Reading

CEPCP

Professional Development: Shortness of breath

Assessing and Managing Shortness of Breath

The Near Drowning

Frank woke up with a start, relieved that it had all been a dream; the slip on the dock, the cold water enveloping his struggling body and then the terror-inducing sensation of drowning. The cold water eventually flowing into his lungs, the breathing getting heavier and the panic of suffocation overcoming him.

Now he was awake but his breathing still laboured. The relief of being awake soon replaced by a sense of dread. The suffocating feeling, the cold wetness...it wasn't going away. He sat up on the edge of the bed. The sleep now clear from his mind, like a drop of water in a hot pan. It was happening again. He tried to calm himself, *'It usually goes away if I sit for a bit'* he thought, acutely aware that this time it was different. He tried to slow his breathing, but it was impossible. It felt like the end of the wind sprints he used to endure in high school under the stern direction of the navy drill sergeant-turned phys-ed teacher. His breathing felt more and more inadequate. A sense of panic washed over him, was this it? He could feel a wet thick frothing from deep within his chest. He tried to cough to clear it, but a vague, metallic taste of blood was all he managed to produce. '*Margaret'*, he gasped, knowing she would be awake in the dark, worried about his restless sleep and laboured breathing. *'You better call an ambulance'*.

When the tones went off, Tony reluctantly left sleep behind and awoke to the dispatchers tinny speaker voice, *'....code four, sixty seven year*

old male, short of breath, diaphoretic'. Although Tony was tired he knew what he had to do and with an unsteady, trembling gait made his way to the truck. His partner followed close behind and got in the driver's seat.

As they pulled out of the base, a lone drop of water slowly rolled down the windshield, the night looked crisp through the newly washed truck window. Once out on the street, the diesel engine reluctantly forced the transmission into second gear with a noticeable jerk.

The dispatcher repeated the call information with the usual monotone, emotional detachment. As they approached the first red light at the end of the industrial street where the base was located, Tony's partner finally spoke, *'I guess we better run it'* he said in a thick, sleep-muffled voice as he flicked the switches on; turning the night into a red and white strobe reflected flicker. The strobes sounding like trapped flies bouncing against their lenses, desperate to escape.

The Challenge

Shortness of breath is one of the most common reasons people call for the ambulance, and also one of the most difficult conditions to assess and treat. Paramedics have different treatment options depending on the cause, so it is important to ensure that the correct working assessment is reached. Now, if you are reading this and think that assessing shortness of breath is easy you are either born with talents that should be explored in a laboratory, or else you may have been taught a very oversimplified version of categorizing short of breath patients. The EMS world is teeming with misleading myths that include;

- If the crackles are bilateral, its pulmonary edema
- If the crackles go away with a cough, its pneumonia
- If the patient has JVD and/or swollen ankles its pulmonary edema
- If they have a history of COPD, then its COPD

Any of these statements, in isolation, is a dangerous oversimplification of very complex processes. If it really was as simple as the common myths suggests, then why would emergency Physicians, armed with chest x-rays, blood work and other diagnostic tests, still get it wrong twenty percent of the time (Ray et al. 2006)? To make matters worse, the same research found that physicians actually went on to provide the wrong treatment to every third short-of-breath patient they treated(Ray et al 2006). Holding on to the belief that we will do better on the road, with a stethoscope and a monitor as our only tools, is a dangerous illusion.

The good news is that awareness of these limitations and a solid knowledge of how to minimize them, is a great leap towards a better understanding of these challenging patient conditions.

This reading will provide a brief overview of the various common causes of shortness of breath. But, more importantly, will focus on how to provide the correct working assessment and treatment plan for these conditions.

Cardiogenic Pulmonary Edema

Cardiogenic pulmonary edema is simply fluid accumulation in the lungs (pulmonary edema), caused by the failure of the left ventricle (cardiogenic). In order to understand the mechanism behind this condition, it is useful to think of the two sides of the heart as two separate pumps. Even though they share a conduction system, beat at the same time and are attached, they really are two separate pumps.

Blood returning from the various tissues and organs throughout the body is pumped by the right ventricle through the lungs into the left side of the heart, which in turn, pumps it back out to the body. If the left ventricle fails, it will not be able to get rid of all the blood that is pumped to it by the right ventricle via the lungs. As the right side pumps more than the left side is able to clear; blood accumulates, backs up, in the blood vessels that lead to the left ventricle.

At first, those large pulmonary veins are able to expand to accommodate the extra fluid, but

as they reach their limit, smaller and smaller vessels become dilated and eventually plasma starts seeping through their thin walls.
As fluid seeps through the vessel walls, it first accumulates in the space between the smallest vessels (capillaries) and the small airspaces inside the lungs (alveoli). This creates fluid accumulation and swelling inside the lungs (edema).

When fluid first starts to back-up into the tiny vessels of the lungs, the lungs become 'water logged' and heavy, which will cause difficulty breathing as well as increase the amount of work required to breath. Imagine the lungs like two soaked sponges, heavy with water. The patient will begin breathing faster (tachypnea) and will definitely 'feel' short-of breath (Ware & Matthay 2005). Because the small airways have become narrowed by the swelling within their walls and the fluid acts as an irritant to the smooth muscle in the bronchial tree, there may be wheezes present. Wheezing caused by early stages of pulmonary edema is sometimes referred to as cardiac asthma.

If the fluid continues to back up, fluid will leak right into the alveoli, essentially 'flooding' the lungs. As air moves through the fluid in the alveoli crackles will be heard and the patient may even start coughing up pink frothy sputum (Ware & Matthay 2005). The tiny vessels in the lungs allow fluid to seep through their wall whilst retaining larger particles like red blood cells. Consequently, the fluid that leaks into the alveoli is mostly plasma, which is why the patient won't cough up bright red blood but pink frothy sputum. Oxygen has a hard time getting from the lungs,through the fluid, into the blood stream, so oxygen saturation will drop and the patient will get very short of breath.

