

# CHAPTER 100

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## Lung Sounds

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Our knowledge of lung sounds is based largely on empirical observation. Laënnec (1) knew that the sounds heard by applying the ear directly to the chest provided useful information, and he was delighted when he found that a device intended to preserve decorum unexpectedly presented better information. With enormous industry, and with access to a large population of pulmonary patients both clinically and in the autopsy room, he took full advantage of his invention, the stethoscope. He compared what he heard at the bedside with what he subsequently saw in the morgue, and his teaching and writing popularized auscultation. His descriptions of the sounds were by simile (e.g., “comme le cri d’un petit oiseau”), and his understanding of their meaning was based on comparison of auscultatory sounds with gross pathology, and intuition based on analogies with familiar sound-producing mechanisms.

Over the next century, isolated efforts at understanding mechanisms of sound production in the respiratory tract were reported. Some of these described ingenious experiments (2 - 4); some applied known principles of acoustics to the respiratory tract (5,6).

Developments in pulmonary physiology allowed the interpretation of lung sounds to be couched in physiological as well as anatomical terms. Forgacs (7—9) led this movement, adding clinical physiological measurements to careful stethoscopic observations. Graphic methods for displaying sound signals were applied to lung sounds by several workers (10,11). Technical advances in recording and analysis of sound signals and the wide availability and appeal of computers have led to an upsurge of interest in lung sounds and their interpretation (12,13). Clinical applications of our better under

standing of lung sounds are limited as yet, but there are great advantages to studying signals that come from the lung spontaneously and are accessible without risk or even discomfort to the patient. The sound signal detected at the chest surface is complex. It is produced by a source within the lung and is transmitted through both airways and parenchyma. The resultant sound is a product of both the generator and the physical characteristics of the tissues through which it passes. Decoding this signal—that is, determining whether the source or transmission or both have been changed by disease—is likely to provide a great deal of useful information. Piirila et al. (14) and Bettencourt et al. (15) have shown that careful observation and analysis of the character and distribution of adventitious sounds can separate diagnostic groups such as idiopathic pulmonary fibrosis, congestive heart failure, chronic obstructive pulmonary disease, pneumonia, and bronchiectasis with a greater degree of accuracy than many important diagnostic tests, comparing favorably for example with chest radiography and computed tomographic (CT) scanning of the head. Recent advances in technology and information processing, and the number of investigators now addressing the problem, ensure that studies of lung sounds will continue to provide powerful new diagnostic tools and better understanding of lung structure and function.

### SOUND PRODUCTION

The respiratory tract brings solids, liquids, and gases into close juxtaposition: that is its purpose. Apply forces to it, as we do 16 or 20 times a minute, and the tissues move, the liquids move, and the gases move. Often they move in ways that can produce sound. Lung tissue is not a good sound conductor. What we hear as we listen at the chest wall reflects sound transmission as well as sound production. Either one can be altered by abnormality or disease.

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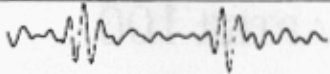
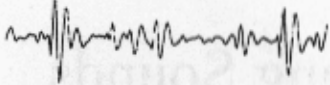

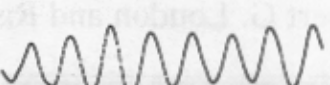
Acoustic Characteristics	American Thoracic Society Nomenclature	Common Synonyms	Laënnec's Original Term	Time Expanded Waveforms
Discontinuous, interrupted explosive sounds (loud, low in pitch)	Coarse crackle	Coarse rale	Rale muqueux ou gargouillement	
Discontinuous, interrupted explosive sounds (less loud than above and of shorter duration; higher in pitch than coarse crackles or rales)	Fine crackle	Fine rale, crepitation	Rale humide ou crepitation	
Continuous sounds (longer than 250 msec, high-pitched; dominant frequency of 400 Hz or more, a hissing sound)	Wheeze	Sibilant rhonchus	Rale sibilant sec ou sifflement	
Continuous sounds (longer than 250 msec, low-pitched; dominant frequency about 200 Hz or less, a snoring sound)	Rhonchus	Sonorous rhonchus	Rale sec sonore ou ronflement	

FIG. 1. Classification of common lung sounds. (From ref. 79.)

