SERIES 'CHEST PHYSICAL EXAMINATION'
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Physical examination of the adult patient with respiratory diseases: inspection and palpation

B. Maitre, T. Similowski, J-P. Derenne

Physical examination of the adult patient with respiratory diseases: inspection and palpation. B. Maitre, T. Similowski, J-P. Derenne. ©ERS Journals Ltd 1995.
ABSTRACT: Inspection of the thorax identifies the breathing position adopted by the patient, the shape of the thorax, the dynamics of respiration (breathing pattern, symmetry of expansion, mechanics and synchrony of rib cage and abdominal movements). Inspection of the neck adds useful information, particularly with respect to the dynamics of breathing. Palpation ascertains the signs suggested by inspection with respect to the mechanics of breathing. It also assesses the state of the pleura and pulmonary parenchyma by studying the tactile fremitus. It integrates extrarespiratory signs, such as enlarged lymph nodes or breast abnormalities. Extrathoracic respiratory signs should also be systematically looked for, including cyanosis, finger deformation, pulsus paradoxus, and pursed lips breathing.

Interobserver agreement about respiratory signs has repeatedly been studied, and generally found to be low, as are clinical-functional correlations. However, some data on chronic obstructive pulmonary disease (COPD), asthma or pulmonary embolism are available. From the description of some signs and the current knowledge about their operative values, it appears that much clinical research remains necessary to better define the precise diagnostic value of a given sign. The impact of training on diagnostic performance also has to be defined. Both of these aspects should allow clinicians to optimize the way in which they use their hands and eyes to conduct respiratory diagnosis, as well as the way they teach respiratory symptomatology.
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Service de Pneumologie et de Réanimation, Groupe Hospitalier Pitié-Salpêtrière, Paris, France.

Correspondence: T. Similowski
Service de Pneumologie et de Réanimation
Groupe Hospitalier Pitié-Salpêtrière
47-83 Bd de l'Hôpital
75651 Paris Cedex 13
France

Signs brought to light by inspection and palpation of the thorax are among the basics of the process of respiratory diagnosis, and make a large contribution to the evaluation of respiratory status. Although they are only part of an ensemble including other respiratory clinical data, chest radiography, pulmonary function tests, and various diagnostic tools, these signs have a major intrinsic value. They can be present notwithstanding normal chest radiography or pulmonary function tests, and they may be sufficient to initiate immediate life-saving actions. For example, removal of air from a tension pneumothorax can be decided on the simple observation of distension and immobility of a hemithorax, in the context of acute shock and impaired venous return in a mechanically-ventilated patient. The decision to start mechanical ventilatory support in a patient with acute respiratory failure (ARF) of chronic obstructive pulmonary disease (COPD) usually relies on the conjunction of physical signs witnessing hypoxaemia (cyanosis), hypercapnia (confusion, flapping tremor), or dysfunction of the respiratory pump (rapid shallow breathing, asynchronous thoracoabdominal motion...). Advanced COPD is associated with a series of changes in the shape and dynamics of the thorax [1, 2]. The corresponding physical signs [3], at times, define a clinical pattern characteristic enough for the clinician who is aware of them to raise the question of COPD after a simple look.

However, physical signs are evaluated on the basis of "norms" stemming from clinical examination of a large number of patients. Therefore, their diagnostic and pronostic value depends upon the physician's training and background. How these factors affect "respiratory" inspection and palpation is ill-known. To begin with, although few studies have addressed this issue in a systematic manner, reproducibility of pulmonary physical examination seems low [4–6]. For example, GJORUP *et al.* [7] reported interobserver variations in the assessment of respiratory signs. They compared the results of independent clinical observations by two physicians. Among the 11 signs studied, none arose from palpation and only one from inspection ("decreased breath movements"). The kappa value (which describes the degree of agreement between several observers evaluating a given population, and ranges from 0=no agreement to 1=full agreement [8, 9]), for this sign was 0.14, the lowest reported in the study. In a similarly designed study, SPITERI *et al.* [10] compared the evaluation of physical signs by six sets of four physicians examining a total of 24

patients. Only 55% of the time were the four physicians in complete agreement, and the more often a given observer departed from the majority with respect to a given sign, the more likely was he to make an incorrect diagnosis. Among signs elicited by inspection and palpation, only finger clubbing had a "correct" kappa value (0.45). Reduced chest movements, cyanosis, prolonged expiration, impalpable apex beat, cricosternal distance, tachypnoea, decreased tactile fremitus, increased tactile fremitus, and displaced trachea were seldom agreed upon, with kappa value from 0.01–0.38 [10].

Furthermore, not only are chest physical signs poorly reproducible from one clinician to another, but their meaning when present is often imprecise. Sensibility and specificity of these signs are generally unknown, and few studies describe correlations between physical signs and physiological measurements or morphological findings. Such information can, in part, be found, principally in patients with COPD [3, 11–16].

The present paper is not intended to describe in detail a comprehensive list of physical signs. Rather, it attempts to stress the clinical significance and diagnostic relevance of the main signs, and to summarize data available in some clinical entities.

