Review Article

Physical signs in patients with chronic obstructive pulmonary disease

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ABSTRACT

We reviewed the various physical signs of chronic obstructive pulmonary disease, their pathogenesis, and clinical importance. We searched PubMed, EMBASE, and the CINAHL from inception to March 2018. We used the following search terms: chronic obstructive pulmonary disease, physical examination, purse-lip breathing, breath sound intensity, forced expiratory time, abdominal paradox, Hoover's sign, barrel-shaped chest, accessory muscle use, etc. All types of studies were chosen. Globally, history taking and clinical examination of the patients is on the wane. One reason can be a significant development in the field of medical technology, resulting in overreliance on sophisticated diagnostic machines, investigative procedures, and medical tests as first-line modalities of patient's management. In resource-constrained countries, detailed history taking and physical examination should be emphasized as one of the important modalities in patient's diagnosis and management. Declining bedside skills and clinical aptitude among the physician is indeed a concern nowadays. Physical diagnosis of chronic obstructive pulmonary disease (COPD) is the quickest and reliable modalities that can lead to early diagnosis and management of COPD patients. Bedside elicitation of physical signs should always be the starting point for any diagnosis and therapeutic approach.

KEY WORDS: Accessory muscle use, barrel-shaped chest, chronic obstructive pulmonary disease, Hoover's sign, physical examination, purse-lip breathing

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a common, preventable, and treatable disease characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.[1] COPD is a major global public health issue because of its high prevalence, morbidity, and mortality.[2,3] The socioeconomic impact of COPD is also substantial.[4] Adeloye *et al*.[2] in a systematic review and meta-analysis reported a global prevalence of spirometry-defined COPD of 11.7%. According to the World Health Organization report, more than 3 million people died of COPD in 2012 and majority of the deaths occurred in developing countries.[3] The mortality due to COPD is rising, and it is expected to become the third leading cause of death globally by 2030.[5] Underdiagnosis of COPD is a global phenomenon. Lamprecht *et al*.[6] had shown that 81.4% of (spirometrically defined) COPD cases remain undiagnosed. Solution to the problem of underdiagnosis lies with proper planning and implementation of strategies

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focused on an early and accurate diagnosis of COPD. Among the various diagnostic modalities, physical diagnosis is rapid, cost-effective, and can lead to the earlier institution of various preventive and treatment strategies. The initial clinical examination (history and physical) helps in developing a rapport with the patients, identifying the severity of symptoms, determining prognosis, and monitoring therapy.[7] Clinical examination also helps in developing a pretest probability of the disease. It is highly relevant to establish the efficacy of clinical diagnosis, as an early diagnosis by clinical criteria will increase the number of patients for spirometric confirmation of diagnosis. There should be judicious use of various diagnostic modalities such as chest radiograph, pulmonary function tests, or other laboratory tests along with the evaluation of detailed history and physical signs. Interpretations of diagnostic tests without clinical findings will lose its importance. In this review, we will discuss various physical signs of COPD, their pathogenesis, and clinical importance.

PATHOPHYSIOLOGICAL CHANGES IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

COPD patients often develop hyperinflation. Hyperinflation occurs due to expiratory flow limitation caused by reduced lungs' elastic recoil and increased airway resistance. Hyperinflation increases during exercise and acute exacerbation. Hyperinflation has a significant negative impact on respiratory muscles, particularly the diaphragm. Studies done in animals, healthy humans, and COPD patients have shown that hyperinflation increases the contribution of rib cage and neck muscles and decreases relative contribution of the diaphragm.[8] The effect of hyperinflation on diaphragmatic length is the main mechanism by which it affects the force-generating capacity of the diaphragm.[8,9] The diaphragm becomes flattened and shorter in emphysema. According to Laplace's law, flattened diaphragm because of its increased radius of curvature has reduced force-generating capacity.[10] Moreover, increase in resistive and elastic load and hypoxemia also causes inspiratory muscle fatigue.[11] The impact of hyperinflation on inspiratory intercostal muscles is substantially less compared to that of diaphragm.[12] Dysfunction of the diaphragm may lead to the development of various clinical signs such as abdominal paradox, Hoover's sign, and tripod position.[13]

ACCESSORY MUSCLE ACTIVITY

Accessory muscles of respiration include the sternocleidomastoid, scalene, trapezius, internal intercostal, and abdominal muscles. Dynamic hyperinflation and air trapping in COPD patients place the diaphragm and intercostal muscles in a mechanically disadvantageous position. Because of this, the diaphragm and intercostals are unable to provide adequate ventilation, leading to the recruitment of accessory muscles. Two important accessory muscles of inspiration are the scalene and sternocleidomastoid. Accessory muscle use is one of the earliest signs of airway obstruction. Use of accessory muscles indicates severe disease and signifies that the forced expiratory volume in 1 s (FEV_1) is decreased to 30% of the normal or less. More than 90% patients with acute exacerbations of COPD exhibit use of accessory respiratory muscles.[14] The activity of these muscles is best judged by palpation. The contraction of scalenes is felt by pressing the fingertips into the floor of the posterior triangle of the neck gently. The sternomastoids are examined by drawing them backward with the thumb and first finger to feel the contraction.

