The Functional Basis of Pulmonary Sounds*

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The sounds radiated by diseased lungs are regarded by many clinicians as signs of relatively low diagnostic value. In comparison with radiographic and laboratory procedures, such sounds appear to lack precision, so that auscultation is often performed as a bedside ritual, in deference to tradition. Most textbooks of pulmonary diseases dismiss lung sounds in a few short paragraphs written in the language of the last century.

The custom of interpreting clinical signs in morbid anatomic terms persisted longer in respiratory medicine than in most other specialties. There is surprisingly little experimental work on pulmonary sounds, and most of the generally accepted theories of their mode of origin do not bear critical examination. The objective of the experiments and clinical observations summarized in this report was to discover how normal and adventitious pulmonary sounds are generated. They are treated, whenever possible, as signals of physiologic events, to be decoded and interpreted in terms of normal or disordered function.

One reason for the neglect of auscultation in respiratory medicine is the confused terminology of adventitious pulmonary sounds. An example is the use of the terms, “rale” and “rhonchus.” Rale was originally a generic term applied to every variety of adventitious pulmonary sound. For Laennec’s patients, “rale” had unpleasant associations with the rattle of secretions in the airways of the dying, and in order to spare their feelings, he used the Greek synonym, “rhonchus,” at the bedside. As a result of translation into English and redefinitions, these terms have come to mean two different varieties of adventitious sound. The confusion deepens when one looks for consensus about the meaning of dry and moist rales, crepitations, wheezing, rhonchi, stridor, etc. None of these terms has been defined in the language of acoustics, so that it is not surprising that opinions differ widely about their characteristics and significance.

In an attempt to restore order, it was suggested by Robertson and Coope1 that the traditional terms, tainted by long misuse, be discarded and that all adventitious sounds be classified as crackles or wheezes. These two terms will be used exclusively in this review. Although their meaning is self-evident, crackles and wheezes are also capable of definition in acoustic terms. Crackles are a sequence of short interrupted sounds with a wide spectrum of frequencies between 200 and 2,000 hertz. They may be high or low pitched (according to whether the lower or higher frequencies predominate within this range), loud or faint (according to the amplitude of oscillations), scanty or profuse (according to the number of individual crackles), and inspiratory, expiratory, or both. Other characteristics of timing, such as regular or random spacing, are also of clinical significance. The qualifying adjectives, “moist” or “dry,” are usually applied to low-pitched or high-pitched crackles, respectively. These terms are not only inaccurate, but they also prejudice the mode of origin of these sounds and should therefore be discarded. The term, “crepitation,” meaning high-pitched crackling, is also superfluous.

Wheezes are musical pulmonary sounds. Their musical character, obvious to the listener, is determined by their spectrum in which most of the energy is contained in harmonically related frequencies. The lowest or fundamental frequency sets the pitch of the note. Musical sounds can also be recognized on the screen of the oscilloscope by the regular pattern formed by a sequence of identical wave forms. Wheezes may be classified as high or low pitched, inspiratory or expiratory, short or long, and single or multiple. Monophonic wheezing consists of a single note or of several notes starting and ending at different times. Polyphonic wheezing contains several notes starting and ending simultaneously, like a chord. The different character of wheezing heard at a distance, or through the chest wall, is explained by filtration through the lung. The source and mode of origin of these sounds is identical; the customary

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Chest, 73: 3, March, 1978
distinction between wheezing and rhonchi is therefore superfluous.

A particularly loud musical sound of constant pitch is known as stridor. Although nothing, except its intensity, distinguishes stridor from a monophonie wheeze, the term is widely used when laryngeal or tracheal obstruction is known or suspected to be the source of the sound. There is no objection to its continued use, provided that its musical character is understood and that stridor is not confused with noisy breathing.

Breath sounds heard through the stethoscope on the chest were originally classified as bronchial or vesicular. These were purely descriptive terms, applied to the sounds heard through the neck or the chest wall, respectively. Although Laennec did not state explicitly how such sounds are generated, the terms themselves and his comments imply that he attributed them to two different sources. There is no evidence in support of this belief. To avoid the implication that the breath sounds heard through the chest of a healthy subject are generated in the alveoli, the term, "vesicular breathing," should be abandoned and replaced by "normal breath sounds."

