Physical Diagnosis of Chronic Obstructive Pulmonary Disease

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Abstract

Among the various diagnostic strategies of chronic obstructive pulmonary disease (COPD), physical diagnosis is the quickest and requires no extra cost. Rapid physical diagnosis of COPD in primary care practice can lead to earlier actions of preventive measures and counseling for patients. Further, rapid physical diagnosis of COPD in an emergency department is also crucial for timely use of potentially lifesaving therapy specific for COPD patients. In this review, we will present a broad scope of physical findings for rapid physical diagnosis of COPD.

Key words: chronic obstructive pulmonary disease, physical examination, inspection, percussion, palpation, auscultation, vital sign, acute exacerbation

Introduction

Chronic obstructive pulmonary disease (COPD) is a major global health concern. However, undiagnosed chronic obstructive pulmonary disease is common in the general population and is associated with impaired health (1, 2). Among the various diagnostic strategies of COPD, physical diagnosis is quickest and less expensive, and this can lead to earlier actions of secondary prevention. Rapid physical diagnosis of COPD is important even in patients free of pulmonary symptoms, since this can lead to more intensive counseling on smoking cessation and vaccination against influenza and pneumococcal infections. Thus, in settings such as primary care practice, home-based, and community-based care, rapid physical diagnosis of COPD is also still useful and important.

Rapid physical diagnosis of COPD is crucial also in an emergency department for timely use of potentially lifesaving therapy with bronchodilators or systemic steroids in patients with acute exacerbation of COPD, whose previous clinical history (indicating the presence of COPD) may be unavailable at the time of arrival to an emergency department. Moreover, the clinical recognition of COPD by physical diagnosis may have time, cost, and convenience advantages compared to pulmonary function testing and computed tomography. Thus, in this review article, we will present a broad scope of physical findings of COPD (Table 1). In addition to physical findings for rapid diagnosis of COPD, we will also present interpretation of vital signs for assessing the severity of acute exacerbation of COPD.

Inspection

Pursed-lip breathing

Patients with COPD tend to exhale with pursed-lips. In this way, they increase expiratory airway resistance to elevate pressure inside the small collapsible airways for preventing alveolar collapse or slow the breathing frequency (3). Because the airways are not at risk of collapse during inspiration, many patients, who do purse their lips, do it unconsciously only during expiration. This is considered as a form of self-administered positive end-expiratory pressure (4).

Use of accessory muscles of respiration

This finding may be accompanied by increased activity of sternocleidomastoid and scalenus muscles in patients with COPD (5). With chronic compensatory use, sternocleidomastoid muscles may develop noticeable hypertrophy and they will be thicker than the patient’s own thumb (6).
the contrary, hypertrophy of scalenus muscles is more likely identified in patients with restrictive lung disease (6).

**Jugular venous distension during expiration**

An inspection of the neck veins usually involves an estimation of jugular venous pressure and an analysis of the venous pulse (7). However, jugular venous pressure may be difficult to assess in patients with severe lung hyperinflation because of marked respiratory variations in intrathoracic pressures (8). Jugular venous distension during expiration indicates an increased positive pressure in the thorax of patients with COPD (9). The presence of a prominent jugular V wave with loss of X decent (CV merger) may indicate cor pulmonale with tricuspid valvular regurgitation (10). Figure 1 presents jugular venous wave forms in patients with COPD with tricuspid regurgitation and in normal controls. Additionally, hepatojugular reflux is a useful adjunct to jugular venous distention in the diagnosis of cor pulmonale with tricuspid regurgitation (11).

**Retraction of supraclavicular fossa during inspiration**

Retraction of supraclavicular fossae during inspiration is due to excessive swings of intrathoracic pressure, and this finding probably results from a phase lag between the generation of negative pleural pressures and the resultant change in lung volume (12). One study found significant correlation between the reduced forced expiratory volume in one second (FEV1) and the presence of this finding (13).

**Short trachea**

The distance between the suprasternal notch and the lower edge of thyroid cartilage is 3 to 4 digits in width in normal individuals, while this may be shortened to about 1 to 2 digits in patients with COPD (12). The configuration of trachea is distorted in patients with COPD and the ratio of the short to the long radius of trachea is smaller in patients with COPD than in normal controls (14).