Chronic use of sternocleidomastoids may lead to the development of noticeable hypertrophy, and they may become thicker than patient's own thumb. The sternocleidomastoid activity can be seen in respiratory distress due to any cause, but their activity is mainly seen in patients with COPD.[15] Sternocleidomastoid activity leads to elevation of the clavicles, and more than 5 mm upward movement of the clavicle is a valuable sign of severe obstruction, correlating with FEV_1 of 0.6 L.[16] Magendie[17] described the cyclical inspiratory contraction of the scalene muscles as "respiratory pulse." The scalene muscle is recruited earlier than the sternomastoid muscles. De Troyer *et al*.[18] had shown that most patients of COPD use the scalenes and not the sternocleidomastoids and trapezii during resting condition, suggesting that both the sternocleidomastoids and trapezii in humans have a very high threshold of activation. Sternomastoids are usually recruited at very high lung volumes and during periods of high levels of ventilation as in exercise.[19] Gandevla *et al*.[20] similarly demonstrated that in stable COPD patients, the inspiratory discharge frequencies were significantly greater for both the parasternal and scalene muscles compared to the controls; however, the sternomastoid muscle is not activated. However, whether scalene should be considered as accessory muscle is a big question as the study had shown its activity even at rest in healthy individuals.[15] Stubbing *et al*.[8] had shown that only contraction of scalene muscles but not the sternomastoid muscles is correlated with the degree of obstruction and the duration of symptoms. Accessory expiratory muscles are the abdominal respiratory muscles (rectus abdominis, transverse abdominis, and external and internal obliques). They augment the passive recoil of the lungs during expiration and also help in inspiration. Their contraction cause lengthening of the diaphragm, thereby diminishing its radius of curvature, which helps in generating greater inspiratory pressure by the diaphragm.[21,22] Dodd *et al*.[22] postulated that abdominal muscle recruitment can store elastic and gravitational energy within the diaphragm which when released during inspiration augments inspiratory pressure generation. Ninane *et al*.[23] demonstrated that many stable patients of severe COPD contract their abdominal muscles during expiration and it is confined to the transverse muscles. Abdominal muscles are active in expiration when the minute ventilation

exceeds 40 L.[24] The accessory muscle recruitment has a likelihood ratio (LRs) of 4.75 (2.29–9.82; $P < 0.0001$) for the diagnosis of COPD.[25]

PURSE-LIP BREATHING

Patients with COPD frequently adopt purse-lip breathing (PLB) pattern either spontaneously or as a part of the pulmonary rehabilitation programs. In PLB, patients tend to exhale through pursed lips. PLB reduces the respiratory rate (both at rest and during exercise), reduces carbon dioxide level, and improves ventilation and oxygenation.[26] PLB decreases the respiratory rate by lengthening the period of expiration. During the PLB maneuver, resistance to expiratory airflow is increased, resulting in the development of a positive expiratory pressure in the airways. This positive intraluminal pressure reduces airway collapse, airway resistance, and residual volume and improves ventilation.[27] Bianchi *et al*.[28] had shown that PLB causes a significant decrease in end-expiratory volume of the chest wall and this decrease is related to baseline $FEV_{1,}$ but not to hyperinflation. PLB also increases the inspiratory muscle strength over time. Lesser inspiratory force is required for each breath which reduces the sensation of dyspnea.[29,30] Due to different time constants, there is disproportionately less delivery of tidal ventilation in lung segments with a higher resistance. Moreover, increase in respiratory rate in COPD patients will further accentuate the uneven distribution. PLB by slowing the respiratory rate improves ventilation into these subdivisions.[27] Overall, PLB significantly reduces the $PaCO_2$ level and respiratory rate, increases the tidal volume, and improves the distribution of ventilation. PLB by slowing the respiratory rate reduces the work of breathing and subsequently carbon dioxide production. PLB decreases the $PaCO_2$ level by 5% compared to patients with normal breathing pattern. An appreciable drop in $PaCO_2$ level occurs after 10 min of PLB maneuver.[27]

