Educational Objectives:

1. Recognize the defining characteristics and global burden of chronic obstructive pulmonary disease (COPD).
2. Identify COPD risk factors and the core concepts of COPD prevention.
3. Describe how to diagnose and treat COPD.

CASE ONE:
Mrs. Sangala is a 60-year-old woman who is seeking care for breathlessness and fatigue. She has become increasingly short of breath with exertion and found herself unable to keep up with her grandchildren when walking. She has had a cough for many years, which has recently become more productive of sputum. Her grandchildren have remarked that her lips sometimes turn blue and that she will tightly press her lips when breathing. She states that she has been healthy for most of her life and had only previously sought medical care for her five uncomplicated pregnancies. This is her first time seeing a healthcare provider in many years.

Questions:

1. What is your differential diagnosis for Mrs. Sangala’s dyspnea? Which diagnoses are most likely given the provided history?

Encourage learners to name multiple etiologies across different organ systems before continuing to the next question. Highlight that a person can have more than one condition contributing to dyspnea. For example, a woman experiencing dyspnea of pregnancy is also at increased risk for pulmonary embolism or worsening asthma associated with pregnancy and that there may be multiple factors explaining her symptoms of dyspnea.

The differential diagnosis for shortness of breath is broad. It is helpful to separate it out by organ system. Table 1 provides a non-exhaustive list of conditions that cause dyspnea.

Mrs. Sangala’s symptoms of dyspnea, chronic cough, and sputum production increase suspicion for a chronic respiratory disease, particularly COPD.
Table 1. Possible Etiologies of Dyspnea.¹

<table>
<thead>
<tr>
<th>Organ System/Physiology</th>
<th>Etiologies of Dyspnea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac</td>
<td>Heart failure cardiomyopathy, valvular disease, myocardial infarction coronary artery disease, arrhythmia, constrictive pericarditis, or intracardiac shunting</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>COPD, asthma, pulmonary infection (e.g. community-acquired pneumonia, pneumocystis jirovecii pneumonia, or pulmonary tuberculosis), airway obstruction (e.g. compressive mass, vocal cord dysfunction, aspiration of foreign body, or anaphylaxis), pleural effusion, pneumothorax, pulmonary embolism, pulmonary hypertension, interstitial lung disease, or bronchiectasis</td>
</tr>
<tr>
<td>Hematologic</td>
<td>Anemia</td>
</tr>
<tr>
<td>Neuromuscular</td>
<td>Guillain-Barré syndrome, myasthenia gravis, or amyotrophic lateral sclerosis</td>
</tr>
<tr>
<td>Psychological</td>
<td>Anxiety</td>
</tr>
<tr>
<td>Other</td>
<td>Pregnancy, obesity, physical deconditioning, metabolic acidosis, thyroid disease, or alternative causes of volume overload, such as end-stage renal disease, chronic liver disease/cirrhosis, and hypoalbuminemia</td>
</tr>
</tbody>
</table>

2. What are the symptoms and underlying pathophysiology of COPD?

The classic symptoms of COPD include dyspnea, cough, and sputum production. Patients may also experience wheezing and chest tightness. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) defines COPD as follows: “COPD is a common, preventable, and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that are due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.”²

COPD develops from an inflammatory response in the lungs that leads to poorly reversible airflow obstruction. Long-term exposure to irritants or gases such as cigarette smoke, automobile exhaust, and smoke from fires (e.g. charcoal, wood, and trash) leads to chronic inflammation of the lung tissue. The inflammatory response leads to destruction of the lung tissue, excess mucus production, and narrowing of the small airways causing air trapping, decreased gas exchange, and increased lung compliance. These irreversible structural changes obstruct the airway and lead to chronic dyspnea, chronic cough, and hypoxia.
3. Based on your knowledge of chronic respiratory disease, what additional history would help determine if Mrs. Sangala’s dyspnea is explained by COPD? What are risk factors for developing COPD?
In addition to the past medical, medication, family, social, and allergy histories, it is important to ask about risk factors and symptomatic triggers in order to obtain a complete history for patients with COPD. This information helps to identify the diagnosis and modifiable factors. Assisting patients to make lifestyle changes like smoking cessation and to recognize environmental symptom triggers is important to control symptoms and progression of COPD.