**Breath Sounds at the Mouth**

One of the most useful signs of chronic obstructive pulmonary disease is noisy breathing. The reason for the neglect of this sign of narrowing of the airways is the belief that breath sounds heard at a distance from the patient are generated in the upper respiratory tract and are therefore irrelevant to the state of the lungs. Nevertheless, it is common knowledge that the breathing of most patients with chronic bronchitis or asthma is noisy. The breath sounds of a healthy subject are inaudible at a distance of a few centimeters from the mouth, unless he is panting, gasping, or sighing, while the resting ventilation of many patients with chronic obstructive pulmonary diseases is accompanied by a noise that can be heard across the room.

This noise is not to be confused with adventitious pulmonary sounds, which have already been defined. The noisy breathing of bronchitic patients, uncontaminated by crackles or wheezes, has a wide spectrum of frequencies, with its energy evenly distributed over a range of 200 to 2,000 Hz. The wave form of this noise displayed on an oscilloscope is irregular, constantly varying in wave length and amplitude. In acoustic terminology a sound with such characteristics is known as a white noise.

The loudness of this noise varies according to the severity of obstruction to the flow of air; for example, in asthma the loudness increases when bronchoconstriction is provoked by exercise and diminishes after the administration of bronchodilator drugs. These variations have nothing to do with changes in the rate of respiratory flow, since they are demonstrable when the sounds are recorded and compared at identical rates of flow (Fig 1).

Measurements of the intensity of this noise during inspiration at rates of flow up to 1 L/sec confirmed the clinical observation that the loudness of the inspiratory breath heard close to the patient's mouth reflects the severity of the obstruction to the flow of air. There is a significant correlation between the intensity of the inspiratory white noise and the forced expiratory volume in one second (FEV1), the peak expiratory flow rate, and other indices of obstruction to air flow. The changes in the intensity of sound during bronchoconstriction and bronchodilatation were also confirmed by objective measurements.

The theoretic basis of this important clinical sign is that the linear velocity of fluid increases when the volume flow rate is held constant and the caliber of the conduit is reduced. The breath sounds become quieter when air is replaced by a mixture of helium and oxygen. This experiment shows that the source of the noise is turbulence in gas flowing through the airways. Laminar flow is silent, but above a critical velocity of flow, turbulence and vortices appear, and the oscillations set up by these irregular patterns of flow radiate sound. The intensity of the turbulence and the noise generated increase with rising velocity of flow.

This explanation, applied to the bronchial tree, offers a clue to the functional basis of the observa-
tions and measurements summarized previously. The noisy breathing of chronic bronchitic asthma patients reflects the high velocity of flow and the increased turbulence in the abnormally narrow proximal intrathoracic airways. The peripheral limit of the turbulent zone is uncertain. It lies somewhere between the lobar bronchi, where flow is known to be turbulent above a critical respiratory rate of flow, and the terminal airways, where flow is always laminar. Between these two regions of the bronchial tree, there is an indeterminate zone where irregular patterns of flow have been demonstrated. It seems probable that noisy breathing in chronic obstructive pulmonary diseases and the correlation between the intensity of the noise and other indices of obstruction to air flow, as well as the silencing effect of bronchodilator drugs, can be explained by widespread changes in caliber in this region of the bronchial tree.

There are two exceptions to the rule that the loudness of the breath sounds heard at a short distance from the mouth is closely correlated with the FEV₁ and other indices of obstruction to air flow. One of these is focal stenosis of one of the principal or lobar bronchi. The noise generated by turbulence at the stenosis and transmitted to the mouth is much louder than predicted from the FEV₁. This sound can be identified by its higher pitch and by the fact that it is often out of phase with the respiratory cycle.

The other discrepancy between the intensity of the breath sounds at the mouth and the conventional indices of obstruction to air flow is the paradoxically quiet inspiratory sound in the presence of severe expiratory obstruction in primary emphysema. In this disease, the inspiratory sound at the mouth is either inaudible, as in healthy subjects, or at most slightly above normal, even when the FEV₁ is reduced to less than 1 L. In chronic bronchitis or asthma, expiratory obstruction of this severity is usually associated with very loud inspiratory breath sounds. The reason for the discrepancy is that in primary emphysema, increased resistance of the peripheral airways, loss of elastic recoil of the lung, and atrophy of the bronchial wall lead to premature dynamic compression of the central airways during a forced expiration, while in contrast to chronic bronchitis and asthma, the caliber of the central airways during inspiration is normal. The paradoxically quiet inspiratory breath sound in primary emphysema is a useful clinical sign of a disease which is clinically often indistinguishable from other varieties of chronic obstructive pulmonary disease.