Breslin[31] demonstrated a shift in the recruitment pattern of inspiratory and expiratory muscles with PLB maneuver. There is a greater activity in the rib cage and accessory muscles compared with diaphragmatic activity. The diaphragmatic tension-time index is reduced. Therefore, PLB by altering pattern of recruitment reduces the possibility of diaphragmatic fatigue and ameliorates dyspnea. PLB also increases recruitment of abdominal expiratory muscle. Expiratory muscle recruitment does not only facilitate expiration but also helps in inspiration by improving the length–tension relationship of the inspiratory muscles. The presence of PLB increases the probability of COPD with an LR of 5.05. The kappa score is 0.45 (moderate interobserver agreement).[25]

HOOVER'S CHEST SIGN

COPD patients with hyperinflation may show various abnormalities of chest wall motion, but the most common abnormality is the paradoxical inspiratory indrawing of the lateral rib cage (costal margin) popularly known as Hoover's sign.[32,33] Normally, the costal margin moves very little during quiet respiration. However, if it moves at all, the direction is outward and upward. In some healthy individuals, at the end of maximum inspiration, they may move slightly inward. However, in COPD patients, there is a gross exaggeration of this inward movement. The indrawing of the lateral rib cage may occur at the end of inspiration or throughout inspiration.[34] It occurs both at rest and during exercise. Gilmartin and Gibson[32] described the following types of paradoxical movement: late inspiratory paradox, a combination of late inspiratory paradox at the upper level and early inspiratory paradox at the lower level or intermittent paradoxical movement. The Hoover's sign is best appreciated by placing the first and second fingers on the costal margin near the anterior axillary line.[35] Occasionally, a biphasic Hoover's sign is seen when the costal margin moves out initially, then in, and then moves out again with the onset of expiration.[35] Paradoxical lateral rib cage movement is seen in both upper and lower rib cages, but it is greater at the lower rib cage level. COPD patients may also show inspiratory indrawing of the lower sternum known as anteroposterior ribcage paradox. It occurs typically in early inspiration and usually occurs along with lateral paradox.

Gilmartin and Gibson[32] demonstrated Hoover's sign in 77% of patients with chronic airflow obstruction. Garcia-Pachon and Padilla-Navas[36] reported the presence of Hoover's sign in 45% of stable COPD patients. The frequency of the sign increases with the severity of airflow obstruction. It was demonstrated in 36%, 43%, and 76% of patients with moderate, severe, and very severe COPD, respectively. Hoover's sign has a good interobserver agreement with kappa statistics of 0.74, which is better compared to other physical signs (wheezes, rhonchi, and reduced breath sounds) and clinical impression. The sensitivity and specificity of Hoover's sign for the detection of airway obstruction is 58% and 86%, respectively, and the positive LR is 4.16.[37] Hoover's sign is associated with a higher level of dyspnea (both at rest and after exercise) and higher use of health-care resources in COPD patients, including hospitalizations, and this finding is independent of the FEV_1 and the body mass index.[38] There is a conflicting report in the literature regarding the relationship between lateral ribcage paradox and FEV_1. Gilmartin and Gibson[32] found a weak correlation, but Stubbing *et al*.[8] observed a correlation between paradoxical rib cage movement with FEV_1 and age.

Hoover's chest sign develops due to the inward pulling of the lateral rib cage by the flattened diaphragm.[39] Hyperinflation leads to the loss of zone of apposition, and the diaphragmatic fibers adopt horizontal orientation. When the horizontally orientated fibers contract, they pull the costal margin inward. However, Gorman *et al*.[40] in an ultrasonography-based study had shown that the zone of apposition length is reduced by 50% at residual volume,

but not completely absent. Therefore, the conventional theory that Hoover's sign is the result of an inward pull of the lower ribs by radially oriented diaphragm muscle fibers is probably not correct. Troyer and Wilson[41] proposed the three-compartment model to explain the chest wall mechanics. During diaphragmatic contraction, pleural pressure (Ppl) falls which exert a caudal and inward force on the entire rib cage. However, via the "insertional force" and "appositional force," the diaphragm exerts forces in the cranial and outward direction on the lower ribs. In the presence of hyperinflation, the zone of apposition is decreased, and pleural pressure becomes the dominant force on the lower ribs and rib displacement is reversed in caudal-inward direction.

BARREL-SHAPED CHEST

The normal chest is oval shaped, with its anteroposterior diameter less than its lateral diameter. The thoracic ratio, thoracic index, or chest index[42] is the ratio of the anteroposterior to lateral diameter and is normally approximately 0.70–0.75 in adults. The upper normal limit is approximately 0.9.[43] In a barrel-shaped chest, the anteroposterior diameter is equal to or greater than its lateral diameter and the thoracic ratio becomes >0.9. The ribs become more horizontal and dorsal kyphosis is present in the majority of cases.[34,44] Other findings are prominent sternum, elevated clavicles, shortened neck, and widened intercostal spaces.[43] Barrel-shaped chest is usually seen in advanced emphysema. Aging can also produce barrel-shaped deformity of the chest without any lung disease.