COPD develops progressively over many years from exposure to noxious substances or from pre-existing pulmonary disease. Risk factors for developing COPD include the following:

- Cigarette smoke, including secondhand smoke, which is the primary cause of COPD worldwide
- Indoor air pollution, particularly use of solid fuel (e.g. charcoal and firewood) for cooking and heating indoors with inadequate ventilation, which can lead to 100 times higher than acceptable levels of exposure
- Outdoor air pollution and occupational exposure to dust and chemicals (e.g. fumes, vapors, slash-and-burn agriculture, trash burning, and exhaust from vehicles)
- Pre-existing pulmonary disease, including poorly controlled asthma, lower respiratory infections in childhood, delays in lung development, and pulmonary tuberculosis
- Genetic susceptibility, such as alpha-1-antitrypsin deficiency
- Age: Generally diagnosed over the age of 40
- Sex: Women appear to be more susceptible to developing COPD than men

Environmental exposures have a significant effect on population health. The prevalence of tobacco smoke can vary greatly by location and population group. In many areas of the world, slash-and-burn agriculture and trash burning are important exposures that can contribute to COPD. In addition, people who work near the roadside are more exposed to fumes from vehicle exhaust which can lead to lung complications over time.

CASE ONE CONTINUED:
On further history-taking, you learn that Mrs. Sangala has been exposed to various forms of smoke throughout her lifetime. She has cooked over a charcoal-burning stove in a poorly ventilated room of the home for most of her life. She lives in an urban neighborhood and regularly walks beside a road with heavy automobile traffic and exhaust fumes when going to the marketplace. She burns the household trash just outside the home, as do her neighbors. While she has never smoked cigarettes, she is regularly passively exposed to cigarette smoke from her husband who is a long-time smoker.

4. What is the global burden of COPD?

According to the World Health Organization (WHO), approximately 251 million people have COPD worldwide and about 3 million people die of COPD annually, accounting for about 5% of all worldwide deaths. Greater than 90% of COPD-related deaths occur in low- and middle-income countries. As a result of growing worldwide tobacco use and aging populations, the WHO predicts that COPD will become more prevalent.

5. What are the characteristic physical exam findings of COPD?
Physical exam findings may initially be normal or mild, typically only demonstrating a prolonged expiratory phase or wheezing in the early phases of the disease. Progression of the disease leads to more pronounced exam findings including the following:
- Decreased breath sounds or wheezing on auscultation
- Prolonged expiratory phase
- Expiration through pursed lips
- Hyperresonance on percussion due to hyperinflation of lungs
- Increased anterior-posterior diameter of the chest (“barrel-shaped chest,” see Figure 2)
- Use of accessory respiratory muscles of the neck and shoulder girdle
- Paradoxical retraction of lower rib cage during inspiration
- Cyanosis (i.e. bluish discoloration of lips or fingers)
- Jugular venous distension due to increased intrathoracic pressure
- Signs of right-heart failure (i.e. distended neck veins and enlarged tender liver) if complicated by cor pulmonale. Cor pulmonale is a phenomenon that occurs when severe COPD and hypoxia results in vasoconstriction of the pulmonary arteries, leading to pulmonary hypertension and ultimately right-sided heart failure. Refer to heart failure modules for more information on this.
- Yellow stains on fingers may indicate nicotine and tar from cigarette smoke
- Cachexia may be a late finding

Figure 2. “Barrel-Shaped Chest” due to Air Trapping and Expansion of Lung Cavity in Patient with COPD.7

Credit: “How to Diagnose COPD”. wikiHow. License: CC BY-NC-SA 3.0.

As shown in Figure 2, the characteristic chest of advanced COPD entails a barrel-like appearance as depicted on the right by the red arrow. Notice the increased anterior-posterior diameter of the chest on the right relative to the normal chest on the left.

**CASE ONE CONTINUED:**
On exam, Mrs. Sangala's respiratory rate is 20 breaths per minute with an oxygen saturation of 91% on ambient air at rest. She is breathing comfortably. She coughs throughout the exam bringing up white-colored sputum. Her auscultated breath sounds are decreased with faint wheezes and a prolonged expiratory phase. Her cardiac exam is normal. You suspect that Mrs. Sangala has COPD and that her disease has progressed over many years. You decide to obtain studies to determine if she has COPD.