General principles

Suitable inspection and palpation of the respiratory system require the patient's chest, neck and abdomen to be appropriately exposed. The front of the thorax is best examined in the supine position, and its back in the sitting or standing position. A patient who has difficulty in sitting upright unaided, can be supported by someone standing at the foot of the bed and holding his/her hands. In patients who are too sick to stand or sit, examination of the back of the thorax requires that the patient be rolled on one side and then the other [17].

Whatever the clinical context in which it takes place, clinical examination of the chest is comparative in nature. Each region of one side should be compared with the same region on the other side. This is of particular importance when characterizing rib cage movements. It has to be borne in mind that, for a correct respiratory diagnosis, physical examination should not be restricted to the thorax and neck: inspection and palpation of the lower limbs are of major importance when pulmonary embolism is suspected, inspection of the hands is at times relevant to the diagnosis of malignant processes developing in the chest.

It should also be noted that "respiratory" inspection is a dynamic process: it starts long before the beginning of physical examination itself, and continues throughout. Some elements appear at first glance, such as obvious respiratory distress, nasal flaring (the outward inspiratory motion of the nares), pursed lip breathing (see below). History taking is propitious to the observation of breathing pattern and various phenomena (such as cough, sighs, expression of pain accompanying respiration or cough, hiccups...) devoid of the influence of the examination itself on these elements.

Inspection and palpation of the thorax

Inspection of the rib cage

Inspection of the rib cage provides three types of information: the breathing position adopted by the patient, the shape of the thorax, the dynamics of respiration.

Position. Orthopnoea is often considered suggestive of left heart failure. However, it is not specific, and patients with asthma or acute airflow obstruction tend to sit and even to bend forward to breathe. Patients with COPD often choose a position in which they can support their arms and fix the muscles of the shoulder and neck to aid respiration. They sometimes clasp the sides of the bed and use the dorsal muscles to help overcome the increased inspiratory burden to respiration.

Shape of the thorax. A large variety of thoracic malformations, most often congenital, can be detected by inspection. Pectus excavatum, or funnel chest, consists of a depression of the sternum with anterior protrusion of the ribs. Pectus carinatum is the reverse deformity, in which the sternum protrudes anteriorly. Kyphoscoliosis is the result of an abnormal curvature of the thoracic spine, with a combination of anteroposterior and lateral deviations. Clinical examination is of little value for a quantitative approach to kyphoscoliosis, which relies on chest radiography and determination of the Cobb angle.

In most cases, identifying such malformation has no particular consequence, but, under certain circumstances, it implies particular actions. For example, doubt about the true congenital nature of pectus carinatus should raise the question of a cardiac malformation [18]. The diagnosis of kyphoscoliosis should be associated with detailed evaluation of respiratory status, to serve as a basis for the subsequent follow-up.

The thorax can also change shape in response to acquired diseases. Acute hyperinflation is a feature of asthma, but its characterization is highly subjective and impossible to quantify. Dynamic elements (see below) are probably better indicators of distension. An increase in the anteroposterior (AP) diameter can be seen in advanced COPD. The AP diameter tends to become close to the lateral diameter, which defines the barrel chest configuration. The ribs lose their normal 45° angle and become more horizontal [1]. Kyphosis often accompanies this deformation in COPD. It should be noted that the barrel chest configuration of the thorax is not specific to COPD, and can be observed as a mere consequence of ageing effects.

Dynamics of respiration. Inspection will 1) specify breathing frequency and pattern, 2) examine symmetry in the expansion of the right and left hemithorax, and 3) characterize the mechanics of rib cage and abdominal linked movements.

1. Breathing pattern. In adults, the normal respiratory rate ranges 10–14 breaths·min^{-1}; the term bradypnoea depicting an abnormally slow breathing frequency, and tachypnoea or polypnoea an abnormally rapid one. Although it is very difficult to estimate tidal volume from observation of the chest and abdomen expansions, particularly when patients mainly use their diaphragm to inspire [17], polypnoea is generally used to characterize rapid shallow breathing. Hyperpnoea, on the other hand, is used when the depth of breathing is markedly increased. Whereas polypnoea can be present in many clinical situations, hyperpnoea seems to be a reliable sign of metabolic acidosis. It is most often combined with a certain degree of tachypnoea (Kussmaul breathing). Certain conditions have been associated with particular breathing patterns. Biot's pattern [19] consists of irregular breathing with prolonged apnoeas. It can be seen as a consequence of increased intracranial pressure, central nervous system lesions at the medullary level, drug-induced comas. Cheyne-Stokes breathing [19] consists of a progressive increase and then a progressive decrease of breathing frequency and depth, alternating with periods of apnoeas. It is associated with metabolic or drug-induced encephalopathies, severe congestive heart failure, and central nervous system damage at the cerebral level.