However, the increased anteroposterior chest diameter may be an illusory finding as Kilburn and Asmundsson[45] demonstrated that the anteroposterior diameter was not different significantly between the three groups: 25 patients with emphysema, 22 patients with other diseases, and 16 normal individuals. Hence, the authors hypothesized that the decreased abdominal diameters due to weight loss seen in COPD may be responsible for an illusory increase in the anteroposterior diameters of the chest. Walsh *et al*.[46] examined the structural changes of the thorax in hyperinflated individuals with COPD and compared it with age-matched normal individuals. They reported no difference in rib cage dimensions between the COPD patients and the controls. The barrel-shaped chest has an LR (95% confidence intervals) of 2.58 (1.45–4.57; $P < 0.001$) for the diagnosis of COPD.[25] The interobserver agreement for barrel chest is good with the kappa score of 0.62.[25] Patients without a barrel-shaped chest are significantly less likely to have airflow limitation.[47,48]

INSPIRATORY RECESSION OF SUPRACLAVICULAR FOSSA AND INTERCOSTAL SPACES

Some patients of COPD may show recession or indrawing of the supraclavicular fossa. It is attributed to a phase lag between the generation of a large negative inspiratory Ppl and a resultant change in lung volume. The phase leg is related to increased airway resistance and reduced FEV_1 level.[49] The same mechanism is also responsible for retraction of intercostal spaces. Godfrey *et al*. reported a correlation between airway obstruction and recession of the supraclavicular fossa.[50]

DYSPNEA-RELIEVING POSTURE

COPD patients often adopt instinctively during episodes of respiratory distress dyspnea-relieving position such as tripod position. In tripod position, the patients are in sitting and leaning forward posture with their outstretched hands on their knees. The forward-leaning position improves dyspnea by several mechanisms. The arm support in tripod position fixes and lifts the shoulder girdle and improves the length–tension relationship of other accessory muscles (pectoralis major and minor) that are attached between the ribs and the upper limb or shoulder girdle.[51] The tripod position by compressing abdominal contents and pushing the short, flattened diaphragm upward helps in restoring the normal dome-shaped appearance of the diaphragm.[52,53] It optimizes the length–tension relationship of the diaphragm and improves its functions. The tripod position also decreases the recruitment of sternocleidomastoid and scalene muscles.[53] The tripod position also improves thoracoabdominal movement.[54] O'Neill and McCarthy had shown that of six different positions, the seated leaning-forward position is the optimum position for the patients to generate maximum inspiratory pressures and to obtain greatest subjective relief of dyspnea.[14]

PERIPHERAL EDEMA

Pedal edema in patients with COPD may indicate right-sided heart failure or cor pulmonale. Right heart failure develops as a result of pulmonary hypertension.[55] However, renal and hormonal abnormalities, manifesting as edema or hyponatremia, are also commonly encountered in patients with COPD.[56] Both hypercapnia and hypoxemia can cause edema, but hypercapnia appears to have more prominent roles than hypoxemia.[57] Kilburn and Dowell had shown that in healthy individuals, moderately acute hypoxemia increases renal blood flow and only severe hypoxemia (PaO_2 <40 mmHg) reduces renal blood flow.[58] Reduction in renal blood flow leads to activation of the renin–angiotensin–aldosterone system, arginine–vasopressin and the sympathetic nervous system, and edema formation.[56,59,60]

INSPIRATORY MUSCLE FATIGUE

Cohen *et al*.[61] had shown the following sequence in the development of inspiratory muscle fatigue: electromyographic evidence of fatigue, tachypnea, respiratory alternans, abdominal paradox, and finally

an increase in $PaCO_2$, associated with a fall in minute ventilation and respiratory rate and worsening of respiratory acidemia. Diaphragmatic failure occurs within 45 min in a healthy person when the target transdiaphragmatic pressure (Pdi) is >40% of maximal transdiaphragmatic pressure.[62] Both the abdominal paradox and respiratory alternans are reliable clinical signs of inspiratory muscle fatigue.[61] Patients with asynchronous breathing have a poor prognosis. They show a significantly higher mortality and a significantly higher requirement of assisted ventilation. They also have significantly lower values for forced vital capacity (VC) and FEV_1 compared to patients with synchronous breathing.[63]

