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Crackles: recording, analysis and clinical significance

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Crackles: recording, analysis and clinical significance. P. Piirilä, A.R.A. Sovijärvi. ©ERS Journals Ltd 1995.
ABSTRACT: Crackles are short interrupted breath sounds usually associated with pulmonary disorders. According to present opinion, a crackle is generated when an abnormally closed airway opens during inspiration or closes at the end of expiration. The timing of crackles in breathing cycles can be assessed with phonopneumography, their duration with time-expanded waveform analysis, and their pitch with analysis of frequency spectra. The timing, pitch and waveform of crackles are different in pulmonary disorders reflecting different pulmonary pathophysiology.

This review deals with the genesis, auscultation, recording and analysis of crackles, with an emphasis on modern signal-processing methods.
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Crackles are short explosive breath sounds, usually associated with pulmonary disorders and heart failure. Crackling sounds have been shown to vary in character in different pulmonary diseases when listened to through a stethoscope. The auscultatory findings have been shown to be related to histological alterations of pulmonary tissue and functional impairment in interstitial pulmonary diseases [1].

The study of crackling lung sounds began with the invention of the stethoscope by Laënnec in 1816. "Auscultation Mediate" a treatise by LAENNEC [2] contains a description of the auscultatory findings of normal and adventitious pulmonary sounds, which he related to autopsy findings. Perhaps the first graphic presentation of recorded crackling sounds was that by CABOT and DODGE [3] in 1925. Studying the frequency characteristics of heart and lung sounds by using bandpass filters, they showed that crackles defined as fine in pulmonary auscultation contained higher frequencies than those defined as coarse. In 1955, MCKUSICK *et al.* [4] displayed sound spectrograms of normal, vesicular, tracheal, bronchovesicular, and asthmatic sounds. They also presented spectrograms of crackles classified in auscultation as moist or dry.

The first phonopneumographic recordings of crackling sounds were presented in the 1970s [5–7]. FORGACS [5, 8] stated that pulmonary crackles were generated during inspiration as a result of sudden opening of the airways. Therefore, the size and properties of the airways could affect the character of a crackle. Since the presentation of this theory, advanced studies on crackling sounds have been conducted using sound spectrography [9, 10], phonopneumography [6, 7], time-expanded waveform display [11–14] and Fast Fourier Transform analysis [11, 13, 14]. These studies have not been able to refute Forgacs' theory.

This review article deals with present theories of the genesis of crackles, methods for auscultation, recording and analysis of crackles, and clinical applications of crackle studies.

Genesis of crackle sounds

In the past, it was commonly believed that crackles were generated by bubbling of air in the airways or from the motion of intraluminal secretions. The persistence of crackles after coughing, and their repetitive nature in consecutive inspirations, contradict this theory [5–7]. Crackles are nowadays thought to be caused by the sudden opening of abnormally closed airways. According to the theory of FORGACS [5, 8], in inspiration, a gradient of gas pressure is developed across airways that are collapsed during expiration. A popping crackle sound is generated when a closed airway suddenly opens during inspiration or closes during expiration, thus inducing a rapid equalization of gas pressures and a pressure wave. Each crackle may represent an abrupt opening or closing of a single airway. This theory has received further support from the work of NATH and CAPEL [7], who found that the individual crackles occurred at similar inspired volumes and transpulmonary pressures during consecutive inspirations.

FREDBERG and HOLFORD [15] have postulated a mathematical theory of stress-relaxation quadrupoles, which is in accordance with the sudden opening theory of

FORGACS [5, 8]. However, their theory assumes that when an airway opens, sudden changes occur in elastic stress, *i.e.* increase in stress and its relaxation in and near the airway walls. Accordingly, dynamic events in the airway wall and in their surroundings rather than differences in gas pressure would create the sound waveform of a crackle, which would spread from its site of generation.

Crackles usually occur when the elastic recoil pressure of the lungs is increased, or there is inflammation or oedema in the lungs. The character of the crackling sounds possibly depends on the diameter of the airways which are closing and opening, and this in turn depends on the pathophysiology of the surrounding tissue [5, 15]. It has been shown experimentally that the characteristics of crackles produced in airway models vary with the size of the airways; smaller airways have been shown to produce crackles of shorter duration [15]. Early inspiratory crackles are probably generated in more proximal airways than late inspiratory crackles. In chronic bronchitis and emphysema, collapse of the lobar bronchi may occur at end-expiration due to loss of elastic recoil and bronchial support [16]. In fibrosis, the process usually involves peripheral airways, the distribution of airway closure being gravity-dependent. The count and distribution of crackles also depend on the disease process and its severity [1].