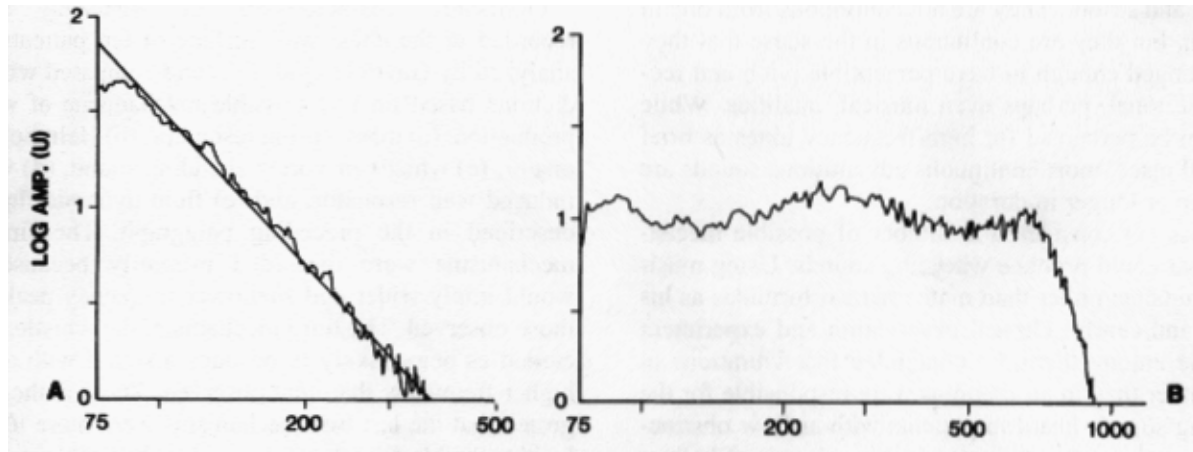
Normal lung sounds are conventionally and conveniently classified as tracheal or bronchial sounds (those heard over large airways) and vesicular sounds (those heard over the chest wall at a distance from large airways). Adventitious or added sounds such as wheezes or crackles usually indicate disease. Adventitious sounds can be subdivided into *continuous* sounds, usually more than 250 msec in duration, more or less musical sounds possessed of perceptible pitch and referred to as wheezes, rhonchi, or stridor; and *discontinuous* sounds, usually less than 20 msec in duration, crackling or bubbling in quality, and referred to as crackles or rales (Fig. 1). These categories of lung sound are produced in different ways. Our understanding of their mechanisms and sites of production is far from complete, but it is improving steadily.

## VESICULAR AND BRONCHIAL SOUNDS

Sounds heard over the lung ("vesicular" sounds) differ from those heard over the trachea or the upper part of the sternum ("tracheal" or "bronchial" sounds) in time—intensity profile and in frequency content. Sanchez and Pasterkamp (16) have shown in children that tracheal sound spectra depend on body height. In models of the upper airways, turbulent airflow has been shown from the larynx to the segmental bronchi, at inspiratory and expiratory flow rates over 0.2 L/sec (17,18). In the more peripheral airways, airflow is generally accepted as being laminar. Olson and Hammersley (19) discussed a variety of mechanisms postulated as vesicular sound generators. They concluded that turbulence in the central airways is the source of most vesicular sounds, with possible additional contributions from vortices developed by the intri-

cate flow pattern in the peripheral airways. Kraman (20) has produced experimental evidence suggesting that the expiratory component of the vesicular sound is produced more centrally than the inspiratory component, and he has shown that when normal subjects breathe a low-density gas mixture, the behavior of the expiratory sound is more consistent with turbulent flow than that of the inspiratory sound (21).

The expiratory component is as loud and as long as the inspiratory component for bronchial sounds but not for vesicular sounds. Bronchial sounds are more hollow in character than vesicular sounds, and the corresponding differences in sound spectrum have been demonstrated in healthy subjects by Gavriely et al. (22). Plotting the logarithm of the sound amplitude against the logarithm of frequency, inspiratory vesicular sounds showed a linear relationship, with log amplitude declining with increasing log frequency; inspiratory tracheal sounds showed a constant log amplitude up to a frequency of about 900 Hz, after which the sound amplitude fell rapidly, as shown in Fig. 2. These authors interpreted their results as compatible with the theory that the tracheal sounds represent the original sounds produced and that the vesicular sounds are the same sounds transmitted to the chest wall surface through a sound filter (small airways, lung, and chest wall) that attenuates the higher frequencies. Hidalgo et al. have shown (23) that changes in frequency content of normal breath sounds in children correlate with increasing height and age. Malmberg et al. (24) described changes in the frequency content of normal breath sounds as a measure of airway hyper reactivity during histamine challenge in adults. Such changes may offer an alternative to the appearance of wheeze or changes in FEV1 (forced expiratory volume in 1 sec) in assessment of response to bron-