**Respiratory paradox (respiratory alternans)**

Normally, the abdominal wall moves passively outward during inspiration, as the descending diaphragm squeezes the intraabdominal contents down and outward. Then the abdominal wall retracts during expiration as the diaphragmatic piston returns to its resting position. By progressive weakness of the diaphragm due to overwork in severe COPD, the weak diaphragm is passively sucked upwards in inspiration as the intercostal muscles do the work of inspiration; the abdominal wall retracts during inspiration (15). This is called the respiratory paradox (16). Patients with this sign usually

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**Table 1. Useful Physical Signs for Diagnosis of COPD**

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COPD: chronic obstructive pulmonary disease.
Figure 1. Jugular Venous Pulsations. Upper; normal wave form: Lower; tracing of patients with tricuspid regurgitation, superimposed on the normal wave form. A=atrial contraction; C=carotid transmission (not visible clinically); X=descent in right atrium following A; V=passive venous filling of atria from the venae cavae; Y=descent during atrial resting phase before contraction. CV merger indicates the merged wave of C and V waves. S1 and S2 denote the first and second heart sounds, respectively (27, 57). Arrow indicates v wave.

have severe ventilatory failure and may be in need of mechanical ventilation.

Muscle wasting

Patients with advanced COPD gradually lose weight and show muscle wasting that is attributed to immobility, hypoxia, or release of systemic inflammatory mediators, such as the tumor necrosis factor (17). Lower body mass index is included into a multidimensional grading system (BODE index) as a significant predictor of hospitalization for COPD (18, 19).

Increased antero-posterior chest diameter (barrel chest or pulmonary kyphosis)

Because patients with COPD undergo significant weight loss, the increased antero-posterior chest diameter may be an illusory finding from an image effect due to a decreased antero-posterior abdominal diameter (20).

Loss of pump-handle and bucket-handle movements of the chest

In the normal respiratory movement by inspection from the lateral side, one will see a pump handle-like movement of the chest with the point of the sternal angle (angle of Louis) as a fulcrum shaft. In addition, by inspection from the front, one will see a bucket handle-like outward movement of the bilateral contours of the rib cage during inspiration. With increased disease severity, patients with COPD may lose these movements, which indicate that %FEV is less than 40% (12, 21).

Peripheral edema

The presence of significant edema may indicate right-sided heart failure or cor pulmonale in patients with pulmonary hypertension from severe COPD. Edema can also be present if pCO2 is elevated over 65 mmHg because of oliguria due to redistribution of renal blood flow from cortical to medullary areas of kidneys (22-25). Hypoxia less than 40 mmHg of PO2 can also cause renal vasoconstriction and subsequent oliguria and edema (23). Patients with COPD and peripheral edema have a 5-year survival rate of only about 30% (26).

Dyspnea-relieving posture

It is important to observe what position the patient assumes for most comfortable breathing to improve respiratory mechanics (27). Patients with more advanced disease may have postures that relieve dyspnea, such as leaning forward against outstretched palms (27).

Hoover’s sign

With the patient supine, the examiner should lightly rest the right hand on the patient’s left hypochondrium, with the thumb on the medial costal margins and the remaining fingers superiorly toward the patient’s head. Then the examiner should lightly place the left hand on the patient’s right costal margin, symmetric to the right hand. The patient should be instructed to take a deep breath. Normally, both hands will swing out symmetrically during inspiration and the thumbs will form a more obtuse angle, returning to a more acute angle with expiration. The hands are not to offer resistance, but only to increase the appreciation of the change in angle. With practice, the examiner can observe this sign without using the hands (27). The change of this angle during inspiration is determined by the balance between two forces: the lateral pull on the costal margins due to the intercostal muscles and the contrary action of the diaphragm normally exerted only at end-expiration when the diaphragm is flat. When the diaphragm is sufficiently flattened in early inspiration, as in COPD, its muscle fibers pull horizontally rather than vertically and might overcome the action of the intercostal muscles and the contrary action of the diaphragm normally exerted only at end-expiration when the diaphragm is flat. This sign has also been called Hoover’s groove, because one can sometimes see a groove as the flattened diaphragm pulls inward. Hoover’s sign may be lost if the patient leans forward, because the increased abdominal pressure causes the diaphragm to take a more convex orientation (30).
Palpation

Palpation of the chest is limited since the bony rib cage hides many abnormalities of the lungs. However, palpation of the chest in patients with COPD may detect abnormal respiratory excursion. Respiratory excursion can be assessed when the patient breathes in and out, either by simultaneous palpation of symmetric areas of the chest or by measurement of the changing circumference (31). In addition, palpation in patients with COPD should also include locating a point of maximum impulse of the heart. Tactile fremitus also seems to be decreased in patients with COPD.