As a result of the left ventricle failing, (remember that is what started this whole mess) the body's compensatory mechanism (fight or flight response), starts working overtime (no doubt also fueled by the shortness-of-breath). As the fight-or-flight response floods the body with epinephrine and nor-epinephrine, surface vessels in the skin are constricted and the kidneys begin to retain fluid volume. The heart starts to beat faster and tries to beat more forcefully, therefore creating even more work for the already failing left ventricle. The end result is more back-up into the lungs, a worsening of the patient's condition and a sense of panic. Due to this strong reflex response, many patients with pulmonary edema have very high blood pressures during their episodes.

The Deadly Half-dozen

The six most common causes of shortness of breath (SOB) seen in the emergency department are;

- Cardiogenic Pulmonary Edema
- Pneumonia
- COPD Exacerbation
- Pulmonary Embolism
- Bronchitis
- Acute Asthma

Adding to the complexity, almost half of the patients that are seen in the ER for SOB have more than one diagnosis.

Pneumonia

Pneumonia is an inflammation or infection of lung tissue. It is a very common affliction among the elderly, in fact the fourth leading cause of death in that age group and has sometimes been referred to as *'the old man's friend'* (Beers & Berkow 1999). Pneumonia can be localized to one particular lobe or be evident in several areas of the lung. The two main types of pneumonia are those caused by a bacteria and those caused by a virus.

Generally viral pneumonia will begin with upper respiratory tract symptoms such as a runny nose, sore throat and a cough (Copstead & Banasik 2005). Even though viral pneumonia may eventually lead to wheezing and crackles, the cough usually remains non-productive. The non-productive cough is a main differentiating feature from typical bacterial pneumonia, which usually has a cough productive of yellow, green and/or blood streaked sputum (Copstead & Banasik 2005). However, many of todays atypical pneumonias typically do not have a productive cough either and may, in fact not have a cough at all.

The inflammation causes the pulmonary vessels to become leaky and fluid leaks into the interstitial spaces around the small airways and alveoli. In bacterial pneumonia, the alveoli often fill with pus as well. The infection usually has predictive systemic effects; fever and chills, aches and night sweats (Hoare & Lim 2006). Although pneumonia can occur in any age group, the elderly and those with underlying medical conditions are at higher risk.

Aspiration

Aspiration pneumonia is a particular category of pneumonia that is commonly encountered by Paramedics. Aspiration pneumonia can develop with relatively small amounts of aspirated materials. The oral cavity is often teeming with bacteria, especially in those with poor oral hygiene. Aspirating even small amounts of oral secretions introduce the bacteria into the lungs and

consequently leads to pneumonia. This is sometimes referred to as *'silent aspiration'* and is related to alcohol, sedatives or narcotics use as well as CVAs or esophageal disorders (Beers & Berkow 1999).

Larger quantities of aspiration occur with conditions that compromise consciousness or in those who have difficulty with swallowing (Beers & Berkow 1999). In these cases, larger amounts of food or fluids can end up in the lungs.

Another related condition is called *'aspiration pneumonitis',* which is caused by stomach contents being aspirated into the lungs. This can occur as a result of active vomiting or passive seepage of fluid from the esophagus. The stomach contents are very acidic and the acids essentially cause chemical burns in the lungs that lead to associated inflammation (Marik 2001). Amounts as small as two table spoons of gastric contents is enough to cause aspiration pneumonitis in adults (Marik 2001)!

Symptoms of aspiration pneumonitis usually develop in two phases; the first phase begins one to two hours after the aspiration and the second phase after four to six hours (Marik 2001).

In summary; infection from aspirated bacteria from the oral cavity leads to aspiration pneumonia, inflammation from the acidic stomach contents leads to aspiration pneumonitis.

COPD Exacerbation

Chronic Obstructive Pulmonary Disease (COPD) is one, or a combination of, emphysema or chronic bronchitis. Various resources may also include asthma in this category but some experts argue that asthma by definition has symptom free periods and is thus not truly a chronic disease. See the table below for a quick summary of the underlying etiologies of COPD.

Since COPD is a slowly progressing, chronic disease, it is unlikely that a COPD patient would call EMS because of their COPD alone. However, because COPD interferes with the lungs ability to defend themselves against infections, COPD patients are prone to lung infections which exacerbates their underlying disease and can render them acutely short of breath.

On an average twice a year, a COPD patient will experience an exacerbation (Stoller 2002). Characterized by increased dyspnea, increased sputum production and/or the sputum turning yellow; COPD exacerbations can render a patient hypoxic and hypercarbic (elevated CO_2).

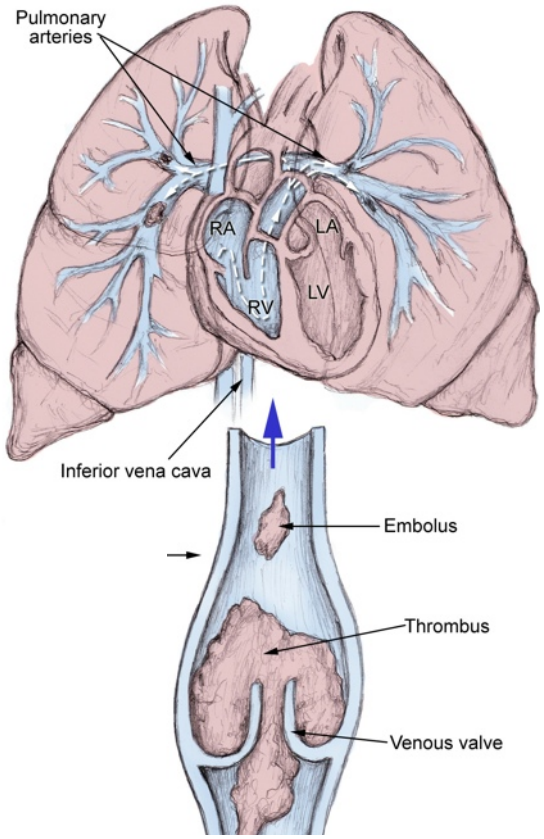
Pulmonary Embolism

Simply stated, a pulmonary embolism is a blockage of a pulmonary artery by an object; which is almost always a blood clot. Blood clots that end up in the lungs must logically come from the venous system or the right atria or ventricle.