**FIG. 2.** A: Sound spectrum of inspiratory vesicular sounds. B: Sound spectrum of tracheal sounds. Log frequency is on the horizontal axis, while log amplitude is on the vertical axis. (From ref. 22.)

choconstrictors or bronchodilators. An important clinical application of the distinction between vesicular and bronchial sounds is in the detection of consolidation of the lung, for example, by lobar pneumonia. Sounds over consolidated lung resemble those heard over the trachea, and it is commonly taught that this is because of better transmission of bronchial sounds to the chest wall through an acoustically homogeneous medium, consolidated lung, which has lost the ability of healthy lung to attenuate the higher-frequency components of the sound signal. The same changes appear to be responsible for egophony—the “E” to “A” change—which tends to accompany bronchial breathing (25).

Granted that consolidation of lung and the change from vesicular to bronchial breathing provide clinically valuable information, what else can we learn from changes in vesicular sounds? Vesicular sounds are louder if a deeper breath is taken. They are reduced in intensity by a variety of disease processes such as emphysema, pleural effusion, and pneumothorax. “Diminished air entry” is a phrase that tends to be equated with reduction in vesicular sound intensity, presumably on the assumption that the latter correlates with regional ventilation. Generalized reduction in vesicular sound intensity has been noted in those with emphysema (26—28), using clinical assessment of lung sound intensity on arbitrary scales. Recordings by microphone allowed quantitation of sound intensity, which was shown to vary at the same site during inspiration with increasing flow, for the same lung volume (29). For the same flow, sound intensity was different at different lung volumes, in a pattern that differed from site to site. Schreur et al. (30) compared lung sound intensity in severe emphysema patients and normal subjects. Sound intensity was strongly influenced by microphone location and by airflow, but when

airflow was standardized, lung sound intensity did not differ between the two groups. This suggested that the diminished breath sounds on physical examination in emphysema resulted from airflow limitation rather than from poor transmission of sound. In more recent studies, the same group (31) used an esophageal accelerometer and showed a wider frequency range for normal breath sounds than either chest wall or tracheal recording sites; the accelerometer shows promise as an investigative tool. When vesicular sound intensity was compared with regional ventilation assessed by radioactive xenon, it was found that the two correlated well if sound intensity was compensated for transmission loss by introducing white sound of known intensity at the mouth and measuring the attenuation of this signal between the site of introduction and the site of measurement at the chest surface (32). The sequence of filling of lung segments in healthy upright subjects is consistent: the upper lobes fill earlier, and the lower lobes accept more air, but later in inspiration. Plotting ventilation at one site against ventilation at another shows the sequence of filling, whether the variable plotted is ventilation measured by xenon scan or measured by lung sound intensity. Ploysongsang et al. (33) used this observation to develop a test that shows nonhomogeneity of parallel-ventilated lung segments by a technique—the lung sounds phase angle test—much simpler than the frequency dependence of lung compliance previously used to measure this abnormality. It is likely that the most important use of knowledge of the characteristics such as intensity and duration of inspiratory and expiratory phases of the normal lung sounds at various sites over the chest will be related to the regional information it provides concerning phenomena such as airway closure and regional ventilation.

## CONTINUOUS ADVENTITIOUS SOUNDS

The loudest of the adventitious lung sounds are those referred to as continuous adventitious sounds: wheezes, rhonchi, and stridor. They are not continuous from breath to breath, but they are continuous in the sense that they are prolonged enough to have perceptible pitch and recognizable tonal, perhaps even musical, qualities. While pitch can be perceived for high-frequency tones as brief as 20–30 msec, most continuous adventitious sounds are 250 msec or longer in duration.

