Dyspnea

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Definition

- The American Thoracic Society consensus statement defines dyspnea as a

  “subjective experience of breathing discomfort ”

  The experience derives from interactions among multiple physiological, psychological, social, and environmental factors

- Dyspnea, a symptom, can be perceived only by the person experiencing it and, therefore, must be self-reported.
- In contrast, signs of increased work of breathing, such as tachypnea, accessory muscle use, and intercostal retraction, can be measured and reported by clinicians.

- Dyspnea is a common
- up to one half of inpatients and one quarter of ambulatory patients experience dyspnea, with a prevalence of 9–13% in the community that increases to as high as 37% for adults aged ≥70 years.
- the degree of dyspnea may better predict outcomes in COPD than does the forced expiratory volume in 1 s (FEV1), and formal measures of dyspnea have been incorporated into GOLD 2017 COPD severity assessment guidelines.
- Also predict outcomes in other chronic heart and lung diseases as well.
- Dyspnea can arise from a diverse array of *pulmonary*, *cardiac*, and *neurologic* underlying causes

MECHANISMS UNDERLYING DYSPNEA

- Complex- a mismatch in the integrative signaling between these two pathways, termed “efferent-reafferent mismatch”

- Afferent signals trigger the CNS (brainstem and/or cortex) and include primarily

- Efferent signals are sent from the CNS (motor cortex and brainstem) to the respiratory muscles,
- In addition, fear or anxiety may heighten the sense of dyspnea through exacerbating the underlying physiologic disturbance in response to an increased respiratory rate or disordered breathing pattern.

- (a) peripheral chemoreceptors in the carotid body and aortic arch and central chemoreceptors in the medulla

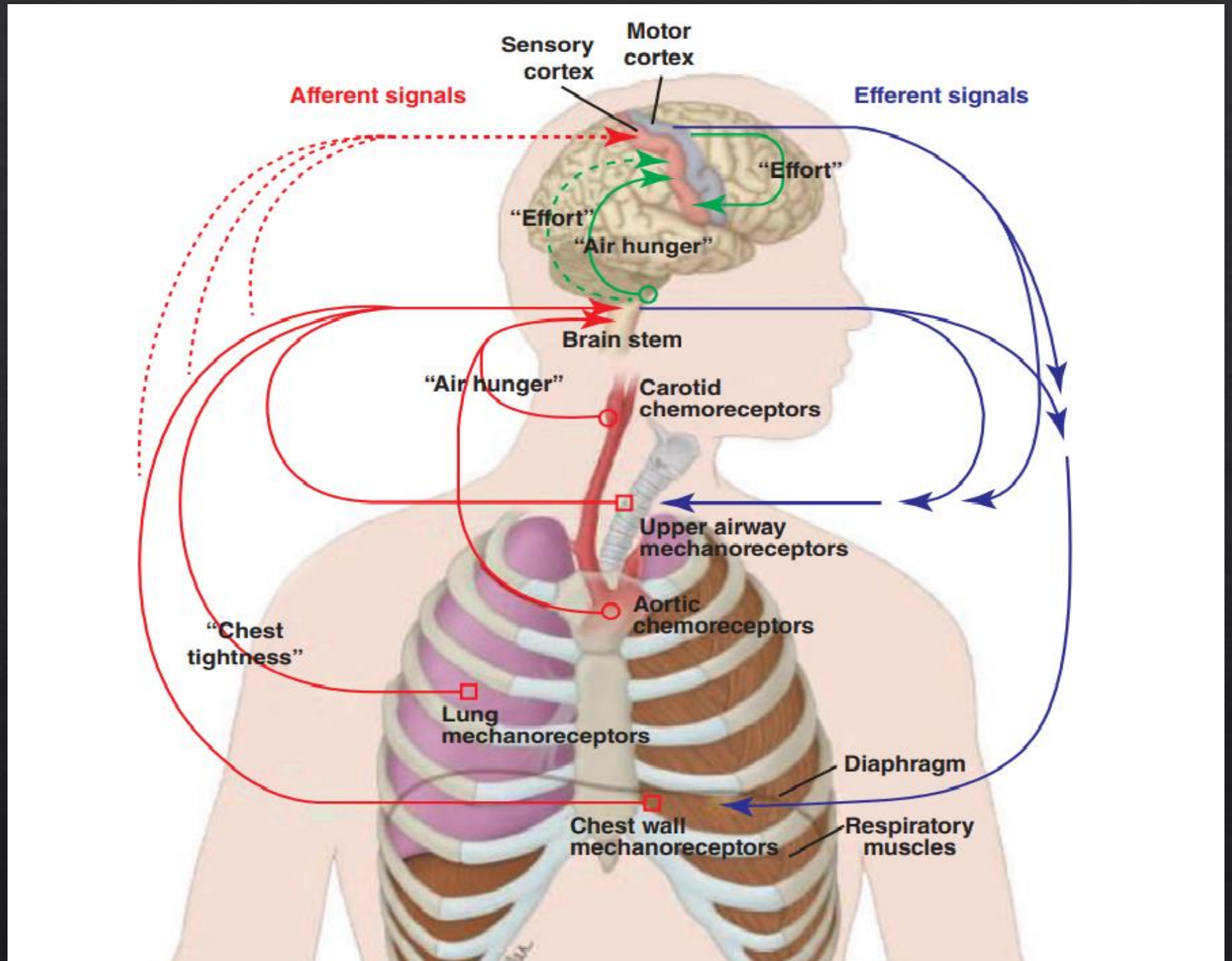
  activated by hypoxemia, hypercapnia, or academia, and might produce a sense of “air hunger”;

