

State of the Art

Lung Sounds^{1,2}

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Introduction

From ancient times limited observations on lung sounds had been made by listening at a distance from patients to such phenomena as wheezing or stridor. Direct application of the ear to the chest increased the number of types of sounds that could be related to diseases. The science of auscultation, however, began with the invention of the stethoscope by Laënnec. This instrument allowed him to describe the major categories of lung sounds that form the basis of the modern classification. His systematic and thorough clinicopathologic correlation of these sounds was a remarkable achievement. It has been argued that he revolutionized the practice of medicine by "altering both the physician's perception of disease and his relation to the patient. In particular, the stethoscope drew the physician into the private world in which signs were directly communicated to him from the patient's body" (1). At the beginning of the 19th century, diagnoses were made principally on the patient's verbal account of his illness and observations with the unaided senses. The stethoscope reoriented physicians to methods for more objective diagnoses, and it paradoxically fell into disrepute for precisely this reason. The

SUMMARY A resurgence of interest in clinical auscultation is in progress. Recent technical advances permit more objective observations than were previously possible. Quantified lung sounds are being correlated with clinical, physiologic, and roentgenologic information. The purpose of this article is to summarize current knowledge on the production, transmission, and clinical significance of lung sounds. The complex sonic signals arising in the lung during respiration reflect its mechanical state. More precise understanding of pulmonary sounds and their clinical correlations could lead to powerful diagnostic tools, which have particular appeal because of their noninvasive nature.

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clear superiority of the chest roentgenogram in diagnosing early pulmonary tuberculosis and lung cancer led to views by prominent chest physicians such as:

The stethoscope is largely a decorative instrument insofar as its value in diagnosis of pulmonary diseases is concerned. Nevertheless, it occupies an important place in the art of medicine. Apprehensive patients with functional complaints are often relieved as soon as they feel the chest piece on their pectoral muscles (2).

Reviews of the literature on lung sounds (3, 4) reveal few fundamental advances in stethoscopes since Laënnec's invention. Nevertheless, clinicians regularly listen to lung sounds, with the recognition that auscultation is subject to a variety of limitations. These include

nomenclatural difficulties, observer variability, inadequate understanding of the basic mechanisms of production of the sounds, and the lack of adequate studies of clinical and physiologic correlations of the sounds themselves. We shall review these in sequence, and show that recent technical advances and research efforts provide the means whereby these limitations may be circumvented. This should help not only in the

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development of new diagnostic and monitoring devices, but also in permitting more precise use of the stethoscope for lung auscultation.

The current status of lung sound nomenclature is best viewed in terms of its history. Laënnec noted that the sounds heard with his "cylinder" were easier to distinguish than to describe (5). He chose familiar sounds to clarify the distinguishing characteristics of lung sounds. For example, his "râle humide ou crepitation" was similar, he said, to the crepitation of salts in a heated dish or the noise emitted by healthy lung when compressed in the hand. His rhonchus or "râle sec sonore ou ronflement" was a snoring noise like the bass note of a musical instrument or the cooing of a wood pigeon. Laënnec's careful classification was modified by later writers. He had used the word rhonchus as the equivalent of rate to avoid saying the word "rate" in the presence of patients, as it might be interpreted as "death rattle." In an English translation of Laënnec's work, the words rhonchus and râle were used differently, introducing confusion that has persisted for more than 150 yr. Examination of current medical literature reveals that different terms are used for the same lung sounds, and the same name is applied to different sounds. An entertaining critique of the sorry state of lung sound terminology was provided by Robertson (6). Bunin and Loudon (7) tabulated terms used to describe lung sounds in published case reports. They reviewed a total of 663 case reports in 7 English language journals. The word "crepitations" in British journals seemed to be virtually equiva-

lent to the American "râles" both by the relative frequency of use and the relative proportion of râles and crepitations described by the qualifying adjectives fine, medium, or coarse. Similar adjectives, such as "wet," "dry" and "crackling," were noted to be applied to both terms, further supporting the idea that they are equivalent. Apparently authors use a variety of qualifying adjectives for lung sounds, despite the lack of precise definitions of the terms or clear understanding of their clinical meaning. They concluded that terminology should be standardized to improve communication. A Committee of the International Lung Sounds Association met in 1976 to attempt to reduce the confusion in terms. Acoustic analysis of sound from tape recordings made by a variety of investigators led to the preliminary formulation presented in table 1 and figure 1, adopted by the American Thoracic Society in 1977 (8). The separation of adventitious sounds into "continuous" and "discontinuous" is used, with further differentiation of these 2 major classes of sounds. The word "crackle" was recommended in place of "râle" because of the multiple and often disparate meanings attached to the latter term. The work of Holford (9) provided an objective basis for using the qualitative adjectives "fine" and "coarse" for discontinuous sounds, as those so distinguished by ear showed significantly different measurements made on time-expanded waveforms of individual crackles (table 1). This was subsequently confirmed by Matsuzaki and coworkers (10). Many observers use the word "medium" to qualify crackles, but

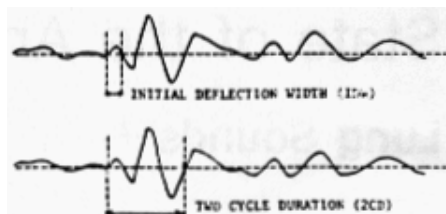


Fig. 1. The IDW or initial deflection width is the time in ms of the first deflection of an identifiable crackle above or below the baseline. The 2CD is the time in ms for 2 S-shaped waves or cycles to occur.

a clear demonstration of the objective features of discontinuous sounds that would allow differentiation of such sounds from "fine" and "coarse" has yet to be reported. The discontinuous sounds associated with secretions in the airways need to be more precisely defined so that they can be separated (if possible) from the other sounds in table 1. Most such sounds can be classified as coarse crackles, but they are also commonly admixed with low-pitched continuous sounds. It may be that the classification will require not only the description of the quality of the individual crackle but also the timing of its occurrence in the respiratory cycle, the number of crackles per breath, the location on the thorax, and other features yet to be described. Correlations with pathophysiologic events will have only limited clinical usefulness until this is done.