Crackles have also been detected in the breath sounds of normal subjects [1, 17, 18]. THACKER and KRAMAN [19] found crackles in the lung sounds of normal volunteers recorded over the anterior chest during inspiration starting from residual volume level, but not when they inspired from functional residual capacity level. These crackles varied considerably in pitch and duration. Thus, crackles may not always be an indication of pathological pulmonary processes, but their occurrence depends also on the expired volumes during auscultation. The basal alveoli of a normal lung, deflated at the residual volume, inflate in late inspiration; the basal airways are the first to close towards the end of expiration [20]. Therefore, crackles will most often appear at the early stage of a disease at basal areas of the lungs. With ageing, when the elastic recoil pressure of the lungs decreases the incidence of crackling sounds tends to increase [21].

Crackles may disappear during pulmonary auscultation procedures repeated several times at short intervals, or during pulmonary function tests after vital capacity manoeuvres. This is possibly due to the effect of lung expansion. When regions of deflated airways open, the number of crackles is decreased.

The auscultation of crackling lung sounds

LAËNNEC [2] described the following types of crackles listened through his wooden cylinder stethoscope: 1) humid or crepitous crackles; 2) mucous or gurgling crackles; 3) dry sonorous crackles; and 4) dry sibilous or hissing crackles. The characterization of crackles according to the "dryness" or "humidity" of the sounds is no longer recommended. The modern classification is based on the pitch and duration of the crackling sounds. In the present terminology of auscultation, the crackles belong to adventitious (not normally occurring) sounds. In contrast to continuous (wheezing) adventitious sounds they are called discontinuous sounds. The terms fine crackles to characterize crackles with high frequency components and short duration, and coarse crackles for crackles with lower frequency and longer duration are recommended by the American Thoracic Society (ATS) (table 1) [22]. Also, the timing of crackles in the respiratory cycles must be characterized. Crackles may appear in early, mid- or end-inspiration or expiration.

Because of the very short duration and often low intensity of the crackles, their discrimination and characterization by normal auscultation is difficult. Moreover, the linguistic descriptions of crackle sounds by different physicians are various. There are differences in physicians' hearing properties as well as in the stethoscopes. The maximal sensitivity of the ear is normally at the frequency range of 1–2 kHz. Below 1,000 Hz, and especially below 500 Hz [23], the sensitivity of the ear falls sharply. The frequency response of stethoscopes varies greatly depending on the type of stethoscope, and there are also great individual differences between them. Stethoscopes have been shown to considerably attenuate all sound frequencies in a nonuniform way in several studies where both the diaphragm and bell chest pieces have been used [24–28].

There are also several other factors which influence the hearing of crackling sounds. The sensitivity of the ear to different sound frequencies depends on the relative loudness of the sound [29, 30]. The ability of the ear is poor in recognizing a tone of very short duration [31]. There are also difficulties in hearing short sounds separated from each other by very short intervals. The minimum audible time interval for the separation of sounds also depend on the frequency composition and intensity of the sounds to be separated [32].

The low pitched components of a complex sound appear more prominent to the observer when the sound is loud, because low pitched sounds mask the higher pitched sounds; and *vice versa*, when the intensity of the sound

Table 1. – The classification of crackles according to ATS [22] based on lung sound analysis results: sound recording and filtration procedure affect the waveform characteristics and crackle sound parameter durations

	Intensity	Pitch	2CD duration	IDW duration	Other terms
Coarse crackles	Loud	Low	About 10 ms	About 1.5 ms	Bubbling rales, coarse crepitations
Fine crackles	Less loud	Higher	<5 ms	About 0.7 ms	Fine crepitations

ATS: American Thoracic Society; 2CD: two cycle duration; IDW: initial deflection width.

attenuates, the high frequency components of the sound accentuate [33, 34]. Moreover, when a sound of relatively great intensity precedes a sound of low intensity, the greater intensity has a tendency to fatigue the ear and the low intensity sound gets masked; the ear, thus, fails to sense some sounds [30, 35].

Semantic factors, clinical routines, and the physician's personality influence the description of auscultatory findings. Therefore, the interobserver variability between clinicians in describing the auscultatory finding of crackles has been great. When determining the presence or absence of abnormal lung sounds in cotton workers, SHILLING *et al.* [36] reported that two observers had an interobserver error of 24%. In a study by SMYLLIE *et al.* [37], the interobserver repeatability of respiratory physical signs was about midway between complete agreement and chance. HUDSON *et al.* [38] reported 47% agreement of three observers to assess whether the crackles were fine, medium or coarse, and moist or dry. In these studies, the observers were not well-trained.

In a study by WORKUM *et al.* [18], especially trained observers assessed the presence or absence of crackles by listening to tape-recorded breathing sounds of asbestos-exposed workers. Two observers' agreement was 89%, and four observers' agreement 81%. In a study by the authors of this review [39], two observers tried to classify crackles by listening to tape-recorded crackling lung sounds which had previously been analysed by computerized waveform analysis; 10 samples included fine crackles of fibrosing alveolitis and 10 coarse crackles of heart failure. The observer agreement on the coarseness or fineness of the crackles was 60%. The percentage of wrong classification was higher, 70 and 90% in samples from patients with fibrosing alveolitis, and 80 and 60% from patients with bronchiectasis, respectively. These studies elucidate the great difficulties and variations in the description of auscultatory findings. In lung auscultation, the agreement is better when there is only a question of the presence or absence of abnormal sounds; the agreement is poorer the more characteristics that have to be taken into account, such as grading or timing of sounds.