2. Symmetry of the expansion of the thorax. Asynchrony and asymmetry of expansion between the two hemithoraces can be observed in diseases of the lung or pleura. It can relate to acute disease, such as atelectasis, pneumonia, or pleurisy. Only in complete pneumothorax or massive atelectasis is it associated with a marked change of the volume of one hemithorax. Chronic atelectasis or long-standing fibrotic process of the pleura can lead to spinal curvature, with concavity to the diseased side and head inclination toward this same side. A local lag during inspiration can be difficult to ascertain during quiet breathing, and is best manifested by asking the patients to breathe in deeply [17].

3. Mechanics and synchrony of rib cage and abdominal movements. Normally, during quiet breathing, rib cage movements are of small amplitude. Expansion of the upper rib cage is not visible, and the costal margin moves outwards and upwards. Most of the displacement corresponding to inspiration is due to an outward movement of the abdominal wall, which is synchronous with the lower rib cage expansion. Normal expiration is passive.

Several changes can be observed in this pattern and co-ordination. Predominant rib cage expansion during inspiration is abnormal, particularly if it is associated with asynchronous thoracoabdominal motion. Abdominal paradox is defined by indrawing of the abdominal wall when the rib cage inflates. It is due to the abdominal pressure becoming negative rather than positive during inspiration, and is a sign of major diaphragmatic dysfunction. Respiratory alternans is a cyclic alteration in breathing movements, with an alternation of "normal" breaths, where the diaphragm seems to have the principal contribution to inspiration (abdominal

expansion) and "rib cage" breaths. This pattern of breathing has been attributed to developing respiratory pump failure [20] (see below). Hoover's sign [21] consists of a paradoxical inward displacement of the costal margin at the end of inspiration or throughout inspiration. A biphasic aspect can be observed, with the costal margin initially going out, then in, and then out again at the onset of expiration [3]. Hoover's sign is observed in chronic airway obstruction. It is interpreted as a reflection of the effects of hyperinflation, which modifies diaphragm action on the lower rib cage *via* a reduction of the zone of apposition. However, Hoover's sign has not, to our knowledge, been systematically studied with reference to pulmonary volume or diaphragmatic function. The loss of the bucket-handle movement of the ribs about the vertebrosternal axis with exaggeration of the pump-handle movement about the axis of the neck, is also observed in airway obstruction with hyperinflation [3]. However, two studies agree on the lack of correlation of this pattern with the degree of obstruction [11, 15].

Inspection of the neck

Inspection of the neck cannot be dissociated from that of the thorax. Accessory inspiratory muscle hypertrophy and activation can be seen when intense. Cyclical inspiratory contraction of the scalene muscles has been, described by MAGENDIE [22], who termed it "respiratory pulse". In their classical paper, BEAU and MAISSIAT [23] used this term in a different context: they applied it to contraction of the sternomastoid, and further stated that during asthma only the sternomastoid and not the scalene muscles were contracting. This does not appear to have been confirmed by recent studies, and does not seem very likely as the scalene muscles appear to be recruited before the sternomastoid muscles [24]. Respiratory pulse may be present in severe chronic airflow obstruction, when obstruction acutely increases, or in any situation where ventilatory needs are markedly augmented.

In patients with chronic airways obstruction in stable state, the sternomastoid muscles can appear to be increased in volume, the skin clinging to their prominences and the floor of the posterior triangle. However, recent electromyographic data indicate that, whereas the scalene muscles are recruited during quiet breathing in most patients (and in normals), the sternomastoid muscles are not [24]. Only in acute severe episodes will the latter be activated. The term "retraction" of the sternomastoid muscle is employed, when visible contraction of this muscle lifts the clavicle during tidal inspiration. An upward inspiratory motion of the clavicle exceeding 5 mm has been associated with severe COPD [25]. In acute asthma, this sign seems to be correlated to the severity of obstruction and appears when forced expiratory volume in one second (FEV_1) falls below 1 L [26].

Excavation (or retraction) of suprasternal and supraclavicular fossae during inspiration is due to excessive swings of intrathoracic pressure and probably result from

a phase lag between the generation of negative pleural pressures and the resultant change in lung volume. Although this sign has been associated with the presence of airways obstruction [11], STUBBING et al. [15] have failed to find a significant correlation with any other parameter than patients' age. Retraction of the intercostal spaces has the same meaning. It should not be confused with Hoover's sign.

Tracheal descent can be seen, but tracheal movements are better assessed by use of palpation (see below).

Neck veins distension can be part of the superior vena cava syndrome, and then associated with oedema of the face, neck, shoulders and hands and, at times, dilatation of veins over the anterior chest wall. It can also be indicative of right heart failure. This sign can be detected at rest or can be induced: firm abdominal pressure is applied for 10 s over the centre of the patient's abdomen with the palm and fingers of the examiner's hand. An elegant study by BUTMAN et al. [27] showed that, in patients with severe chronic heart failure, the presence of jugular venous distension is strongly correlated with an increased central venous pressure, and has a sensitivity of 81% and specificity of 80% to detect an elevation of the pulmonary capillary wedge pressure (>18 mmHg). Jugular venous filling during expiration indicates that a positive pressure is present in the thorax, and can be observed in patients with COPD.