Think of the lungs vascular tree as a large filter where blood from the right ventricle travels down ever finer branches of the pulmonary arteries until they are forced into the capillaries. The capillaries are so small that the red blood cells are literally forced down to a single file. A blood clot that flows into that vascular tree will be caught at some level in that vast network of vessels.

The blood clots that eventually get caught up in the lungs usually originate in the deep veins of the lower legs. Most medics have probably seen the red, swollen calf effected by a Deep Vein Thrombosis (DVT). However, it is important to note that many DVTs are completely asymptomatic. Until they let go that is, and get lodged in the lungs.

Chronic Bronchitis (Blue Bloater)	**Emphysema** (Pink Puffer)
Obstruction is due to narrowing of the airway lumen by mucosal thickening and excess mucus.	Dilation and destruction of air spaces distal to the terminal bronchiole. Decrease elastic recoil of lung tissue causes airway collapse and air trapping.
Productive cough on most days for at least three consecutive months in two successive years.	Dyspnea, tachypnea, minimal cough
Usually overweight and cyanotic.	Usually underweight and barrel chested.



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The effects of a blood clot lodged in a lung will vary widely on the size and location of the clot. A large pulmonary embolism can get lodged in the first division of the pulmonary artery where the main artery that exits the right ventricle splits into the left and right pulmonary arteries. This type of embolus is called a saddle embolus and usually causes the immediate dilation and failure of the right ventricle as it tries to pump blood against a complete obstruction. The patient will die immediately. Basically, large embolisms cause a circulatory problem with hypotension and syncope, and smaller embolisms cause pleuritic chest pain with fast breathing rates and tachycardia.

When the lungs detect a loss of blood flow to a segment, the body responds by closing the airways to that area, since ventilating airspaces is a waste without blood flowing through their adjacent capillaries to be oxygenated. The result of this bronchoconstriction can be heard as localized wheezes. A pleural friction rub can sometimes also be heard over the area as the lung surface becomes inflamed. The patient may also cough up blood (hemoptysis).

Bronchitis

Acute bronchitis is usually caused by a viral infection and causes widespread inflammation of the trachea and bronchial tree. The inflammation causes the blood vessels that surround the small airways to swell and leak, causing a narrowing of the airways. The difference between pneumonia and acute bronchitis is determined by chest x-ray that will show exudates in the lungs with pneumonia, but will look normal with bronchitis. Usually bronchitis is mild and will resolve with no treatment.

Acute Asthma

The most common trigger of an asthma attack is a respiratory infection. Other common triggers are allergens, exercise, cold air, humid air, air pollution, aspirin, dyes and chemicals. The coughing, wheezing and shortness of breath that marks an asthma attack result from two main factors; bronchial smooth muscle contraction and airway inflammation.

The first phase of an asthma attack is caused by the bronchial smooth muscle contraction and can usually be reversed with ventolin. The second phase is caused by inflammation which cause swelling of the airways, mucous plugging and further bronchospasm. The second phase usually requires treatment beyond ventolin administration, such as anti-inflammatories.

This two phase concept clearly has implications for the prehospital setting as patient's may feel that their condition improves enough to not warrant transport to the hospital after an initial treatment with ventolin. As always, it is the Paramedic's responsibility to fully inform the patient of the risks involved in refusing transport. In the case of an asthmatic, the risk of having a secondary attack, which will be resistant to EMS treatment, must be carefully explained to the

asthmatic patient and all efforts must be made to convince the patient to come to the hospital.

It is important to note that during bronchospasm, as the patient inhales, the lungs expand and the narrowed airways open somewhat, letting air flow into the alveoli. As the patient exhales, the lungs constrict and those narrowed airways close, trapping air and causing resistance to exhalation. So, the problem with an asthmatic is getting the air out of the lungs, not in. Unfortunately, the only assistance we can lend with a BVM is with getting the air in. Therefore, using a BVM on an asthmatic person is likely to worsen their situation as more air is forced in to the already overinflated lungs. If enough air gets trapped in the lungs pressures can get so high that blood flow through the pulmonary vascular tree completely stops. This will result in pulseless electrical activity as the empty left ventricle beats fiercely while the right ventricle dilates and eventually fails.

There are times when there is no choice but to assist the asthmatic patient with ventilations, but it should be considered a last resort. Guidelines on when to start assisting ventilation are somewhat vague. AHA suggests intubation if, *'..the asthmatic patient deteriorates despite aggressive management'*. They go on to suggest that the asthmatic patient is ventilated at a slower rate of 6-10 / minute, allowing more time for exhalation. To further assist you in making the determination of when an asthma attack is really severe,Barry E Brenner a PhD and ER physician writes on eMedicine;

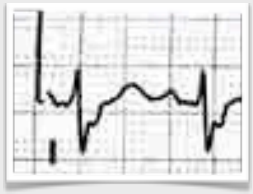
> *'If symptomatology becomes more severe, profuse diaphoresis occurs. The diaphoresis presents concomitantly with a rise in PCO2 and hypoventilation. In the most severe form of acute asthma, the patient may struggle for air, act confused, agitated, pulling off his oxygen stating, "I can not breathe." These are signs of life-threatening hypoxia. With advanced hypercarbia, bradypneic, somnolence, and profuse diaphoresis may be present; almost no breath sounds may be heard, and the patient is willing to lie recumbent.'*.

