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Lung crackles in bronchiectasis

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ABSTRACT The inspiratory timing of lung crackles in patients with bronchiectasis was compared with the inspiratory timing of the lung crackles in chronic bronchitis and alveolitis. In severe obstructive chronic bronchitis the lung crackles are typically confined to early inspiration while in alveolitis the lung crackles continue to the end of inspiration but may begin in the early or the mid phase of inspiration. In uncomplicated bronchiectasis on the other hand, the lung crackles typically occur in the early and mid phase of inspiration, are more profuse, and usually fade by the end of inspiration. In addition in bronchiectasis, crackles are also usually present in expiration, they are gravity independent and become less profuse after coughing.

Lung crackles in inspiration and expiration can be timed precisely by the simultaneous recording of lung sounds and airflow. Forgacs[1] pointed out that the lung crackles of fibrosing alveolitis typically occur late in inspiration and suggested that these sounds coincide with the late opening of the peripheral airways in this condition. Nath and Capel[2] contrasted the characteristically late timing of inspiratory crackles of fibrosing alveolitis and related disorders with the characteristically early timing of the inspiratory crackles in severe obstructive chronic bronchitis and emphysema. In this paper the timing during inspiration of lung crackles in uncomplicated bronchiectasis is compared with the timing of lung crackles in fibrosing alveolitis and obstructive chronic bronchitis. Additional distinguishing features of lung crackles observed in the three conditions are described.

Patients

The 38 patients studied were divided into three diagnostic groups: bronchiectasis, chronic bronchitis, and alveolitis. The patients in the study were identified as members of one or other of three diagnostic groups by the presence or absence of certain clinical, spirometric, and radiological features.

BRONCHIECTASIS

Sixteen adult patients with the clinical features of many years cough and expectoration going back to childhood or adolescence were studied in this group. Three patients had been moderate smokers at some time in the past. Their chest radiographs did not show diffuse lung changes and the mean FEV_1/VC ratio for the group was 71% (table 1). The diagnosis of bronchiectasis in each patient was confirmed by bronchogram.

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CHRONIC BRONCHITIS

Twelve patients with the clinical features of many years of cough, expectoration and breathlessness were included in this group. They had previously been moderate or heavy smokers and showed spirometric evidence of severe expiratory obstruction with a mean FEV_1/VC ratio of 32% (table 2). Their chest radiographs showed no focal or diffuse lung lesion and they were not submitted to bronchographic examination.

ALVEOLITIS

Ten patients studied in this group showed radiological evidence of diffuse lung infiltration. Eight patients had fibrosing alveolitis and the remaining two had pulmonary sarcoidosis confirmed by lung biopsy. Spirometrically there was no airways obstruction with a mean FEV_1/VC ratio of 84% (table 3).

Methods

All the sound recordings were made with the patient sitting. A crystal suction microphone mounted in an aluminium cup was fixed on the chest wall at the point where the lung sounds were best heard. The usual site of recording was at the right or the left lung base. The output of the

Table 1 *Clinical, radiological, and spirometric features of patients with bronchiectasis (UL=upper lobe, ML=middle lobe, LL=lower lobe)*

Patient	*Age (yr)*	*Sex*	*FEV_1 (l)*	*VC (l)*	*FEV_1/VC %*	*Previous smoking history*	*Lobes affected*
1	37	M	3·65	4·60	79	Non-smoker	RLL, LLL
2	25	M	4·20	5·85	72	Non-smoker	LLL
3	38	F	2·15	3·40	63	Non-smoker	LLL
4	63	F	2·45	3·00	82	Non-smoker	RLL, LLL
5	30	M	1·90	3·00	63	Non-smoker	RLL, RML, LLL
6	26	F	3·55	3·85	92	Non-smoker	RLL, LLL
7	49	F	1·60	2·45	65	Non-smoker	RLL, RML, LLL, LUL
8	32	F	1·95	3·15	62	Non-smoker	RLL, RML, LLL
9	58	F	1·25	1·65	76	Non-smoker	RLL, LLL
10	46	F	1·00	2·15	47	Non-smoker	RLL, LLL
11	63	M	1·35	1·75	77	Moderate smoker	RLL, LLL
12	38	F	0·50	1·80	28	Non-smoker	RLL, LLL
13	32	M	3·20	3·55	90	Moderate smoker	RML, LLL
14	56	F	2·15	2·30	93	Non-smoker	RML, LLL
15	59	F	0·65	0·95	68	Non-smoker	RML, RLL, LLL, LUL
16	51	F	1·55	1·85	84	Moderate smoker	RML, RLL, LLL

