Survey of Human Diseases
Module 3/Week 7, Chapter 12
Respiratory System Presentation

>> PROFESSOR: Today's lecture is going to be on chapter 12, the respiratory system. Basically, for the respiratory system, there's two things that happen. Basically there's ventilation and respiration. Okay? What happens is we have to get oxygen from the air into our circulatory system, and of course the circulatory system then transports the oxygen in the bloodstream to the tissues.

Now, the way the lung is made up is it's once again the main highway, interstate 75 is going to be the trachea, and then the trachea branches off to the bronchi, which are slightly smaller in diameter, and then it goes to the bronchioles, and then you have the terminal bronchioles, which are the smallest of the bronchioles, which basically are at the end of the terminal bronchioles, and they have alveolar sacs in the walls of the respiratory bronchioles. All right? And then from the respiratory bronchioles, you go further to the alveolar ducts and sacs, all right, and we have a diagram, which I'll show you. But once again, it's like, you know, the transportation system, I-75, the interstate is the large passageway, and then you start narrowing down. You go from a six-lane highway to four lanes to two lanes, and maybe the alveolar ducts are the equivalent of a small dirt road, a single-lane dirt road. So in the alveoli, there's the exchange of 02 and CO2, and there's a small septum in between the air and the capillary system carrying the red blood cells. Okay?

And for the alveolar sacs to stay open, all right, the -- there are cells in the alveolar sacs that produce surfactant. All right? And we'll talk more about surfactant a little bit later in this lecture, but what surfactant does is serves to reduce the surface tension. An example of reducing surface tension is if you take water, okay, you really can't blow bubbles with water. But if you introduce a detergent into the water, you can blow bubbles. And so the reason why you can make bubbles is because of the fact that the surface tension is reduced. And so surfactant helps to reduce the surface tension so it makes it easier for the alveolar sacs to stay open.

So the lungs divided into lobes, and then smaller units called lobules, which I don't really consider that significant. You don't have to know that type of terminology. So here you have your diagram from the trachea, the bronchi into the bronchioles, and then from the bronchioles, you get the terminal bronchioles, and you get a little further, you see the respiratory bronchioles, and
right above that you see the two little outpouchings and those are alveoli. Okay? And then the other side of the diagram, the multi-colored shows you the alveolar sacs being supplied with oxygenated and venous blood, so you've got that reddish orange, and then that blue. The capillary network, which is on the surface of the alveoli. And then the bottom cluster, what that shows you is the alveolar sacs. Okay? The alveolar duct and the alveolar sacs.

So there's two functions, as I mentioned, respiration the ventilation. And, of course, when you ventilate, you have to take air in, and you have to get rid of air, so it's inspiration, expiration, and it provides for exchange of gas exchange between the alveolar air and the blood in the pulmonary capillaries. Now, the atmospheric pressure at sea level is 700 millimeters of mercury, and basically the partial pressure of oxygen is about 20%. Okay? So 20% of 760 millimeters of mercury comes out to 152 million meters of mercury.

And that's going to be alveolar air. And then in the capillaries, what happens is there's going to be the oxygen pressure of blood in the pulmonary capillaries is going to be 20 millimeters of mercury, so there's going to be a concentration gradient. So there's going to be movement of oxygen into the pulmonary capillaries, and the exact opposite for CO2. The partial pressure of CO2 and capillary blood is around 60 millimeters of mercury, and alveolar air is about 35. So there's going to be transition from the capillary blood, or the CO2 into the alveolar air.

So anyway, requirements for efficient exchange of oxygen, CO2. You need to have a large capillary surface, all right, an unimpeded diffusion across the membrane, and then normal pulmonary blood flow and normal pulmonary alveoli.

All right. Now, there are tests that can be done to evaluate pulmonary function, all right, and they measure the amount of air that can be moved in and out of the lungs, and the term -- I don't really want to go into this in too great a detail. The term "Vital capacity" refers to the maximum volume of air expelled after maximum inspiration. So you take a really deep breath in, and then you exhale all that air, and that's a measure of the vital capacity, and the rest of these, like FEV-1, you don't really have to know.

All right. When you want to measure oxygenation, you need to get arterial gas. All right? Arterial blood gas. So you need to put a needle into the -- into an artery. The common spots for that are the radial artery in the wrist, or in the hospital, in the femoral artery, which is in the groin.