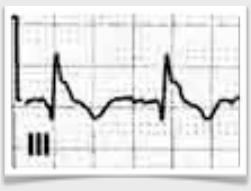
Can a pulmonary embolism be found on a 12 lead ECG?

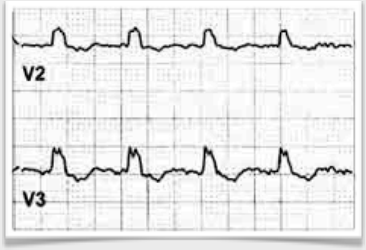
Wouldn't it be nice if we could spot a pulmonary embolism on a 12 lead ECG? At first this may seem like a crazy idea since the clot is lodged in a lung, hence the word 'pulmonary' in pulmonary embolism. Nevertheless, this might not be as crazy as it seems. If the embolism is large enough, it will cause increased resistance to blood flow out of the right ventricle. The obstructed flow will in turn cause the right ventricle to strain, and that strain can be detected on a 12 lead.

So, indirectly we can find evidence of a pulmonary embolism on a 12 lead. In Skejby Denmark, Paramedics have a pulmonary embolism bypass directive where the 12 lead is transmitted to a physician when a PE is suspected. If the physician agrees with the diagnosis, the patient is by-passed to a cardiac care unit where urgent CT scanning, echocardiography and, if required, thrombolysis is performed (Terkelsen et al 2008).

The pattern to look for is called $S_1Q_3T_3$ which stands for negative 'S' waves in lead I, 'Q' waves in lead III and inverted 'T' waves in lead III (Daniel et al. 2001). In addition to the $S_1Q_3T_3$ look for flippedT waves in leads V1 through V4 and right bundle branch blocks (Daniel et al. 2001). As with any other ECG finding these findings need to be considered in the context of the rest of the patient presentation. Most PEs present with normal 12 lead ECGs or sinus tachycardia only.







Assessment Insights

As mentioned in the opening paragraphs of this reading material, arriving at the correct working assessment and hence, the correct treatment plan, with the short-of-breath patient can be very challenging. There are no shortcuts or absolutes, the whole picture needs to be considered before jumping to a treatment plan. The most challenging short-of-breath patient to figure out might be the elderly patient who is severely short of breath with crackles. This type of patient might have acute pulmonary edema, a COPD exacerbation or pneumonia and often has more than one of these issues at the same time, all contributing to their symptoms and presentations. The treatments are different, so EMS providers are forced to make an informed guess as to what is the main culprit in a very limited time, with no diagnostic tools other than a stethoscope, monitor and blood pressure cuff.

Let's walk through a call, from entering the room and first seeing the patient, to making our informed guess, and highlight what to specifically look for and ask about along the way.

General Impression

A ton of important information can be obtained as we first walk into a patient's house. Is the smell of cigarette smoke almost knocking us over? Is there an oxygen machine purring away in the living room? The presence of either, makes the probability of COPD much higher. Where do we find the patient and what position are they in? A patient sitting bolt upright with a look of panic on their face, makes acute pulmonary edema more likely. On the contrary, a patient lying in bed, lethargic and ill-looking would make pneumonia a much more likely culprit.

The skin colour and condition also provide important clues. Pale skin with diaphoresis is typically seen with acute pulmonary edema. A flushed appearance is more typical of pneumonia.

Jugular Venous Distention (JVD)

Jugular Venous Distention (JVD) is typically associated with pulmonary edema. However, a word of caution is in order here. The presence of JVD when a patient is in a sitting position is a reliable sign of elevated venous pressure, but what this truly indicates is a back-up of fluid behind the right ventricle. Any problem 'downstream' from the right ventricle could be to blame. A pulmonary embolus might be obstructing flow through the lungs causing JVD.

COPD causes chronic pulmonary vasoconstriction from the constantly low oxygen levels. This widespread vasoconstriction within the lungs cause impeded flow and leads to elevated venous pressure and resultant JVD.

A right ventricular MI would also cause JVD as fluid backs up. So, even though the JVD could be caused from a failing left ventricle that has fluid backing up through the lungs, right ventricle and into the venous system, there are many other possibilities to consider.