Forgacs (7) considered a number of possible mechanisms that could produce wheezing sounds. Using musical instruments rather than mathematical formulas as his models and careful clinical observation and experiment to decide among them, he concluded that vibrations in tissue rather than in air columns were responsible for the wheezing sounds heard in patients with airflow obstruction. Wheezing patients were given a mixture of helium and oxygen to breathe, and the pitch of wheezes was unchanged, although their intensity was reduced. This, and calculation of the sound frequencies that could be produced by resonating air columns of the length possible in the airways, ruled out the possibility of a simple resonating air column as causing wheezes. By blowing air through an excised bronchus and pinching it lightly until the opposing walls were virtually touching each other, Forgacs could produce sounds not unlike those of a wheezing patient. He proposed the toy trumpet, whose metal reed vibrates to produce a note and whose horn is decorative but not resonant, as the most closely analogous musical instrument. More recently, Shabtai-Musih et al. (34) described forced expiratory wheezes by five normal adults, after breathing air, He-O<sub>2</sub>, and SF<sub>6</sub>-O<sub>2</sub> mixtures. Spectral analysis showed no consistent changes in the wheezes with gas density, although speech tones were markedly changed in the usual way.

The mathematical models developed by Grotberg and Davis (35) and by Grotberg and Reiss (36,37) involve interaction between a flowing gas column and flutter in the walls of the airway through which it flows. The pressure and viscous forces of the gas and the viscoelastic forces of the airway wall can combine to generate oscillations that will occur at a critical gas flow velocity. The variables in the proposed equations are gas density and viscosity, and the thickness, density, resistance to bending, longitudinal tension, parenchymal support elastance, and structural damping of the airway channel walls, and the distance between them. By using estimates for the various values, or measurements when these are available, the behavior of the model can be predicted in terms of critical gas flow required to produce flutter and the resultant oscillatory frequency. The critical flow necessary to induce flutter is reduced by a narrower channel, heavier walls, or a decrease in bending resistance, elastance, or longitudinal tension of these walls. The frequency of the oscillation produced will increase with a narrower channel, lighter walls, or increased bending

resistance, elastance of parenchymal support, or longitudinal tension of these walls.

Frequency characteristics of wheezing sounds recorded at the chest wall surface of ten patients were analyzed by Gavriely et al. (38) and compared with predictions based on five possible mechanisms of wheeze production: (a) mass—spring resonator, (b) Helmholtz resonator, (c) whistle or vortex shedding sound, (d) vortex-induced wall resonator, and (e) fluid dynamic flutter as described in the preceding paragraph. The first two mechanisms were discarded primarily because they would imply wider and shallower frequency peaks than those observed. The third mechanism, the whistle, is discarded as being likely to produce a sound with a much higher frequency than that observed. These authors suggested that the last two mechanisms were more likely to be responsible for wheezing sounds than other proposed mechanisms.

An important implication of the fluid dynamic flutter model for wheeze production is that the appearance of wheeze will be associated with flow limitation at the airway where the sound is produced. The reverse is not true:

flow limitation is not necessarily associated with sound production. By studying the generation of wheezes during forced expiration in normal subjects, Gavriely et al. (39) demonstrated a correlation between the onset of wheezes and the sudden change in transpulmonary pressure, which in their experimental system corresponded to the onset of flow limitation. Flow limitation preceded wheeze by about 100 ml. Administration of bronchodilators did not affect this relationship. The frequency spectra observed were similar to those seen in patients, but the differences between the forced expiratory wheeze of healthy subjects and the wheezes heard in patients with airway disease have still to be defined. Forgacs (8) has pointed out the sequence of increasing numbers of wheezes heard with increasingly forceful expirations in patients with airway disease and contrasted this with the sudden appearance of the full complement of wheezing sounds in the healthy subject whose lungs have uniform mechanical properties, and in whom the dynamic compression of all the flow-limiting bronchi occurs simultaneously.

Support for the theory that airway wall vibration is related both to sound production and to flow limitation was provided by a series of experiments done on dried animal lungs held in the expanded position in a plastic shell, with holes drilled to allow continuous airflow in a mouthward direction (40). Pressure—flow relationships were studied, and wheezing sounds were frequently noted when flow-limiting conditions were reached. When the isolated lung was frozen, making the walls rigid and unable to flutter, flow limitation and wheeze production both disappeared, to reappear when the preparation was thawed.

