam</th>
<th>Basic exams(^2)</th>
<th>NYS/DOH Written(^3)</th>
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<td>10:00 exams</td>
<td>18:00 exams</td>
<td>all at 18:00</td>
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<tr>
<td>January</td>
<td>1/1/16</td>
<td>1/20/16</td>
<td>1/15/16</td>
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<tr>
<td>February</td>
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<td>2/17/16</td>
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<tr>
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<td>7/18/16</td>
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<td>11/14/16</td>
<td>11/16/16</td>
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<td>December</td>
<td>12/1/16</td>
<td>12/14/16</td>
<td>12/12/16</td>
<td>12/14/16</td>
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\(^1\) **REMAC Refresher examination** is offered for paramedics who meet CME requirements and whose REMAC certifications are either current or expired less than 30 days. To enroll, go to the REGISTER link under “News & Announcements” at [nycremesco.org](http://nycremesco.org) before the registration deadline above. Candidates may attend an exam no more than 6 months prior to expiration. Early testing is strongly encouraged; there is no loss of certification time.

\(^2\) **REMAC Basic examination** is for initial certification, or inadequate CME, or certifications expired more than 30 days. Seating is limited. Registrations must be postmarked by the deadline above. Exam fee by $100 money order to NYC REMSCO is required. **All Basic candidates must meet new education requirements**. Email [Christopher.Swanson@fdny.nyc.gov](mailto:Christopher.Swanson@fdny.nyc.gov) for instructions.

\(^3\) **NYS/DOH exam dates** are listed for information purposes only. Scheduling is through your paramedic program or contact NYS DOH for more information.