Observer Variability

Observer variability has been studied in terms of the ability of different observers to agree on specific diagnoses or specific physical findings. There are also some studies that record lung sounds objectively and compare them with other parameters, but these will be discussed separately.

Hudson and coworkers (11) found that the qualitative descriptors "fine," "medium," "coarse," "moist," and "dry" in examinations of 100 consecutive patients admitted to a medical service in the United States were not useful in distinguishing between disease categories, such as congestive heart failure, chronic lung disease, and pneumonia. No doubt some of the difficulties of observer variability reflect nomenclature problems. Observers in the study *cf* Hudson and coworkers agreed on qualitative adjectives to describe crackles in less than half the patients. Most of the few other studies of observer variability in terms of specific diagnosis have shown considerable disagreement. For example, in

TABLE 1
CLASSIFICATION OF COMMON LUNG SOUNDS

Acoustic Characteristics*	American Thoracic Society Nomenclature	Common Synonyms	Laënnec's Original Terms
Discontinuous, interrupted explosive sounds Loud, low in pitch; Average values: IDW = 1.25, 2CD = 9.32	Coarse crackle	Coarse râle	Râle muquex ou gargouillement
Discontinuous, interrupted explosive sounds Less loud than above and of shorter duration; higher in pitch than coarse crackles or râles; average values, IDW = 0.92, 2CD = 6.02	Fine crackle	Fine râle, crepitation	Râle humide ou crepitation
Continuous sounds longer than 250 ms, high pitched; dominant frequency of 400 Hz or more, a hissing sound	Wheeze	Sibilant rhonchus	Râle sibilant sec ou sifflement
Continuous sounds longer than 250 ms, low pitched; dominant frequency about 200 Hz or less, a snoring sound	Rhonchus	Sonorous rhonchus	Râle sec sonore ou ronflement

Definition of abbreviations: IDW = initial deflection width; 2CD = two cycle duration.

* See figure 1 for waveforms.

1952, Fletcher (12) reported marked variation and error by specialists attempting to detect emphysema. Studies focusing on specific physical findings have also shown considerable disagreement. Schilling and associates (13) reported that 2 observers disagreed 24% of the time about the presence of abnormal sounds in 187 cotton workers. This study was done by relatively inexperienced observers, but mean observer error for adventitious sounds and other physical signs was similar to that of the more experienced physicians reported by Fletcher (12), as was the interobserver variability among 9 observers in assessing râles and rhonchi in 20 patients reported by Smyllie and coworkers (14). The mean variability in the study of Schneider and Anderson (15) was 29% for the presence of decreased breath sounds. Such variability appears unacceptable for diagnostic and therapeutic decisions, but it is about the same as that found in clinical data collection in general (16), including chest roentgenographic interpretation (17). Fortunately, physicians rarely use single observations such as these in clinical decision-making.

Origin of Sounds

Precise localization of the sources of lung sounds has been impaired by lack of knowledge of the degree to which they are veiled by unknown transmission effects. Although much speculation exists, this review will focus on the few investigations that have been done to validate hypotheses concerning the origin and transmission of normal breath sounds and major categories of adventitious sounds.

Normal Breath Sounds

Laënnec believed that normal breath sounds were caused by friction of air against the lining of the airways. Skoda (18) agreed with Laënnec, attributing vesicular sounds to the friction of air against the walls of the finer bronchial tubes and the air cells. On the other hand, Beau (19) suggested that the sounds were produced by movement of air at the back of the mouth and transmitted to the chest wall, being modified by passage through the airways and lungs. Bushnell (20), 90 yr after Beau, suggested that the sounds were produced at the larynx; this opinion is still expressed in several textbooks of physical diagnosis (21, 22). Some observations made in unusual clinical situations

have provided evidence that the vesicular sound is at least in part produced locally.

deficiency in Bullar's observations, but his experimental findings still have considerable

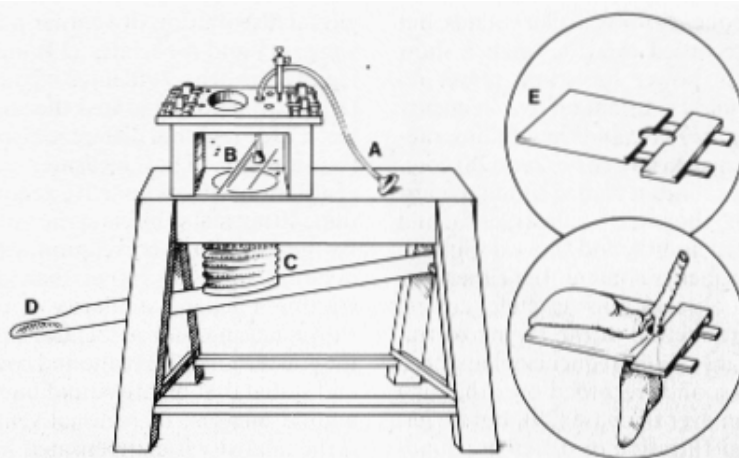


Fig. 2. Artificial thorax for studying lung sounds (Bullar 1884). A flexible stethoscope (A) allowed auscultation of sheep or calf lungs placed in a chamber (B). The lungs could be inflated or deflated by a bellows (C) operated manually by handle (D). The chamber was made airtight using a sliding frame (E). When the left lung was placed in the chamber and the attached right lung was outside, vesicular sounds were heard over the ventilated left lung, while bronchial sounds were heard over the unventilated right lung. With the trachea plugged the sounds in the right lung became vesicular (see text).

Sahli (23) observed vesicular sounds over herniated lung as it inflated with the glottis closed during a Valsalva maneuver. Fahr (24) noted that vesicular sounds are present in laryngectomized patients.