Wheezing is a common symptom. Dodge and Burrows (41) reported questionnaire responses from a population sample in which the point prevalence for asthma was

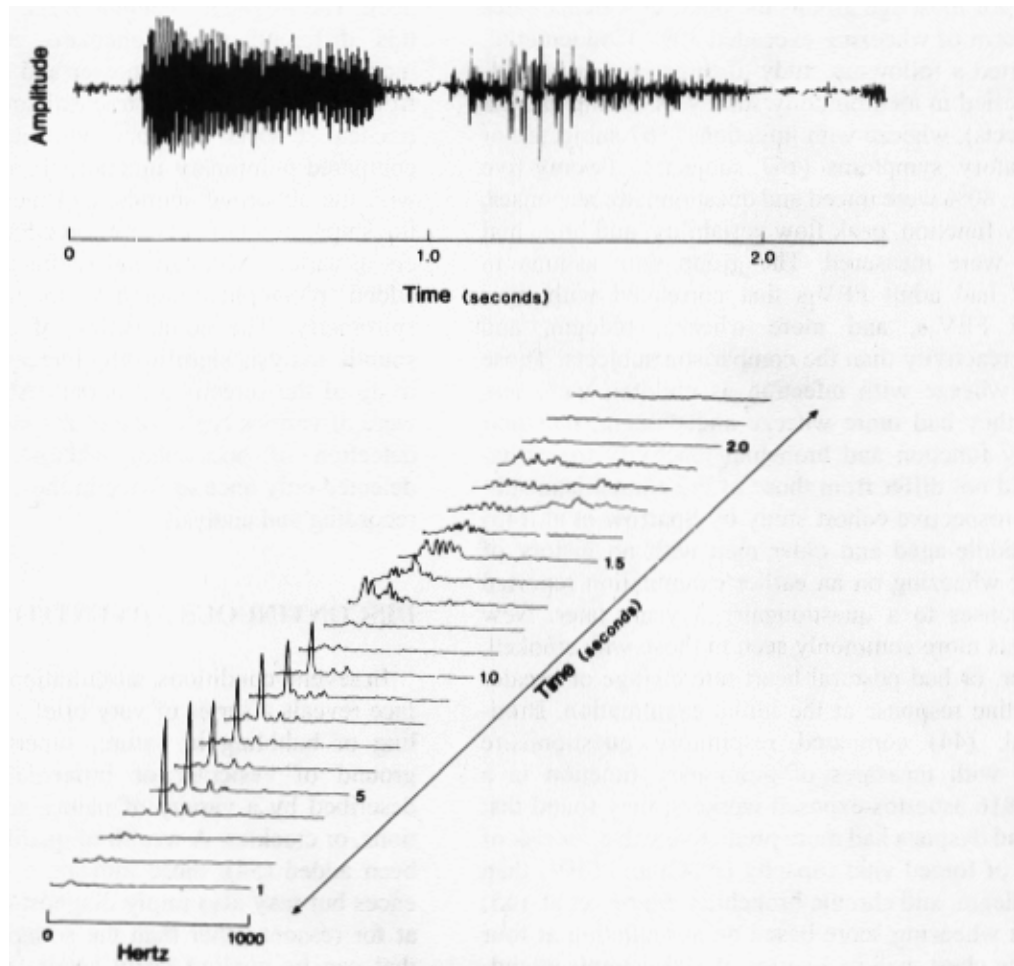
6.6%, but for most age groups the point prevalence rates of some form of wheezing exceeded 30%. Godden et al. (42) reported a follow-up study of three groups of children classified in a community survey as having asthma (121 subjects), wheeze with infections (167 subjects), or no respiratory symptoms (167 subjects). Twenty-five years later, 80% were traced and questionnaire responses, ventilatory function, peak flow variability, and bronchial reactivity were measured. The group with asthma in childhood had adult FEV<sub>1</sub>s that correlated with their childhood FEV<sub>1</sub>s, and more wheeze, phlegm, and bronchial reactivity than the comparison subjects. Those who had wheeze with infection as children were less affected: they had more wheeze and phlegm, but their ventilatory function and bronchial reactivity to methacholine did not differ from those of the comparison subjects. A prospective cohort study by Sparrow et al. (43) of 624 middle-aged and older men with no history of asthma or wheezing on an earlier examination reported their responses to a questionnaire 3 years later. New wheeze was more commonly seen in those who smoked, were older, or had postural heart rate change or greater methacholine response at the initial examination. Brodtkin et al. (44) compared respiratory questionnaire responses with measures of pulmonary function in a group of 816 asbestos-exposed workers; they found that wheeze and dyspnea had more predictive value for risk of reduction of forced vital capacity (FVC) and FEV<sub>1</sub> than cough, phlegm, and chronic bronchitis. Marini et al. (45) reported a wheezing score based on auscultation at four sites on the chest wall in a series of 100 patients attending a pulmonary function laboratory. This score correlated with the severity of airflow obstruction, but the presence of wheezing was more useful as a predictor of response to bronchodilators, particularly in those with a history of asthma. A study of several of the measurable characteristics of wheezes in 20 patients presenting with acute asthmatic attacks showed that the duration of wheeze as a proportion of the total respiratory cycle duration (Tw/Ttot ratio) (Fig. 3) was the measurement that correlated best with the FEV<sub>1</sub> at the time of presentation. Reduction in this same ratio (Tw/Ttot) and reduction in the sound frequency correlated best with improvement in FEV<sub>1</sub> as a result of treatment with bronchodilators (46). Other clinical applications involving wheezing lung sounds include monitoring of patients with nocturnal asthma to determine the timing of changes in airflow without waking the patient (47), and detecting a response to inhaled bronchoconstrictors, for example, in children too young to cooperate in pulmonary function testing (48–50). Differentiation between stridor resulting from upper airway narrowing and wheezing associated with asthma was helped by the use of recording and spectrum analysis techniques (51). The major differences were in the timing of the sound—predominantly inspiratory in stridor—and in the prominence of the sound over the

