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A M E R I C A N C O L L E G E O F
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Mechanism of Inspiratory and Expiratory Crackles*

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Objective: Although crackles are frequently heard on auscultation of the chest of patients with common cardiopulmonary disorders, the mechanism of production of these sounds is inadequately understood. The goal of this research was to gain insights into the mechanism of crackle generation by systematic examination of the relationship between inspiratory and expiratory crackle characteristics.

Methods: Patients with a significant number of both inspiratory and expiratory crackles were examined using a multichannel lung sound analyzer. These patients included 37 with pneumonia, 5 with heart failure, and 13 with interstitial fibrosis. Multiple crackle characteristics were calculated for each crackle, including frequency, amplitude, crackle transmission coefficient, and crackle polarity.

Results: Spectral, temporal, and spatial characteristics of expiratory and inspiratory crackles in these patients were found to be similar, but two characteristics were strikingly different: crackle numbers and crackle polarities. Inspiratory crackles were almost twice as numerous as expiratory crackles ($n = 3,308$ vs $1,841$) and had predominately negative polarity (76% of inspiratory crackles vs 31% of expiratory crackles).

Conclusion: These observations are quantitatively consistent with the so-called *stress-relaxation quadrupole hypothesis* of crackle generation. This hypothesis holds that expiratory crackles are caused by sudden airway closure events that are similar in mechanism but opposite in sign and far less energetic than the explosive opening events that generate inspiratory crackles. We conclude that the most likely mechanism of crackle generation is sudden airway closing during expiration and sudden airway reopening during inspiration. (CHEST 2009; 135:156–164)

Key words: acoustics; breath sounds; crackle mechanism; crackles; lung mechanics; lung sounds; rales; respiratory sounds

Abbreviations: CHF = congestive heart failure; CTC = crackle transmission coefficient; IPF = interstitial pulmonary fibrosis; T1 = half period to the left of the highest peak; T2 = half period to the right of the highest peak; T2/T1 = ratio of the second half period to the first half period

Crackles are intermittent short-lived sounds that emanate from the lung and are associated with pulmonary disorders including interstitial pulmonary

fibrosis (IPF), congestive heart failure (CHF), and pneumonia.¹ The mechanism underlying crackle generation is not well understood, however, and the spectral, temporal, and spatial characteristics of crackles have not been well quantified. In this article, we characterize crackles in patients with IPF, CHF, and pneumonia who had a significant number of both inspiratory and expiratory crackles. We quantified these events using multiple microphones placed on the chest surface, and we focused in particular on differences between crackles generated during inspiration vs expiration.

These data were then used to address a particular hypothesis of crackle generation that was suggested many years ago, the *stress-relaxation quadrupole*