Restricted chest expansion

Restricted expansion can be quantitated by measuring the difference in circumference of the chest between end-expiration and end-inspiration with a tape measure placed at the nipple line (32). The normal value has been stated to be 5 cm: An expansion of 3.8 cm or less is considered impaired (33). As a general rule, a single measurement of less than 2.5 cm is definitely abnormal, while a measurement of more than 7.6 cm is normal (27).

Exaggerated bulging of the intercostal spaces

Normally, the intercostal spaces bulge inward during inspiration and outward with expiration. An exaggeration of the inspiratory retraction occurs in patients with COPD. The mechanism of this finding is likely due to an imbalance between the ability of the respiratory muscles to create a negative intrapleural pressure and the impaired ability of the lungs to expand (27). Exaggerated expiratory bulging of the intercostal spaces also results from a mechanism similar to that of heightened inspiratory retraction (34). Diffuse expiratory bulging suggests that the lungs are not being emptied because of an increased expiratory airway resistance.

Subxiphoid shift of a point of maximum impulse of the heart

Patients with COPD may not have a point of maximum impulse of the heart (PMI) in the expected place (35, 36). This is also called an “absent” apical impulse. PMI may be found in the subxiphoid region, which suggests that %FEV1 is less than 50% (12). In addition, a systolic para-sternal heave may indicate the presence of right ventricular hypertrophy (7).

Percussion

Chest hyperresonance

The chest of patients with COPD should be percussed to determine the quality of the sound that resonates. If the sound is more hollow than normal, the chest is called hyperresonant (37). Generalized and symmetrical hyperresonance on percussion is a valuable finding suggestive of COPD.

Drop heart (microcardia)

On percussion, the heart of patients with advanced COPD may be noted in small and vertical shape (38). This finding is sometimes referred to as a drop heart (shaped like a teardrop) or as microcardia, which should suggest strongly the presence of subclinical emphysema (38).

Auscultation

The recommended terms for adventitious lung sounds, based on their acoustic characteristics, are crackles for discontinuous sounds and wheeze or rhonchus for continuous sounds (39). Wheezing will sound like high-pitched musical tones, while rhonchi will sound like lower-pitched wheezes (40). In auscultation, patients with COPD often present with diminished lung sounds, prolonged expiratory time, and expiratory wheezing that initially may occur only on forced and unforced expiration (27, 41). One may hear coarse crackles beginning with inspiration (early inspiratory crackles). Cardiac auscultatory signs of COPD include distant heart sounds, sometimes best heard in the epigastrium.

Diminished lung sounds

Although breath sound intensity is insensitive to mild degrees of ventilatory impairment (42), definitely reduced intensity is a strong indicator of obstructive pulmonary disease, and normal breath sounds virtually exclude the possibility of severe COPD (43, 44). Another study stated that diminished breath sounds are also the best predictor of moderate-to-severe COPD (36).

Early inspiratory crackles

Crackles in the early inspiratory phase correlate with obstructive lung disease involved with medium to large airways, while late inspiratory crackles are associated with interstitial lung disease (40, 45). Early inspiratory crackles occurring in patients with COPD generally mean that the %FEV1 is less than about 40% (45). Early inspiratory crackles can radiate through the mouth of COPD patients so that one can hear it by holding the stethoscope in front of the opened mouth (23). In contrast, crackles from congestive heart failure do not radiate through the opened mouth (7). Early-to-mid inspiratory crackles indicate the presence of bronchiectasis (46).