neck. The frequency content of the sound did not help in this differentiation. Yonemaru et al. (52) reported increased peak spectral power and mean spectral power from 600 to 1300 Hz in tracheal sounds in patients with tracheal stenosis, compared with normal subjects. They compared pulmonary function changes and CT findings with the abnormal sounds, and they described the relationships. In a lung disease screening program for workers in various occupational settings, Gavriely et al. (53) added phonopneumography to a questionnaire and spirometry. The combination of spirometry and lung sounds analysis significantly increased the overall sensitivity of the screening program. Abnormal lung sounds were of various types: one of the more common was the detection of occasional wheezes—in some subjects detected only once or twice in the 12 to 15 min of sound recording and analysis.

## DISCONTINUOUS ADVENTITIOUS SOUNDS

In several conditions, auscultation at the chest wall surface reveals a series of very brief sounds that are crackling or bubbling in nature, superimposed on a background of vesicular or bronchial sounds. They are described by a variety of names such as rales, crepitations, or crackles. A wealth of qualifying adjectives have been added (54); these indicate a perception of differences but may also imply diagnostic preferences arrived at for reasons other than the sound heard. Descriptions that can be applied fairly accurately and reproducibly include the loudness, profusion, and timing of the adventitious sounds with respect to the respiratory cycle; the location at which they are heard; and whether they are affected by changes in posture, deep breathing, or cough. Other observations of importance in distinguishing the causes of crackles are the tendency for the same pattern to repeat in successive breaths and the audibility of crackles at the open mouth (55). Crackles are commonly referred to as “fine” and “coarse,” and this distinction has been shown to be reasonably uniform within and between observers and to have a basis in measurable differences in the acoustic signal (56).

As is the case with wheezes, crackles are almost always an indication of abnormality. As is the case with wheezes, by adopting unusual maneuvers it is possible for healthy subjects to produce crackles. Workum and associates (57) noted crackles in more than half of 56 young women, all but six being nonsmokers, when the subjects were asked to lean forward in a chair, to exhale to residual volume, and then to breathe in slowly and deeply. Crackles were most frequently heard over the lower lung zones anteriorly. In five healthy subjects, Ploysongsang and Schonfeld (58) studied the circumstances in which crackles were produced. The subjects were seated in a body box to allow volume measure-



**FIG. 3.** Time-amplitude waveform is shown at top. Burst of activity is characteristic for wheeze. Each line represents 250-msec sound, analyzed over spectral range from 1 to 1000 Hz. Line 1 represents beginning of expiration. On line 4, peak is identified at 250 Hz. This peak corresponds to wheeze heard on auscultation and seen on time—amplitude waveform. Peak is seen on lines 4 through 10. This means wheeze was present for 700 msec. Total duration of breath was 2.4 sec, so  $T_w/T_{tot}$  is 0.292. Amplitude of peak can be measured, and this is linearly related to intensity of sound. We can measure frequency, intensity, and duration of wheezes.

ment, with a balloon measuring esophageal pressure, and with microphones attached to the anterior chest. They then conducted a series of maneuvers: breathing air or 100% oxygen at a normal tidal volume, or breathing air or oxygen in shallow fashion at a lung volume below functional residual capacity. After each of these four maneuvers, quasistatic pressure—volume curves were obtained, and during the entire process sound signals, esophageal pressure, and volume change were recorded for later analysis. After low-volume breathing, the residual volume fell, particularly after breathing oxygen, and the first few deep breaths from low volume produced crackles. Their number correlated with the reduction in lung volume. This supported the commonly accepted belief that some crackles are associated with the reopening of atelectatic peripheral lung units, particularly at the bases.