6. What is a peak expiratory flow rate and how can it help confirm the diagnosis of COPD? What diagnostic methods can be used to differentiate COPD from asthma?

COPD and asthma are both obstructive lung diseases resulting in airflow limitation. The severity of airflow limitation in obstructive lung diseases, such as COPD and asthma, can be estimated by measuring a patient’s peak expiratory flow rate (PEFR). PEFR is a patient’s maximum speed of expiration and can be measured by a peak flow meter, a small, handheld device that a patient exhales into forcefully. The peak flow meter measures the airflow through the bronchi and, thus, estimates the degree of obstruction. Normal PEFR values vary by age, height, and sex as shown in Figure 3.

When diagnosing a patient with obstructive lung disease, it is important to establish a baseline, or a “personal best” PEFR value. This level can then be compared with readings on future visits to estimate the level of obstruction and assess if the disease has progressed or is responsive to treatment.
COPD and asthma are differentiated by the reversibility of airflow obstruction. COPD causes irreversible airflow obstruction while asthma causes reversible airflow obstruction. The reversibility of airflow obstruction can be assessed by measuring a patient’s PEFR before and after administration of a bronchodilating beta-2 agonist medication. Beta-2 agonists (e.g. salbutamol) are a class of medications that work by binding to bronchial beta-2 adrenergic receptors, resulting in bronchial smooth muscle relaxation and dilation of the airway passages. While these medicines are used to treat both asthma and COPD, patients with asthma are more responsive. Increase in a patient’s PEFR by ≥20% after administration of a beta-2 agonist is more consistent with asthma. Alternatively, if the PEFR does not correct or only corrects slightly, the diagnosis is more consistent with COPD. It should be noted, however, that in well-controlled asthma the PEFR may also not increase with bronchodilators.
Spirometry is a test method used for diagnosing COPD and asthma, but it may not be available in some low-resource settings. Briefly, spirometry measures the forced vital capacity (FVC) and the forced expiratory volume in 1 second (FEV₁). FVC is the total amount of air that can be forcibly exhaled after maximum inhalation. FEV₁ is the amount of air that the patient can forcibly blow out in the first second of the test. These values can be compared to the predicted normal of a healthy person and used to differentiate forms of lung disease. A FEV₁/FVC ratio between 0.70 and 0.80 is normal in adults. However, a FEV₁/FVC ratio below 0.70 is indicative of obstructive pulmonary disease, such as COPD or asthma.

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) provides a framework for staging the severity of airflow limitation in COPD by comparing a patient’s FEV₁ value with the predicted normal of a healthy individual.²,⁹ In COPD, FEV₁ is best assessed after the use of a bronchodilating beta-2 agonist medication to minimize variability. FEV₁ values can be interpreted as delineated in Table 2.

### Table 2. Classification of Airflow Limitation Severity in COPD for Patients with FEV₁/FVC <0.70²,⁹

<table>
<thead>
<tr>
<th>Classification</th>
<th>Post-bronchodilator FEV₁</th>
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<tbody>
<tr>
<td>GOLD 1: Mild</td>
<td>≥80% predicted</td>
</tr>
<tr>
<td>GOLD 2: Moderate</td>
<td>50 to &lt;80% predicted</td>
</tr>
<tr>
<td>GOLD 3: Severe</td>
<td>30 to &lt;50% predicted</td>
</tr>
<tr>
<td>GOLD 4: Very Severe</td>
<td>&lt;30% predicted</td>
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7. Describe how you would teach this patient to use a peak flow meter.
Figure 4. Use of Peak Flow Meter: (A) Set the Sliding Cursor to Zero. (B) Blow Forcefully into the Meter.¹⁰,¹¹

(A) ![Use of Peak Flow Meter: (A) Set the Sliding Cursor to Zero.](image)

(B) ![Use of Peak Flow Meter: (B) Blow Forcefully into the Meter.](image)

Credits: (A) “How to Use a Peak Flow Meter - Step 3: Set the cursor to zero”. wikiHOW. License: CC BY-NC-SA 3.0. (B) “How to Use a Peak Flow Meter - Step 6: Blow forcefully into the meter”. wikiHow. License: CC BY-NC-SA 3.0.