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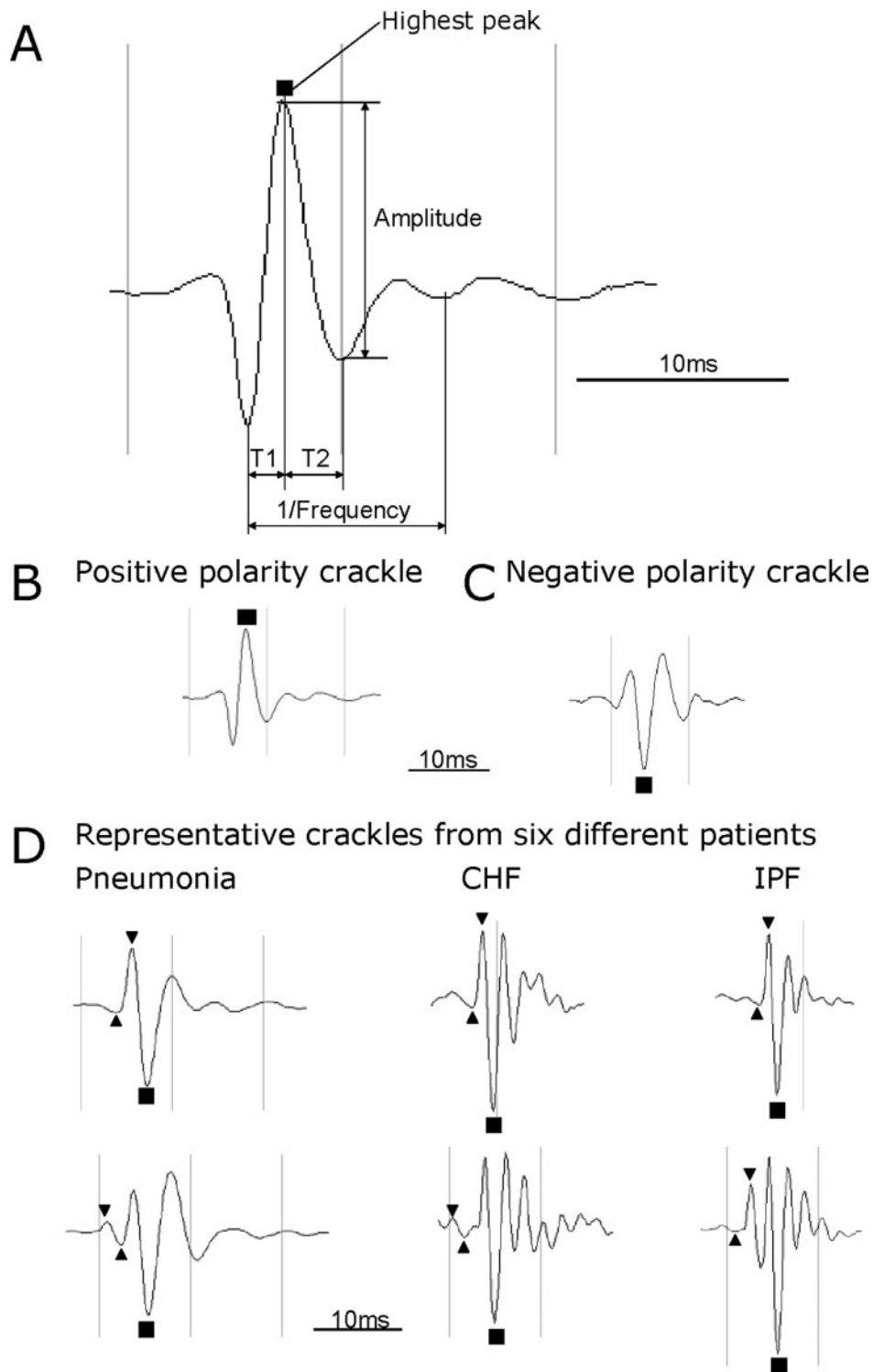


FIGURE 1. The waveform of a typical crackle. *Top, A:* The crackle analysis starts by identification of the crackle's highest deflection, the highest peak. The half period to the left of the highest peak is marked as T1. The half period to the right of the highest peak is marked as T2. Crackle frequency is calculated from four consecutive half periods, with T1 as the first half period. Crackle amplitude is marked "amplitude." *Center, left, B:* Crackle polarity is defined as positive if the highest peak is upward. *Center, right, C:* Crackle polarity is defined as negative if the highest peak is downward. *Bottom, D:* Six representative crackles recorded from six different patients. Squares mark the highest deflection. Triangles mark the candidates for initial deflection. The definition of the highest deflection is unambiguous. The definition of the initial deflection is highly subjective because it might be very small and comparable to the noise floor.

hypothesis, as described below.² This hypothesis makes a series of *a priori* mechanistic predictions that we test here. The hypothesis predicts the following: (1) the existence of both inspiratory and expiratory crackles; (2) that the energy of expiratory crackles should be substantially smaller than the energy of inspiratory crackles, and hence far fewer expiratory crackles should be detected; more critically, however, the hypothesis also predicts that (3) crackles of both positive and negative polarity should be observed during inspiration; (4) crackles of negative polarity should predominate during inspiration and correspond to roughly 90% of the total; and, conversely, (5) crackles of both polarities should be observed during expiration with positive polarity predominating. To account for inspiratory crackles, other mechanisms have been proposed in the literature.³ However, these other mechanisms have led to no comparable testable predictions, and they do not address at all the existence of expiratory crackles.

MATERIALS AND METHODS

Patient Selection

Patients were selected for this study from a pool of patients who had undergone lung sound analysis as a part of a broader study of the correlation of disease processes with lung sounds patterns. To select patients into this study, we identified hospitalized and outpatients of a community teaching hospital who had a specific cardiopulmonary disease diagnosed or were considered to be normal by their caregivers. These patients were drawn from > 1,000 for whom we have both the diagnosis and the lung sound analysis. The diagnostic category for each patient was that of the clinician caring for the patients. This was reviewed by the senior author to be sure they were consistent with established criteria.