Palpation

Cardiac impulse should be looked for during respiratory physical examination of the chest [28]. Impalpable apex beat is classically cited among clinical means to diagnose emphysema [12, 29], but apex beat is not palpable in up to 50% of normal individuals. Deviation of the location of the cardiac impulse is a sign of mediastinal shift, whatever its mechanism. A heave in the left parasternal region denotes right ventricular hypertrophy.

It should be noted that palpation of the thorax is also of major interest in detecting enlarged lymph nodes, breast abnormalities, cutaneous nodules, subcutaneous emphysema, *etc.* Intercostal spaces and ribs should be palpated to detect tumour masses and elicit pain to pressure. Nodes and masses in the neck and supraclaviculars areas are best assessed with the examiner positioned behind a sitting or standing patient.

From a more specifically respiratory point of view, palpation of the thorax and neck provides two different types of information. On the one hand, it helps confirm the information suggested by inspection, essentially with respect to contraction of the scalene muscles and thoracic mechanics. On the other hand, it gives information relevant to parenchymal and pleural diseases by analysis of the tactile fremitus.

Dynamics of respiration. A suspected lag detected on inspection of the chest is easily confirmed by placing a hand on each hemithorax and asking the patient to breathe deeply. Most of the aforementioned signs of mechanical dysfunction of the respiratory pump are best detected and characterized by palpation. Tenuous abdominal paradox and Hoover's sign can be sensed by placing the hands flat on the abdomen or the costal margins and letting them move with the patients' respiration. Palpation of the abdomen also helps distinguish abdominal paradox from abdominal muscle contraction, which is present not only when ventilatory needs are acutely increased but also in many stable COPD patients [30]. A discordance is possible between electrical activity of abdominal muscle and the impossibility to perceive it by palpation. This generally disappears when the contraction becomes intense enough, namely when the respiratory system has to face a markedly increased demand. CAMPBELL [3] has very precisely described the contribution of palpation to the description of movements of the upper ribs. To clinically detect the pump-handle movement of the upper rib cage (see above), he recommends "spreading the first and second fingers of each hand and locking them on the top of the second or third rib anteriorly, placing the second finger as far laterally as the rib can be felt before it disappears under the shoulder muscles. Normally, the second (outer) finger swings outwards and upwards around the first finger during inspiration and the first finger does not move much. In patients with chronic airway obstruction the outer finger loses its movement relative to the inner; in extreme cases the inner finger moves upwards more than the outer". Another method of assessing the pump handle movement of the rib cage is to place the palm of one hand flat on the sternum; during inspiration in severe COPD, the hand moves upward rather than anteriorly as in normal subjects.

The respiratory pulse (contraction of scalene muscles during inspiration, see above) can be felt by gently pressing the finger tips into the floor of the posterior triangle of the neck. The sternomastoid muscles are examined by drawing them backwards with the thumb and first finger to feel the contraction. In the study by STUBBING et al. [15] (see below), contraction of scalene muscles closely correlated with the degree of obstruction and the duration of symptoms, but not with the contraction of sternomastoid muscles, which is not present in most patients with severe COPD when they are in steady state.

Palpation of the trachea. The position of the trachea can be determined by placing the index finger in the suprasternal notch and moving it slightly until the tracheal cartilage is felt. Lateral deviation of the trachea can bear witness of mediastinal shift. The distance between the thyroid cartilage and the sternal notch varies from three to four fingerbreadths in normal subjects, and is reduced or even null in severe chronic airways obstruction [3]. Interobserver concordance for this sign seems extremely low [10], as is its correlation with the degree of obstruction [15].

Tracheal upward mobility is assessed as follows (tracheal tug): the patient's head being slightly flexed and supported at the back by one hand, the other hand is placed parallel to the trachea with the palm facing out; the middle fingers slide into the crycothyroid space, and the larynx is pushed upward; the larynx and trachea

normally move about 1–2 cm. Decreased tracheal mobility can be related to fixation of the mediastinum by carcinoma or fibrosis. An abnormal mobility of the trachea with cardiac systole can be seen in aortic aneurysm.

Tracheal descent with inspiration [31] is best felt by resting the tip of the index finger on the thyroid cartilage. This sign is not specific for chronic airways obstruction and can be present in any respiratory distress. It represents an increased amplitude of the pleural pressure swings during respiration. There is a significant relationship between the presence of Campbell's sign (inspiratory descent of the trachea) and severity of airflow obstruction, age, and duration of symptoms [15].