Recording and analysis of crackling sounds

The lung sound recording systems usually consist of a pneumotachograph for airflow or volume measurements, and microphones and a tape-recorder for sound capturing. In phonopneumography the sound intensity signal is displayed simultaneously with the airflow (or air volume) signal. The crackling sounds can usually be visualized as transient peaks in the sound signal (fig. 1). Phonopneumographic recording serves as the basic method in analysis of crackle sound timing; the time or volume of occurrence of the crackles can be accurately measured. The following timing parameters can be used: beginning of crackling (time interval from the beginning of a respiratory cycle to the first crackle in relation to the duration of the whole respiratory cycle) and end-point of crackling (the distance from zero flow to the last crackle, respectively) [13].
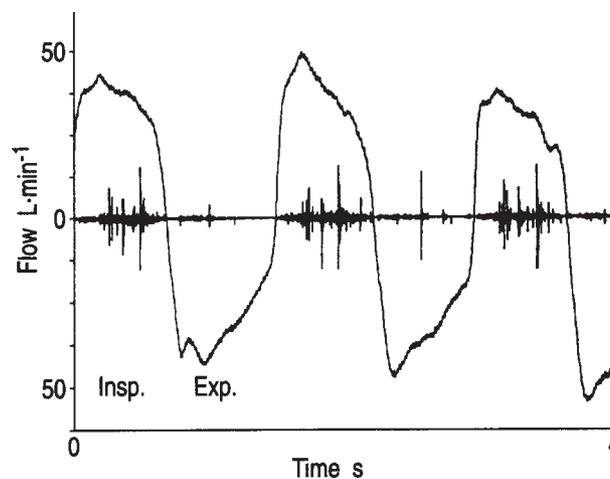

Fig. 1. – A phonopneumogram (simultaneous presentation of lung sound and airflow signals) of the breath sounds of a patient with fibrosing alveolitis, showing late inspiratory (Insp) crackling as sharp peaks in the sound signal. Some expiratory (Exp) crackles are also shown. (Reproduced by permission of *Bull Eur Physiopathol Respir*, DALMASSO *et al.* [11]).

Phonocardiographic microphones have been used in the first lung sound recording systems [6, 7, 40, 41], but their low sensitivity and their narrow frequency response band limit their application for further lung sound analysis. DRUZGALSKI *et al.* [42] found air-coupled condenser microphones suitable for lung sound recordings. At present, piezoelectric microphones are also used [43–46]. The techniques of attachment and placing of microphones have varied; some researchers have used a belt [13] or rubber tape attachments [46, 47], and some hold microphones by the hand [48].

Sound signals are usually recorded on magnetic tape or stored in digital form in computer memory. In lung sound analysis, the frequency response band of the whole recording and signal processing equipment is highly important. Direct mode tape-recorders have been used in some studies [49–53]. The frequency response of direct mode recorders is variable; they attenuate low frequency components like a high-pass filter with a cut-off frequency of 50–200 Hz. In many studies, frequency modulation (FM) tape recorders are used, with adequate frequency response both at low and high frequency bands.

High-pass filters are frequently used in lung sound recordings to diminish low frequency noise, possibly generated by muscles, large blood vessels and the heart [48, 54, 55]. There has been a controversy about the clinical significance of the low frequency sounds recorded, since some researchers have suggested that the low frequencies may also originate in the lungs [56, 57]. The high-pass cut-off frequency has varied markedly in recent studies: no prefiltration [56, 57], >50 Hz [48], >75 Hz [44], >100 Hz [13, 58], >200 Hz [59], and >600 Hz [6, 7]. In several studies, different bandpass filters have been applied: 50–2,000 Hz [43], 100–3,000 Hz [10], 150–350 Hz [47], and 150–700 Hz [60].

FORGACS [5] used a somewhat expanded time scale for displaying crackling sounds. In this way, late and early crackles and their repetitive nature could be demonstrated. MURPHY *et al.* [49] expanded the time scale to

800 mm·s^{-1}, thus allowing a detailed analysis of the waveform of crackles. They demonstrated the waveforms of normal vesicular and tracheal inspiratory sounds, rhonchi, wheezing sounds and inspiratory crackles. At the moment, time expansion up to 2,000–6,000 mm·s^{-1} is commonly used in lung sound research.

In the sonographic analysis, a frequency spectrum of the sound is displayed in time domain [4, 9, 10, 51]. This method has not yet been widely used in the analysis of crackling sounds. Recently, however, computerized sonogram analyses of crackling sounds have been published [46, 61–63]. In sonographic display, a crackle appears as a sharp and high intensity peak with frequency components up to 4 kHz [11, 61, 62].