A peak flow meter will allow you to determine the patient’s peak expiratory flow rate (PEFR). In general, lower PEFR values indicate more obstruction. The following steps ensure accurate peak flow meter readings:¹²

1. The sliding cursor or marker on the peak flow meter should be set to zero or its lowest setting as depicted in Figure 4A.
2. Stand or sit up straight.
3. Take in as deep a breath as possible.
4. Place the peak flow meter in the mouth, with the tongue under the mouthpiece.
5. Close the lips tightly around the mouthpiece as depicted in Figure 4B.
6. As shown in Figure 4B, blow into the meter as hard and fast as possible. Do not move the head forward while blowing out. Repeat the test if the tongue partially blocks the mouthpiece or if coughing or spitting occurs during the test.
7. Breathe a few normal breaths and then repeat the process two more times. Write down the highest number obtained.

CASE ONE CONTINUED:
Mrs. Sangala has a chest x-ray film and her PEFR is measured using a peak flow meter. The chest x-ray demonstrates hyperinflation of the lungs and flattening of the diaphragm. Her initial PEFR is 340 L/min. Repeat PEFR testing 15 minutes after receiving bronchodilator therapy with a salbutamol inhaler showed no significant change in her PEFR value. You note that she is a 60-year-old woman and 167 cm tall and use this information to compare her personal PEFR to a normal PEFR for her age and height.

8. What do you expect to see on a COPD patient’s chest x-ray (XR)? How does this differ for asthma?
In asthma, the patient’s chest radiograph can look normal. However, patients with later stages of COPD develop abnormalities visible on chest radiograph. As shown in Figure 5, characteristic chest radiograph findings of advanced COPD include hyperinflation of the lungs and flattening of the diaphragm. Notice how lung enlargement and flattening of the diaphragm can pull the heart into a more vertical position.

**Figure 5.** A Chest Radiograph with Classic Features of COPD.\(^{13}\)

Credit: Radiopaedia.org.

9. Reference Figure 3 and discuss how her PEFR of 340 L/min compares to the normal expected PEFR for her age and height. Are Mrs. Sangala’s test results consistent with COPD?

Mrs. Sangala’s chest x-ray and PEFR findings suggest a diagnosis of COPD. Her chest x-ray demonstrates hyperinflation and flattening of the diaphragm which are consistent with COPD. Her PEFR of 340 L/min is significantly decreased from an expected normal PEFR of about 390 L/min for her age and height indicating obstructive airflow limitation. Her decreased PEFR was irreversible with bronchodilator therapy which distinguishes her COPD from asthma.
CASE ONE CONTINUED:
Mrs. Sangala is concerned about her breathing. It has affected her day-to-day life and has prevented her from having the energy to enjoy time with her grandchildren or work. She does not want her breathing to worsen and asks if there is anything that she can do to prevent progression of her COPD.

10. What lifestyle modifications would you suggest to Mrs. Sangala, and how would you discuss these modifications with her?

Mrs. Sangala’s history brings up multiple modifiable risk factors, including exposure to smoke from the indoor charcoal-burning stove, walking by exhaust fumes on a daily basis, trash burning, and secondhand cigarette smoke exposure. An essential part of her COPD care will be to reduce these exposures as much as possible.

The WHO suggests the following to prevent or slow progression of COPD:6,14
- Stop smoking and avoid dust and tobacco smoke.
- Keep cooking areas well ventilated by opening windows and doors. If possible, move wood- or charcoal-burning stoves outside to increase ventilation.
- Limit work in areas with occupational dust or high air pollution. If you must continue working in that environment, wearing an appropriate mask may help.
- Wear an appropriate mask when walking in areas with high exhaust fumes, burning trash, or participating in slash-and-burn agriculture.

CASE ONE CONCLUSION:
Mrs. Sangala established care with the clinic and was prescribed a salbutamol inhaler to use as needed for her shortness of breath. The medical student taking care of her used motivational interviewing methods and discussed strategies for reducing smoke exposure, including moving her stove outside and wearing a mask. She followed up in clinic one month later and her symptoms were improved, giving her more energy to care for her grandchildren.

CASE TWO:
Mr. Banda is a 64-year-old man with a significant smoking history who presents to your clinic with worsening shortness of breath. He experiences shortness of breath with walking and physical work that resolves when he is sitting or resting. He is breathing at a respiratory rate of 20 breaths per minute with an oxygen saturation of 92% on ambient air at rest and 88% when walking. His lung exam is significant for a prolonged expiratory phase with faint wheezing. His cardiac exam is normal. You appropriately rule out cardiac and infectious causes and diagnose him with COPD.