Breathing Mechanics

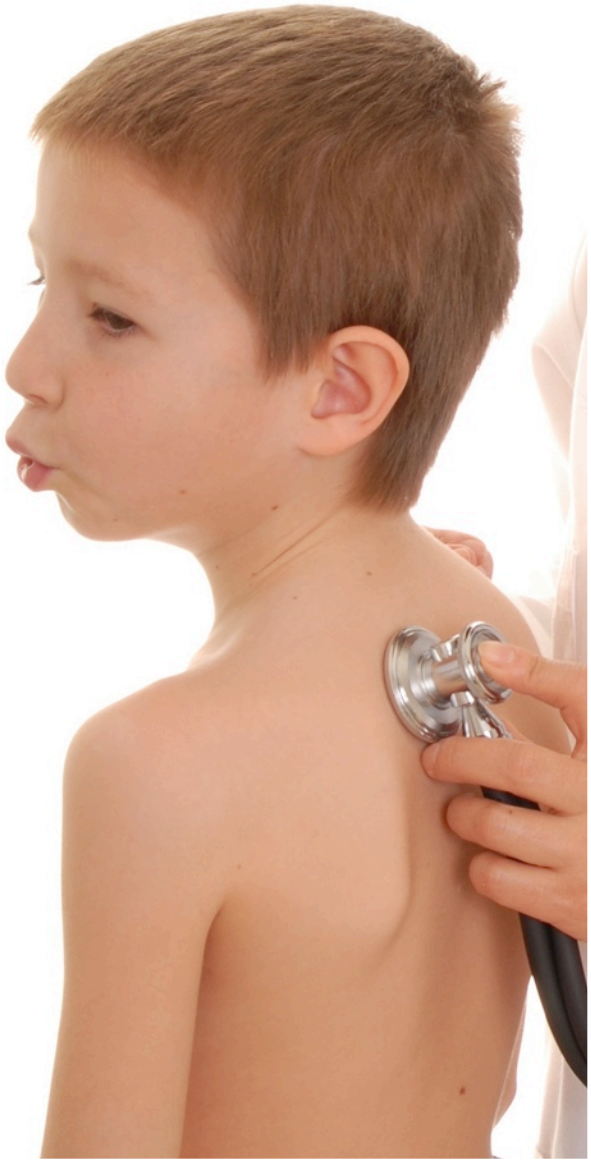
The breathing mechanics is another factor worth taking note of. A forced, prolonged expiration is a sign of lower airway (bronchial) obstruction. Unfortunately, this breathing pattern is seen in most of the conditions that we are attempting to tell apart.

Difficulty with inspiration, especially if accompanied by stridor, should alert you to the possibility of an upper airway obstruction. An often overlooked, but important objective assessment finding is the respiratory rate. A baseline respiratory rate will help determine how severe the condition is and also, give us a comparison to measure the effectiveness of our interventions.

Lung Auscultation

Lung auscultation is a useful assessment tool but it does have some significant limitations. Several studies have shown that medical providers have difficulty with accurately assessing breath sounds.

Researchers in Switzerland conducted a study to determine how much lung auscultation contributed to the final diagnosis in patients who presented to the ER with shortness of breath. In the article, published in 2005, they found that



auscultation only increased the accuracy of their diagnosis by one percent and actually worsened it in three percent of the cases (Leuppi et al. 2005). To make matters worse, research from 1995 showed that Paramedics were not as good as Physicians at identifying breath sounds, regardless of the experience level of the Paramedic (Wigder et al. 1995). Interestingly, if the Paramedics knew the patient's medical history, the accuracy increased significantly (Wigder et al. 1995). Furthermore, in the back of a moving ambulance, even with lights and sirens off, the ability to hear breath sounds drop from ninety percent, (in a quiet environment) to nine percent (Brown et al. 1996).

The research is unequivocal about emphasizing good technique in order to glean the limited benefit that auscultation can afford. It is also imperative to consider the breath sounds in the context of the medical history and not base the entire working assessment on auscultation alone.

To stack the odds in favour of accurately hearing breath sounds, do not engage in what was dubbed by one Physician, 'AWFUL' auscultation (Auscultation With Fabric Upon Lungs) (Sherman 2009). Listening to a patient's lungs through layers of clothing is not uncommon among any level of health care provider.

Proper landmarking, placing the stethoscope on the patient's bare skin and listening with focused intent are all habits that will help improve the chances of an accurate auscultation. Instruct the patient to take slow, deep breaths (Kiyokawa et al 2001).

Most Paramedics are very familiar with crackles and wheezes. However, a more subtle breath sound that may be present in pneumonia or some other, rare conditions is *bronchial* breath sounds when they are heard in the periphery of the patient's lungs. Bronchial breath sounds are the tubular, harsh sounding air movements that can normally be heard over the trachea, or bronchus. When those sounds can be heard in the periphery of the lungs it is a sign that the airspaces are filled with fluid or pus, indicative of pneumonia (Metlay et al 1997). For an in-depth review of advanced chest assessment check out some of our past mail-out packages on our website www.cepcp.ca.

Pneumonia typically presents with decreased air entry, bronchial breath sounds or crackles. The sounds are usually localized to the area of the lung affected (Hoare & Lim 2006). However, patients may have bilateral pneumonia, so it is not possible to rule out pneumonia based on bilateral crackles alone.

Pulmonary edema will often present with expiratory wheezing in the early stages and then progress to inspiratory crackles as fluid floods the alveoli.

Chronic bronchitis and asthma will usually present with expiratory wheezing. Non-exacerbated emphysema will usually have generally diminished, distant breath sounds that may be very difficult to decipher. Since COPD exacerbations are usually caused by a lung infection, we can expect to hear the same types of sounds, as in pneumonia in cases of COPD exacerbation.

Dependent Edema

Dependent edema is also associated with congestive heart failure and hence, is often linked to pulmonary edema. However, like with JVD, dependent edema indicates a chronic fluid back up behind the right ventricle and might not be caused by the left ventricle.

Long-standing COPD can lead to right ventricular failure due to increased flow resistance through the diseased lungs (cor-pulmonale) with resultant dependent edema. It is a good idea to assess the ankles, and lower legs for edema ask if they have been more swollen lately and use this information in the context of the rest of the picture. As far as differentiating COPD exacerbation from pulmonary edema, leg edema is of limited usefulness.

Incident History

The incident history is one of the most important factors in determining the cause of a patient's shortness of breath. The OPQRST-A mnemonic is one useful tool to ensure nothing is missed. Another method is to use a chronological approach to questioning;

'How long have you been short of breath?'
'What were you doing when it started?'
'*Did it start suddenly or over a period of time?'*
'How severe is this episode in comparison to past episodes?'
'Does anything make it better?'
'What made you decide to call 911 now?'