Patients were examined with a 16-channel lung sound analyzer (model STG1602; Stethographics; Westborough, MA), which was described in detail by Bergstreser et al⁴ and Murphy et al.^{5,6} In short, the lung sound analyzer uses electret microphones mounted in stethoscope chest pieces to record data on a personal computer. Fourteen microphones are incorporated into a soft-foam microphone pad. The microphone pad is positioned on a stretcher or a plastic reclining chair positioned at a 45° angle. In

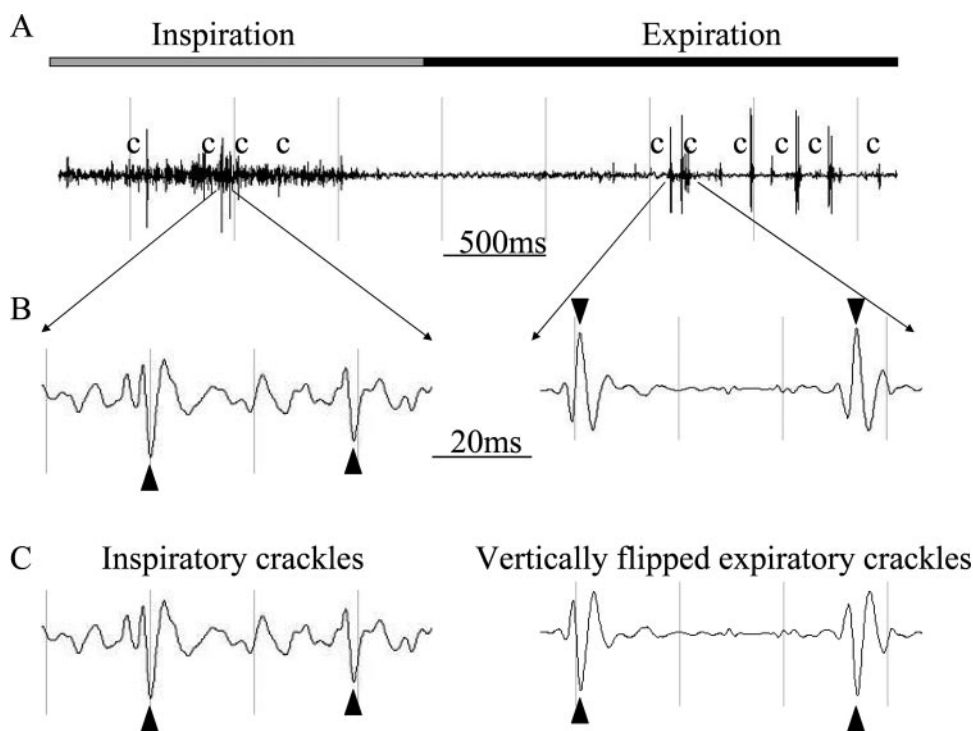


FIGURE 2. A time-amplitude plot of a sound recorded from a patient with pneumonia in the retrocardiac region. The acoustic sensor was located on the left posterior chest over the area of roentgenologic opacification. Waveforms are presented in both the unexpanded (*top*, A) and expanded (*center*, B) modes. *Top*, A: The unexpanded waveform shows one full breath. The solid bars above the unexpanded wave mark the respiratory phase; the light bar indicates inspiration and the dark bar indicates expiration. The normal inspiratory sound can be seen as having an almost random waveform fluctuations. Crackles, marked "c," look like spikes on an unexpanded waveform. *Center*, B: The expanded crackles waveforms reveal the negative polarity during inspiration and positive polarity during expiration (triangles). In this patient, all inspiratory crackles (total of 11 crackles or 2.8 inspiratory crackles per breath) had negative polarity, and all expiratory crackles (total of 55 crackles or 13.8 expiratory crackles per breath) had positive polarity. *Bottom*, C: Vertically flipped expiratory crackles have waveforms nearly identical to that of inspiratory crackles. These observations were typical of the crackles detected in our study.

this study, subjects were instructed to lie in a recumbent position on the microphone pad. Subjects were instructed to breathe more deeply than normally. Typically, three to six full breaths were captured in a 20-s recording.

Crackles were defined in accordance with accepted criteria.^{7,8} The lung sound analyzer software automatically identified crackles in all full breaths. The validation of the use of the device as a crackle counter has been reported.⁹ The goal of this study was to compare inspiratory crackles to expiratory crackles. Accordingly, we only accepted into the study those patients who had both inspiratory crackle counts more than two crackles per breath and expiratory crackle counts more than two crackles per breath. A single 20-s recording contained three or more breathing cycles. Therefore, patients accepted into the study had at least six inspiratory and six expiratory crackles in the 20-s recording. These patients included 37 patients with pneumonia, 5 patients with CHF, and 13 patients with IPF. The study was approved by the Institutional Review Board of Faulkner Hospital.