Tactile fremitus [19]. Posterior palpation of the thorax during speech elicits the perception of vibrations that are called tactile fremitus. Their analysis relies heavily on left to right and top to bottom comparisons. Tactile fremitus, which provides information about the density of the underlying lung tissue and chest cavity, can be evaluated in two ways. The examiner can place the ulnar side of the hand against the patient's chest wall and ask the patient to say something. The use of a repeated number that causes good vibrations is traditional (most often "ninety nine" in English speaking countries, "trente-trois" in France...). This has the advantage of "standardizing" the "input" to the "system", *i.e.* the thorax, that determines tactile fremitus, the "output". Finger tips can be used instead of the ulnar side of the hand. By moving the hand from side to side and from top to bottom, differences can be detected in the transmission of the sound. Increased tactile fremitus reflects increased density of the lung, *e.g.* consolidation by pneumonia or atelectasis. Conversely, an excess of fat tissue on the chest, the presence of air or fluid in the pleura, or distension of the lung, decreases tactile fremitus. Two studies suggest that agreement about tactile fremitus being increased or decreased is little better than chance [5, 10], with kappa values of 0.11 and 0.01, respectively [10].

Extrathoracic respiratory signs (inspection and palpation)

Cyanosis

Cyanosis is due to incomplete oxygenation of arterial blood [32]. It appears when the concentration of reduced haemoglobin exceeds 5 g·dL^{-1} of capillary blood [33]. The capillary location of the reduced haemoglobin producing cyanosis is crucial, and was very clearly stated by LUNDSGAARD and VAN SLYKE [33] in their early description: "It is the blood in the capillaries, and possibly in the arterioles and venules of the subpapillary plexus as well, which produces the cyanotic skin color. The arteries and most of the veins are so far away from the skin that their content cannot influence the skin color". Overlooking this location and considering arterial instead of capillary blood has led several authors to mistakenly state that the 5 g·dL^{-1} value was far too high [34–36].

Indeed, cyanosis generally appears at an arterial oxygen tension (P_{a,O_2}) around 8.0 kPa (60 mmHg) (see below), that corresponds to a reduced arterial haemoglobin concentration of about 1.5 g·dL^{-1} [36]. A detailed account of this debate has recently been given by MARTIN and KHALIL [37].

Cyanosis usually reflects severe hypoxaemia, whatever its cause. However, anaemia can prevent cyanosis from appearing, and polycythemia can be responsible for cyanosis with moderate hypoxaemia.

Detection of this blue or bluish-grey colouration of the skin and mucous membranes require adequate light (natural daylight is ideal when available and standards for clinical lighting have been defined [38, 39]) and is easier in the nailbeds or buccal mucosa. Cyanosis can be particularly difficult to appreciate in coloured skins. It is usual to distinguish central cyanosis, where the arterial blood leaving the left heart is inadequately oxygenated, and peripheral cyanosis, where haemoglobin desaturation occurs in tissue capillaries because of extreme slowing of the bloodstream due to reduced cardiac output or vasoconstriction. Clinically, nailbeds are usually deep blue and the skin warm in central cyanosis, whereas peripheral cyanosis is usually associated with cold skin and livid nailbeds. Central cyanosis can be present in numerous different diseases, congenital (*e.g.* right-to-left cardiac shunts) or acquired, acute (*e.g.* severe pulmonary oedema or pneumonia) or chronic (*e.g.* COPD). Peripheral cyanosis is seen in the majority of conditions with compromised haemodynamic status, with the exception of septic shock, and given the level of haemoglobin is sufficient for cyanosis to appear. It is also a feature of diseases where peripheral vasomotor activity is impaired, such as scleroderma or Raynaud's disease. In these cases, it may disappear with warming of the cyanotic area. The administration of oxygen can correct central but not peripheral cyanosis.

Atypical coloration of the skin, particularly if there is no clear cause for cyanosis, should lead to the consideration of methaemoglobinaemia or sulph-haemoglobinaemia.

Identification of cyanosis is at times difficult. COMROE and BOTELHO [40] long ago reported that a majority of observers did not detect cyanosis until the arterial oxygen saturation (S_{a,O_2}) had fallen to 80%, one quarter failing to perceive it at S_{a,O_2} levels as low as 70%. A summary of data available on this specific point was given by MARTIN and KHALIL [37]. The study by GOSS *et al.* [36], albeit its discussion appears flawed by the aforementioned confusion on the location of the reduced haemoglobin responsible for cyanosis, provides interesting and recent data. Among 80 patients, these investigators found that presence of central cyanosis was well agreed upon by two independent observers (who diverged in only three out of 80 cases). All cyanosed patients (n=29) had a P_{a,O_2} below 10.7 kPa (80 mmHg), the average value being 7.6±1.4 *vs* 10.3±1.7 kPa (57±10.4 *vs* 81.3±12.7 mmHg) in the noncyanosed patients. Haemoglobin concentration was comparable in the two categories of patients. Cyanosis correctly identified all patients with a P_{a,O_2} lower than 8.0 kPa (60 mmHg).

