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The Art and Science of the Medical Auscultation: Respiratory Sounds

Alcibey Alvarado[1*] and Isabel Arce[2]

[1]Internal Medicine and Neumology, Clínica de Diagnóstico Médico, San José, Costa Rica.
[2]Medicine and General Surgery, Medicine School, University of Costa Rica, San José, Costa Rica.

Authors' contributions

This work was carried out in collaboration between both authors. Authors AA and IA contributed equally in the planning, data collection, data analysis, writing and critical review. Both authors read and approved the final manuscript.

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ABSTRACT

Auscultation of the chest is a very old diagnostic method. Originally, it was performed by the examiner directly hearing into the patient's chest, and this practice dates back to the time of Hippocrates. The original description of lung sounds was based on comparing acoustic phenomena that was heard in the chest with gross lesions autopsy. Subsequently, the interpretation of these findings is based on functional rather than anatomical analysis. Auscultation became an invaluable diagnostic method. With the advent of radiological images, increasingly sophisticated, computerized tests of lung function, rapid arterial blood gas analysis, endoscopic studies airway and percutaneous biopsies of pleura and lung; the practice, with method and timing of chest auscultation, has lost presence. In addition, the nomenclature of respiratory sounds is sometimes confusing and the terminology proposed by international committees, little known. One objective of the article is to give a pathophysiological basis of noise based on modern computer-assisted studies which have enabled accurate recording and sound analysis techniques. The other objective is to provide a practical and useful tool to understand and correlate what you hear, with the pathophysiological basis, the underlying condition that generates the phenomena and streamline the diagnostic work.

**Corresponding author: E-mail: alcialvagonza@yahoo.com.mx;*

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1. INTRODUCTION

René Theophile Hyac Laënnec invented the stethoscope and introduced the first classification of respiratory sounds in 1816 [1]. Paul Forgacs subsequently reclassified and reinterpreted them no longer based on macroscopic observations autopsy as done by Laënnec, but based on functional findings [2,3]. Since then, confusion has occurred in the terminology of breath sounds, not only in terms of its anatomical origin and the mechanism that generates, but also their clinical translation. The problem has been perpetuated or increased when translating a term from one language to another. In addition, recent acoustic research sometimes can't find the explanation for a term that is supposed to have a pathophysiological basis, but that has been passed down through generations with medical over time.

Since the traditional nomenclature of lung sounds suffers from imprecision and ambiguity, several attempts have been made to sort the issue. For example, the ad hoc committee of the International Lung Sounds Association replaces the term "rale" by "crackle" as the adjectives often used to qualify rales (e.g., "moist" or "dry") can be misleading with respect to the mechanism of production. "Crackle" can be defined acoustically and does not suggest any means or site of generation [4].

The same applies to the "vesicular" murmur. The idea that the "vesicular" sound is produced by air entering the alveoli ("vesicles") is incorrect. Gas molecules in the lung periphery migrate through facilitated diffusion by the Brownian motion of the particles and this is a silent process. For this reason the term normal lung or respiratory sound is preferred [5].

Wave frequency, time, amplitude (distance from the baseline to the peak) and wavelength (λ = distance between two peaks) are fundamental concepts in recording, analyzing and explaining the genesis of sounds, and help to understand what is being auscultated; however, neither stethoscope nor the human ear can discern this kind of quantitative characteristics; only qualitative can be appreciated (for example pitch, which is a subjective perception of sound frequency or loudness is the subjective perception of amplitude). Therefore, experience and repetition are essential in the process of listening and is where the tool has a lot of art.

2. NORMAL RESPIRATORY SOUNDS

2.1 Sounds of the Mouth and Trachea

It is very likely that the sounds heard in the mouth and the trachea have the same origin and translate the same phenomena, but there is no absolute consensus on this. The sound in the mouth is panting, continuous (inspiration and expiration), of a tone (auscultation) not well defined, with a frequency distribution of 200-2000 cycles per second (Hertz = Hz) and is produced by airflow turbulence at the level of the mouth, pharynx, larynx, trachea and bronchi higher to the tenth generation. The turbulence in the airways appears when the airflow exceeds a certain critical speed [6].