Crackle Parameters

Crackle analysis started by identification of the highest deflection or peak of the crackle (Fig 1, top, A). The half period to the left of the highest peak is marked as T1. The half period to the right of the highest peak is marked as T2. Crackle frequency is calculated from four consecutive half periods, with T1 as the first half period. Crackle amplitude is measured in arbitrary units. Crackle polarity was defined as positive if the highest peak was upward (Fig 1, center left, B). Crackle polarity was defined as negative if the highest peak was downward (Fig 1, center right, C).

In this study, crackle polarity was determined by the highest wave deflection, while in previous reports it was often determined by the initial wave deflection. In our experience, the definition of initial wave deflection is highly subjective because it might be very small and comparable to the noise floor (Fig 1, bottom, D). The highest wave deflection, however, is a more objective parameter.

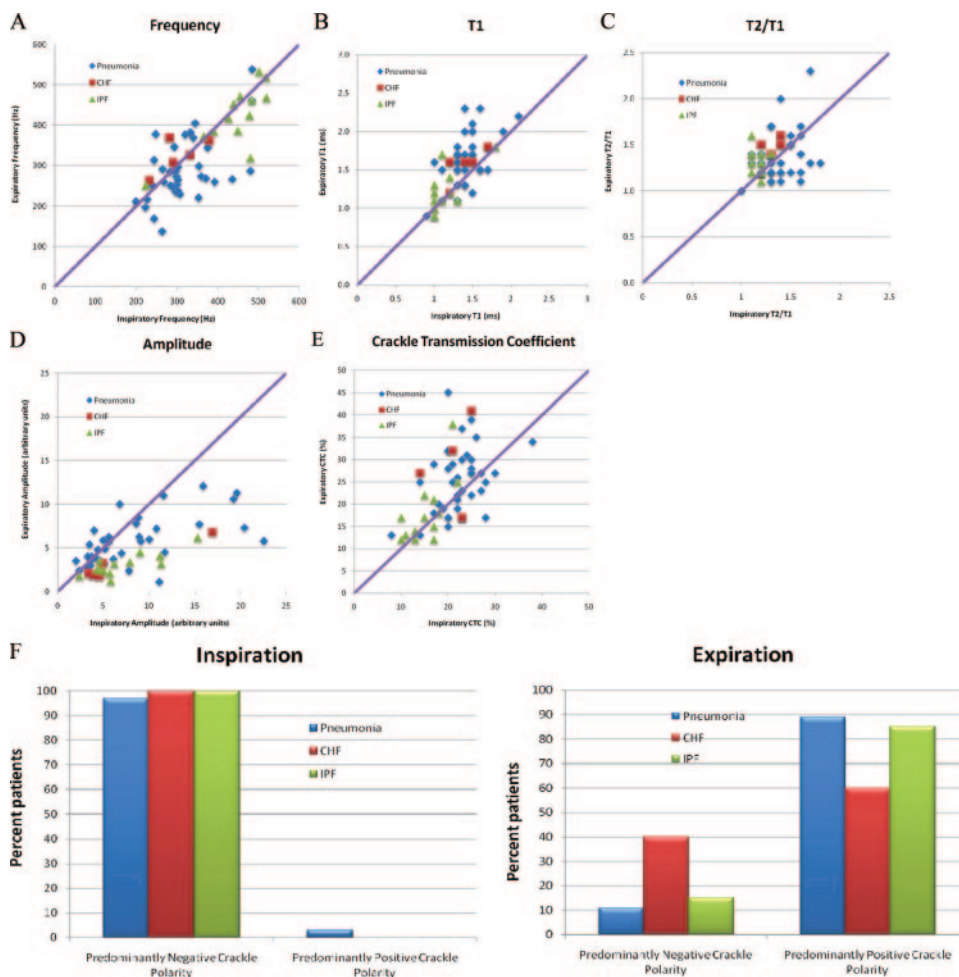


FIGURE 3. Comparison between inspiratory and expiratory crackle waveform characteristics. Each data point corresponds to one patient. SD of each data point was approximately 30% (not shown). *Top left, A:* Mean frequency of inspiratory crackles as a function of mean frequency of expiratory crackles; one data point per patient. The solid line indicates the line of equality. *Top center, B:* Mean T1 of inspiratory crackles as a function of mean T1 of expiratory crackles. *Top right, C:* Mean ratio of T1 over T2 of inspiratory crackles as a function of mean ratio of T1 over T2 of expiratory crackles. *Center, left, D:* Mean amplitude of inspiratory crackles as a function of mean amplitude of expiratory crackles. *Center right, E:* Mean CTC of inspiratory crackles as a function of mean CTC of expiratory crackles. *Bottom F:* Predominant crackle polarity in inspiration (left) and expiration (right).

