State of the Art

Respiratory Sounds
Advances Beyond the Stethoscope

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INTRODUCTION

In many ways, the sounds of respiration have remained timeless since Laënnec (1) improved their audibility with the stethoscope. Indeed, 30 yr ago, Forgacs (2) characterized the field by stating that "the sound repertoire of a wet sponge such as the lung is limited." Why then is there a growing interest in the acoustics of respiration, as evidenced by recent editorial comments in leading pulmonary and physiology journals (3–5)? Also, why is there a multinational effort, funded by the European Commission,[1] to standardize computerized respiratory sound analysis?

It is precisely the application of computer technology that has provided new insights into acoustic mechanisms and new measurements of clinical relevance since the last State of the Art review on lung sounds in this Journal was published 13 yr ago (6). A closer bridging of pulmonary acoustics with traditionally measured lung mechanics, e.g., air flow and volume, and the use of digital techniques to extract information on average sounds under standardized conditions were major steps that have advanced the utility of lung sounds beyond the stethoscope. Lung sound analysis to detect flow obstruction during bronchial provocation testing, for example, has drawn much attention (3) because it does not require maximal breathing effort and can therefore be used with young patients. Respiratory acoustic measurements have also shown promise in the investigation of upper airway pathology, e.g., in patients with obstructive sleep apnea or with tracheal narrowing.

This review begins with the current understanding of the thorax and upper airways as an acoustic system. Some details about the methods for lung sound measurement are provided, but interested readers will find more comprehensive information in a recent monograph (7). The following sections focus on the present state of knowledge about normal and adventitious lung sounds, their origin, and their clinical relevance. Finally, a view on the likely areas of practical application for respiratory sound analysis is presented.

SOUND AT THE BODY SURFACE

Stethoscopes

Despite the high cost of many modern stethoscopes, these instruments remain simply conduits for sound conduction between the body surface and the ears. Stethoscopes are rarely tested, rated, or compared and are often chosen for their appearance, reputation, and inadequately supported claims of performance. They are less than ideal acoustic instruments because they do not provide a frequency-independent, uncolored transmission of sounds. Rather, they can selectively amplify or attenuate sounds within the spectrum of clinical interest. Amplification tends to occur below 112 Hz and attenuation at higher frequencies (8). This feature is inherent in the design of the stethoscope that often places convenience and clinical utility ahead of acoustic fidelity. Amplification at low frequencies is appreciated by cardiologists since heart sounds are in this frequency range, which is poorly perceived by the human ear. Auscultation of the lung, however, could benefit from a more faithful representation of sounds than present stethoscopes provide.

Sensors for Lung Sound Recording

Two types of transducers are in common use for lung sound recording and research: the electret microphone with coupling chamber and the accelerometer (9). Small electret microphones are widely available for speech and music recording. When coupled to the skin by a sealed chamber, similar to a stethoscope bell, this type of microphone is a sensitive lung sound transducer. Different sizes and shapes of coupling chambers have been found to affect the overall frequency response of this coupling. Those arrangements with smaller, conically shaped chambers are more sensitive to higher lung sound frequencies (10, 11), but also highly susceptible to ambient noise. Contact accelerometers are also popular in lung sound research and can be calibrated on a vibration table so their output is quanti-

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fied. However, they are typically more expensive than electret microphones, are often fragile, and may exhibit internal resonances near the lung sound frequencies of interest.

SOUND TRANSMISSION

Models and Predictions

Many factors that influence auscultation, including the response of the stethoscope and psychoacoustic phenomena, have contributed to concepts that are now widely taught to students in the health care professions. These concepts include: that there is relatively little bilateral asymmetry of sound amplitude and that asymmetry indicates disease, that sounds on the chest surface are primarily filtered versions of those detected over the trachea or neck, and that flow effects are of little diagnostic importance as long as near- or above-normal rates are attained. Although these and other concepts have proven useful in many clinical circumstances, recent acoustic investigations with high-fidelity measurements indicate that considerably more information of clinical utility can be gathered from respiratory sounds. This information often cannot be obtained by auscultation, and some of the new findings can only be interpreted by taking an acoustical perspective and extending or even breaking down a few traditional concepts. For example, it has become clear that inspiratory sounds measured simultaneously over the extrathoracic trachea and at the chest surface contain highly unique regional information that can only be reproducibly extracted with a knowledge of the breathing flow rate. Such realizations are stimulating the investigation of the acoustic properties of the respiratory system to improve their use for diagnostic, screening, and monitoring purposes.

The respiratory tract consists of the vocal tract, which has been studied extensively, and the subglottal airways, which are now the topic of more detailed acoustic investigations. It is the combined effect of these two components that gives rise to the highly unique properties of the overall tract. The branching airways in the thorax have been modeled by a number of investigators to assess the structural determinants of sound reflection and transmission measurements (12–18). Although the exact branching structure is important at high audible frequencies (14), at the relatively low frequencies and long wavelengths associated with lung sounds the system possesses primarily two distinct features: the large airway walls vibrate in response to intraluminal sound (16, 18), allowing significant sound energy to be coupled directly into the surrounding parenchyma; and the entire branching network behaves to a first approximation as a single nonrigid tube that is open at its distal end to the relatively large air volume in the numerous smaller airways and alveoli (18, 19). This tubelike behavior in concert with wall vibration yields airway resonances with a fundamental frequency near 650 Hz for the subglottal system, as measured in tracheostomized patients (13), and at a lower frequency when the entire respiratory tract is patent. At higher audible frequencies, the airway walls become effectively rigid because of their inherent mass (20), allowing more sound energy to remain within the airway lumen and potentially travel farther into the branching structure.

The lung parenchyma consists primarily of alveoli and small airways, capillaries, and supporting tissues. At frequencies in the audible range below about 10,000 Hz where the sound wavelengths significantly exceed alveolar size, the parenchyma has been modeled as a foamlike substance that is a homogeneous mixture of air and waterlike tissue, assuming that no gas exchange occurs because of the sound wave propagation (21). Here the composite density is dominated by the tissue component and the composite stiffness by the air, resulting in a mixture with a relatively low sound speed (on the order of 50 m/s) and therefore short wavelength at a given frequency as compared with propagation in air or tissue alone (with respective sound speeds of roughly 350 and 1,500 m/s). These relatively shorter sound wavelengths suggest that more regional information concerning lung structure can be obtained from low-frequency acoustic measurements than had first been hypothesized. In addition, the effect of changing the amount or type of gas in the mixture on parameters such as sound speed is significantly less than if propagation were through the gas alone. To estimate the losses associated with sound propagation through the parenchyma, the parenchymal mixture has been represented as air bubbles (alveoli) in water (lung tissue) at both low (18) and high (22) audible frequencies. These models suggest that the absorption of sound energy is highly frequency-dependent even for this simple geometry, with very large losses at higher frequencies where the wavelength approaches alveolar size. More complicated theoretical approaches that include the effects of small airways (23) have also been used to predict the frequency dependence of sound speed and other properties over a wide frequency range.

The encasement of the lung parenchyma by the chest wall is an important factor that affects sound propagation to the chest surface. The chest wall, although relatively thin compared with the extent of the parenchyma, is significantly more massive and stiff. In addition, the heterogeneous composition of bone, muscle, skin, and other tissues makes it a complex surface upon which to make acoustic measurements, with a potential for surface waves to travel between transducers on the skin and poor transmission to areas overlying bones such as the scapulae (24, 25). It has been estimated that the mechanical/acoustic impedance mismatch between the parenchyma and the chest wall can account for as much as an order of magnitude decrease in the amplitude of sound propagation (25) and significant alterations in the timing and waveform shape of adventitious lung sounds such as crackles (24).