- (b) mechanoreceptors in the upper airways, lungs (including stretch receptors, irritant receptors, and J receptors), and chest wall (including muscle spindles as stretch receptors and tendon organs that monitor force generation)

  activated in the setting of an increased work load from a disease state producing an increase in airway resistance that may be associated with symptoms of chest tightness (e.g., asthma or COPD) or decreased lung or chest wall compliance (e.g., pulmonary fibrosis).

- (c) metaboreceptors: Other afferent signals that trigger dyspnea within the respiratory system can arise from pulmonary vascular receptor responses to changes in pulmonary artery pressure and skeletal muscle

sense changes in the biochemical environment


Sensory cortex
Motor cortex
Afferent signals
Efferent signals
"Effort"
"Effort"
"Air hunger"
Brain stem
"Air hunger"
Carotid chemoreceptors
Upper airway mechanoreceptors
Aortic chemoreceptors
"Chest tightness"
Lung mechanoreceptors
Diaphragm
Chest wall mechanoreceptors
Respiratory muscles

ASSESSING DYSPNEA

TABLE 33-1 An Example of a Clinical Method for Rating Dyspnea: The Modified Medical Research Council Dyspnea Scale[a]

GRADE OF DYSPNEA	DESCRIPTION
0	Not troubled by breathlessness, except with strenuous exercise
1	Shortness of breath walking on level ground or with walking up a slight hill
2	Walks slower than people of similar age on level ground due to breathlessness, or has to stop to rest when walking at own pace on level ground
3	Stops to rest after walking 100 m or after walking a few minutes on level ground
4	Too breathless to leave the house, or breathless with activities of daily living (e.g., dressing/undressing)

[a]Which has been incorporated into the GOLD 2017 guidelines as a possible tool for rating dyspnea in COPD.

Source: Modified from DA Mahler, CK Wells: Evaluation of clinical methods for rating dyspnea. Chest 93:580, 1988.

DIFFERENTIAL DIAGNOSIS

- chronic dyspnea, which is defined as symptoms lasting longer than 1 month and can arise from a broad array of different underlying conditions, most commonly attributable to pulmonary or cardiac conditions that account for as many as 85% of the underlying causes of dyspnea.