Waveform polarity carries information about tissue displacement toward or away from the microphone. In this report, positive waveform deflection corresponds to displacement of the diaphragm toward the microphone. In other words, positive waveform deflection corresponds to positive pressure and is therefore consistent with the sign convention used in the theory of stress-relaxation quadrupoles.²

Crackle Family Definition

The concept of a crackle family was introduced and validated in Vyshedskiy et al.¹⁰ In short, a single crackle event can be detected by multiple microphones located on the chest surface. The group of waveforms corresponding to the single crackle event and recorded by multiple microphones is referred to as a *crackle family*. The channel with highest crackle amplitude is called the *mother crackle*, and the corresponding deflections at other channels are called *daughter crackles*. These definitions are consistent with the theory that the event, which generated a crackling sound, had occurred closer to the mother channel microphone than to the other microphones. Indeed, evidence supporting this theory can be found by examination of the stack plots of the crackles.¹⁰ Each crackle family was characterized by the set of parameters measured in the mother crackle. Additionally, each crackle family was assigned a crackle transmission coefficient (CTC).¹⁰ The CTC characterizes the degree of crackling sound transmission to all ipsilateral microphones. To calculate the CTC, we first expressed each daughter crackle amplitude as a percentage of the mother crackle amplitude by calculating the ratio of the peak of the cross-correlation function to the peak of the mother crackle autocorrelation function. This ratio characterizes the degree of sound transmission from the sound source to the corresponding microphone on the chest surface. We call this *ratio transmission coefficient*. By definition, the mother transmission coefficient is always 100%. The daughter transmission coefficient has a value of 0% in the absence of any transmission and 100% when sound is transmitted equally to the daughter and mother channels.

The CTC was calculated by averaging transmission coefficients of all daughter crackles. The CTC has a value of 0% in the absence of any transmission (only the mother can be observed) and 100% when there is equal transmission to all ipsilateral channels.

RESULTS

In a time-amplitude plot, crackles look like spikes. Figure 2, *top, A*, shows lung sounds recorded during a single breath from a patient with pneumonia. The automated computer algorithm identified four inspiratory crackles and six expiratory crackles (Fig 2, *top, A*; crackles are marked by “c”). Expansion of the time axis allowed detailed observations of crackle waveform characteristics (Fig 2, *center, B*; crackle peaks are marked by triangles). The comparison of inspiratory to expiratory crackles showed that the waveform of expiratory crackles looked nearly identical to the waveform of inspiratory crackles but of opposite polarity (Fig 2, *bottom, C*).

To confirm this observation, we compared inspiratory and expiratory crackles in multiple patients. Each crackle waveform was characterized by six parameters: frequency, T1, T2/T1, amplitude, CTC, and polarity.

We analyzed 5,149 crackles in 55 patients (approximately 94 crackles per patient or 16 crackles per breath per patient). Crackle characteristics varied randomly in each patient and even in each breath. However, each patient’s crackle characteristics (frequency, T1, T2/T1, amplitude, and CTC) followed a Gaussian distribution. Thus, in order to compare inspiratory parameters to expiratory parameters, means and SDs were calculated separately for all inspiratory crackles and for all expiratory crackles. Averaged crackle parameters varied significantly between patients. To study the correlation between inspiratory and expiratory waveform characteristics, we plotted average inspiratory crackle frequency, T1, T2/T1, amplitude, and CTC as a function of corresponding expiratory frequency, T1, T2/T1, amplitude, and CTC (Fig 3; each point corresponds to a single patient; a solid diagonal line indicates a unity line). If all crackle characteristics were to fall on the unity line, we would have concluded that inspiratory crackles were identical to expiratory crackles.