Most crackles are heard in patients with lung abnor-

malities, such as the interstitial lung diseases, chronic obstructive lung disease, pneumonia, and congestive heart failure. Forgacs (7) described a repetitive pattern seen in successive breaths, where a series of crackles occurred with similar timing and relative sound intensity, in patients with fibrosing alveolitis (interstitial pulmonary fibrosis), and he deduced from the repetitive pattern that the source must involve solid tissue with a fixed structure, rather than gas bubbling through liquid, which would be expected to produce a random pattern of sounds. Nath and Capel (55,59) confirmed the repetitive pattern for the late inspiratory crackles of fibrosing alveolitis and presented graphic representations of the crackles and the airflow pattern associated with their appearance. They subsequently measured the airflow rates, lung volume, and transpulmonary pressures associated with

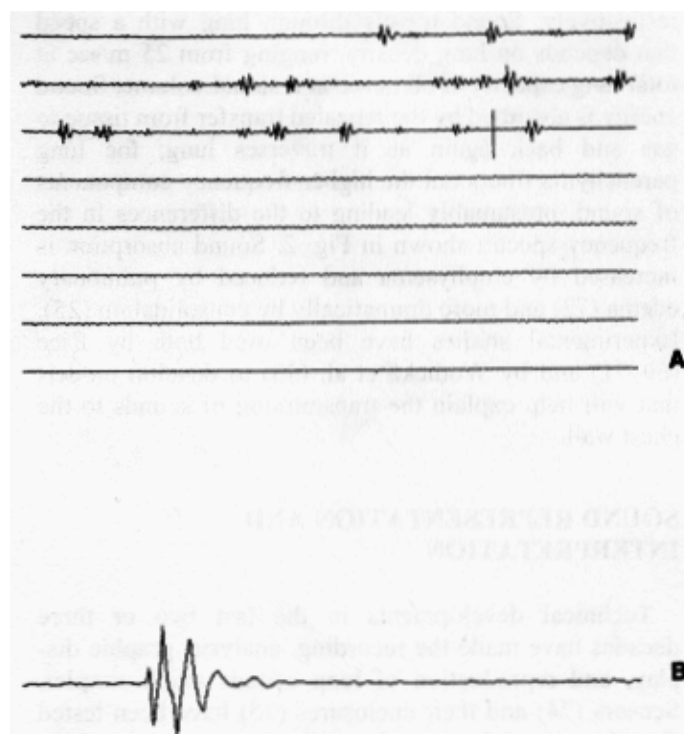
the production of these crackles and showed that the appearance of an individual crackle, which could be recognized from breath to breath, was determined by lung volume and the closely correlated transpulmonary pressure, rather than by flow rate or timing. This supported the concept that these crackles were the result of forces developed and suddenly released in fixed tissue. These observations related to the late inspiratory crackles of interstitial lung disease, and the same authors were able to demonstrate several differences between such crackles and those associated with obstructive lung disease. Crackles of interstitial lung disease tended to show late inspiratory predominance, they tended to be fine in character, they were frequently repetitive in pattern from breath to breath, and they were rarely audible at the open mouth (which most crackles associated with obstructive lung disease were).

Forgacs (7) suggested that the crackles of interstitial lung disease are caused by the sudden opening of small lung units as they inflate, with sudden equalization of pressure on the two sides of the site at which the airway was formerly closed. Surface tension forces presumably tend to hold an airway closed until it pops open with equalization of pressures on the two sides of the previously closed segment. Fredberg and Holford (60) have suggested that energy might be stored in the tissue surrounding a collapsed airway in such a way that slow expansion of the lung would lead to a discontinuity of stress distribution and the sudden assumption of a new configuration with a step-function liberation of mechanical energy. Whatever the method of sudden release of tissue energy, it appears likely that the crackles heard at the surface represent a series of step-function or impulse-function signals derived at a point within lung tissue. The energy produced is transmitted to the chest wall surface through lung, and the sound heard and the waveform associated with it reflect the circumstances of its production and also the acoustic characteristics of the lung and other tissues between the site of production and the site of auscultation.

