Expiratory Lung Crackles in Patients with Fibrosing Alveolitis

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Inspiratory lung crackles are a diagnostic feature of interstitial pulmonary fibrosis, but expiratory crackles are not well documented. In a phonopneumographic study of 13 patients with fibrosing alveolitis, expiratory crackles were audible with the stethoscope in 12. Phonopneumographic analysis of these 12 patients showed the crackles to be fine with the initial wave deflection of the expiratory and inspiratory crackles in opposite directions. They were few in number, occurred predominantly in mid- and late expiration, and were not affected by varying the volume history or by breath holding maneuvers. These observations support the theory that some crackles are produced by vibration of the walls of peripheral airways. In addition, this group of patients showed a significant correlation between the number of expiratory crackles and the reduction in predicted transfer factor, suggesting that expiratory crackles may be a clinical indicator of the severity of disease in fibrosing alveolitis. (Chest 1990; 97:407-09)

Lung crackles are characterized by their quality (coarse or fine) and where they occur in the respiratory cycle.1 Coarse early inspiratory crackles are typical of chronic airflow limitation, while showers of fine late inspiratory crackles are a relatively constant feature of fibrosing alveolitis.2 Coarse expiratory crackles are described in patients with chronic airflow limitation,3 but neither coarse nor fine expiratory crackles have been documented in fibrosing alveolitis. It was our clinical impression that expiratory crackles do occur in fibrosing alveolitis and we therefore undertook a clinical and phonopneumographic assessment of lung crackles in patients with this disorder.

Patients and Methods

Patients

Successive patients attending the chest clinic with a clinical diagnosis of fibrosing alveolitis were studied. All patients had a history of progressively increasing breathlessness associated with fine inspiratory lung crackles, a restrictive pulmonary function defect, impaired gas transfer, and diffuse pulmonary shadowing on chest x-ray film. In two, the diagnosis was confirmed by open lung biopsy. Patients with heart failure, sputum production, wheezing, or significant exposure to asbestos were excluded.

After consent, each patient gave a full history. The presence or absence of expiratory crackles was noted on physical examination. Lung volumes, spirometry and the gas transfer factor were measured.

Thirteen patients with a mean age of 67 years (range 52 to 83 years) met criteria for the study. Ten had cryptogenic disease and three had fibrosing alveolitis secondary to autoimmune disease (two rheumatoid arthritis, one systemic sclerosis). There were seven men; eight were ex-smokers, and six had finger clubbing.

The mean FEV₁ (± SD) of the 13 patients was 1.68 (± 0.37) L, 66 percent of predicted values, while the mean FVC was 2.3 (± 0.45) L, 61 percent of predicted. As a group, the patients did not have airflow obstruction; the mean FEV₁/FVC value was 73 percent (± 10). Five patients had an FEV₁/FVC % below 70, two with values of 69, the other three being 63, 61 and 53 percent respectively. Gas transfer was impaired in all cases with a mean of 48 (± 16) percent of predicted.

Methods

Lung sounds were recorded using a high sensitivity crystal microphone placed at the right lung base posteriorly, with the patient sitting upright. The signal was passed through a high gain amplifier (Leatham amplifier module, Cambridge Instruments). Air flow was measured at the mouth with a Fleisch pneumotachograph (type 17212, Gould), and both signals were stored on separate channels of a Hewlett Packard instrumentation FM tape recorder (model 3960) running at 15 cm/s. The signals were later played back on a Schwarzer C3000 chart recorder for analysis. By varying the paper speed of the chart recorder and the playback speed of the tape recorder, it was possible to produce time-expanded signals from which crackle waveforms could be analyzed.4

Like other workers, we have defined fine crackles as having an initial deflection width of <0.9 ms and a two cycle duration of <6.0 ms.5

Recordings were made during four breathing patterns:
1) Tidal respiration (tidal breathing).
2) Deep breathing to and from FRC (FRC breathing).
3) Repeated ventilation from TLC to RV and back (maximal breathing), to examine the effect of volume history on the timing and presence of crackles.
4) Maximum ventilation (as in 3), but with breath holding after inspiration and expiration (breath holding), to eliminate inequalities in regional ventilation resulting from variations in time constants of parallel lung regions.

Results

Clinical Detection of Expiratory Crackles

All patients had fine late inspiratory crackles; 12...
patients also had fine expiratory crackles easily audible with the stethoscope. One of these patients was too breathless to perform the breath holding maneuver, and another did not produce satisfactory tracings during the FRC breathing maneuver. The patient with no expiratory crackles was a 65-year-old woman who had fibrosing alveolitis secondary to rheumatoid arthritis. She had no other features which distinguished her from the rest of the study group.

Phonopneumographic Characteristics of Expiratory Crackles

When present, expiratory crackles were detected in all patterns of respiration employed during the study. However, unlike inspiratory crackles, expiratory crackles were intermittent, discrete, and varied in number from breath to breath (Fig 1).

Tracings of time expanded crackles were reproduced from all patients to allow waveform analysis. Representative crackles are shown in Figure 2. The initial deflection width was <0.9 ms and the 2-cycle duration was <6.0 ms in all cases, fulfilling the criteria for fine crackles. The initial pen deflection was in the opposite direction in inspiration compared to expiration (Fig 2), and this was a constant finding in all our patients with expiratory crackles.

During tidal breathing, 70 percent of expirations contained fine crackles, with a mean of 1.3 crackles per expiration. These values did not change significantly with the other breathing patterns used during the study (Table 1). Expiration was divided into thirds in order to analyze the time distribution of fine crackles. Eighty-four percent occurred in the middle and last third of expiration during tidal breathing, and their timing did not change significantly with the other breathing patterns studied (Table 1).