TABLE 33-2 Differential Diagnosis of Disease Processes Underlying Dyspnea

SYSTEM	TYPE OF PROCESS	EXAMPLE OF DISEASE PROCESS	POSSIBLE PRESENTING DYSPNEA SYMPTOMS	POSSIBLE PHYSICAL FINDINGS	POSSIBLE MECHANISMS UNDERLYING DYSPNEA	INITIAL DIAGNOSTIC STUDIES (AND POSSIBLE FINDINGS)
Pulmonary	Airways disease	Asthma, COPD	Chest tightness, tachypnea, increased WOB, air hunger, inability to get a deep breath	Wheezing, accessory muscle use, exertional hypoxemia (especially with COPD)	Increased WOB, hypoxemia, hypercapnia, stimulation of pulmonary receptors	Peak flow (reduced); Spirometry (OVD); CXR (hyper-inflation; loss of lung parenchyma in COPD)
	Parenchymal disease	Interstitial lung disease[a]	Air hunger, inability to get a deep breath	Dry end-inspiratory crackles, clubbing, exertional hypoxemia	Increased WOB, increased respiratory drive, hypoxemia, hypercapnia, stimulation of pulmonary receptors	Spirometry and lung volumes (RVD); CXR and chest CT (interstitial lung disease)
	Chest wall disease	Kyphoscoliosis, Neuromuscular (NM) weakness	Increased WOB, inability to get a deep breath	Decreased diaphragm excursion; atelectasis	Increased WOB; stimulation ofpulmonary receptors (if atelectasis is present)	Spirometry and lung volumes (RVD); MIP and MEPs (reduced in NM weakness)

Differential diagnosis of interstitial lung disease includes idiopathic pulmonary fibrosis, collagen vascular disease, drug or occupation-induced pneumonitis, lymphangitic spread of malignancy;

Pulmonary and cardiac	Pulmonary vasculature	Pulmonary Hypertension	Tachypnea	Elevated R heart pressures, exertional hypoxemia	Increased respiratory drive, hypoxemia, stimulation of vascular receptors	Diffusion capacity (reduced); ECG; ECHO (to evaluate PA pressures)[b]
Cardiac	Left heart failure ——— Pericardial disease	Coronary artery disease, cardio-myopathy[c] ——— Restrictive pericarditis; Cardiac tamponade	Chest tightness, air hunger	Elevated L heart pressures; wet crackles on lung examination; pulsus paradoxus (pericardial disease)	Increased WOB and drive, hypoxemia, stimulation of vascular and pulmonary receptors^	Consider BNP testing in the acute setting; ECG, ECHO, may need stress testing and/or LHC
Other	Variable	Anemia Deconditioning Psychological	Exertional breathlessness Poor fitness Anxiety	Variable	Metabo-receptors (anemia, poor fitness); chemoreceptors (anaerobic metabolism from poor fitness); some subjects may have increased sensitivity to hypercapnia	Hematocrit for anemia; exclude other causes

APPROACH TO THE PATIENT WITH DYSPNEA

- For patients with a known prior pulmonary, cardiac, or neuromuscular condition and worsening dyspnea, the initial focus of the evaluation will usually address determining whether the known condition has progressed or whether a new process has developed
- For patients without a prior known potential cause of dyspnea, the initial evaluation will focus on determining an underlying etiology.

HISTORY:

- The patient should be asked to describe in his/her own words what the discomfort feels like as well as the effect of position, infections, and environmental stimuli on the dyspnea, as descriptors may be helpful in pointing toward an etiology
- symptoms of chest tightness :possibility of bronchoconstriction
- the sensation of inability to take a deep breath: dynamic hyperinflation from COPD.
- Orthopnea :congestive heart failure (CHF)

- mechanical impairment of the diaphragm associated with: obesity, or asthma triggered by esophageal reflux.
- Nocturnal dyspnea :CHF or asthma.
- Acute, intermittent episodes of dyspnea : MAY reflect episodes of myocardial ischemia, bronchospasm, or pulmonary embolism, WHILE
- chronic persistent dyspnea : COPD, interstitial lung disease, and chronic thromboembolic disease
- platypnea—i.e., dyspnea in the upright position with relief in the supine position: Left atrial myxoma or hepatopulmonary

PHYSICAL EXAMINATION

- Initial vital signs
- the presence of fever :underlying infectious or inflammatory process
- the presence of hypertension :heart failure might point toward diastolic dysfunction;
- the presence of tachycardia : cardiac dysfunction, and deconditioning;
- the presence of resting hypoxemia : hypercapnia, ventilation-perfusion mismatch, shunt, or impairment in diffusion capacity
- Inability of the patient to speak in full sentences before stopping to get a deep breath : condition that leads to stimulation of the controller or impairment of the ventilatory pump with reduced vital capacity.