A patient with diffuse large tract obstruction, by deformity or narrowing, will need a higher flow speed to transport the air, generating greater turbulence, and consequently, an increase in the intensity of auscultated sound in the mouth during inspiration. Theoretically, this could have diagnostic value in the face of chronic obstructive pulmonary disease (COPD), because in a patient with only chronic bronchitis, the sound would be increased in the mouth and lung sound would be auscultated in the chest, increasing or with abnormal aggregated sounds (see later), while in a pure emphysema the sound would be normal in the mouth (as the caliber of the major routes is normal) and would be absent or greatly diminished in the chest due to gas entrapment. Probably the biggest semiotic advantage is comparing the auscultated breath in the mouth against the breathing sound in the chest in pure emphysema. The practical value is questionable as most patients with COPD have airflow obstruction, particularly during expiration, produced by mixing higher airway injury and destruction of lung parenchyma [7].

As in auscultation of the mouth, the trachea is also infrequently performed. Whether the doctor auscultates on the side of the neck or the supra-sternal notch, the sound has large amounts of energy, it´s loud and continuous, with a frequency range of 100-5000 Hz, and therefore, can be easily heard. It is produced by turbulent air flow in the pharynx, glottis and subglottic region [8].

Auscultation over the trachea can be helpful in certain specific situations. The tubular quality of the trachea is a good model to teach the "bronchial breathing" (see later). The trachea drives sounds from within the lung, unfiltered by the ribcage. The acoustic characteristics are similar to the abnormal bronchial breath of a consolidation. Tracheal breath sounds may be helpful in detecting obstruction of upper airway (UAO). The sound becomes loud in the presence of UAO. Extrathoracic UAO produces stridor, whereas, intrathoracic UAO is associated with wheezing sound; if just the chest wall is auscultated, it can be confused with asthma [9,10].

Tracheal sounds analysis can be helpful in monitoring patients with sleep apnea-hypopnea syndrome although monitoring can't be performed by auscultation with a stethoscope [11,12]. While a child with croup stridor is easily recognizable, stridor in adults (tracheal stenosis or bronchial or tumor central air) it is more subtle and may go unnoticed if just the chest wall is examined, but it will be obvious when you auscultate the trachea or larynx [13].

In developing, with high PTB (pulmonary tuberculosis) prevalence countries, it is not uncommon to find patients with chronic sequelae with apical fibrosis and atelectasis of the ipsilateral upper lobe. This will produce retraction of the trachea and the anterior chest auscultation (subclavian region) or rear-upper chest, or supraclavicular fossa, the full tracheal sound will listen as if heard on the neck [14].

2.2 Normal Lung Sound

Also known as normal respiratory sound, is the right term that replaces the "vesicular murmur". Not all types of air flow produce breath sounds. The laminar flow, which takes place in the small airways (<2 mm diameter), is silent. The current air flow is parallel to the walls, being parabolic in shape with a central layer, which moves faster than the peripheral with little or no transverse flow. There is little mixing or collision between the layers of gas and no sound is generated. Therefore, again, the passage of air through the alveoli does not generate buzz [9].

The mechanisms that generate normal lung sounds are several. They include the turbulent flow, vortices and other still unknown [15-17]. There are actually studies that support the idea of a double origin, with the inspiratory component generated within the lobar and segmental airways and the expiratory component coming from more central sources [5]. Turbulent flow occurs when air passes at high speed through large diameters of the airways, particularly with irregular walls, for example trachea and bronchus or with sudden airway branching. Turbulent flow is disorganized and of chaotic nature. It depends on the density of air more than viscosity. In fact, if a mixture of air with helium (low-density gas) is used, breathing turbulent flow is reduced, the laminar flow increases, and intensity of sounds in the mouth, paravertebral region and in the lung bases is reduced. The fact that the sound is decreased despite the increase in laminar flow, supports the idea that the sound does not occur at the alveolar level [18,19]. The normal lung sound is different from the voice sound transmitted. Lung sound is generated within the lung, voice sound by the larynx.

It is obvious that the sound heard on the surface of the thorax is markedly influenced by the anatomical structures that are between the source site and the site auscultation. We must remember that the waves become weaker in amplitude as they move away from their point of origin. It is clearly heard during inspiration, but only in the early phase of exhalation, is smooth, not musical and the frequency range from 100-1000 Hz [20].

The most common abnormality is decreased intensity. Mechanically, this loss may be due to decreased intensity of sound energy at the site of generation, transmission altered, or both [21]. Sound generation can be reduced when there is a drop in inspiratory airflow, which can result from short range cooperation to take a deep breath, to a depression of the central nervous system drug overdose. Blocking the airway by a tumor or a foreign body and narrowing by obstructive airway diseases (asthma and COPD) as well. The phenomenon may be permanent (pure emphysema) or reversible as asthma [22].