In 1884, Bullar (25) reported a sequence of experiments designed to determine the origin of breath sounds. He used an airtight, rigid box with a bellows attached to ventilate fresh calf or sheep lungs while listening with a stethoscope to sounds produced. One of the pair of lungs, both still attached to the trachea, was placed inside the box and the other lay on top (figure 2). A flexible stethoscope was used to auscultate either lung. The lungs could be ventilated via the trachea, or by the bellows, with the trachea open or plugged. With the trachea open, operation of the bellows ventilated the lung in the box; the lung outside the box lay still and collapsed. When the trachea was plugged, air moved from one lung to the other with no air movement in the trachea. Variations in the loudness or nature of sounds were noted, depending on the method of ventilation. Bullar concluded that sounds were produced at those parts of the respiratory tract where the air passes from a narrower to a wider space, that during expiration sounds were produced at the glottis only, but that during inspiration additional sounds were produced in the lung itself. The absence of numerical expression of results, inevitable in the absence of quantifying instruments, may seem to the modern reader to reflect a

relevance. He was one of the first to adopt an experimental approach instead of relying on intuition or on previous pronouncements.

Another early experimental study of interest was reported by Hannon and Lyman in 1929 (26). They wished to resolve the controversy between those who felt that breath sounds were tracheal in origin and those who believed that they were produced more peripherally. Using a graphic recording method involving a microphone, filters, and a string oscilloscope, they measured the effect on tracheal sounds of transmission through sheep's lung placed over the subject's trachea at varying degrees of inflation. Sounds recorded directly from the subject's chest wall had a definite but low intensity signal passed by the 130-Hz low pass filter, whereas tracheal sounds transmitted through the sheep's lung had none. They concluded that movement of air in and out of the lung was necessary to effect the transmission of vibrations of low frequency.

Technical advances in sound recording and sound analysis have allowed measurements and comparisons of sound intensity and frequency content to be made more accurately. Gavriely and associates (27) showed that the spectrum of sound recorded over the trachea differs from the spectrum of sound recorded at the chest wall in normal subjects. Power declines exponentially with increasing

sound frequency for vesicular sounds, but sounds recorded over the trachea show a different power spectrum, power remaining fairly constant up to a frequency averaging 920 Hz, and then falling rapidly. Druzgalski and coworkers (28) compared the sounds recorded simultaneously over the thorax, over the trachea, and at the open mouth, and showed similarities of frequency content. In neither case did the observations exclude central sound production at the larynx or trachea. Higher peak frequencies have been noted in sounds recorded over the lung apex than over the base (29), but different sound-filtration or different sound-producing characteristics could explain these observations equally well.

Gavriely (30) subsequently studied sound generation and transmission by measuring the transfer function and coherence between sound signals recorded at the trachea and at the lung surface in an inflated, dried, dog lung model. His observations suggest that during expiration the surface sounds are derived from tracheal sound, but that during inspiration the surface sounds are generated closer to the lung surface.

Sound waves recorded from 2 separate sites on the chest wall a few centimeters apart are likely to be relatively similar if produced at a distant common source, and less so if produced locally, near each microphone, by separate sound sources. This argument is used by Kraman (31), who derived a "subtraction intensity index" or "cancellation index," the ratio of the subtraction signal amplitude to the addition signal amplitude for 2 sound signals recorded from adjacent chest wall sites. When the subject said "Ee" cancellation was nearly 100%; in a patient with a pleural friction rub it was less than 50%. He concluded that the inspiratory phase of vesicular sound is produced in the lung, not in the upper airways, not in the alveoli, but somewhere between, and that the expiratory phase is produced more centrally than the inspiratory phase. He also found, in a group of 23 subjects with varying degrees of air-flow obstruction, that the site of generation of inspiratory vesicular sounds seems to move towards the chest wall with increasing degrees of generalized air-flow obstruction.

Breath sound intensity at different chest wall sites was related to lung volume, air-flow rates, and body position in 8 normal subjects by LeBlanc and coworkers (32) using a microphone and phonocardiograph. The regional intensity of normal breath sounds varied in ways characteristic of the variation in re-

gional distribution of ventilation. Ploysongsang and associates (33) measured regional breath sound intensity in a similar way, but compensated the sound intensity for regional differences in sound transmission. They measured regional ventilation with radioactive xenon in the same 10 normal subjects at the same time. By measuring the correlation within individual subjects, rather than demonstrating a general similarity of pattern, Ploysongsang and associates amended the conclusion of LeBlanc and coworkers and stated that breath sound intensity is a good measure of regional ventilation if the intensity is compensated for local differences in sound transmission.

Correlation between regional breath sounds and regional ventilation favors regional production of vesicular breath sounds; if the inspiratory vesicular breath sounds were produced centrally, one would have to postulate that sound transmission to the periphery of the lung correlates with gas transport to the periphery of the lung. It would seem more likely that regional production of sound rather than regional transmission of sound would depend on regional ventilation.

Bronchial Sounds

The tracheal or bronchial breath sounds heard by listening over the large airways have a different character from vesicular sounds. The sound has a hollow or "tubular" character. Laënnec said that the sound clearly showed that the air was passing through a roomier channel than the air cells. In the early part of this century, investigators in Germany and in the United States concerned themselves with objective methods for distinguishing the frequency content of bronchial and vesicular sounds because of the importance of the bronchial sound in indicating pulmonary consolidation. Mueller graphically demonstrated that the bronchial sound contained higher frequency components than vesicular sounds. Using models made with rubber tubes as well as observations on humans, Martini and Mueller (34) provided evidence that the site of bronchial breath sounds was in airways 4 mm or larger in diameter. They concluded that the presence of bronchial sounds in abnormal locations signified a continuous infiltration of lung tissue from the periphery 3 to 5 cm inward toward the hilum, depending on body size and location on the chest wall. Fahr (24) confirmed these observations, and demonstrated that the presence of the larynx was un-

necessary for the generation of the bronchial sound.