How the airways, parenchyma, and chest wall interact to produce the measurable acoustic properties of the thorax is the topic of considerable interest, debate, and investigation. With the knowledge of relatively short sound wavelengths in the parenchyma, earlier models (26) served as the precursors to more recent approaches that treat the thorax from an acoustical perspective like a large cylindrical drum (18, 27). Wodicka and coworkers (18) represented the respiratory tract at low frequencies as a single nonrigid tube that was at the center of the drum, was open on its distal end, and was assumed to be the source of an outgoing cylindrical sound wave into a surrounding parenchymal mixture that included losses. The predicted amplitude on the surface of the drum (the chest wall) of sound originating from the tube (the central airways) compared well as a function of frequency with transmission measurements performed on healthy human subjects (28). The model highlighted the importance of tubelike resonances of the respiratory tract and of propagation losses in the lung parenchyma and chest wall. A model by Vovk and coworkers (27) also predicts a preferential transmission to the chest surface at low frequencies not unlike that of lung sounds, although it does not allow for regional sound generation in the branching airways.

Sound Transmission Measurements

To measure the response of the thorax to sonic perturbations of known quality, a number of investigations have focused on the transmission of sound from introduction at the mouth to

detection on the chest surface. In this manner, the static and even dynamic properties of the system can be measured and compared with model predictions and other hypotheses. Because the thorax has been shown to respond in a linear fashion to low-level sonic perturbations at frequencies as high as 1,500 Hz (29), chest surface responses relative to a reference measurement over the extrathoracic trachea have been used to determine the amplitude and phase delay of transmission. In general, the observed responses have been complex, including drumlike resonance behavior that indicates intrathoracic sonic reflections and spatial heterogeneity that reflects the underlying anatomy.

Sound on the chest surface relative to the trachea is lower in amplitude, is loudest at low frequencies (near 135 Hz), and decreases in amplitude with increasing frequency (30). These characteristics are consistent with a number of studies where the chest surface measurements were not referenced to a tracheal site (31–35). This knowledge has been used to compensate for transmission characteristics and thereby use breath sounds as quantitative indices of ventilation (36) or make direct comparisons to model predictions (28). The preferential transmission at low frequencies is consistent with the observed thoracic properties during speech production and portrays a drumlike response (30). The frequency-dependent decrease in amplitude agrees with models of the thorax that account for parenchymal losses (18, 25). These losses limited the frequency range of early transmission studies to roughly 600 Hz. Recent improvements in sound generation, measurement, and processing have extended the bandwidth to more than 1,500 Hz to encompass the lung sound frequency range (37) and into the high audible range when techniques such as cross-correlation are employed to enhance the effective signal to noise ratio (22).

A strong spatial dependence of sound transmission from the mouth to the chest wall was reported by Kraman and Austrheim (38) and Kraman and Bohadana (39). They found that the amplitude at low frequencies to sites overlying the right lung was significantly greater than that measured at corresponding locations over the left lung. These findings, which were later corroborated (40, 41), indicate the preferential coupling of sound to the right lung because of the massive mediastinum that is adjacent to the left side of the major airways. Incorporation of these mediastinal effects into a model of transmission can account for the bilateral asymmetry at low frequencies (40). At frequencies above a few hundred Hz no amplitude asymmetry of transmitted sound is observed, whereas inspiratory lung sounds at higher frequencies show a lateralization to the left over the posterior chest (41). This suggests that the primary pathways for transmitted sound change with frequency from direct coupling from the large airways to the surrounding parenchyma via wall motion to a predominantly airway lumen route, bypassing the effect of the mediastinum. Lung sound asymmetries, on the other hand, may also reflect differences in regional flow turbulence and sound generation.

The lack of gas density effects on the amplitude of low frequency sound transmission to the chest surface was first observed by Kraman and Bohadana (39) and later confirmed by Mahagnah and Gavriely (42). This insensitivity to inhaled gas composition means that the bulk of the low-frequency sound propagation to the chest wall occurs in the lung parenchyma, which behaves as a composite mixture with acoustic properties that are relatively weakly determined by the gas.

Transit time of sound through the lungs has been measured using both cross-correlation (43, 44) and spectral techniques to estimate the phase and subsequently phase delay (29, 37, 42, 45) of sonic transmission from a tracheal reference site to the chest surface. The interpretation of the phase delay as an index of the transit time is complicated by a number of factors, including the presence of thoracic resonances, which indicate internal reflections at low frequencies and the potential for multiple propagation pathways to a single measurement site.

As depicted in Figure 1, the phase delay of sound propagation to the posterior chest surface is frequency-dependent, with shorter delays of approximately 1 ms at frequencies near 1,500 Hz compared with roughly 2.5 ms or more at frequencies below a few hundred Hz (45). This finding supports the hypothesis that transmission at low frequencies occurs at slower speeds primarily through parenchyma, whereas higher frequency sound traverses a faster, presumably more airway-bound, route, as is the case in isolated lung preparations where the parenchyma and trachea are not in contact (46).

At frequencies below approximately 300 Hz, the phase delay increases as the measurement site on the chest surface moves caudally (29, 42, 44, 45), suggesting that the propagation takes a relatively direct route. At higher frequencies, however, this correspondence between phase delay and sensor distance is not observed, indicating a change in propagation pathways with frequency (45). In addition, the phase delays exhibit a bilateral asymmetry with relatively decreased delays to the left posterior chest, as compared with the right (45), that loosely coincide with the observed asymmetry in amplitude. Thus, the heterogeneous anatomy of the thorax affects both the amplitude and phase delay of sound transmission in a manner that is frequency-dependent.

As is the case for the amplitude of sound transmission to the chest wall at frequencies below approximately 300 Hz, the transit time and phase delay are only weakly affected by the inhalation of an 80% helium-20% oxygen ($He-O_2$) mixture

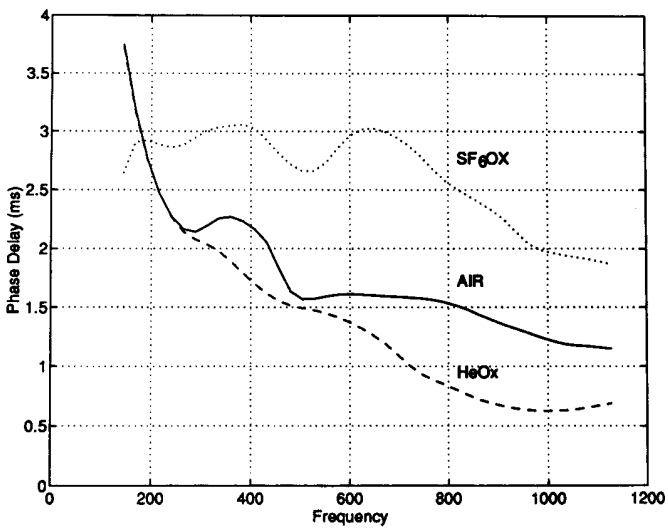

Figure 1. Phase delay (in milliseconds) of sound transmission from the trachea to the lower right posterior chest wall, estimated from measurements performed on a healthy subject, as a function of the sonic frequency (in Hertz) and the inhaled gas composition (SF60x = 80% sulfurhexafluoride/20% O_2; HeOx = 80% helium/20% O_2). The longer delays at low frequencies that are relatively insensitive to inhaled gas composition indicate primarily parenchymal propagation, whereas the more gas-dependent and shorter delays at higher frequencies imply a more air(way)borne route. Such findings suggest that chest wall sounds at low frequencies provide information mostly on lung parenchyma and thoracic cavity. A wider bandwidth is required to fully extract airway properties. (Reproduced with permission from Reference 45.)