All right. Now, so the pleural cavity, very interesting. The lung sits within the pulmonary cavity, okay, and really,
the lungs flow in the pulmonary cavity. They're really not attached, okay, so the question is why do they stay suspended?

All right. Well, the reason why they stay suspended is that there's a slight vacuum within the pulmonary cavity. The wall of the chest is lined with a very thin saran wrap layer, I've talked about it before, derived from mesothelial cells. They're flat, squamous, epithelial cells. One cell thick. And it's called the pleura. And the lining that covers the chest inside the chest cavity is called the parietal pleura, p-a-r-i-e-t-a-l. And then there's also a layer that covers the outside of the lungs. That's called the visceral pleura, v-i-s-c-e-r-a-l. And the reason for these pleural coverings is they're very smooth like saran wrap, so it reduces friction when the lungs move. Okay? Now, so then what happens is that the diaphragm at the bottom -- is the floor or the bottom of the thoracic cavity. All right? So when you go to take a deep breath, what happens is the diaphragm moves down, the ribcage expands. And the deep breath occurs because in between the visceral pleura and the parietal pleura, there's a very small vacuum, okay, just a couple of millimeters of mercury vacuum. When you increase the space in the chest cavity by your diaphragm moving down, ribs moving up, you're increasing the vacuum. When you increase the vacuum, in other words, you make it more negative, it sucks the lung tissue open, so as a result, it opens up, more air comes in. Then when you exhale, what happens? Exhale all your air. Ribcage goes down, diaphragm moves up and the capacity of the thoracic cavity, chest cavity, decreases. So as a result, the lung tissue then is pulled in, and naturally, the fibers want to suck it in, pull it in, and that's how you exhale. All right? So the pleura is the potential space from the lungs and chest wall.

All right. Let's see. So a pneumothorax, okay, pneumo means air, thorax the chest. What happens is there is the loss of vacuum in the pleural cavity, and this can occur from trauma, a knife stabbing, a fractured rib due to a car accident, and if so, people develop a pneumothorax. Now, when they're admitted to the hospital, how is a pneumothorax treated? What do they do? Chest tube. Right. The purpose of the chest tube gets inserted in the chest, in the back, above the diaphragm, and the purpose of the chest tube is gradually to remove the air, okay, gradually move the air in the pleural cavity and help to re-expand the lung. Okay? By removing the air, you create a negative pressure, a slight vacuum. All right? So a stab wound penetrating injury to the chest, atmospheric air enters the pleural space, or you can have -- I had one patient who had
used to get spontaneous pneumothoraces and what that was is they have blebs on their lungs, and, like, it's just like a pocket of air. It's like a blister, okay? They're from birth. And what happens is they can occasionally rupture. When they rupture, they get a pneumothorax. So initially what happens is pneumothorax are admitted to the hospital, chest tube inserted. After several incidences like that, what happens is there's enough scarring that the lung doesn't really collapse, because there's scarring between the lung tissue and the wall. So as a result, it doesn't really collapse. They can have some symptoms, they've had a pneumothorax, but it doesn't involve the whole lung. So pneumothorax, chest pain, shortness of breath, reduced breath sounds, and as a result, what happens is the lung collapses, okay, and there's in the pleural cavity. Tension pneumothorax, all right, what happens is that there's then a shift into the -- a shift to the mediastinum. So then, like, for example, when positive pressure develops in the pleural cavity. Air flows through a perforation, but then when you inspire. But then when you exhale or expire, it gets sealed, so there's a buildup of tension within the cavity, and as a result, it may cause the mediastinum to shift. Now, in the mediastinum is the heart. All right? The heart, the aorta, the vessels. So that can be a real medical and surgical emergency. Collapse of the lung refers to atelectasis, and typically, atelectasis, maybe it's not the whole lung, maybe just a portion of the lung, and it's usually caused by mucus plugs, mucus obstruction. It can also be by tumor, foreign object. Anybody know what's the most common foreign object to cause atelectasis? Block an airway? Foreign object? Shouldn't be in the lung? Anybody? >> STUDENT: Food? >> PROFESSOR: Right. What type of food? What? Peanuts. How many times you go to a bar or pub and they've got peanuts there and you see people flipping them up in the air and catching them with their mouth? That's how it happens. So anyway, so any time you see that again, you'll remember this lecture. But so atelectasis, obstruction of one of the airways, and as a result, you can't get air and it gradually collapses so it becomes obstructed. Reduced volume of the pleural cavity. It can happen with a mediastinal structure shift. Can develop as a post operative complication. That's why if anybody's had major abdominal surgery, the doctor comes in, they listen to the lungs. Why do they listen to the lungs? Because they want to make sure there's good aeration in both lung fields because you don't want them to develop an atelectasis. If they develop an atelectasis, in other words if they have abdominal surgery
and they have pain, incisional pain, they don't want to take deep breaths because it hurts, what happens then is they develop atelectasis. If they get atelectasis, they can get a fever from the atelectasis, and then you've got a post-op patient with a fever and your concern, is it really atelectasis, or is it incisional infection or is it at the site of the surgery? So you really want to follow patients, make sure they take good, deep breaths post-surgery. And also, if you've ever visited someone after surgery, if they have incentive spirometers, usually at the bedside. And what those are, it's usually a plastic cylinder and it's got a ball in it, plastic ball, with a mouthpiece. A little tube and a mouthpiece, and the patient is instructed to take deep breaths. And the idea is as they take deep breath, they can move the ball up the cylinder. And it's visual so they can see the ball move up. And so it's an incentive for them to try to get the ball all the way to do that and to do that they have to take really deep breaths. By taking deep breaths, it reduces the risk of atelectasis. Caused by shallow breathing.