Bronchial breath sounds heard over consolidated lung almost certainly represent improved transmission to the periphery of sound produced in the large airways. Consolidated lung acts like a continuous acoustically conducting medium that does not attenuate the transmission of sound, as does inflated lung. It may be presumed that the physical finding of bronchial breathing over consolidated lung represents the replacement of locally produced vesicular sound (abolished by consolidation and consequent absence of local ventilation) by sound transmitted from a more central sound generator. The sound over consolidated lung may differ qualitatively from that heard over the trachea, likely because of the differences in the filtering characteristics of the intervening tissues, but the specific differences have not been delineated.

Breath sounds intermediate in characteristics between vesicular and bronchial sounds are called bronchovesicular. Presumably the mechanisms responsible for their production represent a combination of the mechanisms discussed above. In healthy persons, they likely represent a combination of sounds arriving at the site of auscultation from different sources. In disease, partial change in filtration characteristics may result from partial consolidation also producing sound with frequency characteristics intermediate between bronchial and vesicular. The variations in the frequency characteristics of the 2 conditions have not been clarified.

Although it seems probable that bronchial and vesicular sounds each arise from a range of airways rather than from a point source, the extent to which these ranges may overlap, or the generations or dimension of airways involved, have not been precisely defined. Unfortunately, at this time the terms "bronchial" and "bronchovesicular" refer to tubular sounds with along, loud expiratory phase independent of whether they arise in normal lung or are due to increased transmission of centrally generated sound through peripheral consolidation. Work on the subject is now receiving more attention after a half century of emphasis on roentgenographic rather than auscultatory diagnosis.

Continuous Adventitious Lung Sounds

"Adventitious" lung sounds are superimposed on the breath sounds in certain

circumstances and usually indicate disease.

Wheezes, or continuous adventitious lung sounds, are usually louder than the underlying breath sounds. The word "continuous" in this context implies a duration of more than 250 ms rather than meaning a sound that continues throughout the respiratory cycle. Several wheezes may be present simultaneously. They are often audible at the patient's open mouth, or by auscultation over the larynx, and may, for example in a patient with asthma, be audible to the unaided ear at some distance from the patient.

The mechanism producing wheezing sounds in the intrathoracic airways appears to involve the airway walls interacting with the gas moving through the airway. Forgacs (3) states that these sounds are produced when the caliber of the airways is narrowed to the point where its opposite walls touch one another, and he proposed that the best model among musical instruments is the toy trumpet with a metal reed whose vibration is responsible for the sound production and whose horn is purely decorative. By experimenting with excised bronchi, he found that high-pitched musical sounds could be generated in lightly compressed airways of any size. The pitch of the wheeze indicated the mass, the elasticity, and, above all, the flow velocity through an airway on the point of closure, but not its length, caliber, or mechanism of closure.

Recent efforts have been directed toward a more fundamental understanding of wheezing. Mathematical models representing flow of an inviscid and incompressible fluid through a two-dimensional channel with flexible walls have been developed by Grotberg and Davis (35). Oscillation can be predicted by the models in certain circumstances and related to structural and physiologic variables. These investigators tabulated the clinical situations in which parameter changes occur that are likely to be associated with stable and with unstable oscillations. The mathematical models are accepted as being incomplete, but have characteristics that could serve as models for sound-producing and for flow-limiting conditions. Features of interest that have been claimed for models proposed by Grotberg and Davis (35) and Grotberg and Reiss (36, 37) include an expectation that wheezing will always be accompanied by flow limitation, but that flow limitation will not

necessarily be accompanied by wheezing, an explanation for a transient rise in pitch

towards the end of expiration, and a possible means for determining which airway generation is wheezing, based on pitch. Although such mathematical formulations may appear abstruse to clinicians, they are necessary to quantify the information so that pathophysiologic causes of changes in sound can be understood.

The transmission of wheezing sounds through airways is better than transmission through lung to the surface of the chest wall. Akasaka and associates (38) recorded sounds simultaneously with an intrabronchial and a chest wall microphone, and showed reduction in the chest wall high-frequency components when compared with endobronchial sounds. Takezawa and coworkers (39) recorded wheezing sounds simultaneously at several surface sites in asthmatic patients, and also found that higher frequency sounds (higher than 1,000 Hz) were more clearly detected over the trachea than at the chest wall (39). These studies emphasize the importance of listening over the trachea in asthmatic patients. Circumstances suitable for the production of continuous adventitious lung sounds are encountered in narrowing of airway caliber caused by bronchospasm, by mucosal thickening or edema, by the presence of a tumor, foreign body, or secretions, or by pressure from without by a tumor mass or dynamic airways compression. The last of these can be used by most healthy subjects to produce wheeze during a forced expiratory maneuver.

Discontinuous Adventitious Lung Sounds
Adventitious lung sounds with individual components lasting for less than 20 ms are heard as a series of brief explosive sounds. Theories on the mechanisms that produce these sounds are so far supported only by indirect evidence. Crackles are probably produced by more than one mechanism. Two different mechanisms are generally regarded as probable: one is the sudden opening of a succession of small *airways* with rapid equalization of pressures causing a sequence of implosive sound waves, and the other is the bubbling of air through secretions. The first of these 2 mechanisms is the likely cause of the crackles heard in patients with interstitial lung disease, such as fibrosing alveolitis or asbestosis; it is probably also the cause of the crackles heard in congestive heart failure, at least in its early stages. The sounds heard in patients with secretions in their airways range from the coarse bubbling sounds heard in patients who have copious

secretions in their larger airways - approximating the "râle gargouillement" of Laënnec or "death rattle"—to the sounds heard in patients with pulmonary edema or with obstructive lung disease.

Crackles generated by the sudden opening of a previously closed airway may be considered as arising at a point source. Whether the sound arises from the sudden equalization of gas pressures when the airway opens, or with the release of tissue tensions is not certain; either or both could theoretically produce the sounds heard. A theory of sound transmission called the stress relaxation quadrupole model has been described by Fredberg and Holford (40). According to this theory sound waves radiate from the point source in 4 directions or poles rather than in an expanding sphere. Initial observations on the waveforms of lung sounds of patients are consistent with this theory (9, 40). The waveform shapes predicted by this model varied with airway size, with those predicted for larger airways corresponding to those observed with coarser crackles.