Amphoric breathing (jar breathing; cavernous breathing)

Amphoric breathing resembles tracheal breathing in that the two phases (inspiratory and expiratory) are much closer to each other in amplitude and duration than in normal vesicular sounds. However, amphoric breathing has a more resonant and harmonious timbre than tracheal breathing, and it is heard where vesicular breath sounds are expected (27). Amphoric breathing indicates a cyst, bleb, bulla, or other air-containing space in the lung, which is in communication
with the bronchial system. Cavities with rigid and inflexible walls can produce the best amphoric breath sounds, whereas a resilient and deformable cavity would produce no amphoric sounds due to the lack of vibrations (27). Once a cavity appears and produces amphoric breathing, it should be permanent. Thus, disappearance of a previously noted area of amphoric breathing may suggest that something has filled the cavity, such as blood, pus, or aspergilloma (27).

**Accentuated P2**

Signs of cor pulmonale in COPD include splitting of the 2nd heart sound with an accentuated pulmonic component, which is an indication of pulmonary hypertension (7). Further, tricuspid regurgitation murmur may indicate the presence of right ventricular dilatation. This murmur can be differentiated from left side murmur by noting an increased intensity of the tricuspid regurgitation murmur during inspiration, described as Carvallo’s sign (11).

**Special Maneuver**

**Forced expiratory time (FET)**

The bell of a stethoscope should be placed over the trachea in the suprasternal notch and a stopwatch should be set to zero. The patient should be instructed to take in the deepest breath possible and then to blow it all out as fast as possible. As the patient begins to exhale, the stopwatch should be started. As soon as audible expiration is no longer heard, the stopwatch should be turned off. An FET of more than 6 seconds indicates considerable expiratory airflow obstruction with %FEV1 <50% (47). Three trials with averaging the results are usually recommended. This clinically measured forced expiratory time correlates well with the forced expiratory time measured by spirometry, the latter being almost one second greater on the average (47).

**Match test (Snider test)**

The patient is asked to huff a match out. Huffing is performed with the mouth and lips as wide open as possible. The normal individual can huff a match out at 15 to 20 cm on the first try (27). Patients with moderate-to-severe COPD have great difficulty with this test (48). Some require multiple trials to huff out the match at 15 cm, and some with severe disease are unable to accomplish this. Using a cutoff of 10 cm is a good test to rule in moderate severity of COPD (36). Use of therapeutic oxygen in patients with COPD is a contraindication to this test.

**Vital Signs for Assessing the Severity of Acute Exacerbation of COPD**

**Blood pressure**

Hypercapnia may cause transient hypertension with wide pulse pressure, sometimes along with hot hands (5 mmHg or a higher increase from the baseline value in PCO2), flapping tremor (15 mmHg or a higher increase from the baseline value in PCO2), and cold sweat (49-51). Symptomatic hypotension with oliguria can also be induced by an extremely severe hypoxia (PO2 <20 mmHg).

**Heart rate**

Both hypoxia and hypercapnia can induce tachycardia by an increased release of catecholamine (49). Severe hypoxia or hypercapnia can cause arrhythmia such as multi-atrial tachycardia, atrial fibrillation or ventricular tachycardia (52, 53).

**Respiratory rate**

The respiratory rate increases proportionally to disease severity. Rapid and shallow respirations of more than 40 per minute may cause respiratory muscle fatigue, and this finding along with respiratory acidosis suggests indication for mechanically assisted ventilation (49, 54). A respiratory rate of less than 7 per a minute with hypercapnia indicates an impending respiratory arrest (49).

**Body temperature**

Temperatures over 38.5 or under 35.5 Celsius require careful attention, particularly considering the possibility of sepsis. Shaking chills, or moderate chills along with tachycardia or tachypnea, are a hallmark of sepsis (55).

**Pulsus paradoxus**

While measuring blood pressure, one can determine whether there is pulsus paradoxus. During tidal breathing, the cuff is inflated to above the systolic blood pressure. The cuff pressure is slowly released until the first Korotkoff sound is heard only during expiration; this value of systolic blood pressure is then noted. The cuff pressure is further reduced until the first Korotkoff sound is heard throughout inspiration; this value of systolic blood pressure at this point is also noted. The systolic blood pressure is normally lower during inspiration than during expiration. If the difference between these two pressures is at least 15 mmHg, the patient has pulsus paradoxus, and %FEV1 is likely to be 25% or lower (23, 37). Moreover, this difference is more accentuated, as the patient has a more severe degree of acute exacerbation (56).

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