Basically asking questions about the onset, through to the current symptoms.

The timing of the onset is quite important. Pneumonia or COPD exacerbations rarely come on all of a sudden, but acute pulmonary edema often does.

Another key question, is if the patient has had a productive cough, and if they have, what the colour of the sputum has been. A dry night-cough is suggestive of developing pulmonary edema (Stein 1998; Jaski 2000). In the later stages, the cough might become productive of frothy, pink sputum (Stein 1998; Ware & Matthay 2005).

On the other hand, if the patient tells you that he/she has been coughing up yellow or green phlegm, it is much more likely to be pneumonia or a COPD exacerbation that is causing the patient's symptoms (Anthonisen 2002; Barnes 2000).

Clearly, a fever indicates an infectious process, so specifically ask about night sweats and chills.

Orthopnea and Paroxysmal Nocturnal Dyspnea (PND) are two related but quite separate phenomenas. Orthopnea is defined as shortness of breath that occurs, or gets worse, in the supine position. Orthopnea is believed to be caused by fluid shifting from the lower extremities to the pulmonary vasculature as the patient assumes the supine position. Asking how many pillows the patient normally sleeps on is a great way to establish if they have orthopnea, which usually suggests chronic CHF (Ware & Matthay 2005). If they do have orthopnea, it is helpful to determine if it has gotten worse lately. The answer can be reported as, *'The patient reports three pillow orthopnea for the past two weeks...'*. PND on the other hand, is defined as waking up in the middle of the night acutely short of breath. The shortness of breath usually resolves after the patient sits up for awhile. PND is strongly associated with heart failure as well (Ware & Matthay 2005; Jaski 2000; Stein 1998).

Asking the patient whether they have chest pain or not and to describe the chest pain if they are having it, is another important line of questioning that can help clarify the picture. Chest pain that is ischemic; (steady, pressure, tightness, radiating to neck/shoulder), may indicate that the patient is suffering from angina or an MI. Either condition can cause the left ventricle to fail and directly cause heart failure (Jaski 2000; Ware & Matthay

2005). However, if the patient reports pleuritic chest pain (sharp, increasing with cough/breath), it is much more likely to be pneumonia or a pulmonary embolism that is causing the patient's shortness of breath (Fedullo & Tapson 2003; Halm & Teirstein 2002; Moffa & Emerman 2004).

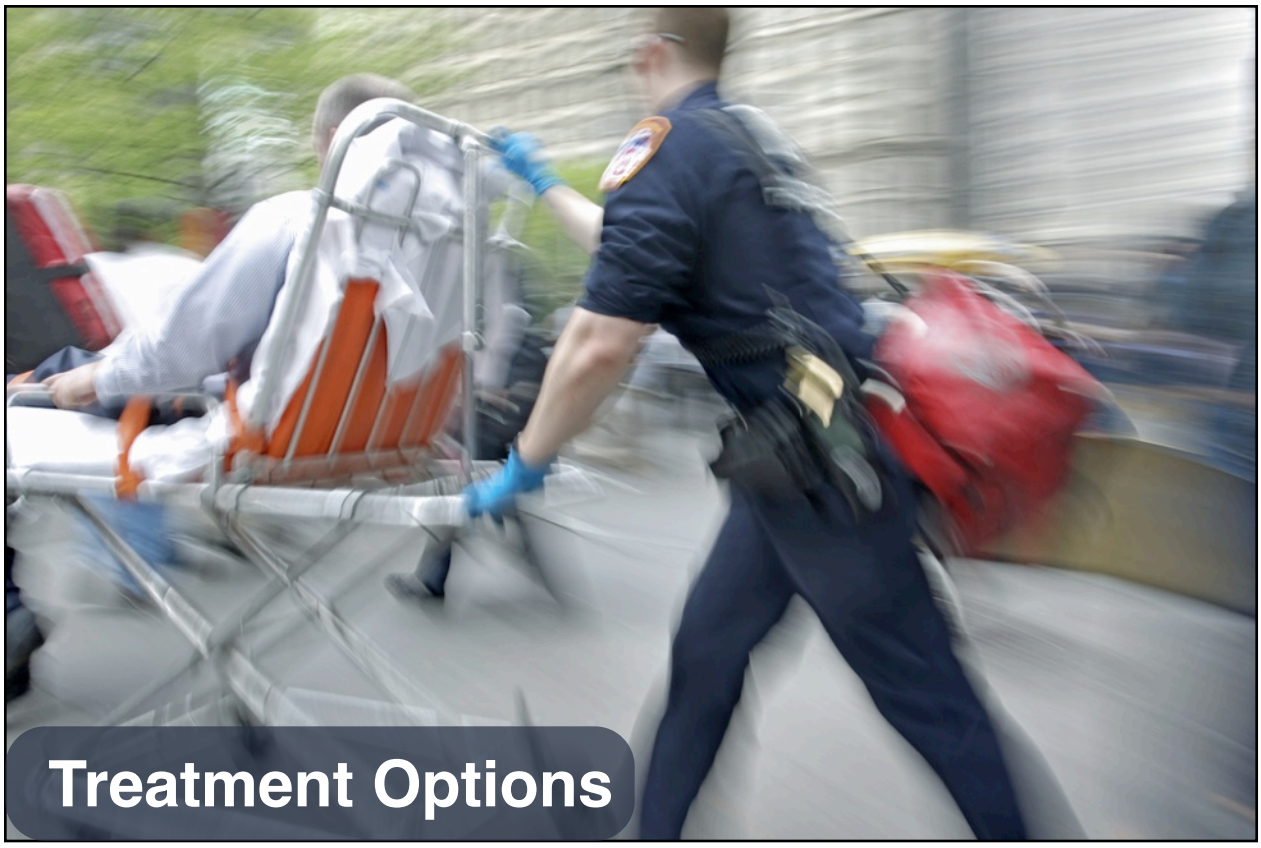
Another simple set of questions that can yield a wealth of useful information, is simply asking the patient if they have had anything similar happen in the past. If they have, what was the diagnosis, and how the current episode compares to past episodes. Keep in mind that pulmonary edema is often referred to as 'water on the lungs' by patients. Asking if the patient has ever been admitted to the ICU and/or have had to 'have a tube put down into their lungs to help them breath' will give you an idea of how severe past episodes have been.