Table 2 *Clinical and spirometric features of patients with chronic bronchitis*

Patient	*Age (yr)*	*Sex*	*FEV_1 (l)*	*VC (l)*	*FEV_1/VC %*	*Previous smoking history*
1	54	M	0·80	3·80	21	Heavy smoker
2	64	F	0·50	1·30	39	Moderate smoker
3	75	M	0·59	1·55	38	Moderate smoker
4	62	M	1·10	2·75	40	Moderate smoker
5	68	M	0·65	2·35	28	Moderate smoker
6	47	F	0·30	1·20	25	Moderate smoker
7	56	M	0·70	3·30	21	Moderate smoker
8	58	M	0·70	1·80	39	Moderate smoker
9	57	F	0·45	1·70	27	Moderate smoker
10	62	M	0·70	2·10	33	Moderate smoker
11	51	M	0·50	1·70	29	Heavy smoker
12	50	F	0·65	1·65	39	Heavy smoker

Table 3 *Clinical and spirometric features of patients with diffuse lung disorder*

Patient	*Age (yr)*	*Sex*	*FEV_1 (l)*	*VC (l)*	*FEV_1/VC %*	*Diagnosis*
1	73	M	2·40	3·00	80	Fibrosing alveolitis
2	65	M	2·25	2·45	92	Lymphangitis carcinoma
3	73	F	1·30	1·40	93	Fibrosing alveolitis
4	53	M	1·90	2·15	88	Fibrosing alveolitis
5	68	M	2·60	4·25	61	Fibrosing alveolitis
6	48	F	1·70	2·10	81	Fibrosing alveolitis
7	51	F	2·15	2·45	88	Pulmonary sarcoidosis
8	48	F	1·10	1·20	92	Fibrosing alveolitis
9	38	M	3·50	3·85	91	Pulmonary sarcoidosis
10	67	F	1·00	1·30	77	Fibrosing alveolitis

microphone was amplified and the lower frequencies up to 250 Hz filtered out to eliminate unwanted noise. The signal so obtained was monitored by headphones and displayed on one channel of an oscilloscope screen (Telequipment DM64), UV recorder (SE Type 3006), as well as the sound channel of an FM tape recorder. The flow rate was simultaneously recorded at the mouth with a Fleisch pneumotachograph coupled to a differential pressure manometer (Furness-Micromanometer MDC), and the output displayed on the second channel of the oscilloscope screen, UV recorder, and the tape recorder (fig 1). The flow rate signal at zero flow was superimposed on the sound signal.[2] Thus the start and finish of inspiration was marked off by the flow signal as it crossed the sound signal. The patient was asked to breathe normally and then to take slow vital

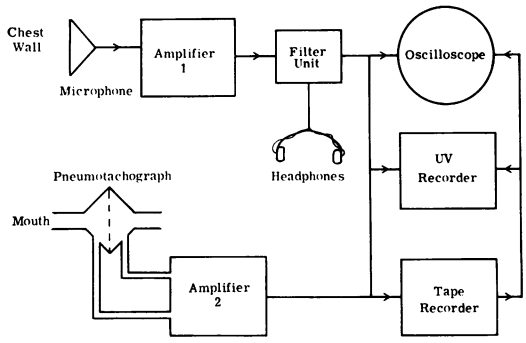

Fig 1 *Instrumentation for simultaneous recording of lung crackles and flow rate.*

capacity breaths. In some patients the recordings were repeated after coughing and on bending forwards without altering the location of the microphone. The direct graphic recordings of the flow rate sound tracings were subsequently compared with the playback recordings obtained from the tape recorder.