Paradoxic pulse

Inspiratory decline of the arterial pulse appeared in the description of asthma given by FLOYER at the beginning of the 18th century [41]. Subsequently, Kussmaul described it as a major manifestation of pericarditis [42]. Currently, the term paradoxic pulse refers to an inspiratory decrease in systolic arterial pressure, which allows it to be quantified. It has been noted in various conditions, including cardiac tamponade due to tense pericardial effusion, acute pulmonary embolism, acute airways obstruction. The mechanisms of paradoxic pulse may differ between these clinical settings. In cardiac tamponade, it is thought that paradoxic pulse is a result of competition between the right and left ventricle for filling [43]. Indeed, inspiration increases right ventricular filling which in turn restricts left ventricular filling. In severe acute asthma, JARDIN et al. [44] have shown that large intrathoracic pressure variations, from a markedly negative level during inspiration to a positive level during expiration, make an important contribution to paradoxic pulse by increasing impedance to right ventricular ejection, which results in a dramatically impaired left ventricular stroke output via concomitant reduction of left ventricular preload.

We did not find any data evaluating the interobserver variability of paradoxic pulse determination. Its correlation to severity is discussed below (see *Pulmonary embolism*).

Finger deformation [45]

Clubbing and hypertrophic osteoarthropathy (HOA) are separate entities which have many similarities.

Clubbing is the painless uniform focal enlargement of the connective tissue in the terminal phalanges of the digits, especially on the dorsal surface. It has little specificity, being present in various intrathoracic diseases (*e.g.* bronchial cancer, cystic fibrosis, pulmonary interstitial fibrosis, pneumoconiosis, and cardiovascular diseases, such as congenital cyanotic diseases and endocarditis) but also in some extrathoracic disorders and, in particular, hepatic or gastrointestinal diseases (*e.g.* primary biliary cirrhosis, inflammatory bowel diseases). A comprehensive list of clubbing aetiologies can be found in the review by SCHNEERSON [45]. Different types of clubbing have been described depending on the distribution of the soft tissue swelling, and termed parrot's beak, watchglass or drumstick. Diagnosis of clubbing is relatively reliable. SPITERI et al. [10] reported that clubbing was one of the five physical signs upon which interobserver agreement was best, with a kappa value of 0.45. However, early clubbing can be difficult to ascertain, and it has been suggested that some "objective" indices could be helpful. For example, the phalangeal depth ratio (between the distal phalangeal depth and the interphalangeal depth) has been proposed. Its clinical usefulness seems vague.

HOA is not as clearly defined as clubbing. It is a systemic disorder, affecting the bones, joints and soft tissues, that develops in association with other diseases. HOA shares many causes with clubbing, to which it is very often associated, but may be more indicative of intrathoracic neoplasm. HOA is often painful, and, in contrast to clubbing, associated with radiological abnormalities, such as periosteal new bone formation, arthritis-like changes in the joints and periarticular areas, as well as with increased thickness of the subcutaneous soft tissue in the distal parts of the legs and arms and neurovascular changes of the hands and feet.

Pursed lips breathing

It has long been shown that pursed-lips breathing as a breathing exercise in patients with emphysema has beneficial effects on blood gases and ventilatory measurements: it decreases respiratory rate, increases tidal volume and lowers arterial carbon dioxide tension (Pa,CO_2) [46, 47]. Spontaneous pursing of the lips during expiration is a common feature in patients with severe COPD and emphysema. It is often interpreted as a means to increase intraluminal pressure of the airways and, therefore, prevent their collapse at low lung volumes. Its reliability as a physical sign of COPD remains to be assessed.

Forced expiratory time [48, 49]

This is a simple and reproducible test to detect airways obstruction. The patient is asked to take a full breath in, and then to exhale with his mouth wide open. Airflow cessation is judged from breath sound listened to at the mouth, or tracheal auscultation. Normal expiratory time is below 4 s in normals. It can be much prolonged in patients with chronic airflow obstruction. Interobserver agreement about forced expiratory time has been assessed in several studies [7, 14, 50]. SCHAPIRA et al. [50] and GJORUP et al. [7] reported kappa values reaching 0.71, which is considered "substantial" agreement [9]. However, the results of BADGETT et al. [14] are less optimistic, with kappa values of 0.23 for a forced expiratory time longer than 11 s ("fair" agreement). The reason for this difference is not clear. Indeed, physicians in the first study were untrained, whereas they were trained in the second. Among the factors affecting the variability of forced expiratory time, MCDONALD et al. [51] considered that interobserver agreement was of little importance. The operating characteristics of forced expiratory time have been studied [50, 52]. SCHAPIRA et al. [50] have built a receiver operating characteristic (ROC) curve of the forced expiratory time as a diagnostic test for obstructive airways disease in a study of 400 subjects (49% of whom had COPD). They reported an area under the ROC curve of 0.61. At a cut-off value of 6 s, the sensitivity of the test was 74% and specificity 75%. There seems to be a significant correlation between this sign and the degree of obstruction [11, 49, 50].

Illustration: physical signs in some clinical entities

Teaching of respiratory medicine associates signs or groups of signs to diseases. Having described some inspection and palpation signs of respiratory disease, it seems interesting to ask the question of how indeed they relate to functional or morphological data. Few such designed studies are available, and most pertain to COPD. The following few examples are intended to give a rapid overview of the value of signs with respect to their physiological foundations.