Since the 1970s, computer-aided systems have been used for lung sound analysis using Fast Fourier Transform (FFT) spectrum analysis [64]. One of the first studies using FFT spectrum analysis was conducted in 1973 by MURPHY and SORENSEN [17], who studied crackles in asbestosis patients. In FFT spectrum analysis, the amplitude of the sound signal is presented in frequency domain. Several studies of crackles have been carried out on the FFT analysis [11, 13, 14, 65]. Serial FFT spectra have clearly demonstrated high frequency components of crackles, *e.g.* in patients with pneumonia [14].

The variation of the second quartile frequency of averaged FFT spectrum (F50) in lung sound samples recorded 1–3 days apart, has recently been shown to be slightly higher in patients with fibrosing alveolitis, having crackles in their breath sounds, than in healthy subjects [66].

Crackle waveform

Time-expanded waveform analysis has revealed that a crackle begins with a short initial deflection followed by deflections with greater amplitude. A true crackle has an amplitude of deflection which is two times as large as the amplitude of the background [67]. HOLFORD [12] studied crackles classified into coarse or fine by a pulmonary specialist. He measured the duration of the initial deflection width (IDW) and the two cycle duration (2CD) determined as the time from the beginning of IDW to the point where the crackle has completed two cycles (fig. 2). IDW and 2CD have been used to classify crackles as coarse or fine (fig. 3). Suggestions for reference values for fine and coarse crackles have also been presented [22, 65, 68]; according to ATS suggestion [22], the mean durations of IDW and 2CD of fine crackles are 0.7 and 5 ms and those of coarse crackles 1.5 ms and 10 ms, respectively (table 1).

HOEVERS and LOUDON [69] have suggested a third crackle waveform index, the largest deflection width (LDW). This is defined as the duration of the deflection with the largest amplitude in the crackle waveform. They found that LDW was a good parameter in classifying crackles into fine and coarse, even better than IDW. LDW has also proved to be a good index to classify crackles for diagnostic purposes [13], or in the follow-up of a pulmonary disease [14]. Sometimes, it is difficult to determine
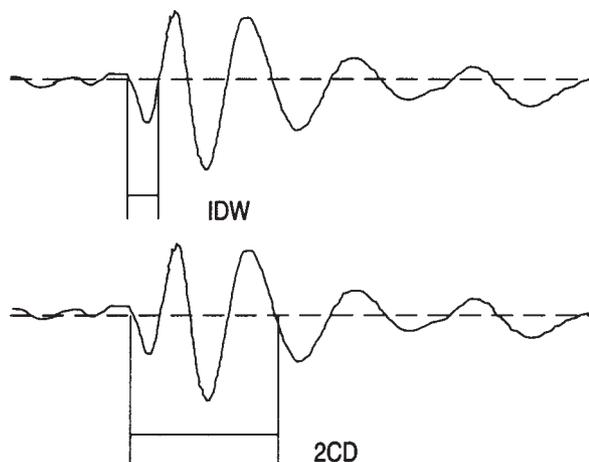

Fig. 2. – The morphology of one crackle in time-expanded waveform display in high-pass filtered recording. The crackle parameters: IDW = initial deflection width; 2CD = two-cycle duration, are presented. (Reproduced by permission of the Massachusetts Institute of Technology, Holford [12]).
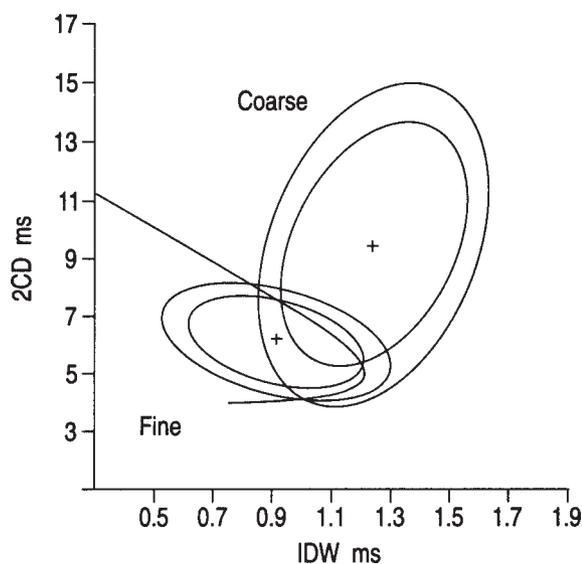

Fig. 3. – The discrimination of crackles into fine and coarse by using two-cycle duration (2CD) and initial deflection width (IDW). (Reproduced by permission of the Massachusetts Institute of Technology, Holford [12]).

the exact beginning of a crackle needed for the measurement of IDW and 2CD. This difficulty does not arise in the measurement of LDW.