ABDOMINAL PARADOX OR RESPIRATORY PARADOX

The abdominal or respiratory paradox is defined by indrawing of the abdominal wall when the rib cage moves outward. Normally, during inspiration, the abdominal and thoracic wall move synchronously, both expanding in inspiration and contracting in exhalation. The diaphragm is attached to lower ribs via the zone of apposition, and its fibers are directed upward, parallel to the rib cage.[64] During inspiration, the descent of the diaphragm causes outward bulging of the abdominal wall and increase in abdominal pressure (Pab). The increased abdominal pressure causes displacement of the lower rib cage via the zone of apposition. COPD patients may develop diaphragmatic fatigue due to a mechanically disadvantageous position of the diaphragm and overwork. The pressure gradient (Pdi) produced by the diaphragm is zero. Since Pdi is the difference between Pab and Ppl,[21] in this condition, Pab equals Ppl. Therefore, with each inspiration, the fall in Ppl caused by the contraction of intercostal muscles sucks upward the fatigued diaphragm and abdomen moves inward. This is called abdominal or respiratory paradox. The best way to demonstrate abdominal paradox is bimanual palpation with one hand over the patient's chest and one over the abdomen. Palpation of the abdomen also helps in differentiating abdominal paradox from abdominal muscle contraction, which is present in many stable COPD patients.[23] The paradoxical movement may not be apparent in upright posture if the expiratory muscles contract and push the diaphragm upward during expiration, as during subsequent inspiration, the diaphragm returns to its resting position passively. However, in the supine position, paradoxical movement becomes obvious. The asynchronous rib cage and abdominal movement are more common in patients with severe COPD. Braun and Rochester[65] had shown that moderate and severe COPD patients with inspiratory muscle weakness do not retain carbon dioxide (CO_2) when the maximum inspiratory mouth pressure (PImax) is > -50 cm H_20. Tobin *et al*.[66,67] on the other hand proposed that abdominal paradox develops due to increase in respiratory load, rather than muscle fatigue.

RESPIRATORY ALTERNANS

Another sign of respiratory muscle fatigue is respiratory alternans. Patients with respiratory alternans exhibit alternate use of either the diaphragm or chest wall cyclically, so that most of the respiratory movements are abdominal for a few breaths, followed by another series of breaths that occur due to the displacement of the rib cage.[68] In some patients, it is seen only in the erect posture. When they lean forward, the increased intra-abdominal pressure can restore the dome of the flattened diaphragm. This may improve diaphragmatic efficiency and respiratory alternans disappears.[13] Both abdominal paradox and respiratory alternans are associated with or followed by the rise in $PaCO_2$ level, but the development of severe respiratory acidemia occurs late.[61]

JUGULAR VENOUS DISTENSION DURING EXPIRATION

Neck veins are inspected for estimation of the jugular venous pressure and an analysis of the venous pulse. Jugular venous distension during expiration indicates that the intrathoracic pressure has become excessively positive due to airway obstruction.[35] Due to the large swings in the intrathoracic pressure, the jugular venous pressure is often difficult to assess in COPD patients.

LOSS OF BUCKET-HANDLE MOVEMENTS OF THE CHEST

During inspiration, ribs undergo pump-handle and bucket-handle movement. Due to hyperinflation and elevation of the sternum in COPD patients, there is a loss of the bucket-handle movement about the vertebrosternal axis with retention or even exaggeration of the pump-handle movement. The bucket-handle movement of the lower rib cage is lost due to two factors: loss of zone of apposition and medial orientation of the diaphragm fibers. However, Godfrey *et al*. demonstrated a lack of correlation between the loss of bucket-handle movement with the degree of obstruction.[50]

LARYNGEAL HEIGHT OR TRACHEAL LENGTH

This is the distance between the top of the thyroid cartilage and suprasternal notch. The positive LR of laryngeal height is 5.21, and when it is combined with lung function questionnaire, the positive LR becomes 29.06.[69] In the Straus *et al*. series, maximum laryngeal height of ≤4 cm has an LR of 3.6 for the diagnosis of COPD.[70] The laryngeal height is shorter in COPD due to two reasons: clavicles and sternum are placed at a higher level due to hyperinflation. Second, the forceful diaphragmatic contraction may pull the trachea abnormally downward. Laryngeal descent is the difference between maximum and minimum laryngeal

heights. Maximum laryngeal height is measured at the end of expiration and minimum laryngeal height is measured at the end of inspiration. Laryngeal descent was not found to be useful in ruling in or out obstructive airway disease. COPD patients may also develop distortion of tracheal shape. The ratio of the short to the long radius of trachea is a better parameter than tracheal index in detecting distortion.[71]

TRACHEAL DESCENT WITH INSPIRATION

Patients with chronic airflow obstruction may show downward displacement of trachea during inspiration. This sign is called Campbell sign and it is different from tracheal tug seen in patients with an aortic aneurysm (pulsation of aorta palpable through the trachea). Campbell sign is best felt by placing the tip of the index finger on the thyroid cartilage.[72] Campbell sign is probably produced by the downward pull of the depressed diaphragm.[35] Godfrey *et al*.[50] had demonstrated that tracheal descent was correlated significantly with FEV_1 and specific airway conductance. However, this sign is not specific for chronic airways obstruction and can be present in respiratory distress of any cause.