With a thorough incident history you can hopefully categorize the patient's shortness of breath and treat it accordingly. However, often a patient with COPD will also have heart failure. If that patient develops pneumonia it can be enough to cause the patient to develop pulmonary edema. Clearly a patient with a history of all those conditions and a mixed bag of signs and symptoms that does not neatly fit into any category are the most challenging to treat.

Past Medical History

Although spending time determining a patient's past medical history might seem a bit inappropriate when they are struggling to breathe, it is very important and will provide you with a much fuller picture of why the patient may be short of breath. Any heart related history (angina, heart attacks etc), puts the patient at greater risk for developing pulmonary edema (Jaski 2000; Ware & Matthay 2005). Hypertension and diabetes are two other common underlying medical problems in patients with pulmonary edema (Jaski 2000).

On the contrary, if the patient has a history of COPD and/or frequent lung infections, it becomes much more likely that the shortness-of-breath is a complication from those conditions, rather then pulmonary edema. In addition, always consider aspiration pneumonia if a patient is bedridden or otherwise unable to swallow well. The majorities of pneumonias in nursing homes are due to aspiration and can occur without the patient ever coughing or gagging during feeding (Anderson 2004).



Treatment Options

Oxygen

The treatments available to Paramedics for patients with shortness of breath is oxygen, assisted ventilations, CPAP (in some areas), nitroglycerine or ventolin. Let's briefly review these options and see which are appropriate for which condition.

Oxygen, goes without saying, should be given to any patient with acute shortness of breath, the BLS PCS clearly establishes this as a standard of care. When treating patient's who are clearly having an asthma attack it may seem appropriate to forgo supplemental oxygen administration and focus efforts on delivering salbutamol instead. Nevertheless, treating a hypoxic asthma patient with salbutamol alone, without supplemental oxygen is dangerous and could result in a further drop in oxygen saturation and even cardiac arrest (Burggaaf et al 2001). The reason for this potentially life threatening reaction to salbutamol is not well understood but appears to be related to the cardiovascular effects of salbutamol leading to shunting (Burggaaf et al 2001). To avoid this effect, ensure supplemental oxygen is administered before and along with, your salbutamol treatment.

BVM Ventilations

Deciding to assist a patient's respirations with a BVM is a difficult decision that requires careful consideration of the entire patient presentation and underlying pathology. The BLS-Patient Care Standards simply state;

> *'Ventilate or assist ventilation if the patient is apneic or breathing appears inadequate as evidenced by signs of hypoxia (e.g. decreased LOC, cyanosis) according to current HSFO Guidelines.'*

A simplified statement that really doesn't address the issue in sufficient depth to assist much in the decision making. It is imperative to keep in mind that a patient's oxygenation may be adequate despite poor ventilation. Ventilation is more related to CO_2 levels than O_2 levels. In other words, a

patient may need to be ventilated despite adequate SpO_2 levels. For example, in the setting of acute pulmonary edema, a patient works harder to breathe causing rising CO_2 production. At the same time, it is difficult for the patient to breathe which limits CO_2 elimination and there is also a rise in dead space, all contributing to rising CO_2 levels (Estenne & Yernault 1984).

Look for signs that the patient is getting tired of breathing and seems to be giving up. If you decide to assist ventilations, explain what you are doing to the patient and warn them that it might feel confining at first. Once you apply the mask, apply very light pressure on the BVM to be able to feel the 'give' in the bag as the patient inhales. Every second or third breath 'chase' their inhalation with the bag and try to increase the tidal volume at the end of their inhalation, starting gently then adding a bit more volume as the patient gets used to the sensation.

Boussignac CPAP Device

Continuous Positive Airway Pressure (CPAP)

CPAP will probably replace assisted ventilations to a large degree. In a 2008 landmark study researchers in British Columbia and Nova Scotia undertook a large, well designed pre-hospital study to investigate the benefit of pre-hospital CPAP (Thompson et al 2008). The results were incredible. The number of intubations dropped by 31 percent, and the mortality was 21 percent lower in the group treated with CPAP (Thompson et al 2008). Very few interventions have been able to demonstrate such a high success rate with almost no complications.

The total cost (including training) of CPAP is relatively low (~$ 89 USD / patient) (Hubble et al 2008). One study calculated a cost of $495 USD per life saved (Hubble et al 2008). The cost to the community vastly offset by the lower hospitalization costs (Hubble et al 2008). As a result, most EMS systems in Ontario are now incorporating CPAP as a treatment modality for patients in acute respiratory distress from COPD exacerbation or acute pulmonary edema.

Nitroglycerine

Nitroglycerine, once reserved for patients suffering chest pain, has now become a well established, effective first line treatment for acute pulmonary edema. Nitroglycerine is an effective venodilator that reduces the amount of fluid that enters the heart (pre-load), which reduces workload and the back-up that is occurring during acute pulmonary edema.

In addition,many cases of acute pulmonary edema are thought to be caused by mitral valve prolapse, that essentially causes the left ventricle to pump blood backwards into the lungs. Nitroglycerine is also effective in reducing this backwards flow (Pierard & Lancelloti 2004).