All right. So let's see. Also, other reasons for atelectasis can be -- okay. Fluid, air or blood in the pleural cavity. Once again, trauma.

All right. Let's see. So here's an example before atelectasis, and then after atelectasis of the left lung. Okay?
And as a result, what you see is increased density of the left lung. Also, if you notice, the left side of the lung, that darkened spot at the base showed you -- that's the stomach bubble. That's going to be elevated. Okay? The diaphragm is elevated so the stomach is pushing up a lot higher than the diaphragm level on the right.

All right. Now, inflammation of the lung, pneumonia, the exudate spreads through the lung and fills the alveoli, and if it becomes very solid, the exudate becomes sort of solid, we call that consolidation. If it extends all the way out to the pleural surface, it irritates the pleura, which has a lot of nerve endings, and when it extends out to the pleura, we call that pleuritis, and it's been shortened to pleurisy before. Anybody heard of the term pleurisy before? No? Okay.

Anyway, it's inflammation of the pleura.

So let's see here. Etiology, okay, what you causes pneumonia? You have bacteria, you have viruses, fungi, chlamydia can, mycoplasma and Rictettsia. Anatomic distribution, it can affect the lobe of the lung. All right? Legionnaire's disease is going to be a gram negative rod that is transmitted. It can be found in soil or water, typically water has to be inhaled.

Let's see, predisposing factors, any conditions associated
with poor lung ventilation, and retention of bronchial secretions. Post-op pneumonia, accumulation of mucus secretions in the bronchi. Aspiration pneumonia. What that refers to is people aspirate, okay, I mentioned about peanuts. That can cause pneumonia also. Atelectasis, but also pneumonia. The peanut oil, sometimes as they throw it up in the air, little molecules, bits of peanut oil, can be inhaled and cause a pneumonitis. So typically for pneumonia, the classic pneumonia features, they present with a fever, cough. They bring up sputum, phlegm that has pus in it and they get pain and respiration due to the pleurisy and they get shortness of breath.

All right. So pneumocystis pneumonia. Pneumocystis carinii, this is the opportunistic infections associated with immunocompromised patients. It's another name for the pneumocystis that's present in AIDS, and basically, the organisms attack and injure the alveolar lining. Patients get a cough, dyspnea, and they get pulmonary consolidation. All right. Tuberculosis, it's caused by what we call an acid fast bacteria. It's a type of staining. It's acid fast, and the organism has a waxy capsule, all right? TB has a waxy capsule. It makes it very resistant to destruction by the body's immune system. So what the body's immune system does is it walls it off, okay, so you get a thick wall of -- and basically it's a cell-mediated immunity, so a thick wall of immune cells, the T lymphocytes and they form what you call a granuloma. All right. It gets walled off. And it becomes basically it contains live particles, but it's kinda walled off and protected from the body. And transmission is through airborne droplets. You inhale it. And you have to have -- usually, you know, you have to have repetitive inhalations of TB, organisms to get TB. It's not like someone sneezes with TB, you walk by once and you inhale at the time they're sneezing and you're going to be infected. Usually it's not that way. Okay?