**Breath Sounds Transmitted to the Chest Wall**

Comparison of voice and breath sounds recorded at the mouth and through the chest wall shows that the lung behaves like a low-pass filter with a steep cutoff of frequencies above 200 Hz. Consolidated lobes transmit voice and breath sounds almost unattenuated and unfiltered, so that the sounds heard over the trachea and the chest wall are very similar. This is the acoustic basis of the familiar clinical signs of consolidation: bronchophony, whispering pectoriloquy, and bronchial breathing.

Experimental observations on excised lungs confirm that sounds generated in the larynx and trachea travel at first through gas contained in the large bronchi. More peripherally, where the caliber of the airways is too narrow for conduction of sound in gas, transmission continues through pulmonary tissue and thence to the chest wall. A variable amount of energy is lost during passage between the lung and the chest wall, depending on the acoustic matching of these two media. The attenuation at this interface is slight in children and thin adults but is considerable in obese subjects. Fluid or air in the pleural cavity forms a complete acoustic barrier; however, a thin layer of fluid (for example, at the upper limit of an effusion) does allow the higher frequencies to pass, so that the transmitted voice sounds acquire a nasal, bleating quality, known as egophony.

The close correlation between the rate of respiratory flow at the mouth and the loudness of the breath sounds heard at a distance also applies to the breath sounds transmitted through the chest, but the intensity of the breath sounds heard through the stethoscope on the chest wall also varies in parallel with the instantaneous rate of flow into the underlying lung.

In some patients the intensity of the inspiratory breath sounds over the left lower lobe rises and falls synchronously with the heart beat. Contraction of the left ventricle and the corresponding expansion of the adjoining alveoli accelerates the rate of inspiratory flow into the left lower lobe. This is accompanied by a systolic crescendo of the inspiratory sounds over the base of the left lung, while their intensity elsewhere remains unchanged.

These observations indicate that the breath sounds heard through the chest wall are dominated by sounds generated in the underlying territory of the lung. Their source is presumably in the lobar or segmental bronchi, with a possible contribution from more peripheral branches where patterns of flow are irregular. The terminal airways and alveoli of a healthy subject are silent. This is confirmed by the observation that no breath sounds can be
heard in the neighborhood of the heart during breath-holding, in spite of the large movements imparted to the adjoining tissues and gas by the contraction of the ventricles. Most of the noise generated by turbulence in the upper respiratory tract and the trachea is lost in transit, while the sounds originating in the lobar and segmental bronchi, having a shorter distance to travel, reach the chest wall.

**Wheezes (Rhonchi)**

A widely accepted but misleading model of the genesis of wheezing attributes these sounds to a mechanism similar to that of an organ pipe. The gas contained within a narrowed bronchus is supposed to be set into oscillation in much the same way as the stream of air blown across the lip of a flue pipe, producing a musical note whose pitch is determined by the dimensions of the pipe. It follows from this model that a simple reduction of caliber or a spur facing the stream of gas in the airways is sufficient to generate musical sounds and that the pitch of the note depends on the length of the adjoining airway. High-pitched wheezing is therefore often attributed to peripheral bronchial spasm.

This theory of wheezing is contradicted by several observations. The longest axial pathway in the bronchial tree is less than one foot in length, while the frequency of some low-pitched wheezes is in the range produced by pipes four to eight feet long. Unlike the sound of organ pipes, the pitch of a wheeze, known to originate from a stenosed bronchus, is not fixed but may differ by as much as an octave between inspiration and expiration. In experiments with excised bronchi, no musical sound can be produced by a simple reduction in caliber until the opposite walls of the compressed bronchus are brought into contact. The frequency of the note can then be varied over several octaves by tightening the compression or by increasing the blowing pressure. The decisive argument against a model resembling the organ pipe and orchestral wind instruments is that the pitch of all these instruments rises when blown with helium, while that of a wheeze remains constant when the inspired air is replaced by a mixture of helium and oxygen.

The correct model is a simple uncoupled reed, represented by a bronchus narrowed to the point of closure, whose opposite walls oscillate between the closed and barely open positions. Reduction of caliber short of the point of closure increases the white noise of respiration but cannot generate a musical note. The mechanism responsible for wheezing is similar to the detached mouthpiece of an oboe or the reed of a child's toy trumpet, which deliver a note of constant frequency and are unaffected by the density of ambient gas.

The pitch of the note is determined by the mass and elastic properties of the solid structures set into oscillation and is not affected by the length of the column of gas in the adjoining airway. It is therefore incorrect to attribute high-pitched wheezing to short peripheral airways and low-pitched wheezing to long central bronchi. While it is true that the oscillations of a large mass, such as a tumor occluding a main bronchus, usually produces a low-pitched note, high-pitched wheezing may be generated centrally or peripherally, depending on the tightness of the stenosis. As in the experiments mentioned earlier, the pitch may rise or fall during a single respiratory cycle in parallel with variations in the tightness of compression.