Sound transmission can be impaired by intrapulmonary o extrapulmonary factors. The latter includes conditions such obesity, chest deformity (e.g., kyphoscoliosis), and abdominal distention due to ascites. Intrapulmonary factors, which can be harder to recognize, include disruption of the mechanical properties of the lung parenchyma as hyperdistention and parenchymal destruction in emphysema, or the interposition of a medium between the sound source and stethoscope (pleural effusion,

pneumothorax, pleural thickening and intrapulmonary masses).

Normal breathing sound comprises various frequencies. The fundamental or primary frequency is a low frequency sound wave and determines the pitch of the sound. Frequencies higher than the fundamental are called overtones. Harmonics are overtones whose frequencies higher are whole number multiples of the fundamental frequency. The air in the lung and chest wall absorb high frequencies (overtones) allowing only the passage of the fundamental. Hence the acoustic characteristics of lung sound or normal breathing and so in emphysema noise is very poor. The air is a poor transmitter overtones (acoustic insulation) [9]. In a condensation, for example pneumonia, if the airway within the parenchyma condensate is blocked (inflammation or viscous secretions) lung sound is diminished or absent. But if the way is permeable, the sound is increased because there is no air to filter high frequencies and reach the stethoscope with the fundamental frequency and overtones, (i.e., unfiltered). This sound has a strong expiratory component, and is an intermediate sound between normal sound and tracheal, crisper, clearer, having no frequency filter. It is known as "bronchial breathing" or “bronchial breath” and corresponds to tubal murmur of pneumonia in classic French semiotics. Really what results is a condensation of the parenchyma without bronchial obstruction and corresponds to air bronchogram on chest radiographs [5]. The "amphoric" terms and "cavernous" are variants of this auscultatory phenomena. Were described in caves and open pneumothorax in which the walls of the pleural cavity or parenchymal amplify high frequency (overtones), increasing the intensity of sound. Very little use today in modern semiotics [9].

3. ABNORMAL RESPIRATORY SOUNDS

Also they are known as adventitious sounds. They are additional respiratory sounds superimposed on normal breath sounds and they can be divided into musical and non-musical.

3.1 Musical Sounds

3.1.1 Stridor

Stridor is a musical high-pitched sound produced by turbulent flow passing through a narrow segment of the upper respiratory tract [23]. Usually intense, audible distance without stethoscope and characterized by a sinusoidal oscillation, regular, with a fundamental frequency of 500 Hz, and generally accompanied by several harmonics. It differs from wheezing for several reasons. Heard stronger in the neck on the chest wall. It is mainly inspiratory, it can also be expiratory or biphasic. In extrathoracic obstruction of the airway (laryngeal edema or ruptured vocal cords) and after extubation prompt intervention is required. Other causes include acute epiglottitis, anaphylaxis, foreign body inhalation, laryngeal and tracheal tumors and thyroid carcinoma [5].

In the case of vocal cord dysfunction (paradoxical vocal cord motility, episodic laryngeal dyskinesia) it is often confused with asthma, causing hospitalizations, intubations and medical costs increased by misdiagnosis. What happens in this disease is an inappropriate adduction of the vocal cords with airflow limitation at the level of the larynx, accompanied by stridor [24,25].

3.1.2 Wheeze

It is the adventitious sound easier to recognize [3]. It is long-term (> 100 msec), and musical quality can be recognized by the human ear. The wave is a sinusoidal oscillation with a range of 100-1000 Hz power and harmonics that can exceed 1000 Hz [26].

An important prerequisite for the production of wheezing is the airflow obstruction although airflow limitation may occur in the absence of bronchospasm [27]. Wheezes are generated in the branches between the second and seventh branch of the bronchial tree, coupled oscillation of gas and air walls that have been clogged to the point of apposition (about to be in contact) by a variety of mechanical forces [27,28].

This perspective deserves several comments. Severe asthma (status asthmaticus) is a condition in which the airways have impaction of mucus and the flow of air is so small that it can’ t provide enough energy to generate wheezes or any other sound, producing a clinical picture known as " silent chest "of poor prognosis. As clogging is reduced and the airflow increases, wheezing and normal breathing sound reappear. Therefore, wheezing after a period of silent chest is a sign of improvement [9]. It is incorrect to credit high-pitched wheezes obstruction of peripheral airways and the low tone of central airway obstruction. Finally, tone wheeze be

determined by the thickening of the wall of the airway, bending stiffness and longitudinal tension, but not by the diameter [27].