(42, 44, 45), indicating a primarily parenchymal propagation. At higher frequencies, phase delays are significantly affected by low (43) and high density gas mixtures (45), which confirms that gas density does affect the acoustic transmission over the frequency range of lung sounds and highlights the strong frequency dependence of the acoustical properties of the thorax (*see* Figure 1).

Effects of Pulmonary Pathology

The changes in lung structure that occur in disease affect the amplitude and timing of sound transmission from the airways to the chest surface. In patients with emphysema, a decrease (47) and larger variability (48) of transmitted amplitude at low frequencies was observed, which is qualitatively consistent with the common auscultatory finding of decreased lung sound intensity. In contrast, cardiogenic pulmonary edema was found to increase the amplitude of sound transmitted to the chest wall in dogs in a linear fashion over a wide frequency bandwidth relative to postmortem wet to dry weight ratios (49), a finding consistent with that of bronchial breathing heard over consolidated lung. Other mechanisms of sound introduction into the thorax such as percussion of the sternum and measurement of the transmission to the posterior chest surface, have also been investigated (50, 51). Through analysis of the transmitted amplitude, large pleural effusions could be detected, but deeper intrapulmonary masses could not, presumably because the majority of the transmission was through the bony chest wall rather than through lung tissue.

RESPIRATORY SOUNDS

Classification and Nomenclature

Lung-sound nomenclature has long suffered from imprecision and ambiguity. Until the last few decades, the names of lung sounds were derived from the originals given by Laënnec (1) and translated into English by Forbes (52). These names carried the implication of the pathologic mechanism of their production, e.g., humid or dry rales, or the character of the sound, e.g., hissing rale. The need for a more objective naming system has long been recognized (2, 53). In 1985, at the 10th meeting of the International Lung Sounds Association, an ad hoc committee agreed on a schema that included fine and coarse crackles, wheezes, and rhonchi (54). Each of these terms can be described acoustically and does not assume a generating mechanism or location. These terms are now widely accepted, although the term "rale," generally meaning "crackle," is still frequently used (55).

Further classification of lung sounds is still vague. No single characteristic distinguishes perfectly between the fine and coarse crackles although combinations of features provide adequate discrimination (56). The fact that fine and coarse crackles tend to appear at different times within the inspiratory cycle assists in their differentiation (57). Wheezes occur within a broad frequency range. Rather than separating low pitched wheezes as "rhonchi" it may be more useful to apply the term "rhonchus" to repetitions of complex sound structures that have a tonal, snorelike characteristic and are likely related to airway secretions and collapse. The nomenclature of the normal lung sound, also called the breath sound (*see* also *Other Respiratory Sounds* in RESPIRATORY SOUNDS section) or vesicular sound, has not attracted much attention. These terms are usually synonymous and we will refer to the normal lung sound in this review, recognizing that "normal" in this context refers to the basic breath sound without implication on the normality of the lung (*see* Table 1).

Normal Lung Sounds

The breathing-associated sound heard on the chest of a healthy person is called the normal lung sound. It is a noise that peaks in frequency below 100 Hz (58), where it is mixed with and not easily distinguished from muscle and cardiovascular sounds. The lung sound energy drops off sharply between 100 and 200 Hz (59), but it can still be detected at or above 1,000 Hz with sensitive microphones in a quiet room (60). The normal lung sound spectrum is devoid of discrete peaks and is not musical. It appears well established that its inspiratory component is generated primarily within the lobar and segmental airways, whereas the expiratory component comes from more proximal locations (61–66). Air turbulence is presumed to generate the normal lung sound. However, tur-

TABLE 1

CATEGORIES OF RESPIRATORY SOUNDS*

Respiratory Sound	Mechanisms	Origin	Acoustics	Relevance
Basic sounds				
Normal lung sound	Turbulent flow vortices, unknown mechanisms	Central airways (expiration), lobar to segmental airway (inspiration)	Low-pass filtered noise (range $<$ 100 to $>$ 1,000 Hz)	Regional ventilation, airway caliber
Normal tracheal sound	Turbulent flow, flow impinging on airway walls	Pharynx, larynx, trachea, large airways	Noise with resonances (range $<$ 100 to $>$ 3,000 Hz)	Upper airway configuration
Adventitious sounds				
Wheeze	Airway wall flutter, vortex shedding	Central and lower airways	Sinusoid (range $\sim$ 100 to $>$ 1,000 HZ; duration, typically $>$ 80 ms)	Airway obstruction, flow limitation
Rhonchus	Rupture of fluid films, airway wall vibrations	Larger airways	Series of rapidly dampened sinusoids (typically $<$ 300 Hz and duration $>$ 100 ms)	Secretions, abnormal airway collapsiblility
Crackle	Airway wall stress-relaxation	Central and lower airways	Rapidly dampened wave deflection (duration typically $<$ 20 ms)	Airway closure, secretions

* This table lists only the major categories of respiratory sounds and does not include other sound such as squawks, friction rubs, grunting, snoring, or cough. Current concepts on sound mechanisms and origin are listed but these concepts may be incomplete and unconfirmed.

bulence is a density-dependent phenomenon, and the behavior of lung sounds in response to low-density gas breathing is peculiar. Austrheim and Kraman (67) found that breathing He-O_2 diminished the tracheal sound amplitude by 45% while the simultaneously recorded sound over several locations on the chest decreased by only 13 to 16%. Pasterkamp and Sanchez (68) found a 17% decrease in lung sound amplitudes on He-O_2 below 300 Hz, where most of the acoustic energy resides, but 40% attenuation above 300 Hz. They concluded that flow turbulence produced the sound in the higher frequency range (*see* Figure 2). Although vorticeal airflow is well known to exist in airways, and this has been postulated as a cause of normal lung sounds (69), the mechanisms that produce the normal lung sound, at least at frequencies to 300 Hz, are not understood.

Lung sound amplitude differs between persons and different locations on the chest surface, but primarily varies with the square of the air flow (59, 65, 70). The effect of lung volume on lung-sound amplitude has been studied relatively less. Kraman (71) found minor effects of volume changes on lung sounds and only over the upper lobes, although this study was limited by musculoskeletal noise at the extremes of the vital capacity. Investigations by Vanderschoot and Schreur (72) have suggested that lung volume effects on normal lung sounds can be separated from the more prominent effects of airflow.

Normal lung sounds exhibit noticeable amplitude variation across the chest. The extent of this variation and its cause have been investigated during the past three decades. Nairn and Turner-Warwick (73), using radioactive xenon lung scanning, found a strong positive relationship between ventilation and lung sound amplitude, and they concluded that diminished lung sound intensity correlated with poor ventilation. Leblanc and coworkers (64) and Ploysongsang and colleagues (36, 74) also found a relationship between lung sound amplitude and regional ventilation as assessed by radionuclide lung scanning. They concluded that the sounds were loudest over the best ventilated lung units, after correction for sound transmission through the lung. These studies, however, included few locations on the chest and were limited to sounds below 300 Hz.