Results

The timing of the inspiratory crackles in the three groups of patients was different. In obstructive chronic bronchitis, the inspiratory crackles, always few in number, were confined to the early phase of inspiration (fig 2). By contrast, the inspiratory crackles in fibrosing alveolitis continued to the end of inspiration but could start in the early or mid phase of inspiration (fig 3). The lung crackles in bronchiectasis showed a pattern distinct from those in chronic bronchitis and fibrosing alveolitis. In bronchiectasis, the inspiratory crackles started early in inspiration, continued to mid inspiration and faded by the end of inspiration (fig 4, table 4). As a rule the inspiratory crackles of bronchiectasis were more profuse in early and mid inspiration and when present in late inspiration the crackles did not continue to the end of inspiration. Of the 16

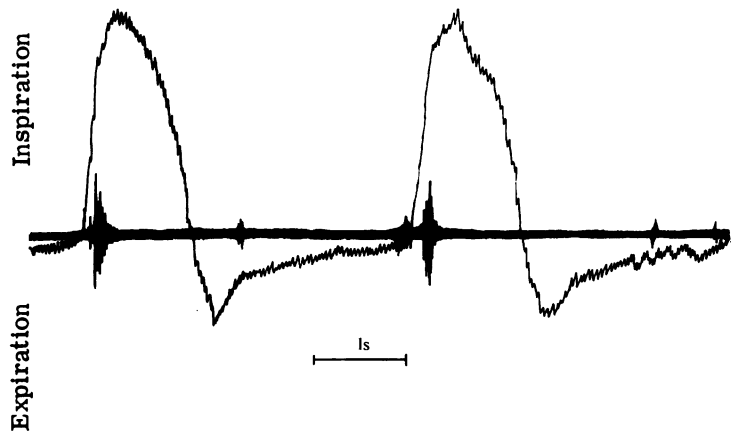

Fig 2 *Phonopneumogram of early inspiratory crackles. This is a simultaneous recording of inspiratory crackles and airflow rate.*

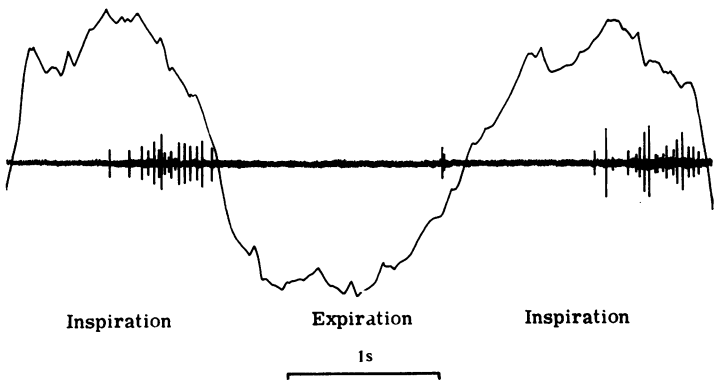

Fig 3 *Phonopneumogram of late inspiratory crackles: inspiratory crackles are seen mid and late in inspiration.*

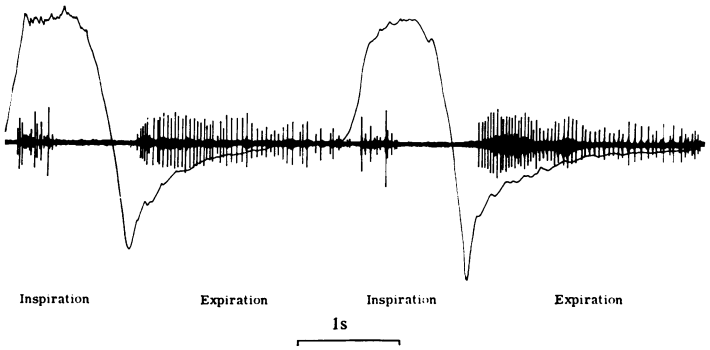

Fig 4 *Phonopneumogram of early and mid inspiratory and expiratory crackles recorded from a patient with bronchiectasis.*