Wheezes may be inspiratory, expiratory, or biphasic. They can be diffuse as asthma, COPD, heart failure ("cardiac asthma"), bronchiectasis, an others, are not pathognomonic of any particular disease. They can be localized, produced by a local event as an aspiration of a foreign body, impacted mucus or tumor.

Monophonic wheeze consists of a single musical note that starts and ends at different times and usually translates a local obstruction. Stridor could be considered a high-pitched monophonic wheezing, inspiratory. Monophonic wheeze can be seen in asthma. Polyphonic wheeze consist of multiple musical notes that start and end at the same time and usually produced by dynamic compression of large, central air and are confined to the expiratory phase and obstructive airways disease [3].

The roncus is a variant of wheezing that differs from them because it has a low tone, about 150 Hz, which is responsible for the acoustic resemblance to snore during sleep. The production mechanism is considered the same as wheezing, but unlike high-pitched wheeze, may disappear after coughing, suggesting that the secretions may play a pathogenic role. Many authors prefer to use the term low-pitched wheeze instead of roncus. The classification would be: high-pitched wheezes or low pitch (rhonchi) [3].

Wheezing is a nonspecific finding and may even be detected in a healthy person toward the end of expiration after forceful expirations [29].

3.2 Nonmusical Sounds

3.2.1 Crackles

Of adventitious lung sounds, crackles are probably the most helpful for clinical diagnosis [30]. Are not musical, short sounds, explosives, discontinuous heard in inspiration and sometimes expiration, of a repetitive nature and appear in the same phase of successive respiratory cycles [3]. Repeatability suggests that crackles occur when certain conditions of pressure and volume are repeated in the lung [6]. They are classified into coarse and fine according to their duration, volume, pitch, time in the respiratory cycle, relative to cough and body position change [9]. Fine crackles on auscultation usually heard in mid to late inspiration, perceived well in the dependent lung regions, and are not transmitted to the mouth. They are not influenced by coughing, but by gravity, and change or disappear with changes of position (e.g., lean forward). Forgacs description is vivid and eloquent regarding its production mechanism and physiological significance: "Crackles are miniature explosions, heard much more often during inspiration than during expiration. Their pattern is remarkably constant and cannot be destroyed by coughing. Except in focal lung disease, the lowermost part of the lung is nearly always the richest source of these sounds. Clearly, these explosive sounds cannot be generated by some evanescent substance-they must come from some structure built into the lung, which is brought into play by a recurrent pressure or volume event, and can be modified by gravity" [28].

Coarse crackles tend to appear early in inspiration and expiration through and have a "popping" quality. They can be heard in any region of the lung, usually transmitted to the mouth, and change or disappear with coughing, and are not influenced by changes in body position. Compared with coarse crackles, fine crackles have a shorter duration (5 msec vs. 15 msec) and a higher frequency (650 Hz vs. 350 Hz) [31,32].

The most likely to generate fine crackles inspiratory mechanism is a sudden opening of small airways that were closed during the previous exhalation. This closure is due to surface forces by a partially deflated lung. During inspiration, a pressure gradient in the segment before and after the collapse is established. The explosive and sudden opening of collapsed airways induces rapid equalization of gas pressures on both sides resulting in oscillations of the gas column, generating crackles [33]. Opening to go, one after another, closed airways, a cascade of crackles occurs [6].

Coarse crackles probably occur by the passage of bowling gas through proximal bronchi that opens and closes intermittently, narrowed by inflammation mediated by loss of cartilaginous support [34]. With the exception of the crackling sounds heard in moribund patients or in patients with abundant secretions (e.g., bronchiectasis), often crackles are probably not produced by secretions [5].

The evaluation of crackles can help the differential diagnosis. Fine crackles lung seen in diffuse parenchymal lung disease (DPLD) (idiopathic pulmonary fibrosis, asbestosis and pulmonary fibrosis associated with connective-tissue disorders). Epler et al., in a study involving 272 cases of DPLD documented by lung biopsy, reported bilateral fine crackles in 60% of patients with idiopathic pulmonary fibrosis (IPF) and asbestosis, with pathological diagnosis; and 53% of patients with desquamative interstitial pneumonitis [35]. However, fine crackles are less common in diseases of intra-alveolar filling (alveolar proteinosis, eosinophilic granuloma and granulomatous diseases such as sarcoidosis and miliary tuberculosis). They listen only 20% of sarcoidosis and other granulomatosis. This is because the fibrosis in sarcoidosis is less extensive than in IPF [36]. In addition, IPF predominates in the bases of the lungs unlike sarcoidosis is more common in the upper lobes and peribronchial location. Crackles are more frequent with the honeycombing and sub-pleural fibrosis and both are more common in IPF. IPF crackles appear first on the bases and extend into the pulmonary apexes with the progression of the disease.