And the granuloma, you have a giant cell with central necrosis, so you have these inflammatory cells usually due to the cell mediated immunity. The giant cells are multi-nucleated giant cells. They're bacteria-infused monocytes, and they're at the periphery of the lymphocytes. You see them when you look at the granuloma underneath the microscope. Okay?

Now, the concern is these granulomas are formed. They're walled off. The TB organism is walled off. Then these granulomas break down. They can erode into the kidneys. Well, when they erode or break down, then you liberate the organism into the arterial system or into the venous system. As a result, the organism gains access to the blood, and it can spread to the kidneys, bones, uterus, fallopian tubes, other locations.
So basically, when the infection is arrested, then the granulomas heal with some scarring. All right? And so the Mantoux test, which is the test for TB, basically you want to see if there's exposure to, if you've been previously exposed to tuberculin organisms and as a result, the Mantoux test, they inject a little bit of protein into the skin to see if there's the immune response, which occurs two or three days after. All right? And people don't respond to the Mantoux test, basically means they have not developed antibodies against TB so they have not been exposed to them.

All right. So typically what happens is the infection is walled off, and then later on it becomes re-activated. And it progresses to pulmonary TB.

All right. So sometimes on a chest x-ray, you can see a large granuloma, walled-off area of the lung. Also, to diagnose it you can do a sputum test. You get phlegm from the patient. You can give them a respiratory treatment to loosen up mucus, collect it, and then do a stain for TB organisms in the sputum.

All right. And then so basically, the reactivated tuberculosis, active TB in adults, occurs from reactivation of an old infection. All right?

Now, Miliary TB refers to multiple foci of disseminated tuberculosis Miliary comes in seeds, small little seeds, and large numbers of organisms are disseminated in the body, and when there's a massive, tuberculous, inflammatory tissue, it then erodes into a large blood vessel. Okay?

So who's at risk? AIDS and immunocompromised individuals, since cell mediated immunity is very significant in dealing with TB.

Now, the treatment for TB is is going to be antibiotics and it's difficult to treat because the fact that very often the organism can develop resistance to antibiotics, number one, okay? These people have to be placed on more than one antibiotics sometimes. There can be side effects to the medication, and the third reason is they have to take medication for like six months sometimes. So it's not like you get a strep throat, you take penicillin for five to seven days and you're done. So patient compliance is also an issue.

So the course of treatment is prolonged. And there's, you know, drug resistant tuberculosis developing.

So here you see the granuloma at the top, okay? Granuloma, the tuberculosis, central necrosis in there, the arrow is pointing to the central necrosis, and then you see the periphery, and then you see the multinucleated giant cell. You see that ring of all nuclei in the cell, and that's a multinucleated giant cell associated with tuberculosis. All right. And you have pulmonary tuberculosis. There's
more consolidation in the right lung field than the left. And of course, you know, when you look at it, it's on your left, but it's the patient's right, okay, and if you get confused, you always see the heart, you know the heart's on the left side.

Chest x-rays are very tricky to read. They're fun, but they're tricky to read.

All right. So bronchitis and bronchiectasis. Bronchitis is inflammation of the tracheobronchial mucosa. You can have acute and chronic bronchitis. Acute bronchitis caused by an infection. Treated with antibiotics. Chronic bronchitis results from chronic irritation of the lining of the airways. Typically we call it chronic if it occurs for at least three months for two consecutive years. Okay? So the term bronchiectasis refers to a chronic infection of the walls of the bronchioles become weakened by inflammation and they become enlarged so the bronchioles actually dilate. When they dilate, they become distended and retain secretions. So these people develop a chronic cough. They develop sputum with pus in it and they get recurrent infections.

And once you have a bronchiectasis, it's really difficult to get rid of and sometimes the only way to do it is surgical resection of some of the affected areas of the lung.