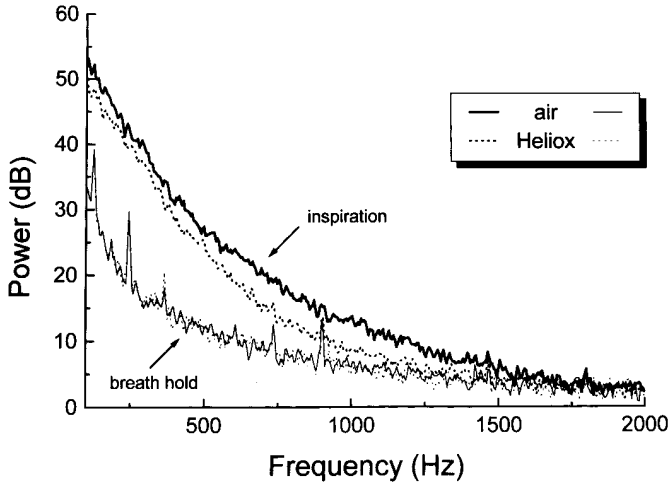

Figure 2. Power spectra of normal lung sounds in a healthy adult male subject, recorded with a contact sensor over the superior right lower lobe. Inspiratory sounds at flows of 1.5 to 2.0 L/s were averaged. Background noise spectra were obtained during breath-hold at end-expiration. The subject breathed air and then Heliox (80% helium/20% O_2). Lung sounds are measurably above background noise at frequencies as high as 1,000 Hz. The most prominent effect of lower gas density and presumably lesser flow turbulence is seen at frequencies above 500 Hz.

The extent of the spatial inhomogeneity of the inspiratory sound was not defined until the early 1980s when researchers began to use chest surface mapping. O'Donnell and Kraman (75) produced linear maps of inspiratory lung sounds in 2 cm increments and found the amplitude to increase toward the base posteriorly, decrease toward the base anteriorly and to remain approximately stable in the horizontal plane. They also found significant right-to-left differences and small scale variations in amplitude, and marked subject-to-subject variation. Dosani and Kraman (76) mapped inspiratory and expiratory lung sounds across 20-cm-square grids over the right and left posterolateral chest wall. This study revealed a heterogeneous distribution of lung sound over the chest and different patterns for expiratory and inspiratory sounds. Although demonstrating these amplitude variations in detail, neither this study nor any other explained the causes for these phenomena.

Body size affects respiratory sounds. Children have a distinct quality of lung sounds, which is generally attributed to acoustic transmission through smaller lungs and thinner chest walls. Laënnec introduced the term of "puerile respiration," which referred primarily to increased sound intensity (77). Acoustic measurements have shown higher median frequencies of normal lung sounds in infants than in older children and adults (78, 79). Pasterkamp and coworkers (60) compared normal lung sounds at flows normalized by body weight in 29 infants, children, and adults. They found that higher median frequencies in infants were explained by less power at low frequencies, whereas the decrease in power toward higher frequencies was similar at all ages. They suggested that the different resonance behavior of a small thorax or less contribution of low frequency muscle noise may explain the difference of normal lung sounds in young children.

The changes in lung sounds imposed by obstructive pulmonary disease are interesting and clinically helpful. Pardee and coworkers (80) used the subjective assessment of four trained examiners to estimate the loudness of lung sounds in 183 patients undergoing pulmonary function testing. They found a strong correlation between the perceived lung sound intensity and the percent-predicted FEV_1. Although lung sounds were insensitive to mild degrees of ventilatory impairment in this study, definitely reduced intensity was a strong indicator of obstructive pulmonary disease, and normal lung sounds virtually excluded the possibility of severe reductions in FEV_1. Recent observations on changes of normal lung sounds during induced airway narrowing (*see Wheezes* in RESPIRATORY SOUNDS section) illustrate that milder degrees of flow obstruction may be detectable by objective acoustic measurements.

It is uncertain what causes the apparent decrease of inspiratory lung sound amplitude in obstructive airway disease. In emphysema, parenchymal destruction could decrease the lung's ability to transmit sound, and diminished airflows could produce less sound than expected. It would seem that the latter explanation is less likely because airflow limitation in emphysema is an expiratory phenomenon. However, in a study to address this question, Schreur and coworkers (81) measured lung sound intensity at equal airflow rates in eight healthy men and in nine men with severe emphysema. They found no significant differences between the lung sound intensity of the two groups and concluded that the perceived decreased lung sound intensity on auscultation of emphysematous patients is due to airflow limitation. This appears to contradict the common finding of a "silent" chest in patients with emphysema who presumably have little inspiratory airflow obstruction. The use of a relatively shallow filter by these investigators may

have permitted muscle sound to influence their results. Nevertheless, these findings are interesting and deserve confirmation.

Gavriely and coworkers (82) evaluated the addition of computerized lung sound analysis to a questionnaire and spirometry in a respiratory health screening program of 493 subjects. Previously defined measurements were applied to detect those lung sound spectra that were outside the normal range. The investigators found that the sensitivity for detection of respiratory disease rose from 71 to 87% by adding the lung sound information. Interestingly, of 24 subjects who had only abnormal lung sounds during the initial screening and were reexamined 12 to 18 mo later, three had developed heart or lung disease. Half of the subjects with normal spirograms but whose questionnaires indicated chronic bronchitis had abnormal lung sounds characterized by sporadic wheezes.

Ploysongsang (83) described a technique of comparing the phase of the lung sound envelope recorded simultaneously from different areas on the right lung. He found that the lung sounds occurred synchronously, with a phase angle less than 5 degrees in all healthy nonsmokers. In 11 of 15 smokers, the phase angle between the sounds exceeded 5 degrees, which was interpreted as reflecting inhomogeneous intraregional ventilation and therefore small airways disease. Jones (84) reported a physical finding in 10 patients with partial stenosis of a main, intermediate, or lobar bronchus. He noted that breath sounds were faint or absent over the lung supplied by the affected airway, whereas transmitted voice sounds were normal at the same location. He attributed this phenomenon to the nondependence of voice sounds on airflow and on the differences in transmission path of the lung and voice sounds.

Normal Tracheal Sounds

Tracheal sounds, heard at the suprasternal notch or at the lateral neck, are currently the topic of significant interest. The tracheal sound signal is strong, covering a wider range of frequencies than lung sounds at the chest wall, with distinctly separate respiratory phases and a close relation to airflow (85). Except in infants and some older subjects with short necks, the placement of sound sensors at the trachea is relatively easy and there is less interference from body hair, garments, etc., compared with chest-wall recording sites. Pulmonary clinicians are interested in tracheal sounds as indicators of upper airway flow obstruction and as the source for qualitative and potentially quantitative assessments of airflow.

The generation of tracheal sounds is primarily related to turbulent air flow in upper airways, including pharynx, glottis, and subglottic regions. Flow turbulence and jet formation at the glottis cause pressure fluctuations within the airway lumen. Sound pressure waves within the airway gas and airway wall motion are likely contributing to the vibrations that reach the neck surface and are recorded as tracheal sounds. Because the distance from the various sound sources in the upper airways to a sensor at the neck is relatively short and without interposition of lung tissue, tracheal sounds are often interpreted as a more pure, less filtered breath sound. Sources within the oropharynx and the hypopharynx contribute to the sound measured at the trachea. Even a pneumotachograph can affect the measurement of tracheal sounds although no significant alteration of lung sounds is detectable at the chest wall (86).