- Evidence of increased work of breathing : increased airway resistance or stiffness of the lungs and the chest wall.
- assess the respiratory rate and measure the pulsus paradoxus :the systolic pressure decreases by >10 mmHg, the presence of COPD, acute asthma, or pericardial disease
- Examination of the chest should focus on symmetry of movement; percussion and auscultation

- The cardiac examination :jugular venous distention, edema, accentuated pulmonic component to the second heart sound); left ventricular dysfunction (S3 and S4 gallops); and valvular disease (murmurs)
- the abdomen with the patient in the supine position: paradoxical movement of the abdomen ,inward motion during inspiration is a sign of diaphragmatic weakness, and rounding of the abdomen during exhalation is suggestive of pulmonary edema
- Clubbing : interstitial pulmonary fibrosis or bronchiectasis, and joint swelling or deformation as well as changes consistent with Raynaud's disease may be indicative of a collagen-vascular process that can be associated with pulmonary disease

- Patients should be asked to walk under observation with oximetry in order to reproduce the symptoms. The patient should be examined during and at the end of exercise for new findings that were not present at rest (e.g., presence of wheezing), and for changes in oxygen saturation

CHEST IMAGING

- lung volumes:

hyperinflation is consistent with obstructive lung disease

low lung volumes suggest interstitial edema or fibrosis, diaphragmatic dysfunction, or impaired chest wall motion

- pulmonary parenchyma: interstitial disease, infiltrates, and emphysema
- Prominent pulmonary vasculature: in the upper zones indicates pulmonary venous hypertension, while enlarged central pulmonary arteries may suggest pulmonary arterial hypertension
- Bilateral pleural effusions : CHF and some forms of collagen-vascular disease.
- Unilateral effusions raise the specter of carcinoma and pulmonary embolism but may also occur in heart failure or in the case of a parapneumonic effusion.

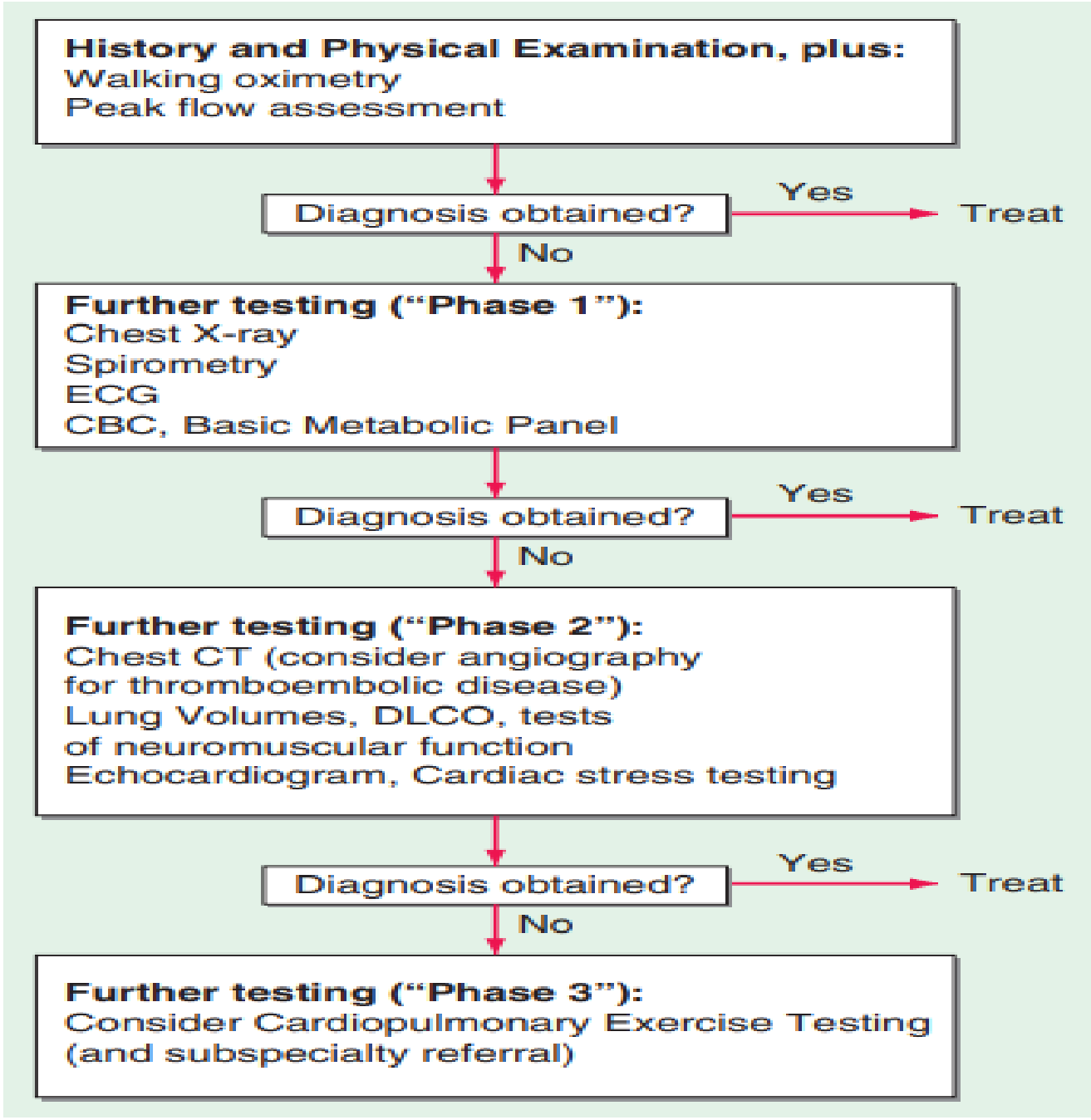
- CT of the chest is generally reserved for further evaluation of the lung parenchyma (interstitial lung disease) and possible pulmonary embolism if there remains diagnostic uncertainty