11. What system can you use to classify the severity of Mr. Banda’s COPD? Based on the WHO Classification of COPD, does Mr. Banda have moderate or severe COPD?

COPD severity is commonly classified using the Global Initiative for Chronic Obstructive Lung Disease (GOLD) staging system, which classifies patients through a combination of spirometry results, quantified symptoms assessment, and exacerbation history.2 For practicality, this module will focus on the WHO guidelines for management of respiratory diseases.
WHO Classification of COPD Severity:15,16
Moderate: if breathless with normal activity
Severe: if breathless at rest

Using the WHO classification system, Mr. Banda meets criteria for moderate COPD. If available, oxygen saturation, PEFR results, and spirometry can also assess severity.

12. You discuss the diagnosis with Mr. Banda. In addition to modifying behaviors and environmental exposures contributing to his COPD, what medications could be effective in treating his COPD?

A variety of inhaler and oral medications have been developed to treat COPD. While these can decrease symptoms and reduce complications of COPD, they have not been found to reverse or slow the long-term decline in lung function. The most widely available medications are inhaled short-acting beta-2 agonists and oral theophylline, which work by dilating the bronchi making it easier for air to move into and out of the lungs. In healthcare settings with increased drug availability, inhaled long-acting beta agonists (e.g. salmeterol) and inhaled long-acting muscarinic agonists (e.g. tiotropium) are standard of care per the GOLD 2017 guidelines and are recommended if a patient remains symptomatic despite adequate use of short acting medications, though they may have limited availability and greater expense. The WHO guidelines for primary health care in low-resource settings outline the following recommendations.

WHO guidelines for management of stable COPD:15,16

Step 1: Inhaled short-acting beta-2 agonists (e.g. salbutamol) as needed. Inhaled salbutamol is typically prescribed two puffs as needed, up to four times daily. The oral formulation of salbutamol can be used if the inhaled version is not available, though it is associated with more adverse effects including tremors and arrhythmias.

Step 2: Low dose oral theophylline can be added if symptoms are not adequately controlled with a short-acting beta-2 agonist. Theophylline must be used with caution, particularly if unable to monitor theophylline blood levels. Theophylline toxicity can produce nausea, vomiting, abdominal pain, tremors, cardiac arrhythmias, hypotension, and seizures. If available, inhaled long-acting beta agonists (e.g. salmeterol) and inhaled long-acting muscarinic agonists (e.g. tiotropium) have fewer severe side effects and should be used in place of theophylline.

CASE TWO CONTINUED:
Mr. Banda returns to your clinic one month later with worsening symptoms. The inhaled short-acting beta-2 agonist you prescribed helped his breathing initially, but over the past couple of days he became more short of breath, even at rest. He also notes having a worsening cough, increased sputum production, and a change in the color of his sputum. He denies fever, chills or sick contacts. On exam, he is unable to speak a full sentence without becoming dyspneic. He is using accessory muscles to breathe. His respiratory rate is 25 breaths per minute, and his oxygen saturation is 88% on ambient air.

13. What do you think is happening to Mr. Banda? What are the defining characteristics of a COPD exacerbation?
Mr. Banda is likely having a COPD exacerbation. COPD exacerbations are most commonly provoked by infections of the lower respiratory tract or air pollution and are characterized by worsening changes in the following three criteria:

- Cough frequency and severity
- Sputum quantity or change in character (e.g. color, consistency)
- Dyspnea severity

These criteria in the setting of a pre-existing diagnosis of COPD suggest a COPD exacerbation. Physical exam signs that point towards this diagnosis are accessory muscle use, respiratory distress (i.e. increased respiratory rate with difficulty completing sentences), wheezing, decreased breath sounds from baseline, paradoxical retraction of lower rib cage during inspiration, and decreased oxygen saturation. A decrease in the PEFR from the patient’s baseline would also indicate a COPD exacerbation.

It is important to keep a broad differential when assessing Mr. Banda’s acute shortness of breath before settling on COPD exacerbation as your diagnosis. A chest x-ray would be helpful to exclude alternative explanations for Mr. Banda’s worsening dyspnea, such as pneumonia or pulmonary edema. A thorough cardiopulmonary exam should be performed following bronchodilator administration to enhance capacity of auscultation.