A close relation between airflow and tracheal sound intensity has long been recognized (61). Attempts have been directed to measure airflow by acoustical techniques since this may offer a method for continuous monitoring with minimal interference. Lessard and Wong (87) documented a linear increase of mean frequencies in tracheal sounds up to airflows of 0.75 L/s with little change at higher rates. On the basis of measurements of mean frequencies and mean amplitudes of tracheal sounds, Charbonneau and coworkers (88) were able to estimate airflow in six adult subjects with correlations of 0.79 and 0.94 between real and computed values. In our experience one may improve the accuracy of flow estimation when sounds at higher frequencies, i.e., above 1 kHz are analyzed.

Tracheal sounds have been characterized as broad spectrum noise, covering a frequency range from less than 100 Hz to more than 1,500 Hz, with a sharp drop in power above a cutoff frequency of approximately 800 Hz (58). More recently it has been shown that the spectra of tracheal sounds exhibit peaks and troughs that are related to airway dimensions and dependent on gas density, indicating their origin from resonances within the airways. Sanchez and Pasterkamp (89) analyzed tracheal sounds at standardized airflows in children and adults and found a close relationship between the cutoff frequency and body height. Children with shorter tracheal lengths had higher cutoff frequencies than did adults. In another study, these investigators documented that tracheal sound spectral peaks shift upward by approximately 60% when subjects breathe He-O_2 (68). This is comparable to a vocal register shift that causes the "Mickey Mouse" characteristics of speech in helium environments (*see* Figure 3).

The spectral shape of tracheal sounds is highly variable between subjects but quite reproducible within the same person, reflecting the strong influence of individual airway anatomy (90). Because of this variability, the parametric representation of tracheal sounds is more complex than that of lung sounds (91). Measurements of power within narrow frequency bands and principal component analysis have been successfully applied (92).

Measurements of tracheal sounds provide valuable and in some cases unique information about respiratory health. Apnea monitoring by simple acoustical detection of tracheal sounds is an obvious application and has been successfully ap-

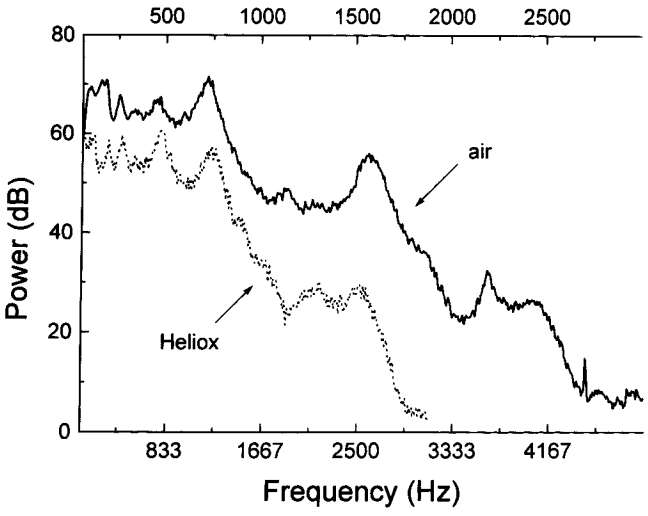

Figure 3. Power spectra of normal tracheal sounds in a healthy man, recorded with a contact sensor at the suprasternal notch. Inspiratory sounds were analyzed as described for Figure 2. Separate frequency axes are used for air and Heliox to facilitate the visual alignment of spectral peaks. Although the change in frequencies with Heliox is not quite linear, the similarity of the spectral shape indicates an upward shift by 55 to 60%, consistent with gas density effects on airway resonances. This is comparable to the well-known effect of Heliox on human speech.

plied in adults (93–95) and in children (96). Tracheal sound microphones have become part of commercial apnea monitoring devices. In most of these applications, however, the focus is on the detection and monitoring of snoring. Pasterkamp and coworkers (90) measured normal tracheal sounds at standardized airflow in awake patients with obstructive sleep apnea (OSA) and in snorers without OSA. Pharyngeal dynamics appeared to be different in the patients with OSA who showed a significantly greater increase of tracheal sound intensity in the supine position. Presumably, this finding is related to structural and functional abnormalities in OSA.

Narrowing below the glottis can also be studied by the analysis of tracheal sounds. Pasterkamp and Sanchez (97) observed that tracheal sound levels reflected the degree of inspiratory flow obstruction in a child with infectious laryngotracheitis. Yonemaru and coworkers (92) found a rise in power at high frequencies in 13 patients with significant tracheal stenosis compared with five control subjects. Greater power at high frequencies was also observed in sounds of airflow through partially obstructed tracheostomy tubes (98).

Adventitious Sounds

Wheezing. Wheezing is probably the most widely used acoustical term in respiratory medicine. Hundreds of publications every year refer to wheeze as an indicator of airway obstruction in infants, as a parameter to gauge the severity of asthma, or as a classifier in epidemiologic surveys, to name just a few examples. Considering the clinical importance of this acoustical sign, there have been few objective studies of wheezing.

Wheezes are musical adventitious lung sounds, also called "continuous" since their duration is much longer than that of "discontinuous" crackles. They may not necessarily extend more than 250 ms, as suggested in an ATS proposal for lung sound nomenclature (99), but they will typically be longer than 80 to 100 ms. Their frequency range extends from less than 100 Hz to more than 1 kHz, and higher frequencies may be measured inside the airways (100).

The pathophysiologic mechanisms that generate wheezing are still not entirely clear. Movement of airway secretions may play a role, but the flutter of airway walls is probably more significant. Grotberg and Davis (101) presented a theoretical model that predicts oscillating wall motion in collapsible tubes at critical airway diameters and at gas velocities greater than those of flow limitation. Their model infers that flow is always limited when wheezing is present but also that flow may be limited without wheeze. In a series of investigations, Gavriely and colleagues tested the occurrence of wheezelike sound pressure oscillations in an isovolume, constant-flow model of animal lung (102), in a physical model of collapsible tubes (103), and in healthy adults during forced expiration (104). They found that flow limitation was necessary for wheezing and that critical transpulmonary pressures were required in normal subjects, presumably to flatten intrathoracic airways downstream from the choke point.

The theoretical model of flutter in flow-limited collapsible tubes predicts that factors such as airway wall thickness, bending stiffness, and longitudinal tension will affect the sound frequency of wheezing (101). Because the airway wall mass is much greater than that of the airway gas, effects of gas density on wheezing are predicted to be minor (101). Clinical observations in support of this prediction were described by Forgacs (61). More recently, the lack of gas density effects on forced expiratory wheezes was confirmed in normal subjects who were breathing at comparable pressures and flows (105). Although the sound frequencies of wheezing do not appear to change, lower gas density has been found to increase the critical pressure and lower the lung volume at which wheeze occurs during forced expiration in normal adults (106).