The average crackle frequency ranged from 120 to 550 Hz (Fig 3, *top left, A*). As expected, patients with IPF exhibited higher frequency crackles (green triangles) than patients with CHF (red squares) and pneumonia (blue circles). The surprising finding was that for most patients the average inspiratory frequency was

Table 1—Data Summary*

Variables	Pneumonia (n = 37)	CHF (n = 5)	IPF (n = 13)
Inspiratory crackles			
Crackles per breath	9 ± 5	13 ± 06	24 ± 17
T1, ms	1.4 ± 0.2	1.4 ± 0.2	1.1 ± 0.2
Crackle frequency or pitch, Hz	316 ± 71	326 ± 43	441 ± 80
T2/T1	1.4 ± 0.2	1.3 ± 0.1	1.2 ± 0.1
CTC, %	22 ± 5	21 ± 4	15 ± 4
Crackle amplitude, %	8 ± 6	7 ± 6	7 ± 4
Percentage of negative polarity crackles	75 ± 14	76 ± 10	76 ± 9
Expiratory crackles			
Crackles per breath	6 ± 4	6 ± 2	8 ± 5
T1, ms	1.6 ± 0.3	1.6 ± 0.2	1.2 ± 0.3
Crackle frequency or pitch, Hz	289 ± 79	303 ± 55	421 ± 78
T2/T1	1.4 ± 0.3	1.5 ± 0.1	1.3 ± 0.1
CTC, %	26 ± 7	27 ± 10	18 ± 7
Amplitude, %	6 ± 3	3 ± 2	3 ± 1
Percentage of negative polarity crackles	29 ± 18	38 ± 18	34 ± 14

*Data are presented as average ± SD. This study has only included patients with very high number of crackles; this study was not designed to predict average numbers of crackles in the different disease categories. For the information on differences in crackle rate in different disease categories, see Murphy.⁶

very similar to the average expiratory frequency. The points lie very close to the unity line (correlation coefficient 0.76). The first half period (T1) ranged from 0.9 to 2.3 ms (Fig 3, *top right, B*). As expected, T1 in patients with IPF was smaller than T1 in patients with CHF and pneumonia. Most data points lie very close to the unity line (correlation coefficient 0.69). T2/T1 ranged between 1 and 2.3 (Fig 3, *center left, C*). This corresponds to the observation that half periods have a tendency to increase over time. However, we saw many individual crackles that did not follow this rule. T2 had tendency to be closer to T1 in crackles recorded from IPF patients. Most data points lie very close to the unity line (correlation coefficient 0.32).

Crackles tended to be louder (greater amplitude) in pneumonia patients (Fig 3, *center right, D*). Inspiratory crackles had greater amplitude than expiratory crackles (most data points lie below the unity line; correlation coefficient 0.63).

The CTC, a coefficient that characterizes the degree of crackling sound transmission to all ipsilat-

eral microphones, ranged from 8 to 45%. Zero percent corresponds to the absence of any transmission, *ie*, the crackle can be recorded by only one microphone. One hundred percent corresponds to equal transmission to all ipsilateral microphones (Fig 3, *bottom, E*). The surprising finding was that in each patient expiratory crackles were transmitted as far as inspiratory crackles (most data points lie close to the unity line (correlation coefficient 0.53).

Thus, in the majority of patients, inspiratory crackle frequency, T1, T2/T1, and CTC were similar to corresponding characteristics of expiratory crackles; the points lie very close to the unity line. The amplitudes of expiratory crackles were somewhat smaller than those of inspiratory crackles (Fig 3, *bottom, E*). In summary, the observation suggested in Figure 2, that expiratory crackles look very similar to inspiratory crackles, appears to be borne out by the data. This phenomenon was found consistently in multiple patients with a range of lung conditions.