**Monophonic Wheezes**

A simple example of the model of an uncoupled oscillating reed, applied to the lung, is a bronchus almost completely occluded by a tumor. The sound produced by such a stenosis is a single musical note, in other words, a monophonic wheeze. When the stenosis is rigid, the wheeze is audible throughout the respiratory cycle; when the stenosis is flexible, small differences in transmural pressure may silence the wheeze during inspiration or expiration (for example, when the patient is turned from one side to the other). For the same reason the pitch of the wheeze may be constant or vary between inspiration and expiration.

Single or multiple monophonic wheezes are a characteristic clinical sign of asthma (Fig 2A). The number of wheezes is never large; the illusion that innumerable wheezes are heard wherever the stethoscope is applied is due to the wide transmission of a few loud wheezes with varying relative intensity to different points on the chest wall. No conclusion can be drawn from the pitch concerning the source of the wheeze. The high-pitched wheezing in severe asthma is not necessarily due to peripheral bronchial spasm; it is often a sign of tight expiratory dynamic compression.

In asbestosis, fibrosing alveolitis, and other varieties of diffuse interstitial fibrosis, and uncommon type of monophonic wheezing is sometimes heard over the lower zones of the lung. It is confined to inspiration and is often associated with late inspiratory crackling. In some patients, there is a single short wheeze at the very end of inspiration; in others, several short wheezes of different pitch follow one another throughout inspiration.

The observation that late inspiratory crackles and
The polyphonic wheeze is produced by dynamic compression of the central bronchi. This flow-limiting mechanism is brought into play by transmural pressure gradients which force the opposite walls of several central bronchi into apposition, creating a set of self-regulating valves. These bronchi oscillate near the point of closure and generate a cluster of musical notes. The area of compression lies just downstream of the point where the intrabronchial and extrabronchial pressures are equal, usually in the lobar or segmental bronchi. Towards the end of expiration, the equal-pressure point migrates towards the periphery of the bronchial tree. The end-expiratory rise in the pitch of the polyphonic wheeze is probably due to this shift of the oscillating segment of the airway from the massive central bronchi to the flimsy peripheral airways.

The clinical significance of the expiratory polyphonic wheeze as a reliable sign of widespread obstruction to airflow is impaired to some extent by the fact that such wheezing can also be produced by healthy subjects. Admittedly, they can do so only during a violent expiratory effort, while patients with severe obstruction to airflow wheeze even during a gentle expiration. The distinction between physiologic and pathologic expiratory wheezing is still easy in moderately severe obstruction to airflow, when a mildly forced expiration is sufficient to produce a cluster of musical notes; however, in others with slight obstruction, nearly as much effort is required as in healthy subjects to generate this wheeze. The significance of the polyphonic wheeze is then ambiguous.

The difficulty can often be resolved by observing the sounds generated by increasingly forceful expirations. In healthy subjects, these are accompanied by progressively louder breath sounds, which retain their character as a white noise until an expiration, delivered with maximal force, suddenly evokes the full complement of musical sounds contained in the polyphonic wheeze. The explanation of this sudden transition from white noise to polyphonic wheezing is that in a lung with uniform mechanical properties, dynamic compression of all of the flow-limiting bronchi occurs simultaneously. By contrast, in diseased lungs with nonuniform mechanical properties, dynamic compression occurs earlier in expiration and at a lesser expiratory effort in territories where pulmonary compliance and peripheral airway resistance is high. An increased expiratory effort produces dynamic compression first in more severely damaged territories of the lung, and this process continues until the expiratory pressure is sufficient to compress all of the central airways. The acoustic sign corresponding to this

**Polyphonic Wheezes**

Polyphonic wheezing is a common sign of all varieties of chronic obstructive pulmonary disease. Such wheezing is confined to expiration and consists of several harmonically unrelated musical notes, starting and ending simultaneously, like a dissonant chord (Fig 2B). This timing distinguishes polyphonic wheezing from multiple monophonic wheezes, which may be inspiratory or expiratory or both, with the component notes starting and ending at different times.
progressive recruitment of compressed airways is a sequence of musical sounds, starting with a monophonic wheeze at a mildly forced expiration, followed at increased effort by bitonal and multiple wheezes, and eventually followed by the full polyphonic wheeze. Such a series of increasingly complex musical sounds cannot be reproduced by healthy subjects.