Forced expiratory wheezes are reproducible in most normal subjects (107), and they have therefore been used to investigate physiologic mechanisms. The limited number of discrete frequency components in forced expiratory wheezes of normal adults, for example, suggests that the source of these wheezes is in the larger airways (108). Considering the appearance of wheeze during forced exhalations in healthy subjects, however, it is not surprising that forced expiratory wheezing lacks specificity and is not useful for the clinical diagnosis of asthma (109–111).

The generation of wheeze during spontaneous or induced airway narrowing in patients with obstructive lung diseases may be different from that during forced expiration. Objective data on the characteristics of natural occurring wheeze, e.g., in acute asthma or bronchiolitis, are sparse. A wide range of sound patterns has been described in children who develop wheezing during bronchial provocation testing (112). Infants in particular may present with a different type of wheezing that is acoustically characterized by complex repetitive sound waves, more similar to rhonchi or snoring than to the typical wheeze of older patients with asthma (113). It is possible that this type of wheezing reflects a different sound source, e.g., secretions in large airways. Acoustic measurements are needed to define a prognostic significance of different wheeze patterns, i.e., to determine if wheezy infants with complex repetitive sounds rather than typical wheezes may be at a lower risk for future manifestations of asthma.

Spontaneous wheeze is often present during inspiration in adults (114) and children (115) with asthma, a situation that healthy subjects cannot reproduce even during forced maneuvers. Regional flow limitation during inspiration is a possibility but has not been proven. Spontaneous wheeze may also occur during tidal breathing, with low transpulmonary pressures and at very low airflows (61, 116). This may suggest a generation mechanism of spontaneous wheeze by vortex-induced vibrations, which requires much lower flow velocities and does not depend on flow limitation (117).

The musical sound of wheezing is easily recognized by ear since it stands out from the noise of normal lung sounds. Wheeze of medium to loud intensity is also easy to notice as sharp peaks in the power spectrum of respiratory sounds. Computer-based detection of wheeze is possible with algorithms that relate the amplitude of these spectral peaks to the average lung sound amplitude (105, 118, 119). When wheezing is faint but still audible, the automated recognition by computer becomes more difficult. Digital sonography (*see* Figure 4) translates the acoustic information into graphic images (120), which allows the visual identification of wheezes even at low intensities.

Quantification of wheeze over time has been used to express wheeze severity relative to flow obstruction. The proportion of the breath cycle occupied by wheezing (Tw/Ttot) was inversely related to the FEV_1 in adult asthmatics with moderate to severe flow obstruction (118) and to FEV_1, maximal midexpiratory flow, and specific conductance in adolescents with exercise-induced bronchospasm (121). Wheeze quantification offers a possibility for noninvasive monitoring in nocturnal asthma. The first promising applications were reported more than 10 yr ago (122, 123). Advances in computer technology and improved methods for acoustical pattern recognition are likely to give us lung sound analyzers for extended observations in clinical respiratory medicine.

As can be expected, the computer analysis of lung sounds allows a reproducible quantification of wheezing in contrast to

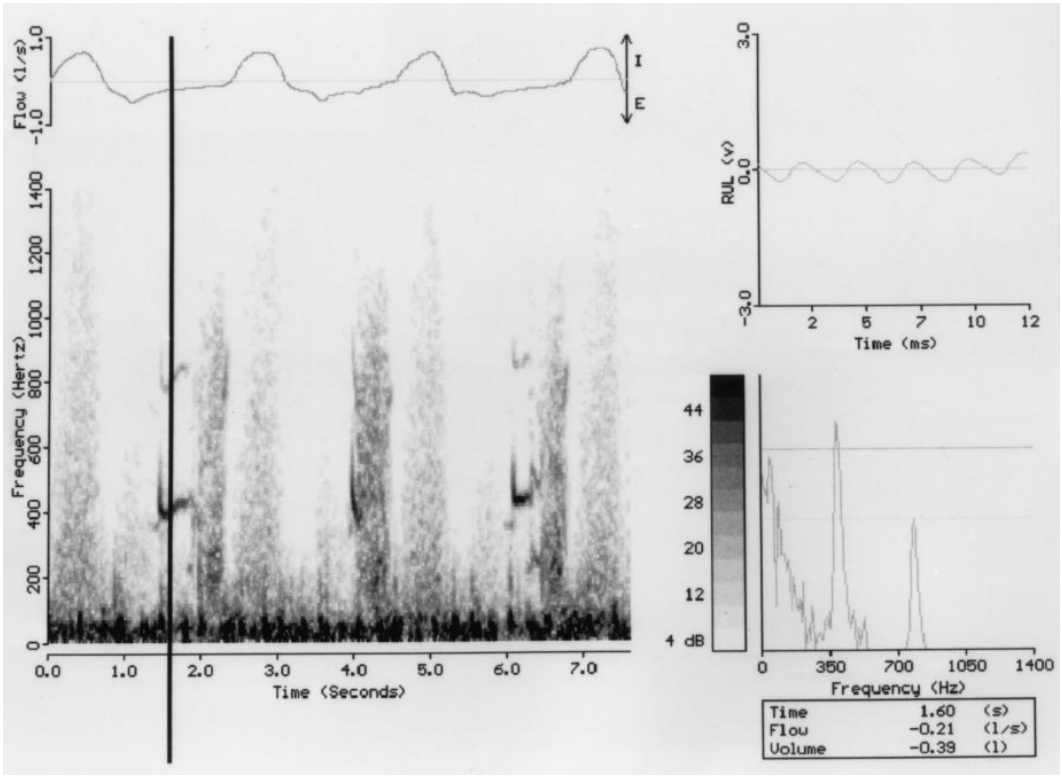

Figure 4. Digital respirosonogram of sounds over the right anterior upper lobe during three complete respiratory cycles in a boy with asthma. Time is on the *horizontal* and frequency on the *vertical axis*. Sound intensity (in decibels) is shown on a gray scale. Airflow is plotted above the sonogram. The *vertical bar* at 1.6 s highlights a segment of expiratory sound that contains wheeze. This segment is shown in detail on the *right*, revealing a sinusoidal wave in the time-amplitude display (*top*) and two harmonically related peaks in the Fourier power spectrum (*bottom*).

subjective auscultation (124). However, the simple detection of wheeze is just as easily achieved by stethoscope. Auscultatory detection of wheeze as an indicator of significant flow obstruction was first advocated in 1988 (125, 126) for bronchial provocation testing of young children. Subsequent reports have also found subjective tracheal auscultation to be useful under these circumstances (127, 128), and a recent editorial in this Journal has emphasized the potential value of lung sound analysis in bronchial provocation testing (3). Beck and coworkers (129) described the use of computerized lung sounds analysis during histamine challenge in 12 children. They found that wheeze appeared in most of these patients before FEV_1 had decreased by 20% or more. Other computer-based studies of lung sounds during bronchial provocation in children with asthma (115) or with cystic fibrosis (130), and in adults exposed to occupational hazards (131) also found wheezing in most subjects who responded positively to the challenge. However, these studies revealed that the sensitivity of wheezing to detect bronchial hyperactivity was only 50 to 75%. Using methacholine inhalation in a recent study of adults with asthma, Spence and coworkers (132) did not find wheeze in three of eight patients, even when the FEV_1 had decreased as low as 44% of the baseline value.