Table 4 *Timing of the inspiratory crackles in patients with bronchiectasis*

Patient	*Early inspiration*	*Mid inspiration*	*Late inspiration*	*End inspiration*
1	Present	Present	Absent	Absent
2	Present	Present	Absent	Absent
3	Present	Present (scanty)	Absent	Absent
4	Present	Present	Present (scanty)	Absent
5	Present	Present	Present	Absent
6	Present	Present	Present	Absent
7	Present	Present	Present	Absent
8	Present	Present	Present (scanty)	Absent
9	Present	Present	Absent	Absent
10	Present	Present	Absent	Absent
11	Present	Present	Absent	Absent
12	Present	Present	Present	Absent
13	Present	Present	Absent	Absent
14	Present	Present	Absent	Absent
15	Present	Present	Present	Absent
16	Present	Present	Absent	Absent

patients with bronchiectasis, the inspiratory crackles in nine were confined to early and mid inspiration. In the remaining seven patients, the inspiratory crackles were present in late inspiration but did not continue to the end of inspiration.

Discussion

The presence of loud crackles is a classical sign of bronchiectasis. Trail[3] suggested that crackles starting in the first phase of inspiration and tailing off in the second and third phase of inspiration indicate fibrosis round the larger bronchi, as may occur in bronchiectasis, crackles in the middle phase of inspiration indicate a combination of bronchial and lung fibrosis while crackles in the third phase of inspiration occur typically in disorders of the acinar structures.

Our findings in this study support Trail's clinical observations. We attempt, further, to distinguish the inspiratory timing of the lung crackles in chronic bronchitis with severe airway narrowing from that in bronchiectasis without severe airway narrowing. From the observations in the present and a previous study,[2] it appears that the early inspiratory crackles of chronic bronchitis occur when spirometrically the airways obstruction is severe with an FEV_1/VC ratio of 45% or less, while in bronchiectasis, the airways obstruction is usually mild. If in patients with obstructive chronic bronchitis crackles are heard not only in early inspiration but also in mid inspiration, then bronchiectasis may be suspected. Such was the case with one patient in whom bronchiectasis inferred from auscultation was confirmed by bronchogram (fig 5). An important feature of the lung crackles in fibrosing alveolitis is that they continue to the end of inspiration but may start in the middle third and even first third of inspiration so that they are then heard throughout inspiration. The timing of inspiratory crackles in one patient with longstanding bronchiectasis and extensive lung fibrosis was compared with the timing in fibrosing alveolitis. Lung crackles in the bronchiectatic patient were loud and more profuse in the early and middle phase of inspiration than in the third phase of inspiration (fig 6), whereas in alveolitis, the lung crackles were fine and became more profuse in the third phase of inspiration (fig 3).

As in chronic bronchitis, the lung crackles of bronchiectasis can commonly be heard at the mouth, suggesting that the crackles are generated in the proximal intrathoracic airways. The observation is in keeping with the bronchographic findings which show abnormality of the larger and the medium sized bronchi. By contrast the crackles which originate in the peripheral airways, as in fibrosing alveolitis, are rarely heard at the mouth.

It seems likely that the crackles of bronchiectasis relate to retained secretions in damaged and dilated bronchi. These crackles are typically present

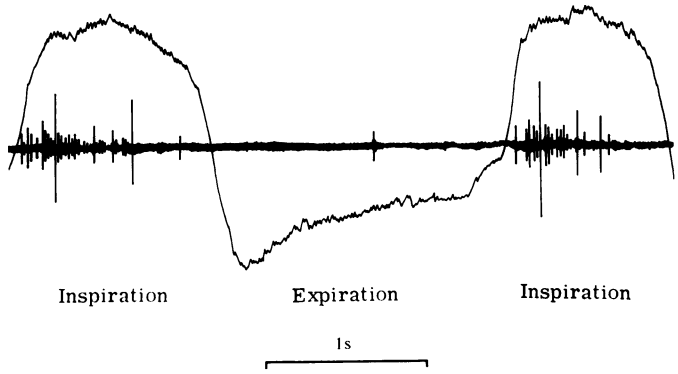

Fig 5 *Phonopneumogram from a patient with chronic bronchitis and bronchiectasis showing early and mid inspiratory crackles. Bronchogram showed cylindrical bronchiectasis of the lower lobe bronchi.*