As airway closure occurs at low lung volumes in normal subjects, crackles caused by opening of previously closed airways might be anticipated. Careful auscultation at the bases during inspiration from residual volume in a group of 56 young nursing students revealed their presence in 63%; they could be detected in 92% when an electronic stethoscope with high-pass filtration was used (41). They were heard only rarely when the subjects breathed at tidal volume. Ploysongsang and Schonfeld (42) studied the frequency with which crackles were heard in a group of normal subjects who breathed air and then oxygen at a low lung volume, and compared the profusion of crackles heard in the immediately subsequent inspiration with the reduction in lung volume and displacement of the quasi-static lung compliance curve, both of which measure airway closure. They found a close correlation between the measured loss of lung volume with low lung volume and the increase in transpulmonary pressure at 25 and 50% of vital capacity, and a significant correlation between these values and the amount of crackles (scored rather than counted) heard at the right base posteriorly during a subsequent inspiration. Crackles were heard regularly at the bases, but very rarely over the upper zone, and they were heard late in inspiration, when a lung vol

ume of more than 50% of vital capacity (usually between 65 and 85%) had been achieved. More reduction in lung volume and more crackles appeared with oxygen than with air breathing at low lung volumes, confirming the findings of Burger and Macklem (43) relating to airway closure, and suggesting that reopening of distal atelectatic lung units generates crackles, and that the more units there are collapsed, the more crackles are generated.

It seems probable that this same mechanism could account for the crackles heard in patients with interstitial lung disease associated with decreased pulmonary compliance and reduced lung volume. Nath and Capel (44,45) have shown in these patients that specific crackles that can be recognized as recurring repetitively from breath to breath, occur at the same lung volume or transthoracic pressure. It would be difficult to ascribe their observations to a mechanism other than one involving tissue forces.

Some light is shed on the site at which crackles of this type are produced by studies of their acoustic waveform, and by recording sounds simultaneously at more than one site. Kunica (46) recorded simultaneously at 2 sites 6 cm apart and at 2 sites 12 cm apart. When the crackles were classified as "coarse" or "fine" by consideration of their waveform, they showed a wider distribution for coarse than for fine crackles. Coarse crackles appeared at 2 sites 6 cm apart in 86% of cases, fine crackles in 22%; the corresponding percentages for sites 12 cm apart were 60 and 6%. Their observations provide evidence that coarse crackles may originate in larger airways than may fine crackles.

Instruments

Lung sounds result from the vibrations within the lung and its airways that are transmitted to the chest wall. Vibration amplitude may be less than 10 μm and is affected by the method by which it is detected or transduced. Microphones employed have included the piezoelectric, condenser, or capacitor and electret types. Both the type of chest piece (or microphone) and the manner of coupling to the chest wall will influence the signal. Mechanical loading by a massive, rigidly applied microphone will limit surface motion and increase surface stress.

The electrical signal is likely to represent a mechanical waveform that combines pressure and displacement. The

compromise achieved between the theoretical limits of acceleration or displacement without loading, or surface stress without movement, will affect the output signal and may, to a lesser extent, influence the input mechanical signal. Impedance matching between the chest wall and the microphone has until recently been empirical, but has been the subject of recent theoretical work by several groups (47). The electret microphone is tending to replace the earlier crystal and capacitor types, being lighter and less expensive. The application of the microphone to the chest wall usually entails a housing with desirable attributes, including stability, freedom from surface noise, exclusion of ambient sound, comfort, and good acoustic coupling. Air coupling is commonly used, and the dimensions of the air space influence the frequency response of the system.

The signal from the lung can also be amplified, recorded, and analyzed by a variety of methods. Since the invention of the stethoscope, understanding of this signal has been affected by the availability of instrumentation to perform these operations, as well as, of course, interest in and availability of other means for medical diagnosis. In any case, lung sounds were transduced to the ear by Laënnec's cylinder more efficiently than by the ancient practice of direct auscultation. Improvements on the simple wooden instrument were accepted slowly. The binaural stethoscope suggested by Comyns (1) was popularized in the United States by Camman (48). Physicians accustomed to listening with one ear complained that "they heard too much." Magnification of lung sounds was of little value except to physicians with hearing problems. This was in part due to poor signal-to-noise ratios of early amplifiers and to the lack of known clinical correlations of the additional sound, a persisting problem. Tape recordings have been useful in teaching and in clarifying nomenclature, and more recently have allowed storage of sounds for subsequent analysis. They have not been commonly used in documenting physical findings because the procedure is too cumbersome to be of practical value.

To record a signal and reproduce it for teaching or reference purposes requires no more than an audio magnetic tape recorder with its associated amplifiers and, perhaps, some filtration capacity or tone control to emphasize the sounds of interest or reduce unwanted sounds or artefacts. Several investigators (49, 50) have described types of equipment suitable for

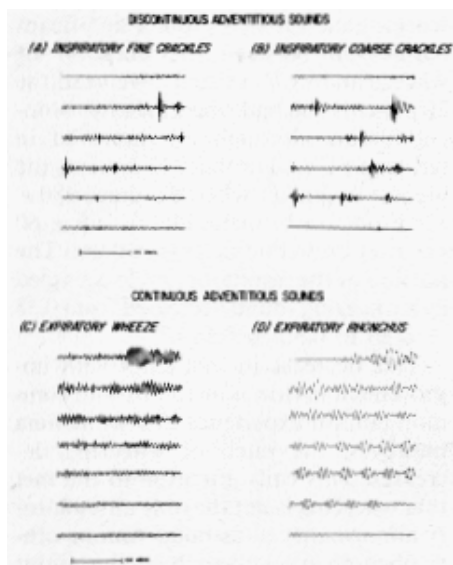
this purpose. The signal-to-noise ratio of lung sounds, particularly vesicular breath sounds, can be sufficiently low that high quality equipment and careful matching of the component parts of a system are necessary.