It seems likely that other mechanisms are responsible for other types of crackle. The bubbling sounds heard in the presence of copious secretions, the rale muquex of Laënnec, are presumably formed by bubbling of air through liquids, which repeatedly form a thin film that breaks explosively. Variations on this basic method of production may occur in patients with chronic obstructive lung disease, in patients with pneumonia, and in patients with pulmonary edema. Such secretion sounds have still to be described in detail. A less commonly heard sound, described by Forgacs as a "chirp," starts with a crackle but continues with a brief musical note, the sequence presumably representing a small airway popping open, but being of appropriate dimensions to produce a brief wheezing sound as the equilibrating airflow induces vibrations in the airway wall for long enough—perhaps 50—100 msec—to possess perceptible pitch. Such

sounds have been noted in patients with extrinsic allergic alveolitis. Earis et al. (61) recorded sounds of this sort, which they described as "the inspiratory 'squawk,'" in 14 patients with diffuse pulmonary fibrosis and squawking sounds. Nine of them had extrinsic allergic alveolitis, seven cases of bird fancier's lung, and two of farmer's lung. Five patients had pulmonary fibrosis from other causes. All of them demonstrated squawking sounds. Those with extrinsic allergic alveolitis had shorter sounds that occurred later in inspiration and were of higher frequency than the sound heard in patients with pulmonary fibrosis. In eight patients, a single loud crackle preceded the squawk. These authors suggested that the sounds resulted from sudden opening of the airways and that the difference between the sounds in the two groups reflected the size of the affected airways.

Visual displays of lung sounds (Fig. 4) and measurement of time intervals and configuration of sounds have been helpful in accumulating information about crackles, which may in time be of considerable value in reaching a diagnosis. Measurements of characteristics of individual crackles, such as the initial deflection width and the two-cycle duration (56), have allowed classification as "fine" or "coarse" by machine, which corresponds accurately with the classification by ear. Time intervals between one



**FIG. 4.** A: Sequence of coarse crackles recorded from a patient with an acute exacerbation of chronic bronchitis. Trace starts with inspiration; trace occupies 1.6 sec. B: Time-expanded waveform of crackle selected by cursor (vertical line) in upper trace. Time scale is 50 msec.

crackle and the next have also been used (62) as a basis for the same dichotomy. The ability to monitor the numbers and character of crackles breath by breath, validated by Murphy et al. (63) and by Kaisla et al. (64), now offers promise of diagnostic and monitoring devices that may be helpful in settings ranging from industrial screening programs for asbestos workers (65—67) to monitoring of patients in an intensive care unit (68).

## SOUND TRANSMISSION

What we hear as we listen at the chest wall reflects lung sound transmission as well as sound production. Either one can be altered by abnormality or disease. In the preceding sections we have concentrated on the known and suspected mechanisms and sites of production of lung sounds, but sound transmission plays an important role in what we hear and how we interpret it. Air-filled, healthy lung is a good acoustic insulator, with sound-filtering properties more complex than those of styrofoam. Rice has measured sound speed in the upper airways (69) and in the pulmonary parenchyma (70). In a review of the transmission of lung sounds (71) he considers four types of wave: longitudinal, transverse, surface, and tube. The first and last of these are the most important, representing the predominant modes of transmission through lung parenchyma and through airways, respectively. Sound travels through lung with a speed that depends on lung density, ranging from 25 m/sec at total lung capacity to 60 m/sec at residual volume. Sound energy is absorbed by the repeated transfer from tissue to gas and back again as it traverses lung; the lung parenchyma filters out the higher-frequency components of sound, presumably leading to the differences in the frequency spectra shown in Fig. 2. Sound absorption is increased by emphysema and reduced by pulmonary edema (72) and more dramatically by consolidation (25). Experimental studies have been used both by Rice (69—71) and by Wodicka et al. (73) to develop models that will help explain the transmission of sounds to the chest wall.

## SOUND REPRESENTATION AND INTERPRETATION

Technical developments in the last two or three decades have made the recording, analysis, graphic display, and reproduction of lung sounds much simpler. Sensors (74) and their enclosures (75) have been tested for this special set of applications, and analytic approaches to sound signals are being pursued with diligence (76—78). This has helped our

understanding of what lung sounds mean and the limits of what they tell us; and so we listen to the chest with more interest and attention. We can teach our students more effectively by supplementing bedside instruction with demonstrations of recorded sound and by complementing sound with visual displays. We shall learn how to disentangle more effectively the information about the structure and function of lung being conveyed economically and safely to the chest wall surface. The stethoscope is still the most widely used diagnostic instrument. It will not be replaced but will become more important as a result of improvement in the scientific foundations of auscultatory chest medicine.

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