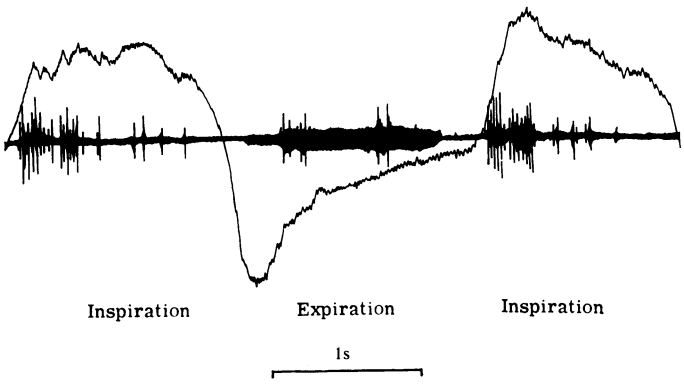

Fig 6 *Phonopneumogram from a patient with bronchiectasis and lung fibrosis. Pan inspiratory and expiratory crackles are seen. Lung crackles are less profuse at the end of inspiratory than in early and mid phase of inspiration.*

in inspiration and expiration, and they are less abundant after expectoration. Bubbling in retained secretions, however, is probably only one of the causes of these crackles. The bronchial wall compliance is increased in bronchiectasis.[4] A bronchus with high compliance lined by mucus will tend to close prematurely in expiration and will open early in subsequent inspiration at a low transpulmonary pressure. The airway opening in inspiration may be abrupt or it may remain lightly closed and open intermittently as a bolus of gas passes when upstream gas pressure rises above a critical level. This mechanism, suggested by Forgacs,[1] is similar to that generating the lower pitched explosive sounds when gas passes through the oesophagus, bowel, or anal sphincter.

Regional studies using radioactive xenon have demonstrated impaired ventilation of bronchiectatic regions irrespective of anatomical type of bronchiectasis.[5] The continuation of lung crackles in mid inspiration may in part be explained by the longer time constant of the bronchiectatic airway where the crackles are generated. The time lag between the instantaneous flow rate recorded at the mouth and the opening of the bronchiectatic airway may make the crackles appear later in inspiration than if there were no such lag. Another mechanism which may operate is suggested by the observation that in bronchiectasis, especially in the varicose and cystic variety, the emptying of dilated sacs is prevented by collapse of the bronchi downstream (towards the mouth) so that the secretions are retained in them. In this situation, early inspiratory crackles may coincide with the opening of bronchiectatic airways and their continuation in the middle phase of inspiration results from bubbling in retained secretions as inspiration progresses.

Although the timing of inspiratory crackles in this study is emphasised, attention to additional features help to enhance the value of this important physical sign at the bedside (table 5). Bronchiectatic lung crackles are present in both phases of the respiratory cycle and are made less abundant after coughing. As in chronic bronchitis, they are well transmitted to the mouth while in alveolitis, even in an advanced state, the lung crackles are rarely heard at the mouth. The late

Table 5 *Common features of lung crackles in obstructive chronic bronchitis, bronchiectasis, and fibrosing alveolitis*

	Obstructive chronic bronchitis	*Bronchiectasis*	*Fibrosing alveolitis*
Typical timing of inspiratory crackles	Present early phase inspiration	Present early and mid phase inspiration	Present end phase inspiration
Typical number of crackles	Always few	Usually moderate	Can be profuse
Effect of cough	No change	Temporarily reduced	No change
Effect of position	No change	No change	Modified or abolished
Intensity	Faint	Loud	Moderately loud
Pitch	Coarse	Coarse	Fine
Expiratory crackles	May be present	Typically present	May be present
Transmission to the mouth	Transmitted	Transmitted	Not transmitted

inspiratory crackles of fibrosing alveolitis are gravity dependent and so can be modified with change in body position. By contrast, the crackles of bronchitis and bronchiectasis are independent of gravity. Thus in bronchiectasis, crackles occur in a pattern which is recognisable and distinct from those of obstructive chronic bronchitis and fibrosing alveolitis.

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