The area of major interest has been the processing of the sound signal either in the frequency or the time domain. As sound filters became available they were used to distinguish the specific frequency content of various acoustic phenomena (34, 51—53). Using the sound spectrograph developed by the Bell Telephone Laboratories, McKusick and coworkers (54) displayed frequency characteristics of a variety of lung sounds. Numerous other workers employed devices of this type (5 5—59). Despite a variety of claims for the value of this phonopneumography, it never enjoyed clinical acceptance. The development and widespread application of the Fast Fourier Transform (FFT) has made frequency analysis of sounds simpler, faster, more accurate, and less expensive, and renewed interest in its application is now apparent. Analysis of sound in the time domain was also inhibited by the capability of available instrumentation (60). Forgacs (61) called attention to the difficulty in studying crackles because they followed one another too rapidly, and that pen recordings were too slow to distinguish wheezes from other adventitious sounds. The availability of instrumentation to make time-amplitude plots of 800mm per second or greater has permitted visual differentiation of the common lung sounds. This has been applied to the quantification of crackles in asbestos workers, the validation of performance of auscultation, and has been helpful in solving nomenclature problems (figure 3). Time domain analysis has been more widely applied to the study of discontinuous sounds, whereas frequency domain has been used to study continuous sounds. Many of the recent advances in signal processing potentially applicable to lung sound analysis have yet to be applied.

Clinical Correlations

From the time of Laënnec, clinicians have made observations on the association between lung sounds heard with a stethoscope and various disease entities. We will review some of the more important of these. It is not practical to discuss all of such associations, but we shall begin with the more common chronic lung

Fig. 3. Analysis of common lung sounds in the time domain. Time-expanded waveform (TEW) of the



common adventitious sounds given in table 1 are shown. The presence of crackles is shown in the TEW by intermittent spikes (A, B), whereas rhonchi and wheezes cause relatively longer undulations of the baseline.

diseases, comment on some acute conditions, and conclude with a discussion of some miscellaneous problems, concentrating on those that have been subjected to scientific investigation.

Chronic Bronchitis and Emphysema

Patients with chronic bronchitis are generally observed to have noisy chests because of the frequent occurrence of adventitious sounds. Rhonchi that clear with coughing and end-expiratory wheezes are common. In obstructive diseases in general, the intensity of the vesicular lung sounds (in regions where they are not obscured by adventitious sounds) decreases (15, 62–65). Indeed, Laënnec's use of the stethoscope led him to believe that emphysema was far more common than he had previously suspected, an observation he confirmed at autopsy (5).

A simple bedside technique using lung sounds to detect or assess air-flow obstruction requires only a stopwatch and a stethoscope (66). The patient is asked to take a deep breath and blow it all out as fast as possible. The bell of the stethoscope is placed in the suprasternal notch. The duration of audible expiration (FET_o) is timed to the nearest half-second. If the FET_o is less than 5 s and air flow has stopped, it can be confidently predicted that the FEV₁/VC,% is greater than 60%. Conversely, if the FET_o is greater than 6 s

and air flow

continues, the FEV₁/VC₀ is less than 40%.

Decreased breath sounds ranked first among 14 signs in order of importance for diagnosing emphysema in the study of Schneider and Anderson (15). Pardee and coworkers (65) demonstrated that breath sound intensity (BSI) at the chest was strongly associated with an abnormal forced expiratory volume in one second (FEV₁). Bohanda and associates (64) used the method of Pardee and coworkers to show that BSI correlated also with specific conductance ($r = 0.759$), maximal flow at 50% of vital capacity ($r = 0.790$), and forced expiratory volume to vital capacity ratio ($r = 0.860$). Correlations with lung volumes were significant but weaker; BSI correlated independently with indexes of both air-flow obstruction and lung distention. In view of the simplicity of the method and potential application in situations where lung function studies are not readily available, a brief description of the method is warranted.

Auscultation is performed on seated patients who are asked to perform fast deep inspirations from residual volume to generate as loud a sound as possible while breathing through the mouth. Sound intensity is assessed in the upper anterior zones, midaxillae, and posterior bases bilaterally. The sounds are classed as follows: 0, absent; 1, barely audible; 2, faint but definitely audible; 3, normal; 4, louder than normal. The final BSI score is the sum of the values for each individual zone. A correlation coefficient for BSI of 0.966 was obtained between 2 observers independently examining 20 patients. The method is not intended to replace function studies or detect mild degrees of obstruction. It requires some practice, but it can be used in patients who for one reason or another cannot undergo lung function assessment.

In order to use the method, adventitious sounds that often have a louder intensity than the normal sound must be disregarded. This limits its application somewhat, but in the stable patient an overall estimate of ventilatory capacity can be determined this way. It can fail to detect mild, pure air-flow obstruction, and its predictive power is impaired in subjects with restrictive disorders. In our opinion, additional work needs to be done to unravel all of the factors that are involved in perceived intensity of the vesicular sound. The studies of Kraman (67) are of particular interest in this regard. They show marked regional variation in lung

sound intensity in otherwise healthy persons.

In addition to comparisons of breath sound intensity with flow rates at the mouth in patients with emphysema, more sophisticated studies have been performed. Nairn and Turner-Warwick (63) showed that weak or absent breath sounds assessed by 2 observers were found to be accompanied by poor regional ventilation, as measured by radioactive xenon. The intensity of the recorded vesicular sound when corrected for transmission is believed by Ploysongsang and associates (37) to reflect the volume of ventilated lung under the microphone. Variation in the time-intensity sequence in inspiratory vesicular sounds at separate points on the same horizontal plane has been shown (68) to correlate with frequency dependence of dynamic compliance, another measure of nonhomogeneity of parallel lung segments and a sensitive measure of early obstructive lung disease.

Sound at the mouth has been reported to be decreased in emphysema while being increased in bronchitis and asthma (69). This has been proposed as a method of distinguishing emphysema from the other causes of obstruction, but has not yet been adequately studied.