A Inspiration

B Expiration

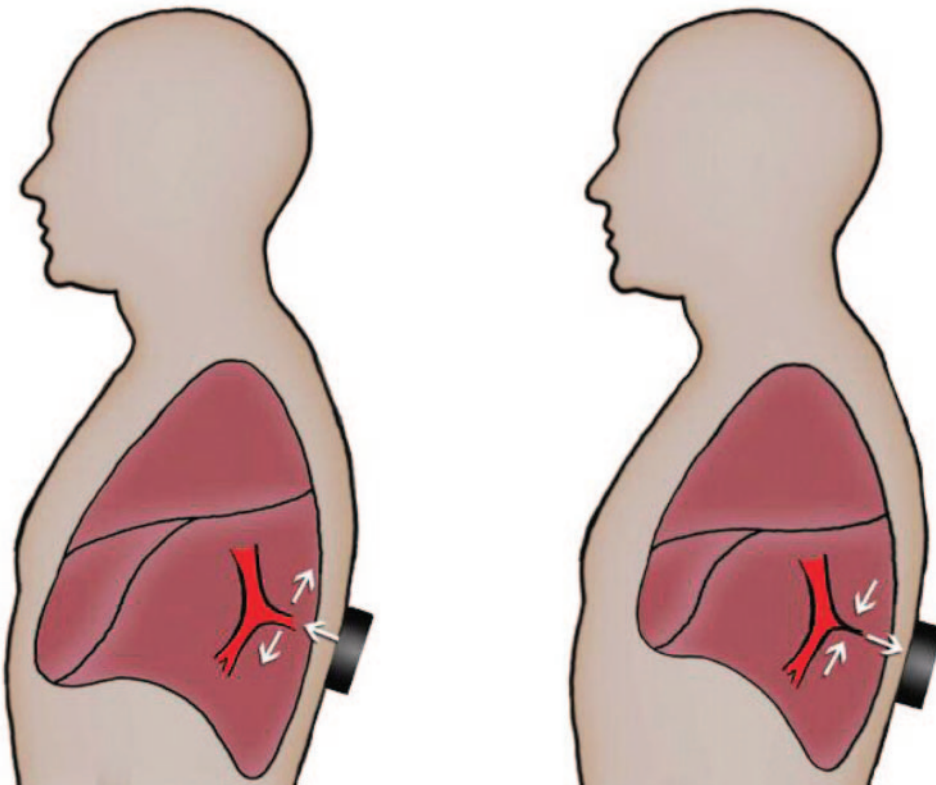


FIGURE 4. Crackle polarity carries information about tissue displacement toward or away from the microphone. Positive polarity in this report corresponds to tissue displacements toward the microphone. *Left, A:* Microphone positioned perpendicular to the airway detects tissue displacement away from the microphone during airway opening (negative crackle polarity). *Right, B:* During airway closing, the microphone detects tissue displacement toward the microphone (positive crackle polarity).

The parameter that was clearly different between inspiratory and expiratory crackles was crackle polarity, Figure 3, *bottom, twin panel*). The majority of patients had predominantly negative polarity of inspiratory crackles (98% of patients) and predominantly positive polarity of expiratory crackles (81% of patients). Seventy-six percent of the 3,308 inspiratory crackles studied had negative polarity. Sixty-nine percent of the 1,841 expiratory crackles studied had positive polarity. Table 1 presents a summary of all data as average \pm SD.

DISCUSSION

There is considerable evidence, summarized by Forgacs,¹ that inspiratory crackles are caused by airway opening. The opening of the airways is expected to be a sudden process, while the closure of the airways is sometimes thought to be a more gradual process of diameter decrease, ending in complete collapse. This study is inconsistent with the view that airway closure is a gradual process. Both the temporal and spectral characteristics of expiratory crackles were found to be similar to those of inspiratory crackles. Thus, the events responsible for crackle generation must be as fast in expiration as they are in inspiration, even if the former are less energetic than the latter.

We observed similar frequency, T1, T2/T1, and CTC during inspiration as compared to expiration, thereby suggesting that similar airway dynamics apply to inspiratory and expiratory crackles. This observation is consistent with the hypothesis that expiratory crackles and inspiratory crackles are generated at the same location in the airways, and perhaps even the same airway.

As noted above crackle polarity was predominantly negative during inspiration but positive during expiration (Fig 2, *center, B*). This observation is consistent with previous reports.¹¹⁻¹³ Crackle polarity carries information about tissue displacement toward or away from the microphone. Positive polarity in this report corresponds to tissue displacements toward the microphone. The predominant polarity of the highest deflection of inspiratory crackles was negative. Therefore, most inspiratory crackles were associated with tissue displacements away from the microphone, as is predicted by the stress-relaxation quadrupole theory² for the opening of an airway positioned broadside (perpendicular) to a microphone (Fig 4, *left, A*). In fact, most small airways of the bronchial tree are expected to be perpendicular to the microphones positioned on the chest. The predominant positive polarity of the expiratory crackles indicated that airways were closing during expiration. The closure of the airways generated tissue displacement toward the microphones during expiration (Fig 4, *right, B*).