COPD

Because COPD is a major public health concern, quick and inexpensive methods to detect it could lead to earlier actions of secondary prevention. Therefore, questioning the efficacy of clinical examination with respect to the diagnosis of COPD is highly relevant. As will be seen, correlations between clinical examination and functional data are scarce and seem weak. Nevertheless, clinicians used to dealing with COPD patients share the experience that physical examination, and principally inspection, is a powerful first line tool.

To be useful, a given sign must first be recognizable by different observers. As mentioned throughout the corresponding sections of the present paper, several studies have assessed interobserver reliability of respiratory physical signs [5, 7, 10, 14, 53, 54].

Another approach to the evaluation of clinical signs is to relate their presence to functional parameters. In this respect, COPD is a particularly interesting model because clinicians have objective and reproducible means not only to ascertain the diagnosis but also to appreciate the severity of the disease (e.g. pulmonary function tests or computed tomodensitometry of the lungs). After an early work by SCHNEIDER and ANDERSON [12], four main studies are available in which the presence of one of the aforementioned physical signs has been related to the results of pulmonary function tests. In the first, GODFREY et al. [11] found significant correlation between FEV1 and tracheal descent on inspiration, scalene and sternomastoid muscle activities, and recession of supraclavicular fossae on inspiration. As in the initial description by LAL et al. [49], FEV1 correlated with expiratory time. That, in this study, external jugular filling was less closely associated to FEV1 than to specific airways conductance suggests a relationship with hyperinflation. It is, therefore, possible to hypothesize that this sign could reflect the presence of intrinsic positive expiratory pressure, but it could also be due to contraction of expiratory muscles.

In the second study, by STUBBING et al. [15], the physical signs assessed were first agreed upon by two trained respiratory physicians contributing to the work (kappa values 0.50–0.62), and then correlated to age, FEV1 and functional residual capacity (FRC). There was a significant correlation between FEV1 and tracheal descent during inspiration, scalene (but not sternomastoid) contraction, costal margin movement, and what was called "cardiac position" (a combination of the impalpable apex beat and impaired cardiac dullness at percussion). This latter sign was the only one that correlated not only with FEV1 but also with the degree of hyperinflation as assessed by FRC (measured using the helium dilution technique). Supraclavicular fossae recession, intercostal recession, upper rib cage movement and tracheal length were not correlated with FEV1 or FRC.

The third and the fourth study, by BADGETT et al. [14] and HOLLEMAN et al. [55], provide operating characteristics of various signs, e.g. wheezing, cough, decreased breath sounds, subxyphoid impulse, chest expansion, forced expiratory time, etc. In a series of patients with a 16% prevalence of COPD, BADGETT et al. [14] reported negative and positive predictive values of 85 and 54% for decreased chest movements, 85 and 57% for forced expiratory time, and 87 and 71% for displacement of cardiac impulse. This study is also the only one in which multivariate analysis was performed. Its results indicate that a history of smoking and reduced breath sounds were the only parameters significantly associated with COPD. This combination had a sensitivity of 67%, specificity of 98%, and an area under the receiver operating curve of 0.88. Interestingly, in this study, peak expiratory flow rate did not improve diagnostic accuracy, but it appeared in a subsequent model used by the same group [56]. HOLLEMAN et al. [55] found similar results, reporting that the number of years the patient had smoked cigarettes and auscultated wheezing were the only independent predictors of airflow obstruction. It should be noted that signs from inspection and palpation were almost ignored in this study, the only one listed being decreased breath sounds.

In addition to these four papers, it should be noted that DELGADO et al. [16] have shown that occurrence of abdominal paradox during exercise depends on the severity of airflow obstruction.

It appears that we still lack a vast comparison of the many signs and symptoms elicited by inspection and palpation in COPD with functional data. Such work would improve our knowledge of the operative value of clinical examination in the diagnosis of COPD and assessment of its severity, and lead to a better teaching of symptomatology.

Asthma

The diagnosis of acute asthma is generally not a problem. Evaluation of the severity of a given attack and of the risk for relapse can be more difficult.

As stated previously, BEAU and MAISSIAT [23] were early in describing the importance of sternomastoid muscle contraction in asthma, rather than that of the scalene muscles. More than a century later, McFADDEN et al. [26] supported this observation when they established correlations between clinical findings and the degree of airways obstruction in acute severe asthma. They found that only sternomastoid muscle retraction (contraction of the muscle associated with elevation of the clavicle, see above) consistently identified patients with

severe obstruction. Increases of the respiratory rate above 30 breaths·min^{-1} and of cardiac frequency above 120 beats·min^{-1} also seem reliable indicators of acute asthma severity. The amplitude of the decrease in systolic arterial pressure during inspiration is commonly used in clinical practice as an indicator of severity of acute asthma. More than 10 mmHg of paradox in this context seems indicative of reduction in FEV1 below 1.25 L [57], and correlates with the presence of hypercapnia [58]. The predictive value of pulsus paradoxus alone in assessing acute severe asthma has, however, been criticized, in particular with reference to the age of the patients [59]. FISCHL et al. [60] constructed a clinical index taking respiratory rate, cardiac frequency, retraction of the sternomastoid muscle, intensity of dyspnoea, intensity of wheezing, and peak expiratory flow rate. In 205 consecutive patients presenting to an emergency room with acute asthma, the index score correlated with the severity of obstruction and allowed accurate prediction both of the risk of relapse (relapse occurred in 40 patients and had been predicted in 95% of them) and the need for hospitalization (needed in 45 patients and predicted in 96% of the cases). The performance of this index has been challenged by a subsequent prospective study [61]. However, physical signs appear to provide a good initial estimate of the severity of asthma, and therefore adequate therapeutic guidance.