Discontinuous adventitious sounds (crackles) also are commonly heard in obstructive disease. They usually begin with the onset of inspiration, are few in number, and are generally heard at the lung bases. They persist with changes in posture, and are frequently associated with crackles audible at the mouth (70). The mechanism of their production is poorly understood.

Bronchial Asthma

Common clinical experience as well as review of standard physical diagnosis texts confirm that the most prominent auscultatory feature of bronchial asthma is a high-pitched continuous sound. The current American Thoracic Society nomenclature classifies this sound as a wheeze, but medical literature has to be interpreted with caution, as the word has been used differently. Some, for example, restrict the term wheezing to the continuous sound heard at a distance from the patient, calling the same sound a rhonchus when heard through a stethoscope. In any case, most but not all asthmatics wheeze during exacerbations of their disease. In early stages, wheezing may be heard over central airways during expiration, and it usually can be detected over the entire chest and

in both phases of respiration as the asthma becomes more severe. The absence of wheezing in severe asthma is regarded as an ominous sign, presumably because it results from flow rates so low that wheezes are not generated or from mucous plugging that has severely impaired ventilation. Wheezing is so closely allied with asthma that physicians have to be reminded that other causes exist, such as tumor, foreign bodies, aspiration, pulmonary emboli, congestive heart failure (71). Moreover, wheezing is quite common in the general population. As a symptom perceived by the patient, it had at some time been present in over 25% of a population sample studied by Dodge and Burrows (72). The point prevalence of asthma in this population was 6.6% implying that other causes existed for the remainder of the subjects. Conversely, reversible bronchospasm can occur without audible wheezing (73), and clinical asthma can also present without wheezing, and, therefore, be overlooked (74).

The important point is that the complex relationships between wheezing and other adventitious sounds and airflow obstruction (including bronchospasm, bronchial compliance, mucosal edema, and secretions) are not well understood at this time. The fact that wheezing is so readily and noninvasively detectable, often when no other abnormalities are present, has prompted efforts to quantify this phenomenon.

Proceeding along clinical lines, Forgacs (70) observed that wheezing occurred in apparently normal persons on forced expiration. He said that this could be distinguished from pathologic causes by observing the sounds generated by increasingly forceful expirations. In healthy subjects, these are accompanied by progressively louder breath sounds until an expiration suddenly evokes a polyphonic wheeze. This is attributed to dynamic compression of all the flow-limiting bronchi occurring at one time in a lung with uniform mechanical properties. According to his theory, an increased expiratory effort produces dynamic compression first in more severely damaged territories of diseased lung, and the process continues until the expiratory pressure is sufficient to compress all the central airways. He observed patients with obstructive diseases to exhibit monophonic wheezing with a mildly forced expiration followed at increased efforts by bitonal and multiple wheezes and eventually the full polyphonic wheeze. Systematic study of

a large number of patients with and without airways disease to define the limits of this technique more quantitatively have not yet been reported to our knowledge.

A variety of graphic methods have been employed to study wheezing. Using a sound spectrograph, McKusick (48) displayed features of a wheeze from a patient with bronchial asthma that distinguished this sound from other common auscultatory phenomena. In McKusick's patient, a dominant frequency of 240 Hz was present and a secondary harmonic at 480 Hz consistent with the musical quality of the sound. The development of more modern instrumentation permitted a more precise characterization of wheezing. Wooten and Waring (75) used a spectrum analyzer to show by three-dimensional displays how the frequency content of wheezes varied over the course of the respiratory cycle. This provided a very precise way of quantifying a given wheeze at a particular chest site, and such instrumentation is becoming more widely available. Unfortunately, the number of studies that correlate wheezing with other clinical and physiologic parameters is small. Graphic displays of lung sounds have shown clear differences in time-intensity plots of lung sounds in asthmatics (49, 76), but clinical wheeze scores have been less helpful. In a series of 100 patients in a pulmonary function laboratory, a wheezing score based on the severity of continuous expiratory musical sounds at 4 chest sites during unforced respiration was shown to correlate with severity of obstruction ($r = 0.42$). The highest wheezing scores in this study were, however, uniformly associated with moderate or severe obstruction. Of particular importance is the presence of wheezing-predicted bronchodilator response. Twenty-nine of 48 patients with wheezing, but only 3 of 35 patients without wheezing demonstrated 15% or greater improvement in FEV₁ after bronchodilator inhalation ($p < 0.001$). This correlation was higher in the subset of 16 wheezing patients with a history of asthma ($r = 0.65$) than in the 32 wheezing subjects without such a history ($r = 0.30$). Another approach used a fast fourier transform technique of sound ranging up to 1,000 Hz recorded at several sites over the chest, neck, and mouth, allowing quantification of frequency, duration, number, and intensity of wheezes. In 20 patients during spontaneous asthmatic attacks, there was no correlation between the frequency content of wheezes and the FEV₁, but a significant

correlation between the duration of wheeze and FEV₁, existed. Twelve of the 20 patients still had wheezing after bronchodilator, although all improved in terms of FEV₁. For these 12 patients the highest frequency wheeze average 480 ± 128 Hz before bronchodilator, 298 ± 60 Hz after bronchodilator ($p < 0.06$). The portion of the respiratory cycle occupied by a wheezing sound decreased from 0.58 ± 0.20 to 0.30 ± 0.15 (77).

This decrease in frequency with improvement in flow is in accord with common clinical experience that as asthma improves, the pitch of wheezing decreases. This calls attention to the fact that wheezing is not the only auscultatory abnormality of asthma. Among other phenomena, low-pitched continuous sounds or ronchi are heard in some patients, as are squeaking noises. Forgacs called attention to the increase in the intensity of sound at the mouth in asthma, as mentioned previously. It is somewhat surprising that the full range of sounds and their correlates in such a common illness as bronchial asthma has yet to be described. Knowledge has advanced on air-flow dynamics in large and small airways, flow limitation, equal pressure points, and differences in response to sympathomimetic and parasympatholytic agents, as observed by expiratory flow rates and their gas density dependence. Wheezing and lung sound intensity are likely influenced greatly by such phenomena. The tools are now available to study this.

