Lung sounds

In 1819, on the basis of observations with his newly invented stethoscope, Laennec published *L’Auscultation Médiate*.1 In this masterly thesis he described and classified all the major lung sounds and suggested how they might correlate with lung abnormalities. Within three years the Scottish physician John Forbes translated Laennec’s thesis into English1 and by the end of the nineteenth century the subjective analysis of lung sounds was well established in that the stethoscope had become the hallmark of the physician.

Detailed investigation of sound waveforms was impossible until the development of analogue electronics. In the 1960s Paul Forgacs used simple microphones, amplifiers, oscilloscopes, and tape recorders to obtain the first systematic measurements of respiratory sounds. His monograph Lung Sounds2 is still the basis of the modern understanding and nomenclature of lung sounds. With modern digital signal processing techniques sound waveforms can now be reliably analysed by frequency, amplitude, and time. Frequency analysis is most commonly achieved by various implementations of the fast Fourier transform. This technique has proved invaluable but nevertheless does have drawbacks as it analyses a signal by comparing it with a set of preformed sine waves. The frequencies of these sine waves are fixed and only signal components that coincide exactly with these fixed frequencies are clearly identified. Signal components at intermediate frequencies must be represented by combinations of adjacent sine waves, producing the artefact of spectral leakage. The effects of spectral leakage can be reduced by a process of “windowing,” which modifies each signal section before the fast Fourier transform analysis. Newer techniques of spectral analysis, based on sophisticated digital filters, are now being investigated, which have the potential for data compression and modelling the underlying mechanism of the sound production.3,4

Many other electronic techniques are now being used, including noise reduction by a range of digital filters; multiple surface microphones to map the sources of a sound within the lung; automatic pattern recognition; self organising feature maps using artificial neural networks; and the capture, storage, and three dimensional spectrographic analysis of sound and dimensional data.

Nomenclature of lung sounds has been mainly descriptive, frequently relying on the use of onomatopoeic words. Unfortunately, Laennec’s carefully constructed original classification was extensively modified during translation into English and this resulted in the use by other authors of the same terms to describe different sounds. To clarify the position, the International Lung Sounds Association in 1976 agreed a simple classification based on the analysis of the various waveforms, and this was subsequently adopted by the American Thoracic Association.5,6

The origin of low frequency vesicular sounds heard with the stethoscope at the chest wall is still incompletely understood, but it is generally accepted that breath sounds originate from turbulent air flow in larger airways and that the higher frequencies are attenuated by the biological filtering properties of the lung and chest wall.7 Gavriely’s work on sound generation and transmission in dogs suggested that expiratory sounds are from a more central source than the inspiratory sounds8 and this theory is supported by the recent work of Kraman and Wang using lung casts.9

Wheeze is continuous musical sounds lasting more than 250 ms. They are acoustic waves produced by airflow induced oscillation of the airway walls. Forgacs9 showed that the pitch of wheezing was largely independent of the density of inhaled gas and so not related to resonance within the airways. He concluded that the pitch was the result of the mass and elasticity of the vibrating bronchus and surrounding tissue. More recent work, using forced wheeze in normal subjects breathing air10 and helium mixture,11 suggests that the generation of sounds does indeed occur at points of critical airway narrowing and that it is related to airflow limitation. Experiments in which Grotberg and Gavriely12 and Gavriely et al13 used collapsible tubes and mathematical modelling suggest that wheezes are due to complex interaction of fluid and elastic forces and can be explained by the wave speed theory of air flow limitation.

The development of a non-invasive method of estimating airflow obstruction by analysing wheeze or other components of lung sounds has been an attractive goal. Forgacs14 suggested that the number of individual wheezes may correlate with flow rates, but clinical wheeze scoring has been disappointing. Baughman and Loudon15 showed that the duration of wheezing in expiration was significantly related to FEV1, and subsequently this technique was developed to estimate changes in airflow obstruction during sleep in patients with asthma.16 Anderson et al17 investigated patients with asthma undergoing a histamine challenge test and found a loose relation between increasing median frequency of the power spectrum of breath sounds and declining FEV1. Spence et al report a similar relation between FEV1, and the median frequency of tracheal breath sounds on page 680 of this issue of Thorax.18

Other investigators have examined the relation between different levels of airflow at the mouth and the spectral content of breath sounds. In normal subjects Kraman19 found a linear relation between airflow and lung sound amplitude, and Shykoff20 reported that breath sound amplitude varied directly with the square of flow in subjects breathing either normally or through resistors. Soufflet et al21 have related both sound amplitude and mean spectral frequency to airflow in normal subjects. In patients with emphysema the subjective intensity of breath sounds has been correlated with regional ventilation22 and more recently this relationship has been confirmed with objective lung sound analysis.23 In this issue of Thorax (p 674) Schreuer et al24 report that the intensity of breath sounds in normal and emphysematous patients breathing with the same flow rates was similar and conclude that the diminished breath sounds in emphysema can be explained by reduced airflow.

Crackles are intermittent explosive sounds lasting less than 20 ms.11 Time expanded waveform techniques have shown them to consist of an initial deflection followed by a few rapidly decaying oscillations. Crackles can be characterised objectively by measuring various subdivisions of the waveforms (initial deflection width, two-cycle duration,13 and largest deflection measurements)25 and also, as reported recently in Thorax, by their spectral characteristics.25 Such analysis of crackle waveforms is laborious and classification by digital filtering techniques26 and computerised automatic counting27 have been developed. The origin of crackles is still not fully understood. Although the original idea of fluid bubbling through copious secretions may be the explanation in some patients, most crackles are caused by the sudden opening of a succession of smaller airways, the acoustic wave being produced either by equalisation of the downstream and upstream pressures or by sudden altera-
tions in the tensions of airway walls. Fredberg and Holford devised an intriguing mathematical model (the “stress quadrupole” theory) predicting many of the characteristics of crackles.

Clinical detection and characterisation of crackles has proved useful despite the incomplete understanding of their origins. In chronic obstructive airways disease and bronchiectasis crackles are coarse and occur early in inspiration and also in expiration. In contrast, the crackles of interstitial fibrosis are fine and there is evidence that in early disease they are end inspiratory and heard only at the lung bases. As disease severity increases they become more widely distributed and eventually occupy the whole of inspiration.

In fibrosing alveolitis inspiratory crackles have also been described; here the initial wave polarity is reversed, which suggests that inspiratory and expiratory crackles have wavefronts moving in opposite directions. Considerable interest has been generated by crackles in asbestosis; these can be heard very early in the development of pulmonary fibrosis and may provide a useful method of screening exposed workers. In many types of pulmonary fibrosis a short inspiratory musical sound, the inspiratory “squawk,” is occasionally heard. This is particularly common in extrinsic allergic alveolitis, where it acts as a useful clinical marker.

The complex acoustic signal arising within the lungs almost certainly contains much information about the underlying pathophysiology of the lungs and has the potential for being developed into a powerful diagnostic tool. Much has been achieved since Laennec’s original discovery but a multidisciplinary approach is required to enable techniques to be standardised and physical, mathematical, and electronic models constructed to increase basic understanding of the interaction between physiological and acoustic mechanisms. Clinically useful developments may soon be available, including the computerised display and interpretation of lung sounds and the effective long term, non-invasive remote monitoring of asthma and apnoea.

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