The duration of IDW and 2CD has varied greatly in different studies [13, 14, 22, 65, 68]. Since the high-pass filtration in these studies also varied, we conducted experiments in which inspiratory crackling sounds in a patient with silicoasbestosis were recorded in an anechoic room without any high-pass filtration [58]. The waveform of the crackles recorded in this way was simply monopolar or bipolar without any further deflections. When progressively filtered with different high-pass levels, the waveform of the crackles gradually reached the morphology presented in some earlier studies [12, 49] (fig. 4). Sometimes, the polarity of the initial deflections of crackles also changed with filtration of the sound signal. Based on this experiment, it was apparent that the waveform of a
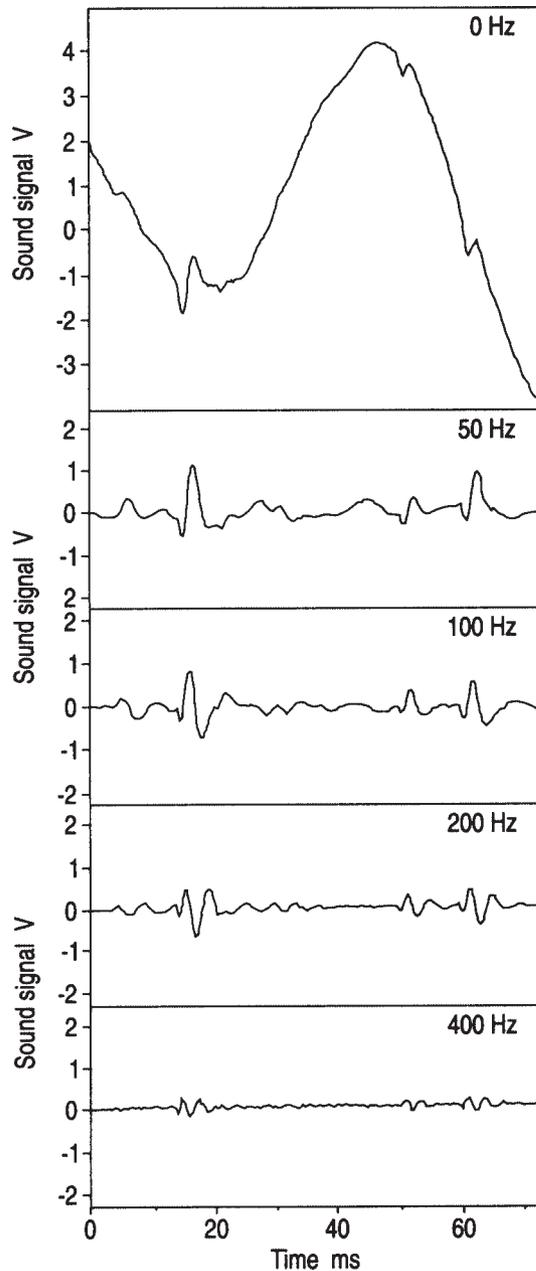
Fig. 4. – The end-inspiratory fine crackles of a patient with silicoasbestosis in time-expanded waveform display without any highpass filtering (0 Hz). The filtration effect of progressive filtering with 50, 100, 200 and 400 Hz analogue filtration is shown below [61].

thus fitting in with Forgacs' theory of the genesis of crackles.

However, it is very difficult to study crackles without any high-pass filtration, due to the slow background oscillation of the sound pressure. International recommendations for filtration have not yet been given. A European Community (EC) project (Computerized Respiratory Sound Analysis (CORSA) project) of seven European countries to develop guidelines for standardized recording and analysis of respiratory sounds, led by A.R.A. Sovijärvi, has recently been started. When the details of filtration and the frequency characteristics of the recording devices are indicated and standardized, high-pass filtered respiratory sound signals may be used to determine IDW, 2CD and LDW for the assessment of different pulmonary diseases. To enhance the diagnostic discrimination power, simultaneous analysis of the waveform and timing characteristics of crackles, *i.e.* two-dimensional discriminant analysis, can be applied, according to our recent discovery [70].

Automatic counting of crackles

The number of crackling sounds possibly indicates the severity of the pulmonary disease at least in fibrosing lung diseases [1, 71]. Because the human ear may fail to detect the right number and character of the crackles, automatic counting methods have been developed [62, 67]. In the sonogram analysis, crackles can be distinguished as narrow peaks of high frequency and short duration. Spectral and waveform differences of the crackle sounds compared to the surrounding vesicular lung sound signal make identification of the crackles possible.

An automatic crackle counting method by MURPHY *et al.* [67] showed a good agreement with the counts obtained by two expert observers who listened to tape-recorded sounds (r=0.74), and with the crackle counts detected by visual inspection in time-expanded waveform display (r=0.85). Another automatic method to detect and count crackles has recently been published by the authors of this review [62]. The method was based on the analysis of spectral stationarity of the sound signal. It was validated by the counting of crackles in time-expanded waveform display by two expert observers. The method indicated a high sensitivity (89% in fibrosing alveolitis and 80% in bronchiectasis) and a high positive predictivity (88% in fibrosing alveolitis and 83% in bronchiectasis). The correlation coefficient between automatic crackle counting and counts by the observers was 0.86 in fibrosing alveolitis and 0.93 in bronchiectasis.

