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Adaptive Yoga 12/21-2/22
Integral Yoga Therapy

Chronic obstructive pulmonary disease (COPD) is characterized by poorly reversible airflow obstruction and an abnormal inflammatory response in the lungs. COPD can be examined from the microscopic cellular changes to the dysfunction of tissues on the gross level. The physiological abnormalities include:

1. Mucous hypersecretion and ciliary dysfunction
2. Airflow obstruction and hyperinflation
3. Gas exchange abnormalities
4. Pulmonary hypertension
5. Systemic effects

COPD is a leading cause of morbidity and mortality worldwide. Age, smoking, biomass smoke and genetic predisposition are the biggest risk factors. Many COPD patients suffer from multiple chronic diseases. As COPD progresses patients are generally considered on a downward spiral.

Cigarette smoking is responsible for about half of COPD etiology. Other causes include environmental pollutants like biomass smoke, fumes, dust, gases and outdoor air pollution. Biomass smoke, that is created by nearly ½ the world's population, is the second largest etiology for COPD. It is thought that 4 million people die prematurely from illness due to biomass smoke (household burning of wood, dung, crop residues).2

The cellular and tissue damage is due to the innate and adaptive immune responses. These immune responses are due to the long-term exposure to noxious particles and gases like cigarette smoke and air pollution. All those exposed to cigarette smoking have some inflammation, but those that develop COPD have an amplified response, that can result in mucous hypersecretion (chronic bronchitis), tissue destruction (emphysema), and disruption of normal repair and defense mechanisms causing small airway inflammation and fibrosis (bronchiolitis). These pathological changes result in increased resistance to airflow in the small conducting airways, increased compliance of the lungs (lack of elasticity-harder to exhale), air trapping, and progressive airflow obstruction – all characteristic features of COPD.

To understand the interventions and treatments, we will look a little deeper at the pathogenesis. Inflammation -activation of the immune system in response to the chemicals leads to tissue destruction, and impaired defense mechanisms that act as a brake on the response, which in turn leads to more destruction and less repair. Two other responses are crucial in the pathogenesis. These two are an imbalance between antioxidants and oxidants and antiproteases and proteases. The lack of antiproteases and antioxidants lead to more pathological changes, including alveolar wall destruction (emphysema), and an increase in mucous from lack of protease inhibitors.

Mucous hypersecretion results in a chronic productive cough-“smoker’s hack”. The cough is characteristic of chronic bronchitis but is not necessarily associated with airflow obstruction. The hypersecretion is a result of the noxious particles and gases. Ciliary dysfunction due to epithelial cell metaplasia, is associated with difficulty in expectorating. The cilia are hair like structures that “sweep” particles and mucus out of airways, mucociliary escalator.

Airway obstruction occurs in the small <2mm diameter conducting airways. It is due to inflammation, narrowing (airway remodeling) and inflammatory exudates.
Other factors in airflow obstruction include loss of lung elastic recoil (compliance) due to destruction of alveolar walls and destruction of alveolar support. During expiration, the airway progressively traps air, resulting in hyperinflation. Hyperinflation reduces inspiratory capacity, resulting in breathlessness and limited exercise capacity typical of COPD. The obstruction can be measured by spirometry and is necessary for diagnosis.

The gas exchange abnormalities result in low arterial oxygen (hypoxemia). The severity of emphysema correlates with the impairment of diffusing capacity for carbon monoxide. These are later in disease and can be measured as well.

Pulmonary hypertension develops late in COPD. When this develops, there is severe gas exchange abnormalities. The structural change in the pulmonary arterioles results in persistent pulmonary hypertension and right ventricular hypertrophy and dysfunction (cor pulmonale).

The systemic features of COPD include systemic inflammation and skeletal muscle wasting (both contribute to further decline exercise), increased risk of cardiovascular disease (associated in increase in C-reactive protein-an inflammatory marker), cachexia, edema, anemia, osteoporosis and depression and anxiety.

COPD often goes undetected. The early symptoms may not add up to much until a multiple of symptoms more severely restricts ones breath and their life activities. The symptoms include; chronic cough, shortness of breath with everyday activity (dyspnea), frequent respiratory infections, blueness of lips or fingernail base (cyanosis), fatigue, phlegm or sputum, and wheezing. Exacerbations can occur from infections, air pollution, change in ambient temperature. An early diagnosis is valuable in successful treatment. Smoking maybe the best clue to the diagnosis.

Comorbidities, other medical conditions that are concurrent with COPD, may be related to the etiology, the disease itself or just another concurrent condition that coexists. The presence of comorbidities is so strongly associated with the management of COPD that the need for thorough attention to them is emphasized by all organizations relevant to COPD. The common respiratory comorbidities are asthma, lung cancer and pulmonary fibrosis. The cardiovascular comorbidities include hypertension, congestive heart failure, coronary heart disease, atrial fibrillation, pulmonary artery hypertension and subsequent right heart failure, venous thrombosis, and stroke. Other comorbidities include diabetes and metabolic syndrome, osteoporosis, cachexia and myopathy, obstructive sleep

apnea syndrome (OSAS), gastroesophageal reflux syndrome (GERD), and chronic renal failure.