Expiratory Crackles and Respiratory Function

There was a correlation ($r = 0.61$, $p<0.05$) between the proportion of expirations which contained fine crackles during the breath holding maneuver and the transfer factor (as percent predicted) (Fig 3). However, when subjected to the same analysis, the other breathing patterns used during the study failed to reach statistical significance. Similarly, no correlation was found between expiratory crackles and the FVC as percent predicted. The reduction in gas transfer factor was independent of both smoking habits and airflow limitation.

Discussion

The presence of fine inspiratory crackles is widely recognized as an important clinical feature of interstitial lung disease, but we have found no data in the published English literature about the occurrence of expiratory crackles.

Many hypotheses have been suggested to explain the occurrence of crackles. Coarse crackles are thought to be produced in the larger airways and in some cases may be due to air bubbling through secretions. Such crackles are well described in both inspiration and expiration and are associated with many disease states (eg, chronic airflow obstruction and bronchiectasis).

In contrast, investigations with stereo microphones and in vitro lung preparations, have suggested that fine crackles are produced by smaller airways. They are well documented during inspiration in patients with interstitial pulmonary fibrosis and in patients with interstitial pulmonary edema. Our results demonstrate that fine crackles also occur during expiration.

Table 1—Distribution and Number of Expiratory Crackles Throughout Different Breathing Patterns

<table>
<thead>
<tr>
<th></th>
<th>Tidal Breathing</th>
<th>FRC Breathing</th>
<th>Maximal Breathing</th>
<th>Breath Holding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percentage of expirations with crackles (± SD)</td>
<td>70 (± 25)</td>
<td>79 (± 18)</td>
<td>79 (± 28)</td>
<td>72 (± 29)</td>
</tr>
<tr>
<td>Mean number of crackles per expiration</td>
<td>1.3</td>
<td>1.4</td>
<td>1.5</td>
<td>1.5</td>
</tr>
<tr>
<td>Percent of crackles in 1st third of expiration</td>
<td>16</td>
<td>17</td>
<td>24</td>
<td>22</td>
</tr>
<tr>
<td>Percent of crackles in 2nd third of expiration</td>
<td>58</td>
<td>46</td>
<td>43</td>
<td>43</td>
</tr>
<tr>
<td>Percent of crackles in 3rd third of expiration</td>
<td>26</td>
<td>37</td>
<td>33</td>
<td>35</td>
</tr>
</tbody>
</table>

Figure 1. Phonopneumographic (upper tracing) and airflow (lower) in a patient with fibrosing alveolitis. Note profuse mid to late inspiratory crackles and intermittent expiratory crackles.

Figure 2. A time-expanded expiratory and inspiratory crackle from the same patient. The initial deflection width is <0.9 ms in duration and the 2-cycle duration is <6.0 ms in each case, showing that these are fine crackles. Note also the reversed initial waveform between the two crackles.
in fibrosing alveolitis, both in the cryptogenic form and in that associated with systemic connective tissue diseases.

Two theories have been developed to account for fine inspiratory crackles. The more widely accepted argues that the crackles occur when small airways snap open during inspiration, the resultant equalization of pressure in the previously separated gas columns producing the cracking sound.\(^8\) The observation that crackles relate to the generation of maximal pressures during inspiration\(^9\) is in agreement with this. Using this model, the occurrence of expiratory crackles is harder to explain, as it assumes that airways initially patent at the end of inspiration close early in expiration and then reopen when the pressure of gas trapped within them exceeds that in the adjacent airways. Although the timing of the crackles is compatible with this, the lack of any effect of FRC or maximal breathing maneuvers on either the number or timing of expiratory crackles is surprising. The forced expirations used with these breathing patterns should increase the number of crackles and produce them earlier in expiration. Likewise, the failure of these breath holding maneuvers (which equalize areas of time-constant inequality in the lung) to affect the subsequent pattern of crackles is contrary to this explanation.

An alternative view of crackle production is the stress relaxation quadrupole theory\(^10\) which suggests that the fine crackle is not produced by the gas column itself, but by vibration in the walls and interstitium of the peripheral airway. This theory predicts what we have observed in our patients, namely: expiratory crackles would be much less frequent than inspiratory crackles, that the initial wave deflection would be in opposite directions, and that variations in respiratory pattern would not affect the distribution or frequency of crackles, as we have found. Further support for this theory comes from work using electronic waveform analysis of crackles which also noted the reversed waveform in expiratory crackles,\(^11\) but did not speculate on their etiology.\(^12,13\)

If fine crackles are due to increased stiffness in the walls of smaller airways, then patients with worse disease would have more crackles. Unfortunately, unless phonopneumographically quantified at multiple sites, inspiratory crackles occur too early in the natural history of fibrosing alveolitis and are too diffuse to be used as a monitor of disease severity. However, the number of crackles heard in expiration in this study were much fewer (mean 1.3-1.5 per expiration) and could easily be counted via the stethoscope. Furthermore, the number of expiratory crackles was found to correlate weakly with the gas transfer factor. Since the latter is an indirect measure of the severity of the mechanical disturbance due to the interstitial fibrosis the presence of expiratory crackles may provide a clinical marker of disease progression, although how useful this is has yet to be determined.

In summary, we have found expiratory crackles to be frequent in patients with fibrosing alveolitis. Their presence favors stress relaxation quadrupole theory\(^10\) of crackle generation.

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