The automatic counting methods enable large sound materials to be studied with the same standardized detecting and counting criteria. Automatic determination of the timing of crackles and the measurement of waveform indices will be possible in the near future. With this type of method, the possibility of analysing lung sounds for differential diagnosis of pulmonary diseases will be enhanced.

crackle with several oscillations is a consequence of high-pass filtration, actually an artefact, and that the original waveform is simpler [58]. Also, the great variability in the duration of the crackle parameters reported in the literature in similar diseases could be explained by the differences in the filtration or recording methods used.

In the filtration study cited above [58], it was also found that, in auscultation, only inspiratory crackling sounds could be recognized. However, in the nonfiltered tape-recorded lung sound signal, there were similar deflections in expiration also but in the opposite direction to inspiration. These expiratory deflections may represent sounds generated by airways closing at the end of expiration,

Clinical applications of crackles

The waveform, timing, number and regional distribution of crackles are associated with the severity and character of the underlying pulmonary disorder. The assessment of crackle is beneficial for early diagnosis and follow-up of the disease. In diffuse interstitial pulmonary diseases, the crackles first appear in the basal areas of the lungs and later, as the disease progresses, also in the upper zones of the lungs. In heart failure with pulmonary congestion, crackles are best heard in the lower zones of the lungs, but in profuse heart failure over the entire chest wall; this phenomenon had already been reported by LAËNNEC [2]. The appearance of crackles has been considered to be an early sign of pulmonary impairment in asbestosis [1, 10, 72]; in early disease, crackles have been described to appear first in the basal pulmonary areas, later successively higher up on the thorax [73]. On rare occasions, *e.g.* in severe heart insufficiency, crackles are audible at the mouth [5].

Crackles in pulmonary fibrosis

NATH and CAPEL [6, 7] showed that late inspiratory crackles occurred in restrictive pulmonary diseases. DALMASSO *et al.* [11] described the timing of crackles in fibrosing alveolitis; the crackles started early or at midphase in inspiration but continued to the end of inspiration. In another study [13], crackling in fibrosing alveolitis occurred late during the inspiratory cycle; there was no overlap with the early timing of the crackles in COPD (fig. 5).

CHOWDHURY *et al.* [74] found spectral differences between normal subjects and patients with fibrotic lesions following tuberculosis. In the study by KUDOH *et al.* [9], the frequency components of crackles in diffuse interstitial pneumonia were as high as 2,000–5,000 Hz measured in sonogram spectrography, whereas the frequency of crackles in chronic bronchitis, bronchiectasis or diffuse panbronchiolitis was lower. DALMASSO *et al.* [11] used FFT spectrum analysis in their study of cryptogenic fibrosing alveolitis, and found the frequency of crackles to range 100–2,000 Hz, the spectral energy being below 1,000 Hz. In our recent studies [13], the crackles in fibrosing alveolitis differed markedly from three other pulmonary diseases, bronchiectasis, COPD and heart failure; the crackles in fibrosing alveolitis were the shortest in duration. The 2CD was below 8 ms and the frequency of maximum intensity was about 200 Hz.

Crackles in asbestosis

In patients with pulmonary asbestosis, fine crackles have been described, assessed by pulmonary auscultation with a stethoscope [5, 17, 72, 75]. The presence of crackles has been shown to correlate with roentgenological and histological honeycombing [1]. The incidence of crackles in asbestosis is also correlated with the duration of exposure to dust containing asbestos [10]. SHIRAI *et al.* [10] demonstrated end-inspiratory timing of the
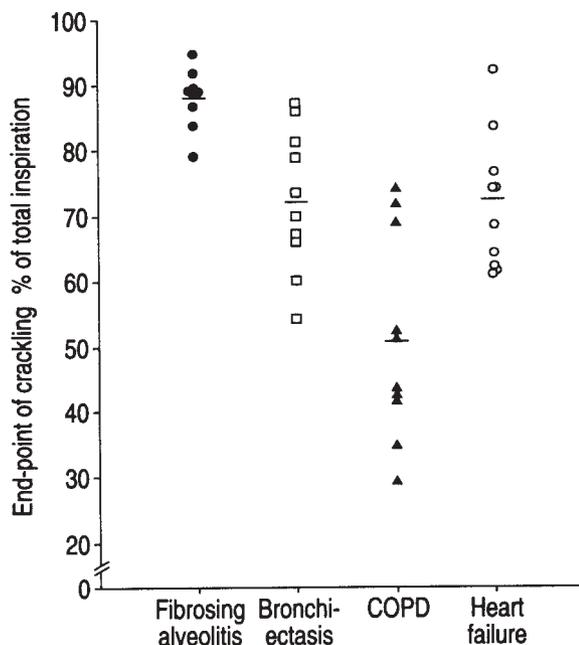