COPD. Very significant. All right? COPD stands for chronic obstructive pulmonary disease, and it's a combination of emphysemic chronic bronchitis and what happens is there's destruction of the alveolar structure of the lungs, okay, and what happens is, let's see -- okay. There's a diagram in this lecture that I want to show you, but we'll get to it.

All right. So what happens with emphysema is the alveoli are very thin-walled, and they're sensitive, and they get destroyed, okay, because of the obstruction in the airways. When you go to exhale, there's mucus in the airways. Usually it's associated with cigarette smoking. There's mucus in the airways. Those mucus plugs obstruct the airway, you retain air, and then these alveoli get injured and when they get injured, they coalesce. All right. So, you know, I told you there's alveolar ducts that lead into alveolar sacs and so when they get injured, the alveoli, the several alveoli that make up that area in the lung off the alveolar duct, become one sac, so destruction of the fine alveolar structure. The destruction tends to begin in the upper lobe and eventually affects all lobes of the lung and then what happens is people get dyspnea. They have difficulty breathing, initially on exertion, later on at rest. You can tell people who have COPD because you'll see them in the food stores and the shopping centers. They have a nasal canula in their mouth -- I'm sorry, not mouth.
Nose. And then they're carrying, along with them, an oxygen tank, and they're breathing in the oxygen because they need to get adequate oxygenation, they need 100% oxygen. All right. Chronic bronchitis is chronic inflammation of the terminal bronchioles, and they get a cough and productive sputum. So there's three main anatomic abnormalities in COPD. Basically, there's inflammation, narrowing of the terminal bronchioles. As a result of the swelling of the mucosa, the airway is narrowed down. All right, reduced caliber of the bronchi and bronchioles. This's also increased secretions. So you've got narrowed down airways, you've got increased secretions, and then as a result, there's increased resistance to air flow. Air enters the lungs more readily than it's expelled, and there's trapping of air. So with the inflammation, the lumen, diameter is increased through secretions which also add to obstruction and then air gets trapped because remember, on exhalation, I told you the diaphragm moves up, the ribcage goes down and as a result the lungs pull in. There's a natural tendency of the lungs to reduce their volume. So that happens on expiration. So gradually, then, there's a dilation and coalescence of some of the pulmonary spaces, talking about the alveolar sacs, and then there's loss of lung elasticity. The lungs no longer recoil normally following expiration. All right. So what causes it? Smoking and inhalation of injurious agents. Okay? Let's see here. So there's swelling of the mucosa, and then the process is with the swelling, all right, air gets trapped in the lungs, the leukocytes accumulate in the bronchioles and alveoli and what happens is they then die there and they can release enzyme, protein-destroying enzymes, that attack the elastic fibers of the lung structural support and reduce the elasticity of the lung. People develop coughing, increased intrabronchial pressure which then destroys the alveoli, causing them to coalesce. And then there's also due to retention of secretions within the pulmonary pathway, the bronchioles. All right? So therefore the lungs get damaged by emphysema. Once that happens, they can't be restored to normal. The way you treat it is you -- first of all, you can give them chest physical therapy to promote drainage of the bronchiole secretions. There's a lot of secretions. They run the risk of infection, and try to decrease the risk of pulmonary infections with aggressive antibiotic therapy. Asthma. Asthma, high incidence in the population, and with asthma, there's contraction of the smooth muscles in the walls of the bronchi. They narrow down. They contract, all right, so they narrow down the bronchi, and then as a
result, there's some difficulty breathing. They call that dyspnea, and there's wheezing on expiration. It's when people exhale that they have wheezing. That's significant. Okay? And of course when they're exhaling, the diameter of the bronchioles is slightly decreased. It's less than what it is on inspiration. So it's a greater impact on expiration than inspiration. What causes asthma? It can be allergens such as dust, pollens, animal dander, or viruses. And so what's the treatment? The treatment is bronchodilators. You can take medication by mouth such as theophylline, or you do a Proventil inhaler. There's the mist treatment where the medication actually gets misted, and you breathe it in that way, or you can take medication that blocks the release of mediators from mass cells.