In COPD, due to the level of inflammation involved, targeting inflammation and addressing all treatable comorbidities, requires a holistic approach to improve outcomes.

Cigarette smoking and exposure to particulates and noxious gases are avoidable. This is the most important tool in prevention. Avoiding the behavior of cigarette smoking would clearly help diminish COPD. Avoiding biomass exposure is more difficult but can be a goal. Air quality improvement in general certainly can be a worldwide goal for international governments. Considering in America, and elsewhere, the high cost of healthcare and chronic lengthy care that is common in COPD, it is fiscally responsible to do more to implement better deterrents to smoking, air pollution and provide clean fuels for use in homes.

Other preventative ideas involve measures that enable a better quality of life post diagnosis by preventing further comorbidities or furthering physiological aspects of their COPD. Protection from further infections may include; vaccination, masks when people visit, washing hands, regular visits to the dentist and practicing good regular dental hygiene. In addition, other lifestyle choices, supplements and yoga therapy that promote greater overall health and diminish inflammation and mucous production are important. Tending to the mental emotional state of patients should not be overlooked. This is a challenging disease that interweaves cause and effect with the physiological causes of mental emotional distress, while patients are facing their mortality.

Once diagnosed with COPD the treatment options are designed to minimize symptoms and slow down the progression of disease. For some, who quit smoking or remove the noxious air insult, they may live a more normal life. To diagnose COPD, spirometry is the most common tool. The test is designed to test lung function, how much air in, out and speed. FVC (forced vital capacity) and FEV1 (forced expiratory volume in the first second of the forceful exhalation) are the 2 important parameters.
Reduction in FEV1 is most common measure examined, due to its relation to airway obstruction. Other tests may include a X-ray, or arteriole blood gases.
COPD medications are different depending on symptoms and stage of disease. The primary two categories of medications include bronchodilators and immunosuppressants. Bronchodilators relax the muscles around the airways, to help keep them open, making breathing easier. Most are delivered through an inhaler or nebulizer to deliver the medicine directly. There is short acting -wear off in a few hours and long acting – wear off 12-24 hours but take longer to act at administration. The 2 most common bronchodilators used in COPD are beta2-agonists and anticholinergics. The first act by relaxing muscles around airway, short and long acting exist. The second prevent muscles around airway from tightening and help to clear mucus from the airway.
The second group of medication is for inflammation. Corticosteroids are generally used in an inhaler to minimize their systemic side effects, however in some cases an oral

corticosteroid may be used short-term. The side effects are commonly weight gain, diabetes, osteoporosis, increased risk of infection and mental emotional issues. Combination medications of corticosteroids and the bronchodilators is common.

Respiratory therapists and pulmonary rehabilitation programs are essential to providing patients with education, nutrition advice, exercise training, counseling and other life skills that really help the COPD patient. Additionally, they help patients navigate the use of supplemental oxygen, which can really help patients extend their time at home and generally have more energy.

Nutritional advice from the COPD Foundation includes the generic advice to increase vegetables and fruit, have whole grains, eat lean meat, eggs, fish or nuts.
They list these three references to further recommend nutritional ideas.

A healthy (mostly plant-based) diet is associated with less depression in COPD. Dinparast, F., Sharifi, A., Moradi, S. et al. The associations between dietary pattern of chronic obstructive pulmonary disease patients and depression: a cross-sectional study. BMC Pulm Med 21, 8 (2021). https://doi.org/10.1186/s12890-020-01383-5

A diet rich in antioxidants (mostly fresh, hard fruits and some vegetables), has been recommended for COPD patients in Role of Diet in Chronic Obstructive Pulmonary Disease Prevention and Treatment (Scoditti et al. Nutrients. 2019;11(6):1357. doi: https://doi.org/10.3390/nu11061357)

An earlier study suggested a positive impact of a diet rich in fiber on COPD. This study a https://bmcpulmmed.biomedcentral.com/articles/10.1186/s12890-020-01383-5#citeasnd other small trials in COPD were reviewed by Michael Greger, MD, in the 2012 blog "Treating COPD with Diet".

Dietary change or nutritional advice is becoming increasingly popular in America’s allopathic landscape. The NIH is funding more studies and an increase in health science by the younger generation, is leading more doctors in their fields to give more dietary advice. For example, a note on the American Lung Associations webpage refers to the metabolism of carbohydrates can lead to higher levels of carbo dioxide, which is the waste product we exhale. With COPD minimizing the carbon dioxide waste can help making breathing easier. To accomplish that, adding healthy fats in the diet and minimizing carbohydrates has been suggested.

An integrative or complimentary physician might exclude dairy due to its nearly ubiquitous ability to increase mucous. Other aspects of mucous forming foods might be examined like IgG food sensitivities. Anti-inflammatory activity might be provided by eliminating animal fats (high in arachidonic acid) and adding fish, flax, and nut oils (omega 3,6,9). Also supplementing with Boswellia serrata. To prevent oxidative stress and cell damage one might include antioxidants like vitamin C and E, NAC and resveratrol. To achieve both anti-inflammatory and antioxidant activity one might include; curcumin longa, green tea, or pycnogenol. To optimize the mitochondria (the

cell energy maker) NAC and CoQ10 a known factor in the Kreb's cycle, can be added. In addition, NAC has been used effectively in COPD and may act as a mucolytic as well. Each practitioner will look at the COPD patient and assess, comorbidities, general health and other factors to determine a plan that fits the individual with COPD.