Fig. 5. – The timing in four different pulmonary diseases: cryptogenic fibrosing alveolitis, bronchiectasis, chronic obstructive pulmonary disease (COPD) and heart failure. The timing parameter is the end-point of crackling as a percentage of the whole duration of inspiration. The timing of the crackles in fibrosing alveolitis was significantly different compared to all other diagnoses (mean/median shown by horizontal bar) and there was no overlap between fibrosing alveolitis and COPD. There was also a significant difference in the timing of COPD compared to the timing of crackles in heart failure and bronchiectasis. The horizontal bars indicate mean values of the groups. (Reproduced by permission of *Chest* PIIRILÄ *et al.* [13]).

crackles in asbestos workers; the duration of crackles was about 10 ms and their highest frequency content ranged 1,000–2,000 Hz. MURPHY and SORENSEN [17] found that the auscultatory scoring of crackling correlated to the duration of exposure in a similar way as the decline of diffusing capacity. They classified the crackles as fine; the frequency components of the crackles were over 500 Hz. AL JARAD *et al.* [76] have found the crackles in asbestosis to be fine in time-expanded waveform display and their timing to be mid- to late-inspiratory. In addition, they have compared the finding in pulmonary auscultation, plain chest radiography, time-expanded waveform display, and high resolution computer tomography (HRCT). They found that signs of early asbestosis which were not visible in the plain chest radiograph could be detected with time-expanded waveform analysis and HRCT with similar frequency [77].

Crackles in bronchiectasis

In bronchiectasis, the walls of the large bronchi are abnormally dilated due to destruction of their elastic and muscular components. In expiration, the bronchial walls collapse followed by sudden opening during inspiration. The crackles in bronchiectasis appear in early or mid-inspiration [6, 7, 11]. Waveform analysis has shown that the crackles in this disease are coarse (2CD >9 ms), and their upper frequency limit is high. An early onset of crackling and its late end-point within the inspiratory

cycle are also typical of bronchiectasis [13]. The crackles in bronchiectasis are distinguished from the early crackles in COPD, since they occur later during the inspiratory cycle and have a longer duration related to the respiratory phase.

Crackles in COPD

In COPD, crackles are most commonly caused by airway secretions. These kind of crackles disappear or their auscultation place changes after coughing [5]. In contrast, crackles can sometimes be heard almost constantly in inspiration as well as in expiration. The main cause of this phenomenon is probably sequential closing and opening of proximal bronchi, narrowed as a result of obstruction, due to loss of their cartilaginous support because of inflammation. In lung sound analysis, the crackles in chronic obstruction have been described to be scanty, early and low-pitched [5–7]. In COPD, the crackles are usually few and coarse (2CD > 9 ms) starting early in inspiration and ending before the mid-point of inspiration [13]. NATH and CAPEL [6, 7] have also documented the early timing of crackles in COPD.

Crackles in heart failure

The crackling sounds in heart failure have been considered to result from the opening of airways narrowed by peribronchial oedema [5]. LAENNEC [2] described the crackles in heart failure as a slight crepitous mucous rattle, which was difficult to distinguish from the crackles in peripneumony. FORGACS [5] found the crackles in heart failure to be late, high-pitched inspiratory and expiratory crackles. MURPHY [78] has described them as "difficult to distinguish from the crackles of interstitial fibrosis, but more transient, more gravity-dependent". In a paper by HUDSON *et al.* [38] heart failure patients were reported to have medium crackles in 65% and dry rales in 88% of cases. Some other authors have reported fine crackles in heart failure [79]. Crackles in heart failure disappear rapidly as the patient recovers.

In computerized lung sound analysis, the crackles in heart failure have been shown to be the most coarse compared to fibrosing alveolitis, bronchiectasis or COPD [13]. Long duration (2CD > 10 ms) and a low upper frequency limit of sound were typical to them; they lasted for a long time during the inspiratory cycle and appeared late [13].

Crackles in pneumonia

In acute pneumonia, oedema and infiltration of inflammatory cells in lung tissue may narrow the bronchi. In resolving pneumonia, inflammatory cells tend to accumulate and oedema diminishes. Consequently, the lung parenchyma becomes drier. Nonuniformly decreased compliance in patients with resolving pneumonia may cause collapse in the airways, and therefore crackles of a different character and timing can be heard.
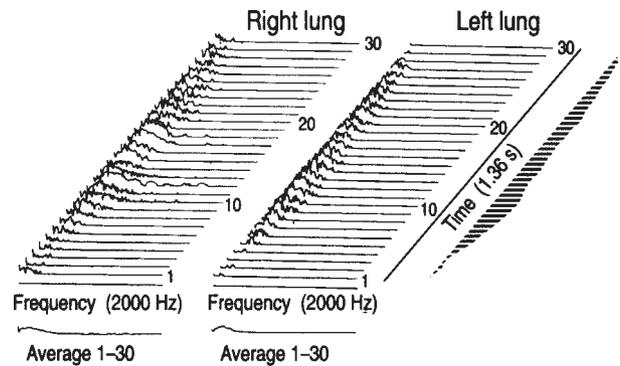

Fig. 6. – Serial spectra of the lung sounds of one patient with unilateral pneumonia. This patient has inspiratory crackling sounds in the pneumonic area of the right lung. The frequency spectrum of the right lung presents significantly higher sound components than the left one, which is healthy. (Reproduced by permission of *Chest* PIIRILÄ [14]).