ASSESSMENT OF CARDIAC POSITION

Patients with COPD may present with an absent apical impulse and an impaired cardiac dullness. The cardiac apex beat in COPD may not be present at the usual location and may be shifted to the subxiphoid area.[73] Both these signs are related to the degree of airflow obstruction (FEV_1) and hyperinflation.[8] Shifting of apex beat to the subxiphoid area suggests a FEV1 of <50%.[74] The subxiphoid region should also be included routinely for the palpation of cardiac impulse in emphysema. Badgett *et al*.[73] noticed the presence of subxiphoid apical impulse in only six patients, and the sensitivity and specificity for moderate COPD are 27% and 98%, respectively. Absent cardiac dullness had a sensitivity of 16%, but specificity for moderate COPD is 99%. The kappa statistic was 0.49 (moderate interobserver agreement). The positive and negative LR of absent cardiac dullness is 16 and 0.8 for diagnosing COPD in patients with a history of smoking or self-reported COPD, respectively. A systolic heave in the left parasternal region indicates right ventricular hypertrophy. Hyperinflation may modify this finding.

Chest hyperresonance

The chest percussion should be done routinely in COPD patients to determine the type of percussion sounds. The percussion sound is hyperresonant, if the sound is more hollow than normal.[75] The characteristic finding in COPD is a generalized and symmetrical hyperresonance note. Oshaug *et al*.[76] showed that hyperresonance to percussion is the strongest predictor of COPD, with a sensitivity of 20.8%, a specificity of 97.8%, and an LR of 9.5.

DIAPHRAGMATIC EXCURSIONS

The diaphragmatic position and its range of movement can be demonstrated by percussion. Diaphragmatic excursion actually measures the movement of the dome as the dome moves more than the peripheral part.[77] The normal diaphragmatic excursion is 4–5 cm, and it is reduced in emphysema patients. However, a normal diaphragmatic movement is less likely useful in decreasing the likelihood of airflow limitation.[73]

DECREASED BREATH SOUND INTENSITY

A reduction in breath sound intensity (BSI) is often seen in patients with COPD. Pardee *et al*.[78] developed a scoring system for BSI. According to this system, the clinician listens sequentially over six locations on the patient's chest: bilaterally over the upper anterior portion of the chest, in the midaxillae, and at the posterior bases. At each site, the clinician grades the inspiratory sound as absent (0 points), barely audible (1 point), faint but definitely heard (2 points), normal (3 points), or louder than normal (4 points). The patient's total score may range from 0 (absent breath sounds) to 24 (very loud breath sounds). A BSI score of 9 or less greatly increases the probability of chronic airflow obstruction (LR = 10.2), whereas a score of 16 or more decreases the probability (LR = 0.1).[78,79] Badgett *et al*.[73] showed that the presence of diminished breath sounds is the best variable for diagnosing moderate COPD. Best strategy would be a combination of history and physical examination. Badgett *et al*.[73] proposed the following combined model: history of smoking more than 70 pack-years, history of chronic bronchitis or emphysema, and diminished breath sounds intensity. The positive LR of COPD diagnosis is 33.5, if answering yes to two of these questions. The kappa score for BSI determination is 0.96 (very good).

BREATH SOUNDS AT MOUTH

Breath sound at mouth is acoustically different from the sounds heard at chest wall. Breath sounds at mouth contain frequencies distributed widely from 200 to 2000 Hz, whereas breath sounds heard at chest wall do not contain frequencies above 200 Hz as they are filtered off by the alveolar air and chest wall.[80] In patients with chronic bronchitis and asthma, breath sound is easily audible even at a distance, and the intensity of the breath sounds at the mouth directly correlates with increased airway resistance, reduced FEV_1, and peak expiratory flow rate (PEFR). In contrast, emphysema patients have quiet breathing at the mouth. This is because emphysema does not cause direct bronchial narrowing. Emphysema patients develop small airway obstruction due to the loss of elastic recoil of the lung.[81] The decreased intensity of breath sounds can be either due to poor sound production or poor sound transmission by emphysematous lung parenchyma.[82] The lung parenchymal tissue is an important conduit for

sound transmission. Therefore, alveolar destruction and air-trapping decreases sound transmission. Ploysongsang *et al*.[83] had demonstrated that regional breath sound intensity in emphysema varies from breath to breath and is correlated with regional ventilation, which suggests that an airflow-dependent reduction in sound generation can explain the decreased intensity. Schreur *et al*. similarly suggest that diminished lung sounds in emphysema are predominantly due to concurrent airflow limitation.[84]

EARLY INSPIRATORY CRACKLES

Early inspiratory crackles appear at the beginning of inspiration and end before mid-inspiration. It is classically seen in COPD. Crackles are usually due to airway secretions within large airway and disappear on coughing. These crackles are scanty, gravity-independent, usually audible at the mouth, and strongly associated with severe airway obstruction. Nath and Capel had shown that among patients with known obstructive lung disease, early inspiratory crackles imply a severe disease (i.e., mean FEV_1/VC 31%).[85] The positive LR of early inspiratory crackles is 14.6.[86-88] The positive LR for detecting severe chronic airflow obstruction is 20.8.[85]

WHEEZING

Wheezes are produced by the vibration of the narrowed walls of airway. The presence of unforced wheezing has an LR of 2.6 for COPD diagnosis.