Yoga therapy has been studied as a modality to treat and improve the quality of life for those living with COPD. In a Meta-analysis study looking at the effect of pulmonary rehabilitation in individuals with COPD using standard pulmonary function tests. The analysis showed that those who were in the yoga or Tai Chi groups had significant improvement in FEV1 values over those in the conventional exercise group. In another meta-analysis of 11 randomized controlled trials with 586 patients were included and revealed evidence for the effects of yoga compared to no treatment. The analysis particularly supported the trials that used breathing focused interventions but not in interventions including yoga postures. This meta-analysis found robust effects of yoga on exercise capacity and pulmonary function in patients with COPD. Yoga, specifically yoga breathing techniques, can be an effective adjunct intervention for patients with COPD. I found an example of the yoga used in a study. Most studies are not very descriptive, so I included this description. This study did have positive outcomes for the yoga group as measured by the FEV1.

"The training in yoga: The yoga practice given to COPD patients (for 3 months) included pranayama and asanas. Yoga includes initial relaxation of external body 5 min, pranayama (25min), asanas (15min), shavasana final relaxation (5 min), meditation (5 min) along with life style changes.17 Pranayama (breathing exercises) Bhastrika:, Anulom vilom:, Kapalbhati:, Bhramari: Asanas (postures) Surya Namaskar, Tadasana (mountain pose), Sukhasana, Paschimotaasana, padahasthasanam, bhavanamuthasanam, vrikshasanam, bhujangasanam, matsyaasanam, shalabhasanam, makarasanam, butterfly, Vajrasanam. (different sets of asanas per session considering the flexibility and safety of patients.) Shavasana: 5min Patients were given a videotape or pamphlets of yoga asanas and were strongly encouraged to practice daily at home. They were asked to document the number of sessions and number of minutes of home practice since the last yoga class during each yoga class session." John, et al. Yoga as an Adjunctive Therapy on the Respiratory Function of COPD

Yoga For COPD

The COPD population is quite varied in ability and they often possess other health issues or comorbidities. Starting, with all interventions including yoga, at the earliest time in their disease would be best. Discussing contraindications for this population really depends on where these people are in their health in general. Those who are not on oxygen and are on medications, have better movement tolerance and the ability to do longer bouts of pranayama. Assessing the COPD patient for movability, koshas and general health is part of the therapy intake. Devising a plan would be based on the individual or the individuals that show up for a class. In the latter situation a therapist must be ready with adaptions for the parameters around that individual.

The study above used 25 min of pranayama and 15 min asana. My design involves pranayama at beginning and at the end of class, with breath cues through the movement sections.

Begin Seated with centering on the physical body, with body scan then breath awareness, bringing attention to the mechanics of breathing. Bring awareness to structures around the thoracic cage, explain diaphragm position, and abdominal use with exhale.
Explore their own capacity for inhale and exhale.
*reminding them at any time during class, if they feel uncomfortable -to rest and with all breath cues to follow their own rhythm of breath, if they feel any level of dyspnea.
3 humm breath
Seated warm up-
Neck Ex: rotate In: back to front 2 x each side.
Lat flex with small shoulder rotation on second rep.
Ext /flex Ex bring chin down In lift chin up stretch through front neck 3rd rep stay1br on last IN
C/C arms extended elbows bent opp hand to elbow
slow Ex: round spine, further cross arms
In: pull shoulders posteriorly, lift chest, fill front body
Lat flex spine, In R arm up Ex: bend elbow over head and side stetch Rpt 2-3 x stay1br Rpt L
Hands or elbows on knees flx hips/extend spine and breath relax scap,shoulders etc lengthen spine
Spinal rotation with R hand on front of L shoulder rotate on ex stay on one side for 4 br
Legs and Feet: move to front of chair
Straighten legs Ankle rotation foot rotate one side toes up, toes down alt feet.
Alt lift knee and opp arm on In and lower on exhale
Stir the pot Ex lean forward In come back both directions
Center- breath - gentle Kapalbhati
FF arms on back or hips to open chest ex forward in up 3x

Standing behind chair hands on top of back chair
1. IN lift R arm and ext L leg ex back In alt rpt 3 x each side
2.Stand Lside to chair and do lat flexion In lift R arm bring over head and ex lengthening hip to tips 3x 3rd stay and inhale into ribs ex lengthen in arm up ex down Switch sides
3.Step back FF extend 90* In: step R foot forward like Vir 1 Ex: back to 90* In Step L etc

Sit get comfy
Anuloma ujjayi teach mudra, inhale both nostrils Ex Left, IN bi-nostril, EX Right etc
Relaxation Guided Imagery tree oxygen to us us carbon to trees basic anat/phys 8min
3 hums ask if they notice a change in tone, capacity or volume from beginning.
Close

Resources

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