Fine crackles in IPF are also called "crackles Velcro" because their sound is similar to the sound produced when gently separate the united bands of adhesive velcro present in the sleeves of blood pressure equipment or jogging shoes [37]. In IPF they can be initiated in the middle phase and even at the beginning of inspiration but continue until the end of inspiration and can be heard from the start but being profuse at the end of inspiration [38]. Unlike COPD and bronchiectasis, crackles in IPF are not transmitted to the mouth and are detected early in the course of the disease.

In IPF and asbestosis fine crackles can be found before the radiological abnormalities and are considered an early sign of lung disorder [39]. Although the presence of crackles Velcro type is not formally considered diagnosis of IPF, auscultation is considered the only realistic and practical means early in the course of the disease [40]. In asbestosis, use of computerized detection of crackles appear to be as accurate as CT in locating disease that is not apparent radiologically and detection of crackles on auscultation may have a role as a noninvasive method of screening in workers exposed to asbestos [40,41]. Advanced computerized acoustic analysis, which involves the use of a multichannel sound-detection device, has made possible the diagnosis of idiopathic pulmonary fibrosis and congestive heart failure, and other cardiopulmonary disorders, with good sensitivity and specificity [42].

Coarse crackles are commonly heard in patients with obstructive disease (COPD, bronchiectasis and asthma) usually in association with wheezes. Also heard in patients with pneumonia and heart failure. In pneumonia, crackles character depends on the phase of pneumonia. In the acute and early phase, crackles are heard in the middle of inspiration and are coarse, in the resolution phase, they can be heard more at the end of inspiration and are shorter, resembling IPF. In acute pneumonia, the reopening of closed airways by edema and infiltration of inflammatory cells produces coarse crackles. During the resolution of pneumonia, lung parenchyma becomes drier and rigid due to the resolution of edema and healing process. Higher pressure and volume to open the airways is needed, therefore, crackles move toward the end of inspiration and become fine [43].

In heart failure, crackles occur due to the opening of the airways obstructed by peribronchial edema [44]. They can be inspiratory and expiratory, and disappear quickly with the resolution of heart failure and are typically posterior and basal. If is supine position, they appear in the anterior face of the chest, an alternate cause must be procured [45].

Fine and coarse crackles can coexist [46].

The presence of persistent crackles in both lungs, after several deep breaths and heard for intervals of several weeks, in subjects over 60 years with dyspnea, should take prompt investigation by interstitial lung disease [40]. Although crackles may appear in healthy people, usually they disappear after a few deep breaths [29].

3.2.2 Pleural friction rub

The normal pleura (visceral and parietal) glide silently one on the other, lubricated by a little amount of pleural fluid. When the surface becomes irregular due to the presence of fibrin deposits or is infiltrated by inflammatory or neoplastic cells, this slip is momentarily interrupted by the frictional resistance offered by the foreign elements [6]. This produces a crackling sound heard as a brush, pleural rub.

This sound is more prominent in the basal and axillary regions than in the higher, possibly because the basal regions are in the steep portion of the pressure-volume curve, while the upper regions on the flat part of the curve. Therefore, for a change in transpulmonary pressure, the basal regions have more expansion [5]. Typically, pleural rub is biphasic, with the expiratory sound sequence mirrored with respect to inspiratory [44]. It is a non-musical sound, explosive, short, squeaky, scratchy, and corrosive [9]. The waveform is similar to that seen with crackles, except for its longer duration and lower frequency. Typically heard in inflammatory diseases (e.g., pleurisy) or malignant pleural disease (e.g., mesothelioma). When the amount of liquid increases significantly, the friction between them disappears and the event is avoided.