All right. Neonatal RDS. Also known as hyaline membrane disease, okay, and basically what that refers to is a lack of surfactant in the lungs. Remember I told you surfactant reduces surface tension. Newborns who are born early, okay, before -- when I say early, three weeks before their due date, okay, are at risk for developing RDS, because the presence of surfactant occurs around 37 weeks of pregnancy. If we say that pregnancy is 40 weeks term. And as a result, if they have inadequate surfactant, it's difficult for them to expand their lungs, and as a result, they get tired out trying to expand their lungs, and sometimes they need help.

So who's at risk? Premature infants. All right? Infants delivered by cesarean section may have slight RDS. The whole deal with cesarean sections is you have to be sure of the due date. Okay? You have to be sure of the due date. If it's an elective section, you gotta know that you're sure about the due date. Okay? Because if you're not really sure about the due date, doing an elective section, if you're off by a week, you could deliver a baby too early and it could have some RDS.

And also, mothers who are diabetic at the delivery. Whether it's they're diabetic before they get pregnant or if they develop diabetes while they're pregnant, they're diabetic at delivery, these babies are at risk for RDS. So if the delivery is going to be induced or done by cesarean section for diabetic mothers, you need to do an amniocentesis to obtain some fluid and check to make sure that the baby will not get RDS. Okay?

If you need to do an early delivery of a baby, and you're concerned about RDS, you can give adrenal corticosteroids to the mother before delivery because it increases the maturation of surfactant. Also, there is certain maternal conditions that accelerate the production of surfactant. Hypertension in pregnancy is going to be one of them.

All right. So here we have neonatal respiratory distress
syndrome, and basically there's something called hyaline membranes, the arrows point to the hyaline that's present on the membranes and that will interfere with oxygenation. All right. Adult RDS tends to occur from shock, and once again, it can be due to decreased surfactant that's present. Okay?

So shock is flow in blood pressure. Reduced blood flow to the lungs. It can be trauma, septic shock, referring to infection, aspiration of acid gastric contents, maybe. That's pretty severe. Inhalation of certain toxic gases, or damage caused by SARS.

And as a result, the capillaries, alveolar capillaries leak fluid and protein and they become damaged and there's impaired surfactant production from the injured or damaged alveolar lining cells so as a result they develop respiratory distress.

So here's a diagram that basically I will not hold you responsible for, but I want you to have an understanding about neonatal RDS and adult RDS. Okay? The pathogenesis in neonatal RDS is inadequate surfactant. All right. Pathogenesis in the adult is damage to the lungs, okay, but then what happens is the damage causes reduced production of surfactant.

And for babies, what happens is once they're delivered, they can be given endotracheal surfactant, which is they put an endotracheal tube in, in the trachea, and they can be given some surfactant. They'll put them in a hood, and hood over their -- a plastic hood over their head so they can breathe moisturized air. The really severely compromised babies with severe RDS, they'll actually put a trachea in and they'll put them on a respirator. Okay? Mechanical respirator to ventilate them.

Pulmonary fibrosis refers to thickening of the alveolar septum from irritating gases, organic, inorganic particles, makes the lungs very rigid, interferes with diffusion of gases, and it causes progressive respiratory disability. Certain substances can do it. Basically, pneumoconiosis refers to injury from inhalation of dust or other particulate material. Silicone is one. Asbestos is another one.

Lung carcinoma, usually it's smoking-related neoplasm, and it's common malignant tumor in both men and women. The mortality from lung cancer in women exceeds breast cancer. That's because it's not caught as early, and we have great success in treating breast cancer statistically. And very often it's referred to as bronchogenic carcinoma. Treatment basically is going to be surgical resection or radiation and chemotherapy.

All right. Treatment of lung cancer is not very satisfactory. There's basically two types, and the second
type is what we call small cell carcinoma. It's a very small type of cell. I think there's a picture in here. All right. Yep. In B, you see the difference of the small cell. All these small little cells, all right, small cell carcinoma, as opposed to the squamous carcinoma. All right. So classification of lung carcinoma, the squamous cell carcinoma, very common. Adenocarcinoma is common. The small cell is very aggressive. All right. Small cell is very aggressive with a very poor prognosis. All right. Here you have squamous cell carcinoma in a bronchus, all right, that white thing in the center is the tumor nodule in a main airway, and then you have the adenocarcinoma, rises from a smaller airway.