OTHER AUSCULTATORY SIGNS

The clinical examination to detect the signs of cor pulmonale in COPD is insensitive due to the hyperinflation of the chest.[89,90] Splitting of the 2nd heart sound with an accentuated pulmonic component and occasionally the murmur of pulmonary valvular insufficiency indicates pulmonary hypertension, but is not a sensitive indicator of pulmonary hypertension in patients with COPD.[60] The presence of a right ventricular gallop sound intensified by a deep inspiration is a reliable index to the onset of right ventricular failure. Tricuspid regurgitation may also develop in patients with right ventricular dysfunction. The murmur of tricuspid regurgitation is holosystolic, best heard in the left fourth intercostal space in the parasternal area. The intensity of the murmur increases during inspiration and is known as Carvallo's sign.[91]

FORCED EXPIRATORY TIME

The forced expiratory time (FET) is a simple, inexpensive, reproducible bedside test to detect airflow obstruction.[92,93] It is the time taken by an individual to complete a forceful exhalation after maximal inspiration. The patient is instructed to take a full breath and then exhale as fast and complete as possible with the mouth wide open. The bell of the stethoscope is placed over the trachea in the suprasternal notch. The duration of audible expiration is measured to the nearest half second with the help of a stopwatch. In normal healthy individuals, 70%–80% of the VC is expelled in the first second of expiratory maneuver and remaining 20%–30% is expelled in further 2–3 s.[94,95] However, in COPD patients, exhalation takes longer time due to airway obstruction. A FET of <5 s indicated FEV_1: VC of more than 60%; whereas, a FET more than 6 s indicates an FEV_1: VC ratio of <50%.[96] The positive LR in patients with the age of 60 years or older is 0.42 for a cutoff of 4–6 s and 4.08 for a cutoff of >8 s. The interobserver agreement is good with kappa score of 0.70.[92] There is a weak correlation with the severity of obstruction as FET depends on FVC and air trapping may reduce FVC in some patients.[1]

MATCH TEST (SNIDER TEST)

It is a bedside test to detect airflow obstruction. The patient is first instructed to inspire maximally and is then asked to expire rapidly and forcefully with their mouths wide open to extinguish the standard cardboard match placed at a distance of 6 inch (15 cm). The ability to blow out the match depends on the velocity of air flow which is affected by airway obstruction.[97] The Snider test correlates with FEV_1 and maximum breathing capacity. In the Snider's series, 80% of the patients with maximum breathing capacity above 60 L/min and 85% of the patients with FEV_1 >1.60 L could extinguish the match. The test is positive if the patient fails to extinguish the match. This test is a simple screening test, and if positive, further pulmonary function test should be performed. The Snider's test can be positive in both obstructive and restrictive lung diseases. This test should not be done in patients receiving supplemental oxygen therapy.

ANCILLARY TESTS

Pulsus paradoxus

Normally, there is an inspiratory fall in systolic blood pressure, but the magnitude is <10 mmHg. Pulsus paradoxus (PP) is defined by an inspiratory fall in systolic blood pressure of >10 mmHg. It is an exaggeration of normal physiologic fall in systolic blood pressure, so the term paradoxical is a misnomer. PP is classically detected in cardiac tamponade, acute asthma, and acute exacerbation of COPD.[98] PP is measured by the following sphygmomanometer-based method: initially, the sphygmomanometer cuff is inflated 20 mmHg above the systolic blood pressure level. The cuff pressure is slowly released at a rate of 2 mmHg/s until the first Korotkoff sound is heard only during expiration, and this value of systolic blood pressure is then noted. The cuff pressure is further reduced until the Korotkoff sounds become audible in both phases of the respiratory cycle. The difference between these two levels quantifies PP. PP of at least 15 mmHg indicates that the FEV_1 level is probably 25% or lower.[75,99]

Clubbing

Clubbing of the digits is not typical in COPD and when present should raise the possibilities of comorbidities such as lung cancer, interstitial lung disease, or bronchiectasis.