Crackles (Rales; Crepitations)

These sounds are generally attributed to bubbling of secretions in the airways. Some low-pitched interrupted musical sounds are undoubtedly generated in this way (for example, in comatose or moribund patients who are too weak to cough). In other diseases where there is no sputum, and when crackling is confined to inspiration, this explanation cannot be true.

Observations on excised lungs have shown that inflation is not accompanied by crackling unless the lung is deflated at the start of the experiment to the point where some groups of alveoli under the pleura are seen to be airless. In these circumstances the lung does not inflate smoothly or evenly, but small groups of alveoli expand suddenly all over the surface. This random piecemeal reinflation is accompanied by crackling, which ceases as soon as the whole lung is re-aerated. The crackling is due to the explosive equalization of pressure that follows the sudden removal of a barrier separating two compartments containing gas at widely different pressures. The barrier in these experiments is a peripheral airway which remains closed until its resistance is overcome by the blowing pressure. The crackling of surgical emphysema under finger pressure is generated by the same mechanism, only here the barrier separating adjoining bubbles of subcutaneous gas is a flimsy layer of connective tissue, which gives way when the gas pressure rises under the palpating finger.

Late Inspiratory Crackling

The crackling heard towards the end of inspiration in diffuse pulmonary fibrosis and other diseases characterized by pulmonary deflation is generated by this mechanism. In deflated territories of the lung, the peripheral airways remain closed until a late stage of inspiration. By then the gas contained in the alveoli supplied by these airways is considerably below atmospheric pressure. When the airway eventually reopens, the sudden equalization of pressure is accompanied by an explosive sound. The profuse late inspiratory crackling over the base of these lungs is the noise of miniature explosions coinciding with the sequential reopening of many peripheral airways.

A recurrent rhythm characterized by similar spacing and relative loudness of successive crackles can often be recognized in several consecutive respiratory cycles (Fig 3). This is an important clue to the genesis of crackling. The recurrent rhythm indicates that the airways responsible for individual crackles open in the same sequence and at approximately the same pulmonary volume. The inference that the reopening of each airway (and, therefore, the timing of each crackle) is closely linked to the transpulmonary pressure was confirmed by simultaneous recording of sound and esophageal pressure. These observations showed that crackles, identified by their spacing and relative loudness, occurred at the same esophageal pressure in several consecutive respiratory cycles.

Deflation of the basal territories in fibrosing alveolitis, diffuse interstitial fibrosis, and interstitial edema is due to a combination of increased elastic recoil and compression by the weight of the overlying lung. Removal of this weight by bending forward or by turning a recumbent patient from side

![Image](http://journal.publications.chestnet.org/pdffaccess.ashx?url=/data/journals/chest/21004/ on 06/26/2017)

Figure 3. Recurrent pattern of late inspiratory crackles in fibrosing alveolitis in three consecutive respiratory cycles.
to side is sometimes sufficient to expand the basal territories to a volume where the airways open in early inspiration. In relatively slight pulmonary deflation the effect of gravity is critical, and its removal is followed by sudden or gradual extinction of crackling. In more severely deflated lungs the crackling is unaffected by changes of posture.

Early Inspiratory Crackling

Crackling at the beginning of inspiration is a common sign of chronic obstructive pulmonary diseases, particularly chronic bronchitis. In contrast to late inspiratory crackles, these sounds are low pitched, scanty, audible at the mouth, as well as over the lower lobes, and cannot be extinguished by changes of posture. Early inspiratory crackles are often associated with similar late expiratory crackles. Another difference is that unlike the random sequence of inspiratory crackling in deflated lungs, this early inspiratory variety of crackling often contains short sequences of equal spacing and loudness.

Early inspiratory crackles and the associated late expiratory crackles are generated by the passage of boluses of gas through an intermittently occluded airway. In this respect, these crackles resemble the familiar gastrointestinal sounds of borborygmi, belching, and the passing of flatus. In these examples the passage of gas is interrupted by a lightly closed segment of the intestine which opens intermittently whenever the upstream gas pressure is sufficient to force a passage. The transit of each bolus is accompanied by an explosive noise, generated by the sudden equalization of upstream and downstream gas pressures.

The site and mechanism of interruption of the flow of gases in chronic bronchitis are uncertain. Transmission of the crackling to the lower ribs at the back and to the mouth suggests that the source of crackling is in the lower lobe bronchus or one of its segmental branches. The cause of the obstruction may be viscid mucus, a fold of mucosa, or a poorly supported bronchial wall. When coughing fails to silence these crackles, obstruction by mucus is unlikely. The timing of the crackles indicates that the mechanism of obstruction operates only in early inspiration and late expiration, when the lung volume is near functional residual capacity.

References


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