Lung sound analysis confirms the well-recognized finding on subjective auscultation (133, 134) that wheezing is absent in many patients with significant airway obstruction. However, other changes in lung sounds during flow obstruction can be recognized on auscultation, most noticeably a decrease in breath sound intensity. Bohadana and coworkers (135) described the close correlation between a breath sound intensity score and objective indices of flow obstruction, e.g., FEV_1 maximal midexpiratory flow, and specific conductance, in hospitalized patients with obstructive airway disease. More recently, Bohadana and colleagues (136, 137) confirmed these observations by objective measurements of inspiratory breath sound intensity during bronchial provocation. They also showed that a decrease of inspiratory breath sound intensity without wheeze was as common as the appearance of wheeze in patients with a positive response to methacholine, that in most subjects the acoustic findings appeared one or more concentrations before FEV_1 had decreased 20% or more from baseline, and that these observations could be reliably made on subjective auscultation (138).

A decrease in breath sound intensity as the maximal bronchial constriction was also apparent in patients with asthma during histamine challenge who were studied by Anderson and coworkers (139). Lung sound spectra during bronchial obstruction were characterized by a redistribution of power toward higher frequencies and a corresponding upward shift of median frequencies. Similar findings were reported by Pasterkamp and coworkers (140) who recorded breath sounds simultaneously at eight sites on the chest and over the trachea of children during methacholine challenge. Airway narrowing in their subjects was accompanied by changes in lung sounds, with a decrease in power at low frequencies during inspiration (*see* Figure 5) and an increase of power at high frequencies during expiration. These changes already occurred at a decrease in FEV_1 of less than 10% from baseline and were fully

reversed after inhalation of salbutamol. Tracheal sounds were not consistently affected in their patients, similar to observations by Spence and coworkers (141).

A reversible increase of expiratory lung sound intensity was also observed by Schreur and coworkers (142) during allergen-induced early and late asthmatic responses in adults. The mechanisms behind the changes in normal lung sounds during airway narrowing are presently unclear. Changes in regional airflow patterns (142) and the effect of stiffer airway walls on sound transmission at low frequencies (140) have been suggested as explanations for the observed sound spectral changes. These mechanisms need to be better understood because the sensitivity of lung sounds to changes in airway diameter suggests important clinical diagnostic applications. Schreur and coworkers (111), for example, found lung sounds in patients with asthma to be different from those of normal subjects even during episodes with normal lung function. Malmberg and coworkers (143) also reported significant differences in baseline lung sounds between healthy subjects and patients with asthma or COPD.

The inverse relation of breath sound intensity and severity of airflow obstruction is less strong when the acoustical analysis includes breaths with wheeze. Inducing airway narrowing by histamine inhalation in adults and children with asthma, Malmberg and coworkers (144, 145) found breath sound intensity at normalized airflows to be less informative than the upward shift of the median frequency as an indicator of flow obstruction. Most of their subjects developed wheezing, which would tend to shift spectral power to higher frequencies, independent of changes in normal lung sounds. Observations of increased tracheal (145) and lung (146) sound intensity in children with airflow obstruction may also be explained by the inclusion of wheezing in the analysis. When airflow is not standardized (146) it is also possible that the measured increase of breath sound intensity is secondary to an increase of mean inspiratory flows during airway narrowing (147).

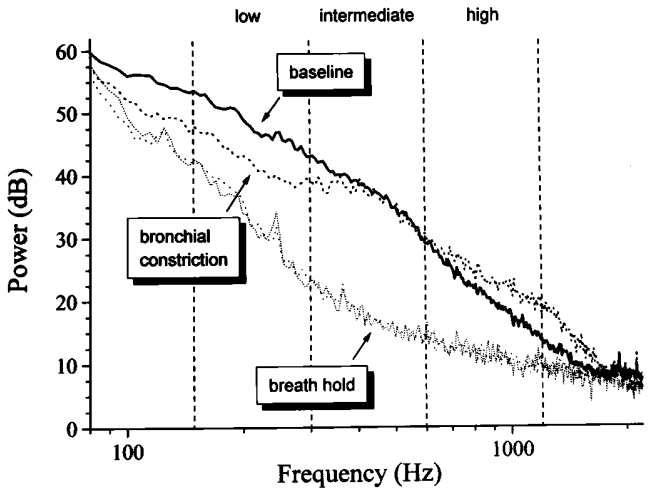

Figure 5. Power spectra of normal lung sounds in a boy with asthma, recorded with a contact sensor over the basal left lower lobe. Inspiratory sounds at flow of 0.8 to 1.2 L/s were averaged. Comparing lung sound spectra during methacholine-induced bronchial constriction (decrease in FEV_1 of 23%) with those at baseline, there is a reduction in power at low frequencies and an increase in power at high frequencies. The changes at low frequencies are orders of magnitude above those at high frequencies and may be perceived on auscultation as decreased breath sound intensity. (Modified from Reference 140.)

Crackles. Of the adventitious lung sounds, crackles are perhaps the most useful for clinical diagnosis. Forgacs (2) described them vividly and expressed notable insight into their mechanism of production and physiologic significance when he wrote: "Crackles are miniature explosions, heard much more often during inspiration than during expiration. Their pattern is remarkably constant and cannot be destroyed by coughing. Except in focal lung disease, the lowermost part of the lung is nearly always the richest source of these sounds. Clearly, these explosive sounds cannot be generated by some evanescent substance—they must come from some structure built into the lung, which is brought into play by a recurrent pressure or volume event, and can be modified by gravity." He was of course referring to what is now generally known as fine crackles. Coarse crackles appear to derive from more pedestrian events, airway secretions, that are perhaps less deserving of eloquent description.

In 1974, Nath and Capel (57) described the salient characteristics of crackles that appear early or late in the inspiratory phase in a group of 56 patients found to have inspiratory crackles and a group of 44 patients selected because they have spirometric evidence of airway obstruction. They found that early crackles tended to be scanty, gravity-independent, usually audible at the mouth, and strongly associated with severe airway obstruction. In bronchiectasis, they tended to be profuse in expiration (148). Late crackles tended to be profuse, gravity-dependent, to rarely transmit to the mouth, and to be associated with restrictive lung disorders. More recently, expiratory crackles have also been documented in patients with restrictive lung disease (149).

In another investigation, Nath and Capel (150) tested the hypothesis that late (or fine) crackles resulted from the explosive opening of airways as Forgacs (2) had proposed. They timed the crackles of six patients with restrictive lung diseases while recording transpulmonary pressure, inspired volume, and inspiratory flow rate. They searched for individual crackles that could be identified on subsequent inspirations (index crackles). Their results revealed a close relationship between the appearance of index crackles and the inspired lung volume and transpulmonary pressure, but not inspiratory time or flow rate. This persuasively supported the theory that explosive opening of small airways, previously held closed by surface forces, causes these sounds. Other investigators, using excised canine lungs (151) and model lungs (152) have also confirmed these findings.

Fredberg and Holford (153) developed a mathematical model of fine crackles in an attempt to predict their behavior. They used simplified assumptions to arrive at a representation that, although idealizing the crackle-generating event and transmission medium, provided a reasonably accurate description of the crackle waveform. Benedetto and coworkers (154) found that each of three microphones attached over the lung bases, separated by approximately 6 cm, recorded only crackles that occurred near it. This suggested that crackles are substantially local phenomena, a conclusion that seems to be consistent with clinical experience.