LABORATORY STUDIES

- hematocrit to exclude occult anemia as an underlying cause of reduced oxygen-carrying capacity contributing to dyspnea
- basic metabolic panel: possibility of carbon dioxide retention that might be seen in chronic respiratory failure
- electrocardiography :ventricular hypertrophy and prior myocardial infarction
- spirometry : the presence of an obstructive ventilatory defect, and suggest the possibility of a restrictive ventilatory defect
- Echocardiography :systolic dysfunction, pulmonary hypertension, or valvular heart disease
- BNP: for CHF in patients presenting with acute dyspnea but may be elevated in the presence of right ventricular strain as well.

DISTINGUISHING CARDIOVASCULAR FROM RESPIRATORY SYSTEM DYSPNEA

- CPET
- If, at peak exercise, the patient achieves predicted maximal ventilation, demonstrates an increase in dead space or hypoxemia, or develops bronchospasm, the respiratory system may be the cause of the problem.
- if the heart rate is >85% of the predicted maximum, if the anaerobic threshold occurs early, if the blood pressure becomes excessively high or decreases during exercise, if the O2 pulse (O2 consumption/heart rate, an indicator of stroke volume) falls, or if there are ischemic changes on the electrocardiogram, an abnormality of the cardiovascular system is likely the explanation for the breathing discomfort.
- Additionally, a CPET may also help point toward a peripheral extraction deficit, or metabolic/neuromuscular disease as potential underlying processes driving dyspnea


History and Physical Examination, plus:
Walking oximetry
Peak flow assessment
Diagnosis obtained?
Yes
Treat
No
Further testing ("Phase 1"):
Chest X-ray
Spirometry
ECG
CBC, Basic Metabolic Panel
Diagnosis obtained?
Yes
Treat
No
Further testing ("Phase 2"):
Chest CT (consider angiography
for thromboembolic disease)
Lung Volumes, DLCO, tests
of neuromuscular function
Echocardiogram, Cardiac stress testing
Diagnosis obtained?
Yes
Treat
No
Further testing ("Phase 3"):
Consider Cardiopulmonary Exercise Testing
(and subspecialty referral)

TREATMENT

- The first goal is to correct the underlying condition
- If relief not fully possible: an effort is made to lessen the intensity of the symptom and its effect on the patient's quality of life.
- Supplemental O2 : if the resting O2 saturation is ≤88% or if the patient's saturation drops to these levels with activity or sleep. In COPD decrease mortality,
- pulmonary rehabilitation
- Opioids have been shown to reduce symptoms of dyspnea, largely through reducing air hunger, thus, likely suppressing respiratory drive and influencing cortical activity. should be considered for each patient individually
- Studies of anxiolytics for dyspnea have not demonstrated consistent benefit.
- inhaled furosemide that might alter afferent sensory information.