Sabutamol (Ventolin)

Salbutamol has been a long standing treatment for patients with bronchoconstriction. Salbutamol works by activating beta receptors which are abundant in bronchial smooth muscle, causing them to relax and the airways to consequently widen.

Salbutamol also has many hemodynamic effects, some which would appear to make it a good choice for acute pulmonary edema patients (peripheral vasodilation, increased alveolar fluid clearance, increased stroke volume). Although it is commonly used for the treatment of acute pulmonary edema in-hospital, it has not been well researched. One multi-center trial in the US established that acute pulmonary edema patients that were given bronchodilator treatment by EMS or in the ED had a higher need for aggressive interventions later in their hospital stay (Singer et al 2007). However, the trial was not designed to assess whether the bronchodilators caused the problems. It is quite possible that the sicker patients were more likely to have been treated with bronchodilators. Regardless, there is not enough solid evidence to support routine use of salbutamol in cases of acute pulmonary edema in the pre-hospital setting.

Salbutamol is most effectively delivered using a metered dose inhaler (MDI) and spacer chamber (Closa et al 1998; Deerojanawong et al 2004; Rubilar 1999). The commonly held idea that nebulized salbutamol is more effective than the MDI/spacer-chamber technique is not supported by any evidence. This long held belief might stem from a poor understanding of how to use the MDI and spacer-chamber effectively. Used correctly, the MDI / spacer-chamber is a safer, more effective delivery method and should be used whenever possible.

Meanwhile...

Frank was on the verge of full blown panic by the time he saw the wintery street outside light up in a welcoming red and white dance of lights. He was surprised at how loud the ambulance sounded and was puzzled by the sound of air brakes hissing after the vehicle came to a stop outside. He couldn't see the driveway from where he was sitting, but he had worked as a driver for Purolator for several years and none of the cube vans had been equipped with air brakes. *'Its the fire department.'* Margaret, peering out the window, said with a mixture of relief and surprise. *'The fire department?'*, Frank gasped. *'Oh, I guess they come too'*.

Margaret pulled her robe tight and quickly made her way downstairs to open the front door. The firefighters were directed to the bedroom and quickly went to work, moving awkwardly in their bulky bunker gear. By the time Tony and his partner arrived minutes later, Frank's medications had been gathered and placed in a large ziplock bag along with his health card. Frank was also being given oxygen via a non-rebreather.

The higher than normal oxygen gradient in the alveoli, provided by the supplemental oxygen, were causing enough of a concentration difference to enhance the diffusion of much needed oxygen through the fluid-filled interstitial spaces and into the blood stream. The higher oxygen levels were reducing Frank's sense of panic and also starting to wind down the sympathetic response that was still causing Frank's skin to glisten with sweat. The changes taking place were hardly enough for Frank to notice though, he was still struggling.

As soon as Tony saw Frank and his level of distress, he instructed a fire fighter to grab the stair chair from the ambulance and get the stretcher ready. He knew they had to move quickly. Tony took notice of Frank's positioning; bolt upright on the side of the bed, his hands on his knees in the classic tripod position. His skin pale and sweaty. The positioning alone told a story of an acute process, less likely to be pneumonia or anything else infectious. Tony also noticed the three pillows

forming a slope between the oak headboard and the mattress, still soaked with sweat. *'Three pillow orthopnea.'* he thought to himself.

He quickly realized that he would have to limit his questions to those that could be answered with a simple 'yes' or 'no' as Frank's shortness of breath prevented him from speaking more than one or two words at a time. Through a combination of asking Margaret, as well as Frank, some focused, direct questions, it was soon clear that Frank had been increasingly short of breath over the past couple of days and had awoken several times lately with a sense of suffocation and a non-productive cough. Tonight's episode was a lot more severe than anything he had ever experienced before and it had come on suddenly.

A quick review of Frank's medications revealed a few antihypertensives, nitroglycerine spray and a cholesterol medication. There were no inhalers of any kind. As Tony's partner obtained a blood pressure, Tony auscultated Frank's chest. By now, Tony already knew what they would find but still ensured that he didn't rush through the assessment. Tony had been around long enough to know that all that quacks, is indeed, not a duck. However; coarse, wet-sounding, inspiratory crackles helped seal the deal.

Tony watched his partner pump the blood pressure cuff, stop, pump a bit more, stop, then pump a bit more. Tony wasn't surprised, he knew Frank's blood pressure was probably going to be sky high and he was right, 230/110 was the final verdict. *'Good..'* Tony thought to himself *'...lot's of room to bring the pressure down with nitro. Nothing worse than a patient in pulmonary edema with no blood pressure'*.

By the time they moved Frank down to the ambulance, it was time for his second dose of nitroglycerine. Tony also moved quickly to set up the CPAP mask, explaining everything to a grateful Frank who was feeling like he had just been thrown a life jacket.

After another five minutes, Tony already saw some improvement in Frank's condition but determined that he was still in a lot of distress. His blood pressure had come down to 190/98. Tony administered another dose of nitro, then reapplied the CPAP mask increasing the pressure to 10 cmH_2O.

The nitroglycerine was dilating Frank's veins, reducing the raging flood waters that had been flowing into his lungs. The CPAP was creating a barrier against the flooding, making the fluid recede back into the vascular spaces. The pressure was also splinting the small airways open, acting like scaffolding allowing the escape of trapped, stale air during exhalation. Frank was starting to feel much better. The sense of panic was subsiding, he finally felt like he might make it.

Later, as the ambulance smoothly raced through the still deserted morning, Tony glanced out the back window where the first hint of a sunrise was starting to be visible. Looked like it was going to be a beautiful day.

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