3.3 Mixed Sound-The Squawk

Also called as "squeak" or "short wheeze", the squawk is a mixed musical and non-musical sound. The sound analysis shows a short wheeze inspiratory, with a sinusoidal oscillation, lasting less than 200 msec, and a fundamental frequency between 200-300 Hz [5]. It usually occurs on inspiration and is preceded by inspiratory crackles [9]. The mechanism that generates it is not clear, but presumably produced by the oscillation of peripheral airways (in deflated lung zones) whose walls remain in apposition enough to oscillate under the action of the inspiratory airflow [44]. Squawks are typically heard from the middle to the end of inspiration in patients with interstitial diseases, particularly hypersensitive pneumonitis [47]. They have also been documented in pneumonia, and bronchiolitis obliterans [48,49]. If a patient has this finding and no evidence of interstitial disease, pneumonia should be suspected, because it is the next most common cause, particularly in lung areas where radiological visualization may be suboptimal, such as below of the diaphragmatic dome or in the retrocardiac region [48].

4. VOICE AUSCULTATION

4.1 Overview

The voice sounds are produced by the larynx. This happens when bowling air passes through the vocal cords, causing vibration. Therefore, unlike breath sounds and adventitious sounds, speech sounds are not produced in the lungs. These sounds are modulated by the filter function of the supralaryngeal airways. These sounds consist of a fundamental note of low frequency (<200 Hz) and several overtones (harmonics). Normally, in a healthy person, due to the filtration exerted by normally aerated lung, vocal sounds are unintelligible (auscultates in the chest wall) because high frequencies are filtered. However, when the alveolar air is replaced by a solid material or has atelectasis (remember atelectasis traction upper lobe tuberculous scar, or complete atelectasis of one lobe or lung by endobronchial obstruction), the vowel sounds are transmitted better and become intelligible and transmitted well, since no air filtration [9]. Patient is requested to say the words "one-two-three" or "ninety-nine" or "thirty-three" (having an adequate distribution of consonants and alternating vowels) and the voice is heard in both hemithorax. A good practice is auscultate first in the mouth to get an idea of the quality of the voice without any filtration.

4.2 Bronchophony

When the patient pronounces the above words, the lung of the hemithorax is compared with the sick hemithorax, and in the consolidated or atelectatic area you listen the words increased in intensity and clarity. This phenomenon is called bronchophony [6].

4.3 Pectoriloquy

Patient is requested to pronounce the words quoted above but in a whispered way. The vocal cords do not vibrate (then no fundamental note), but are very close. This produces a turbulent flow of air flow generated by the whisper, which is filtered and heard very weak or not audible in the chest auscultation. But, in the case of a consolidation, it does not suffer filtration and the whisper is heard clear and distinctive. The phenomenon is called pectoriloquy and constitutes, along with the bronchophony and the bronchial breath, the auscultatory syndrome triad condensation [6].

5. METHODS OF PERFORMING AUSCULTATION

Auscultation should be performed after inspection, palpation and percussion of the chest. It should never be done through clothes and good practice is to make semiotics before

and after seeing the chest radiograph postero-anterior and lateral.

It should be practiced in a quiet room without noise, preferably with the patient sitting as the rest of the thoracic semiotics. If the patient can't sit, you must be rotate from side to side of the bed to examine both hemithorax.

Always warm the cold stethoscope in your hands, by rubbing it, before placing it in the body.

The patient should be induced to take deep breaths with the mouth open, with rhythm and periodicity.

The diaphragm of the stethoscope is used for auscultation, which should begin at the mouth and then in the neck, on either side of the trachea and suprasternal notch.

Subsequently, front faces of pulmonary apexes below the clavicles are heard; and continue down until no breath sounds can be identified. The same is done in the posterior thorax, starting at the apices and moving down. At least one respiratory cycle should be heard at each side where the stethoscope is placed. One hemithorax is heard, then the other and then both, comparing the findings.

Attention should be paid to the quality of the sounds, the intensity and the presence of adventitious sounds.

This procedure, with order and method, consumes no more than 10 minutes of time.

6. CONCLUSIONS

Technology has impacted medical auscultation with computerized equipment that allows to collect, analyze and study sound waves in a realistic way and never before seen. This technological development enables and improvement in auscultation knowledge in order to understand more and better findings.

But this development, should not pretend to cram sophisticated, expensive and unnecessary medical equipment. Auscultation of the lungs with a simple stethoscope, in day to day work and to the bedside of the sick, is part of the physical assessment. We must remember that it is a tool whose effectiveness depends on the rest of the physical examination and medical history.

No other clinical procedure yields much information about the respiratory system. Requires little patient cooperation, it is cost-effective, easy, fast and cheap and available almost universally. It can be repeated as often as necessary, is not invasive and helps making decisions about diagnostic work.

The methodical, repetitive and tenacious practice dye of art a tool that has more science as time goes by.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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