Other mechanisms have been suggested; one mechanism is the "trapped gas hypothesis."^{3,14-18} It is conceivable that some airways collapse during early expiration and trap gas behind the closures. As the expiration continues, the pressure beyond the closure site in the trapped gas region increases. Since the airway proximal to the closure is at near-atmospheric pressure, there will be an increasing pressure difference across the closure as expiration proceeds. As this pressure difference reaches the critical opening pressure of the closure, the airway pops open during expiration. As a result, the waveform generated during expiration is similar to that of an inspiratory crackle. Further, it might be

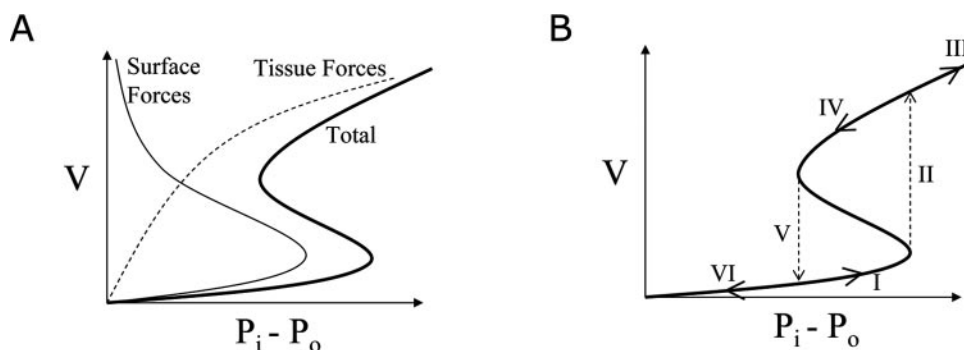


FIGURE 5. Volume-pressure diagram for a small airway or alveolus (adapted from Mead¹⁹). *Left, A:* Interfacial surface forces, tissues forces, and the total forces encountered by a small airway or alveolus. The total force has a clear region of negative compliance, which is intrinsically unstable. V = volume of the airway. P_i = pressure inside the airway; P_o = pressure outside the airway. *Right, B:* Trajectories for inflation and deflation. During inflation, when the system first encounters the unstable region, there will be a sudden opening event depicted by path II; the inflation trajectory is I, II, III. During deflation, when the system first encounters the unstable region there will be a sudden closing event depicted by path V; the deflation trajectory is III, IV, V, VI.

argued that the polarity is reversed because during expiration the trapped region contains higher pressure, whereas during inspiration the trapped region has negative pressure.

The trapped gas hypothesis is in several regards contrary to our observations, however. First, expiratory crackles occur throughout expiration, not just at the end of expiration. We observed many early expiratory crackles with positive polarity, with nearly half of the crackles occurring in the first half of the expiration. These crackles are not explained by the trapped gas hypothesis. More critically, the trapped gas hypothesis predicts tissue displacements toward the microphone during inspiration (due to air rushing from larger airways into smaller airways), and away from the microphone during expiration (due to trapped air escaping toward larger airways). This prediction is opposite in sign to our experimental observations (Fig 4). It is difficult to see how our data can be explained by the trapped gas hypothesis.

Fredberg and Holford² proposed that crackle generation arises from sudden airway opening and sudden airway closing, and that resulting stress waves propagate in the lung parenchyma. In particular, they showed that these events could be modeled quantitatively on the basis of a stress-relaxation quadrupole. This model makes a series of testable predictions that were detailed in the "Introduction," each of which was borne out quantitatively by the results reported here.

Why is airway closure during expiration not a gradual process, but rather a sudden energetic audible collapse? To answer this question, consider the volume-pressure diagram for a small airway or alveolus, as depicted in Figure 5 (adapted from Mead¹⁹). Competing surface vs connective tissue forces lead to hysteresis in the area-pressure relationship for individual airways. As pressure changes during the breathing cycle, the airway volume is forced to follow a hysteretic cycle with unstable regions depicted by dashed lines (Fig 5, *right, B*). As described by Mead¹⁹ long ago, during inflation, when the system first encounters this unstable region, there will be a sudden opening event depicted by path II; the inflation trajectory is I, II, III. During deflation, when the system first encounters an unstable region, there will be a sudden closing event depicted by path V; the deflation trajectory in this case is III, IV, V, VI. Associated critical transitions in the airway volume result in inspiratory and expiratory crackles, but based on this diagram and its pronounced hysteresis, it is clear that sudden opening events require higher pressures, and therefore must be far more energetic, than sudden closing events.

In summary, the observations reported here are quantitatively consistent with the stress-relaxation quadrupole hypothesis of crackle generation. This hypothesis holds that expiratory crackles are caused by sudden airway closure events that are similar in mechanism but opposite in sign and far less energetic than the explosive opening events that generate inspiratory crackles. We conclude that the most likely mechanism of crackle generation is sudden airway closing during expiration and sudden airway reopening during inspiration. Clearer understanding of the mechanism of production of lung sounds offers the promise of improving noninvasive diagnosis of lung disorders.

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