14. How are you going to treat Mr. Banda’s COPD exacerbation?

Management of COPD exacerbations consists of antibiotics, oral steroids, beta-2 agonists, and oxygen. COPD exacerbations may or may not require inpatient care, depending on resource availability of therapy supplies such as nebulizers and supplemental oxygen and the stability of the patient.

The WHO treatment guidelines for COPD exacerbations are as follows:15,16

1. Antibiotics: **SHOULD be given for most COPD exacerbations.** There is evidence that antibiotics reduce the severity of the exacerbation, particularly for those with two or more of the following symptoms: increased dyspnea, sputum quantity, or sputum purulence. Patients with only one of these three symptoms generally do not need antibiotics. Antibiotics should cover common pathogens including Streptococcus pneumoniae, Haemophilus influenzae, and Moraxella catarrhalis, as well as atypical pathogens such as Mycoplasma pneumoniae, Legionella pneumophila, and Chlamydia pneumoniae, but choice may depend on which antibiotics are available. Macrolides, such as azithromycin, clarithromycin, and erythromycin, or doxycycline are all good options for first-line treatment. Fluoroquinolones such as ofloxacina or levofoxacin should be reserved for suspected pseudomonas infections or contraindications to macrolides since they are associated with more adverse drug effects. The duration of antibiotic therapy would range from 3 to 7 days, depending on the specific antibiotic and response to therapy.

2. Oral steroids: A short course of prednisolone or other available oral corticosteroid is recommended for acute exacerbations of COPD. An example regimen is prednisolone 30 to 40 mg daily for 5 days.

3. Inhaled beta-2 agonists: Higher and more frequent doses of salbutamol should be administered via a nebulizer or metered dose inhaler with a spacer. For example, patients should receive 4 puffs of inhaled
salbutamol every 20 minutes for 1 hour. If this is not available, the patient should be given an oral regimen. Refer to the asthma module for patient instructions on how to make a spacer for their inhaler.

4. **Oxygen:** If available, for severe exacerbations in a patient with a decreased oxygen saturation. Oxygen should be administered by a device that controls the concentration at 24 to 28% in order to maintain a target pulse oxygen saturation of 88 to 92%. A high concentration of supplemental oxygen can decrease respiratory drive in some individuals with severe COPD resulting in accumulation of carbon dioxide and respiratory acidosis.

If available in certain healthcare settings, BIPAP (Bi-level Positive Airway Pressure), a type of noninvasive positive pressure ventilation (NIPPV), has been shown to improve oxygenation and ventilation and reduce in-hospital mortality in acute COPD exacerbations. This is an option for patients with severe exacerbations who are still struggling to breath despite the above interventions.

**IV aminophylline is NOT recommended** for routine use in acute exacerbations as it likely only has a small beneficial effect and toxicity can lead to severe adverse effects.

### CASE TWO CONTINUED:
Mr. Banda is admitted to the hospital for a COPD exacerbation. He is treated with nebulized salbutamol six times daily, a five-day course of azithromycin, and a five-day course of prednisolone with good control of his symptoms. Prior to being discharged from the hospital, the medical student visits him to discuss the effects of smoking on his health.

15. **What are some ways the medical student could counsel and motivate Mr. Banda to stop smoking?**

Smoking cessation counseling delivered by physicians has been shown to significantly increase the chance that patients quit smoking. Assisting patients to quit smoking early can help prevent the health consequences of COPD later in their lives. As outlined in Table 3, the Global Initiative for Chronic Obstructive Lung Disease (GOLD) provides a five-step program for intervention to help patients quit smoking.