It is now generally accepted that fine and coarse crackles are associated with different conditions and so have diagnostic importance. Several investigators have examined the waveform of these discrete sounds. Murphy and coworkers (155) first applied this technique and found that fine crackles in patients with fibrotic lung diseases were shorter in duration and period than the coarse crackles of patients with pneumonia. Several investigators have attempted to identify waveform features that would serve to distinguish between fine and coarse crackles. Spectral analysis using the fast Fourier transforma-

tion, which is widely used to study the noiselike lung sounds, is poorly suited to short bursts of sound. Thus, the most commonly used indices are the time duration of the initial deflection and the first two cycles of the waveform, introduced by Holford (156). These parameters appear to do reasonably well in distinguishing fine from coarse crackles even in the absence of knowledge of timing or effect of gravity or cough. Other efforts at refining the discriminatory powers of crackle measurements (56, 157) have not proven superior. It is important to note that sound filters, typically used to suppress low-frequency rumble from muscle noise, can have a major effect on the appearance of crackle waveforms (158).

A number of investigators have examined the usefulness of detecting and classifying crackles to help identify pathologic processes. Although fine crackles have generally been assumed to reflect lung dysfunction, Thacker and Kraman (159) and Workum and coworkers (160) found that about half of the healthy young adults could generate crackles over the anterior lung bases by inhaling slowly from residual volume. Ploysongsang and Schonfeld (161) achieved similar results in subjects breathing air or oxygen at low lung volumes. It is reasonable to conclude that the closure of small airways is the condition that results in crackles. Explosive airway reopening is probably normal once the airway has been closed.

Although many investigators have explored the potential specificity of crackle features and characteristics to certain diseases (151, 162–166), those with established clinical utility appear to be: the presence or absence of crackles to distinguish pulmonary fibrosis (crackles usually prominent) from sarcoidosis (crackles usually scant or absent) (167); fine, late inspiratory crackles indicating fibrotic lung disease and early, coarse crackles indicating obstructive lung disease (162, 168); crackles as an early (perhaps first) sign of asbestosis (169–171), and crackles indicating heart failure (163, 166, 172, 173).

Despite the ease with which an experienced examiner can distinguish fine from coarse crackles by ear, much effort has been expended on developing and validating devices to do this chore automatically (174–180). These schemes have been mostly successful, but none has yet enjoyed wide use, perhaps because they are esoteric, inconvenient, of unproved utility, and not reimbursable, or perhaps clinicians are already satisfied with their ability to classify lung sounds without computer assistance. Nevertheless, there could be a place for such devices in industrial screening, especially in workers at risk for asbestos exposure.

Other respiratory sounds. The terms “lung sound” and “breath sound” are usually used synonymously. However, Forgacs reserved the term breath sound to refer to the sound of breathing heard at the mouth. He and his colleagues (181, 182) described a direct relationship between the loudness of the breath sound and the degree of airway obstruction. They attributed this to the extra turbulence within abnormally narrowed airways of patients with obstructive airway disease. However, breath sounds at the mouth have attracted virtually no further investigational attention since the early 1970s.

The squawk is a short, inspiratory wheeze that occurs primarily in restrictive lung diseases such as idiopathic interstitial fibrosis and allergic alveolitis (183, 184). They have not been well-studied, but squawks appear to always occur along with crackles and are often noted to begin with a crackle, suggesting that they are caused by oscillatory motion in a newly opened airway.

Cough and snoring are not usually considered lung sounds, but they have been studied as such. Cough has been analyzed by many researchers (185–191). These studies defined the components of the normal cough, the effect of asthma or chronic bronchitis on the sound of a cough, and differences in cough sounds in asthma, chronic bronchitis, and acute bronchitis. However, there has been little confirmation of these studies. One could question the usefulness of cough analysis, except that the expanding use of remote telemedicine puts greater emphasis on audio and video for diagnosis. This could prove to be the venue of clinical utility.

Snoring sounds have recently become important as indicators of sleep apnea. In the sleep laboratory, a record of snoring activity can, along with other standard measurements, help distinguish central from obstructive sleep apnea or simple snoring (95, 192, 193). Sound level meters are already part of some in-home sleep apnea monitors (194). More sophisticated devices may be developed to acoustically distinguish between the very common “benign” forms of snoring and those that indicate significant airway compromise.

SUMMARY AND FUTURE DIRECTIONS

Lung sounds have been valuable indicators of respiratory health and disease since ancient times. Laënnec’s stethoscope raised their diagnostic significance but other methods, more sensitive and specific for respiratory assessment, have largely replaced auscultation in clinical pulmonary diagnosis. We are now witnessing the next phase in the evolution of pulmonary assessment by acoustical means. Although the complexity of the respiratory system has slowed the formulation of a comprehensive model of chest and lung acoustics, there have been major advances in understanding lung sounds during the last decade. More powerful yet smaller computers have made digital respiratory sound analysis possible in ambulatory care and at the bedside. The next years will likely bring about an integration of respiratory sound analyzers with more established computer-based spirometry.

The most promising areas for respiratory acoustical measurements are in upper airway diagnosis and monitoring, e.g., in patients with obstructive sleep apnea, in the assessment of lower airway dynamics, e.g., in patients with asthma or bronchiolitis, and in the assessment of regional ventilation. It may soon be possible to determine the site of upper airway obstruction by the analysis of respiratory sounds and to follow the effect of therapy, e.g., the application of continuous positive airway pressure, by acoustical means. Spirometry will remain the standard for assessing lower airway flow obstruction, but lung-sound analysis can extend the assessment to younger patients. Objective characterization of wheezing should improve the epidemiologic understanding of acute and chronic lower airway obstruction, especially in children. Monitoring of regional ventilation by chest surface acoustical topography may now be possible with faster computers that allow the simultaneous processing of sounds from multiple recording sites. The multisite recording of respiratory sounds and of passively transmitted sounds could be particularly useful in critical care, e.g., to monitor regional ventilation and lung water content in intubated patients.

Certain technical challenges need to be resolved before lung sound analysis can enter into routine clinical practice. In particular, a robust and inexpensive sensor for lung sound recording that is relatively immune to ambient noise has yet to be developed. Furthermore, the automated recognition and rejection of artifacts as well as the separate processing of adventitious and basic respiratory sounds need to be refined. However, advances in digital sound processing have already translated into enhancements of the traditional stethoscope, e.g., the use of active noise cancellation in high noise environments (195, 196). Also, the teaching of chest auscultation to

medical students has been enhanced by computer-aided instruction (197–199). Lung sounds of high fidelity can be transferred via telecommunication, as interested readers with access to the Internet can verify.[2]

The sound repertoire of the lung may indeed be limited when heard through a stethoscope, but it clearly exhibits a much wider range of information content when digitally analyzed. Computer analysis is now reaching beyond the capabilities of the human ear, e.g., to resolve changes in respiratory sounds during narrowing of the intrathoracic- or extrathoracic airways. With the disappearance of auscultation as the standard to judge the clinical significance of acoustical findings, it becomes even more important to integrate lung sound analysis and traditional measurements of respiratory mechanics. One should keep in mind, however, that voice recognition of continuous speech, an easy task for the human listener, is still not possible on standard computers after decades of research and substantial investment from industry. Thus, one should not expect that computer-based lung sound analyzers will replace the stethoscope-bearing clinician anytime soon, but they will expand the noninvasive diagnostic capabilities in respiratory medicine.

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[2] The R.A.L.E. Repository. (http://www.umanitoba.ca/faculties/medicine/ILSA/sounds/ralrepos.html)

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