The presence of certain physical signs in individuals with chronic airflow obstruction is related to the degree of airflow obstruction, secondary effects of the airflow obstruction (e.g., hyperinflation), age of the patient, and duration of the disease. Stubbing *et al*.[8] found following physical signs to be significantly correlated with FEV_1: tracheal descent, scalene muscle (but not sternomastoid) contraction, costal margin movement, and cardiac position (a combination of the impalpable apex beat and impaired cardiac dullness at percussion). The cardiac position was found to be correlated not only with FEV_1 but also with the degree of hyperinflation as assessed by FRC. They did not find any correlation between supraclavicular fossae recession, intercostal recession, upper rib cage movement, and tracheal length with FEV_1 or FRC.[8] Godfrey *et al*.[50] similarly found a significant correlation between specific conductance (an index of airway obstruction) and tracheal descent on inspiration, accessory muscle activities, recession of supraclavicular fossae on inspiration, increased resonance on percussion, and FET. There was no correlation between wheezing and airway obstruction and wheezing may be absent in patients with severe obstruction. The costal paradox and tracheal length were more closely related to age or duration of symptoms than to the narrowing of the air passages.

Badgett *et al*.[73] also evaluated the role of history and physical examination in the diagnosis of clinically significant COPD. They also reported the predictive value of various pulmonary signs and symptoms in the diagnosis of COPD. In multivariate analysis, a history of smoking and reduced breath sounds were the only parameters significantly associated with COPD. The sensitivity and specificity of the combination were 67% and 98%, respectively. The positive and negative predictive values for the FET were 57 and 85%, respectively. The sensitivity and specificity for displacement of cardiac impulse were 71% and 87%, respectively, and for reduced breath sound, it was 77% and 93%, respectively.

Holleman and Simel[100] in a prospective observational study reported that the number of years the patient had smoked cigarettes, patient-reported wheezing, and auscultated wheezing were independent predictors of airflow obstruction. FET and PEFR were additional independent predictors of airflow obstruction. However, in this study, other physical signs of airway obstruction were overlooked. The authors subsequently proposed a nomogram using the following criteria: patient-reported wheezing, number of years the patient had smoked, and auscultated wheezing or PEFR. It was validated for the bedside prediction of obstructive airway disease. In a case–control study, Mattos *et al*.[25] evaluated the accuracy of nine clinical variables in the diagnosis of COPD. Majority of patients had severe COPD. All the clinical signs showed high LR for COPD diagnosis: accessory muscle recruitment (LR, 4.75), PLB (LR, 5.05), barrel chest (LR, 2.58), and reduced breath sounds (LR, 7.17). Straus *et al*.[70] demonstrated that only four criteria of the history and physical signs are significantly associated with the diagnosis of airway obstruction in multivariate analysis. The four criteria are the followings: smoking for more than 40 pack-years (LR, 8.3), self-reported history of chronic obstructive airway disease (LR, 7.3), maximum laryngeal height of at least 4 cm (LR, 2.8), and age at least 45 years (LR, 1.3). Patients with all the four criteria have an LR of 220 for obstructive airway disease. Oshaug *et al*.[76] in a cross-sectional study evaluated the role of chest signs along respiratory symptoms and a history of smoking in the diagnosis of COPD. At least one chest sign was observed in 38.7% of the patients. They found hyperresonance to percussion, diminished breath sounds (odds ratio = 5.0), and wheezes as independent predictors of COPD in multivariate logistic regression analysis. Hyperresonance to percussion was the strongest predictor of COPD, with an LR of 9.5. Along with shortness of breath and pack-years, these three chest signs provided significant diagnostic information.

van Schayck *et al*.[101] studied the relationship between the physical signs of the chest and the degree of airflow obstruction in patients with asthma and COPD. They demonstrated following signs to be correlated with the degree of airflow obstruction: a prolonged expiration, low-standing diaphragm, decreased expiratory breath sounds, noisy inspiratory sounds, and decreased diaphragmatic excursions. They also reported a fair correlation between the number of physical signs and the degree of airflow obstruction. Although the sensitivity of the individual sign is low, it was increased with a combination of signs.

However, there are certain limitations of the physical signs. Findings from physical examination had high specificity (>90%), but a low sensitivity.[102] Physical findings are usually normal unless the FEV_1 is <50% predicted.[8] The interobserver agreement about respiratory signs is often highly variable. We definitely need larger and better-designed study related to the role of physical signs in the diagnosis of COPD in future.

CONCLUSION

Legendary physician Sir William Osler once said that "Medicine is learned by the bedside and not in the classroom." Patients will get maximum benefit when a careful history and physical examination is combined with tailored laboratory investigations. The physical examination helps the physician to develop a rapport with the patients, reduces the risk of unnecessary investigations, and renders the physician more autonomous in his or her diagnostic skills. Physical diagnosis should not

be considered a cabalistic rite;[103] it will remain as an important armamentarium for the diagnosis of the patients for years to come.

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Conflicts of interest
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