Pulmonary Embolism

Dyspnoea and polypnoea, together with tachycardia, are usually considered the most sensitive signs of pulmonary embolism. However, they lack specificity, and there are very few well-designed studies allowing firm conclusions to be drawn

From the cohort of patients participating in the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study [62], STEIN et al. [63] have selected 365 patients without pre-existing cardiac or pulmonary disease, 117 of which had pulmonary embolism (PE) as established by pulmonary angiography, the remaining 248 being considered unaffected by PE on the basis of negative pulmonary angiography or ventilation-perfusion scans. They have studied the frequency of a series of clinical signs and laboratory findings in the two groups. If we only consider inspection and palpation, interesting features of this study are as follows: tachypnoea was present in 70% of patients with PE, but had no specificity; cyanosis was extremely rare in PE; leg swelling and Homan's sign were found in only 32% of PE patients, and 24% of non-PE patients. Considering other clinical signs, results of the electrocardiogram, blood gases, and chest radiography, the authors of this study suggested that clinical assessment was unable to allow clinicians to safely diagnose or exclude PE, but that it was useful to decide further investigations. As for other types of data, there does not seem to be any correlation between physical signs and the presence or absence of an embolus in the pulmonary circulation.

Acute inspiratory muscle fatigue

Inspiratory muscle fatigue has been proposed as a mechanism leading to respiratory failure in various clinical situations. However, we lack studies attempting to draw parallels between clinical features and mechanical or electrophysiological indices of muscular dysfunction. To our knowledge, only COHEN et al. [20] collected such data. In 12 patients difficult to wean from a mechanical ventilator, these authors recorded diaphragmatic and intercostal electromyographic (EMG) tracings. They defined "fatigue" as a fall in the high-to-low frequency ratio of EMG power spectrum, and assessed respiratory rate and thoracoabdominal motion after disconnection from the ventilator. Six patients showed EMG signs of inspiratory muscle fatigue. Only in this subset of patients was an increase in breathing frequency observed, followed by respiratory alternans, and abdominal paradox. The onset of the abdominal paradox generally corresponded with a Pa,CO_2 associated with acidosis. Asynchronous thoracoabdominal motion had previously been noted in patients with acute respiratory failure [2, 13, 64], and associated with a poorer prognosis. The study by COHEN et al. [20] further suggested that respiratory alternans and abdominal paradox are valid indicators of respiratory muscle fatigue, and are more specific than tachypnoea and hypercapnia.

Conclusions

In conclusion, throughout this review it has been mentioned many times that physical signs elicited by inspection and palpation of the thorax or other organs relevant to the assessment of respiratory status are extremely variable from one observer to another. In no case does this mean that physical signs do not allow one to make adequate diagnosis, and interobserver variability does not preclude a test's utility. In the study by BADGETT et al. [14], diminished breath sounds among a set of nine clinical signs had the best operative values (sensitivity 65%, specificity 96%, positive predictive value 77%, negative predictive value 93%; this with a 16% prevalence of COPD) in spite of low kappa values.

There is also a marked discrepancy between the fact that physical diagnosis of COPD is generally reported as easy when typical signs are present and the clinician is aware of them on the one hand, and poor clinical-functional relationships on the other. It is important to note that the efficacy of clinical examination can be unrivalled in making a diagnosis, even if its ability to quantify the underlying abnormalities is small. There is, therefore, a need for repeated large scale studies assessing the sensitivity and specificity of respiratory physical signs and groups of signs, together with the effects of the training of observers.

Indeed, intuition suggests that the importance of training is major. It is conceivable that having learned to recognize a sign and to relate it to a disease can increase the usefulness of this very sign for a given individual. This is supported by the differences in kappa values

reported for diminished breath sounds as a sign of emphysema in untrained physicians (0.16) [7] *versus* trained physicians (up to 0.43) [53]. However, in a limited set of examiners, MULROW *et al.* [53] suggested that time spent in training did not, by itself, bestow improved clinical performance. This point clearly warrants reassessment, and once again we lack data concerning inspection and palpation signs of COPD.

It appears that, although caution is needed before taking decisions on the sole basis of physical signs, they remain the starting point of any diagnosis and therapeutic strategy and are impossible to circumvent: the physician's eyes and hands should not be replaced by chest radiograph, pulmonary function tests or other laboratory tests.

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