Interstitial Fibrosis

Usual interstitial pneumonia, fibrosing alveolitis, pulmonary fibrosis. The most striking feature of the chest examination in patients with interstitial fibrosis is the presence of fine crackles. These sounds have a distinctive character and have been described as "close to the ear," cellophane, or Velcro crackles. In instances of slight disease, they are usually confined to end inspiration and are gravity-dependent in that they are best heard at the lung bases with the patient upright, and their distribution can vary with change in position. They may disappear from the posterior lung bases as the patient bends forward. In more advanced stages of the illness, these crackles persist, despite position changes, and are heard at increasingly higher levels above the bases. They may become paninspiratory, often with an end-inspiratory accentuation as the disease pro-

gresses. An end-inspiratory short wheeze beginning with a single crackle may be associated with these sounds (3), presumably caused by air passing through a newly opened but still narrowed airway. As it opens abruptly, it crackles; as it is still narrowed, it wheezes.

Interstitial fibrosis is not the only cause of this type of crackle. The fine crackles of congestive heart failure are difficult to distinguish from those of interstitial fibrosis, but there may be some measurable waveform differences. The differentiation can usually be made on clinical grounds, but a danger exists when diuretic therapy is instituted because crackles caused by interstitial fibrosis may be misinterpreted as being caused by heart failure.

The occurrence of crackles in the pulmonary fibrosis caused by asbestos exposure is of particular interest. As early as 1930, Wood and Gloyne (78) stated that crackles were a common feature of pulmonary asbestosis. Subsequently, reports have varied as to the importance and specificity of crackles. Hunter (79) said they occurred "sometimes," and Wyers (80) said they were "generally" present. Wyers believed that these sounds could be transient in occurrence. Smit hers (81) stated that they were characteristic in their sound and distribution, present *first* at the bases in the midaxillary lines, and tending to spread to the posterior bases. Mitchell and coworkers (82) found crackles more closely related to duration of asbestos exposure than was vital capacity, and recommended that chest auscultation be included as a biologic monitor of the work environment.

Approximately half of the patients with clinical asbestosis have crackles (83—85). In population studies, the prevalences range from about 10 to 20%. Such prevalences depend on the method of auscultation, the age of the population, the severity and duration of exposure, etc. The high frequency in known cases, common occurrence in exposed populations, and increased frequency with increased duration of exposure established their value in monitoring exposed workers. Furthermore, most investigators believe them to be an early sign (86—88). Technicians can be trained to detect these sounds as a screening technique for asbestos-related health effects.

Auscultation has been employed in the study of a variety of other occupationally related chest illnesses. In the study of a detergent-enzyme-induced bronchospasm,

Mitchell and Gandevia (89) noted that with the fall in FEV1 that followed bronchial challenge, sensitized workers developed medium crackles, which he attributed to alveolitis. The descriptions of these sounds, however, appear to be more like what is heard in bronchitis or, perhaps, bronchiolitis. This was an interesting demonstration of an investigational use of auscultatory phenomena that provided otherwise unexpected insights into the pathophysiology of a response to an inhaled agent. The occurrence of adventitious sounds has been of practical value in aiding in the detection of farmer's lung. The initial description of this disease in the English literature by Campbell (90) called attention to the combination of continuous and discontinuous sounds "audible over most of the lungs." In a survey of farmer's lung in the United Kingdom by Staines and For-man (91), who described the clinical picture of 104 cases, 77 had crepitations (crackles), 61 had rhonchi, and 61 had wheezes. The occurrence of numerous crackles in patients with wheezing has helped distinguish farmer's lung from bronchial asthma (92—94) and has suggested that small airways and alveoli were involved, as has been subsequently demonstrated (95, 96). There is little doubt that auscultation performed systematically could aid in monitoring workers exposed to a variety of other potentially hazardous inhalants.

Lung sounds are useful in a variety of other acute and chronic illnesses; congestive heart failure, atelectasis, pneumothorax, displacement of an endotracheal tube into the right main stem bronchus, acute bronchitis in a patient with normal lungs, and foreign body aspiration, to name just a few. We have concentrated on the more common chronic illnesses in our review, as more studies have been directed toward these conditions.

Conclusions

We have been impressed by the overall lack of investigations to provide a scientific foundation for a diagnostic tool that continues to enjoy such common usage. Lung sounds can now be studied objectively. The informational content is rich and likely reflects the pathophysiology of the underlying lungs. We recognize that auscultation may not be the most precise way of studying lung structure and function. Such technical advances as positron emission tomography and nuclear magnetic resonance offer more promise. On the other hand,

auscultation is an approach that is inexpensive, and presents no risk to patients. More accurate knowledge of the clinical correlations of lung sounds can be translated into improved patient management in circumstances where advanced technology is not readily available or accessible. A major reason that lung sounds have not been as useful as they might be is that the amount of information is so great that it overtaxes the clinician's memory. For example, at a single auscultatory site during a single breath, observations may be made on the intensity and duration of each respiratory phase, and also on the presence, timing, profusion, and quality of adventitious sounds. It is possible to make such observations at many sites during many breaths and with differing breathing patterns. Computers provide a tool more suited to handle data of this magnitude. Some applications have already been made. Continuous breath-sound monitoring has been used for detecting apnea in infants at risk of sudden infant death syndrome and monitoring of disturbed sleep. There are potential other applications for monitoring patients in intensive care units. To improve upon these achievements, however, to maximize the potential clinical value, a thorough understanding of the mechanisms of generation and transmission of sound, physiologic factors that affect them, and their clinical correlations is necessary. It will take a concerted multidisciplinary effort on the part of clinicians, engineers, physiologists, psychologists, and other scientists to achieve an adequate understanding of lung sounds that can enhance the clinical use of the stethoscope and provide improved instruments for noninvasive lung diagnosis.

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