According to LAËNNEC [2], the crackles in pneumonia resembled those in pulmonary oedema and haemoptysis. In Osler's Textbook of Medicine [80], the crackles in the early stages of pneumonia were described to be fine, whereas in resolving pneumonia crackles of all sizes occurred. Some authors consider the crackles of pneumonia to be fine at the acute stage of the disease and to become coarser when pneumonia is resolving [81]. FORGACS [5] characterized the pneumonic crackles as late inspiratory crackles, like the ones in fibrosing alveolitis.

In computerized objective sound analysis, the crackles in acute pneumonia have been shown to be mid-inspiratory and fairly coarse (2CD 9–11 ms), resembling crackles in patients with bronchiectasis. During recovery they are more end-inspiratory and shorter in duration, but also greater variation of crackle duration is then characteristic (2CD 6–11 ms) [61]. During recovery, the pneumonic crackles can resemble those in fibrosing alveolitis.

In unilateral pneumonia, simultaneous analysis of sounds from both lungs may be informative; an example of asymmetry in a serial FFT analysis due to unilateral pneumonia-induced crackles is presented in figure 6. In the diseased lung, high frequency sound components due to crackling are clearly seen during inspiration.

Crackles in sarcoidosis

In sarcoidosis, granulomatous lesions are often present in the upper zones of the lungs, but usually no crackles are heard in auscultation of these areas. BAUGHMAN *et al.* [82] have studied the auscultatory findings in sarcoidosis complicated by fibrosis: the crackles were fine and late or mid-inspiratory. Compared to fibrosing alveolitis the crackles were far fewer, and according to HRCT scan results this was related to the different distribution of fibrosis in these two pulmonary diseases; basal subpleural fibrosis favoured crackling sounds.

Expiratory crackles

Coarse expiratory crackles have been reported to occur especially in chronic airway obstruction [5]. However,

expiratory crackling sounds have also been found in many other pulmonary disorders [13, 14, 61]. Fine expiratory crackles have also been noted in fibrosing alveolitis [13, 61, 71]. The polarities of the initial deflections of expiratory crackles have been shown to be opposite to those in inspiration [11, 71, 83]. Expiratory crackles are usually much less frequent than inspiratory ones. Expiratory crackles, according to Forgacs' theory, possibly represent the closing sounds of small airways. Variations in respiratory pattern did not affect their timing or number in the study by WALSHAW *et al.* [71].

Squawks

Squawks are short musical sounds that have been described to occur in fibrotic pulmonary disorders. In the same patients, usually fine inspiratory crackling sounds can be heard [2, 5]. Squawks are very short wheezes with a mean duration of 90–320 ms [84]. FORGACS [5] explained that these sounds are generated when a closed airway suddenly opens in inspiration but for a brief moment the airway walls remain in light contact to each other; thus, the inflowing inspiratory air can cause the walls to oscillate, producing a wheezing sound. EARIS *et al.* [84] have found that the squawks in extrinsic alveolitis were shorter in duration and later in timing than in other pulmonary diseases.

Other crackle-like sounds

Sound from pleural friction can sometimes resemble crackling sounds. Usually, however, the friction sound is very coarse and low in pitch. Ultrafine crackles have been described in subcutaneous emphysema [85]. This review is not dealing with these kinds of sounds.

Summary and future perspectives

The modern concept of the pathophysiology of crackles, opening or closing of single airways modified by the properties of surrounding tissue offer a fascinating basis to the study of acoustic properties of crackling sounds. Results of the modern computerized lung sound analysis can confirm this theory of the origin of crackles. The studies referred to in this review indicate that the timing and waveform characteristics of crackles have a certain discriminating power of pulmonary diseases and heart failure. Therefore, analysis of crackles could be helpful in differential diagnosis, and possibly also in early diagnosis of pulmonary diseases, both in pulmonary and in occupational medicine.

The number of crackles correlates to the severity of a pulmonary disease. Therefore, automatic crackle detection and counting methods have been developed for easy and objective assessment of sound samples of longer duration [62, 66]. In future, automatic analysis of timing and waveform of crackles will also be possible. The development of computerized lung sound analysis enhances the possibility for clinical utilization of the information on crackles both in diagnosis and in follow-up of pulmonary diseases and possibly also in critical care units.

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