**Table 3. Five-step Program to Help Patients Quit Smoking.**²,⁹

<table>
<thead>
<tr>
<th>ASK</th>
<th>Systematically identify all tobacco users at every visit.</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADVISE</td>
<td>Strongly urge all tobacco users to quit.</td>
</tr>
<tr>
<td>ASSESS</td>
<td>Determine willingness and rationale of patient’s desire to make a quit attempt.</td>
</tr>
<tr>
<td>ASSIST</td>
<td>Aid the patient in quitting.</td>
</tr>
<tr>
<td>ARRANGE</td>
<td>Schedule follow-up.</td>
</tr>
</tbody>
</table>

Smoking cessation counseling can be enhanced by incorporating a method called motivational interviewing.¹⁷,¹⁸ According to a meta-analysis by Hettema et al., motivational interviewing is a “client-centered, directive therapeutic style to enhance readiness for change by helping clients explore and
resolve ambivalence.” Motivational interviewing has been shown to have better results than traditional advice in the following areas: obesity, diabetes, substance use (e.g. tobacco, alcohol, and illicit drugs), medication adherence, and asthma control, and there is research supporting its use in low- and middle-income country settings. For the purposes of this module, we will provide a brief motivational interviewing toolkit for you to try with patients.

The Toolkit can be remembered with the acronym OARS. The following four strategies presented in Table 4 are used during motivational interviewing.

### Table 4. The OARS Model: Four Strategies of Motivational Interviewing

<table>
<thead>
<tr>
<th>Strategy</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Open-ended questions</td>
<td>Encourage patients to express their interest or readiness to make a change.</td>
</tr>
<tr>
<td>Affirmations</td>
<td>Acknowledge efforts to make a lifestyle change and provide support and encouragement.</td>
</tr>
<tr>
<td>Reflections</td>
<td>Convey understanding of the patient’s viewpoint and reflect their thought process back to them.</td>
</tr>
<tr>
<td>Summaries</td>
<td>Reiterate you are listening and convey understanding, making sure to highlight the most important points of the conversation.</td>
</tr>
</tbody>
</table>

16. Please read through the following motivational interviewing skit with Mr. Banda and identify which tools are being used by the physician.

**Physician:** Please tell me what you like about smoking. [Open-ended question]

**Mr. Banda:** It relaxes me. When I have not smoked for a while, I get a sensation in the back of my throat and then the thought comes to me that I need to smoke to stop feeling worried or stressed out.

**Physician:** Smoking makes you feel more relaxed and takes away an uncomfortable sensation. [Reflection] What else? [Open-ended question]

**Mr. Banda:** Well, I seem to smoke more when I am sitting around with friends. I like doing it when we are chatting.

**Physician:** What I have heard so far is that you enjoy smoking while socializing and smoking also relaxes you. [Reflection and Summary] Can you think of anything you do not like about smoking? [Open-ended question]

**Mr. Banda:** It sure costs a lot of money, even when I roll cigarettes myself.

**Physician:** You have thought about the amount of money you spend on cigarettes, and it seems you spend more on cigarettes than you want to. [Reflection] Would there be other things that you could do with that money? [Open-ended question]

**Mr. Banda:** Oh yes. The money would certainly help my wife and children.

**Physician:** You seem troubled by this. It does not seem to make sense to you to spend that much on cigarettes if the money could benefit your family instead. [Reflection] What else concerns you about smoking? [Open-ended question]
Mr. Banda: Well, I think it might be hurting me. I have a bad cough most of the time, especially first thing in the morning. And it seems I cannot walk as far as I used to without needing to catch my breath.

Physician: You have a cough and physical limitations due to your shortness of breath. [Reflection] What other concerns do you have? [Open-ended question]

Mr. Banda: I think it might be hurting my eldest son too. He likes to spend time with me. Lately he has been coughing when I smoke near him. It seems that the smoke affects him.

Physician: Unfortunately, both smoking and being exposed to smoke can cause serious breathing problems. It is great that you have already started to think about how this might be affecting you and your son. I know that is a hard thing to think about. [Affirmation] Let me see if I have understood everything you have shared with me. You have reasons why you like smoking – it helps you relax, and it is part of socializing with your friends. At the same time, you have a number of reasons why you believe you should quit – your son’s health, your own health, and the money it could save you. You seem worried about your health and your son’s health. [Reflection and Summary]

Mr. Banda: Yes, I guess that is right. I never thought about it like that before. My health and my son’s health are important for me.

CASE TWO CONCLUSION:
The medical student discusses with Mr. Banda his smoking habits at length using motivational interviewing methods. Mr. Banda is discharged from the hospital in stable condition and agrees to try to quit smoking. Six months later, Mr. Banda visits the clinic and is excited to tell you that he stopped smoking and has not had another COPD exacerbation. You commend his success in quitting smoking.

References:


