Thorax (1971), **26**, 727.

Pulmonary ventilation and gas exchange in bronchiectasis

J. N. PANDE, B. P. JAIN, R. G. GUPTA, and J. S. GULERIA

Respiratory Laboratory, Department of Medicine, All India Institute of Medical Sciences, New Delhi 16, India

The subdivisions of the lung volume, pulmonary mechanics, and resting steady state pulmonary transfer factor were measured in 31 patients with bronchographically proven bronchiectasis. In seven patients the process of gas exchange was further investigated by fractionating the total alveolar-arterial oxygen tension gradient into diffusion, distribution, and true shunt components.

A restrictive type of ventilatory defect with varying degrees of airway obstruction was observed in a majority of the patients; the airway obstruction was partially reversed by a bronchodilator. Dynamic compliance was usually decreased and the pulmonary resistance increased.

Pulmonary transfer factor was decreased in proportion to the number of segments involved. Vital capacity, maximum breathing capacity, and dynamic compliance bore a less significant correlation with the extent of disease. The degree of airway obstruction, as judged by pulmonary resistance, was independent of the extent of disease.

All the patients were hypoxaemic and some had hypercapnia as well. The alveolar-arterial oxygen tension gradient was widened primarily because of distributional abnormalities and, to some extent, by the presence of true right-to-left shunts. The latter amounted to 13·6% of the total cardiac output.

Surgical resection of the affected lobe or segments resulted in a further deterioration of all the parameters of pulmonary function tested.

With the advent of effective antibiotic therapy morbidity and mortality from bronchiectasis and its complications have been considerably reduced in all developed countries of the world. In India the incidence of bronchiectasis is still quite high because of the lack of adequate treatment of acute pulmonary infections. Thus approximately 5 to 12% of the patients attending a non-tuberculous chest clinic in North India suffer from bronchiectasis (Bawa and Guleria, 1957; Guleria and Chitkara, 1966). It is a common cause of chronic cor pulmonale, particularly in female and young adults. Functional abnormalities of the lung resulting from bronchiectasis have been studied by several investigators (Anderson, Bell, and Blount, 1954; Smith, Siebens, and Storey, 1954; Williams, 1954; Cherniack *et al.*, 1959; Cherniack and Carton, 1966; Bass *et al.*, 1968) and a combined restrictive and obstructive pattern of ventilatory dysfunction has frequently been demonstrated. A decrease in dynamic lung compliance and an increase in flow resistance have been observed (Cherniack and Carton, 1966). In earlier studies (Anderson *et al.*, 1954; Cherniack *et al.*, 1959) the transfer factor for oxygen was found to be normal, but more recently Bass *et al.* (1968) have shown a significant reduction in pulmonary transfer factor for carbon monoxide (TL_{CO}) in bronchiectatic patients. Anderson *et al.* (1954) and Cherniack *et al.* (1959) reported an increase in venous admixture but the relative contributions of virtual and true shunts were not evaluated.

The purpose of the present study is to describe the functional abnormalities of the lung in patients with bronchiectasis using the more refined techniques now available for the investigation of pulmonary mechanics and gas exchange.

MATERIAL AND METHODS

Thirty-one patients with bronchographically proven bronchiectasis involving two or more segments formed the material for this study (Table I). Patients having associated diseases like bronchial asthma or pul-

TABLE I

CLINICAL FEATURES

Serial No.	Age/Sex		Duration (yr) Cough and Expectoration	Duration (yr) Dyspnoea	No. of Segments Involved	Location of Diseased Segments	Type of Bronchiectasis
1	40	F	3	1/2	10	Both LL	Cystic
2	45	F	30	1/2	8	RUL, LLL	Cystic
3	55	F	12	1	10	Both LL	Cylindrical
4	45	F	22	6	11	RML, RLL, LLL	Cystic
5	21	F	9	1	8	Left lung	Cystic
6	18	M	2	—	4	LLL	Cylindrical
7	45	M	5	1/2	6	RML, RLL	Cylindrical
8	18	M	7	7	18	Both lungs	Cystic
9	18	M	6	—	4	LLL	Cystic and Cylindrical
10	25	M	13	2	18	Both lungs	Cystic
11	17	M	11	8	8	Left lung	Cystic and Cylindrical
12	31	M	20	7	12	Right lung, lingula	Cystic
13	21	M	10	—	2	Lingula	Cylindrical
14	20	M	16	10	14	Right lung, LLL	Cystic
15	25	M	20	5	18	Both lungs	Cystic
16	29	M	25	5	18	Both lungs	Cystic
17	24	M	15	—	4	LLL	Cylindrical
18	42	F	10	5	4	RUL, RLL	Cystic
19	26	M	15	2	8	Both LL	Cylindrical
20	25	M	—	—	4	LLL	Cystic
21	25	M	3	—	3	Lingula, RLL	Cystic and Cylindrical
22	25	M	1	1/12	5	LLL, lingula	Cystic
23	20	F	5	1	8	Left lung	Cystic
24	27	M	20	—	6	LLL, lingula	Cystic
25	17	M	10	2	4	LLL	Cystic
26	20	M	1	—	8	Left lung	Cystic
27	14	M	3	—	18	Both lungs	Cystic
28	26	M	2	—	2	Lingula	Cylindrical
29	18	M	6	2	8	Left lung	Cylindrical
30	30	M	29	3	8	RUL, RLL	Cystic
31	25	M	10	—	7	RML, RLL	Cylindrical
Mean	27·0		10·1	2·2	8·6		

RUL = right upper lobe; RML = right middle lobe; RLL = right lower lobe; LUL = left upper lobe; LLL = left lower lobe; LL = lower lobes.

monary tuberculosis were excluded after appropriate clinical and radiological examination. There were 24 males and seven females. Their ages ranged from 14 to 55 (mean 27) years. The number of segments involved ranged from 2 to 18 (mean 8·6). The bronchiectasis was of the cystic variety in 19 patients, cylindrical in eight, and a combination of the two in four patients. Three patients had shown signs of chronic cor pulmonale and congestive heart failure. However, at the time of study all the patients were free from respiratory infection and congestive failure. Pulmonary function studies were performed within a week before bronchography to avoid the known adverse functional effects of this procedure (Christoforidis, Nelson, and Tomashefski, 1962; Bhargava and Woolf, 1967).

Vital capacity (VC) and its subdivisions were measured with a 13·5 litre Collins spirometer. Functional residual capacity (FRC) was determined by the 7-minute nitrogen washout technique. Forced expiratory volume in one second (FEV_1) and maximum mid-expiratory flow rate (MMFR) were obtained by fast speed kymography. The results were expressed as per cent of the predicted normal values according to the formulae of Goldman and Becklake (1959) after appropriate correction for use at sea level. Resting pulmonary transfer factor was determined by the modified steady state technique (Bates, Boucot, and Dormer, 1955) using a Rahn and Otis end-tidal sampling device for obtaining alveolar air. It was recorded as per cent of the predicted value according to Bates, Woolf, and Paul (1962). The transfer factor in Indian subjects has been found to be similar to that reported by these investigators (Guleria, Sharma, Pande, and Ramchandran, 1970). Pulmonary mechanics were studied in the sitting position by the oesophageal balloon method (Mead and Whittenberger, 1953). Details of the method used in this laboratory have already been described (Guleria, Talwar, Malhotra, and Pande, 1969; Pande, Jain, and Guleria, 1970). After the initial measurements, 0·25 g of aminophylline (theophylline ethylene-diamine) was given intravenously over a period of 5 minutes. Records of transpulmonary pressure, volume, and flow were repeated 5, 10, and 15 minutes after the injection. Measurements of pulmonary compliance and resistance, both before and after the bronchodilator, were made at a constant respiratory frequency and tidal volume.

In seven patients a polythene catheter was passed into the pulmonary artery to obtain mixed venous samples. Arterial blood samples were obtained from an indwelling needle in the brachial artery. Samples of arterial and mixed venous blood and of inspired and expired air were collected simultaneously during a steady state while the patient inhaled successively room air, 14% O_2 in N_2, and 100% O_2. From these data, the total alveolar-arterial oxygen tension gradient (A-aD O_2) while breathing room air was fractionated into diffusion, distribution, and true shunt components by the method of Ayres, Criscitiello, and Grabovsky (1964). The details of the technique em-

TABLE II

ROUTINE PULMONARY FUNCTIONS

Serial No.	VC[1]	FRC[1]	FEV_1 (%VC)	MMFR[1]	MBC[1]	TLco[1]	Pulm. Compliance (l/cm H_2O)		Pulm. Resistance (cm H_2O/l/sec)	
							Dynamic	Specific	Insp.	Exp.
1	54	132	60	13	32	40	0·065	0·020	4·4	12·0
2	72	130	73	36	37	74	0·158	0·049	1·4	3·0
3	46	120	56	16	20	62	0·088	0·028	8·4	13·5
4	48	138	60	14	35	51	0·034	0·013	16·3	45·5
5	71	110	51	21	40	73	0·155	0·064	1·0	1·5
6	80	96	95	107	69	88	0·299	0·115	0·7	1·0
7	75	135	52	12	23	71	0·158	0·045	3·8	5·7
8	45	112	64	14	21	39	0·033	0·011	7·2	11·7
9	78	120	71	90	85	95	0·112	0·039	1·0	2·8
10	46	90	73	20	28	30	0·045	0·017	6·3	10·5
11	40	91	69	14	17	65	0·060	0·024	4·5	6·5
12	45	80	55	13	24	45	0·044	0·017	8·2	17·0
13	58	59	92	89	50	67	0·135	0·066	1·5	2·5
14	64	112	53	18	28	52	0·059	0·025	7·0	7·0
15	87	82	59	21	42	60	0·124	0·056	1·4	4·2
16	47	76	57	14	33	45	0·134	0·050	0	0
17	51	101	58	18	24	40	0·166	0·051	4·1	5·2
18	88	106	69	43	52	61	0·234	0·113	3·0	5·4
19	50	63	78	30	50	25	0·080	0·039	2·4	3·0
20	76	110	74	50	47	95	0·317	0·097	0·8	0·5
21	73	112	60	36	52	74	0·277	0·071	1·7	3·2
22	64	87	70	44	47	40	0·057	0·020	1·2	3·4
23	63	120	61	12	37	44	0·033	0·012	11·1	12·8
24	75	101	53	18	46	54	0·161	0·058	3·0	4·3
25	59	90	75	40	43	65	0·082	0·033	2·0	4·6
26	56	96	79	47	52	54	0·102	0·066	1·8	0
27	56	113	67	24	36	41	0·185	0·060	1·3	1·9
28	69	91	79	63	48	131	0·185	0·068	1·2	2·3
29	45	51	69	24	36	61	0·161	0·094	3·2	2·6
30	73	98	74	52	62	59	0·139	0·057	3·8	3·8
31	88	93	84	90	72	60	0·250	0·090	0·3	1·6
Mean	62·6	100·5	67·4	35·6	41·5	60·0	0·133	0·051	3·68	6·42
S.E.	2·6	3·9	2·1	4·8	2·5	3·9	0·014	0·005	0·65	1·50

[1]Per cent predicted

ployed in this laboratory for the fractionation of A-aD O_2 have been described elsewhere (Guleria *et al.*, 1971).

Six patients were restudied four months after resection of the bronchiectatic segments. The parameters measured included routine lung volumes, TL_{CO}, and pulmonary mechanics.

Standard statistical methods were employed to assess the significance of change in pulmonary mechanics following the bronchodilator and to calculate the coefficients of correlation between any two measurements (Snedecor, 1956).

RESULTS

The results of routine pulmonary function tests and pulmonary mechanics before and after a bronchodilator are given in Tables II and III. The vital capacity was less than 80% predicted in 28 patients, the mean value in 31 patients being 62·6% (S.E. ±2.6). FRC was increased in four patients and decreased in three while in the remaining patients it was within normal limits (80 to 120% predicted). MMFR and maximum breathing capacity (MBC) showed a striking reduction in a majority of the patients, their mean values being 35.6 (S.E. ±4·8) and 41·5 (S.E. ±2·5%) predicted respectively. TL_{CO} was decreased below 80% predicted in all but four patients [mean 60·0 (S.E. ±3.9%) predicted]. Dynamic compliance (C_L) was abnormally low in 13 patients. The normal range of C_L in the laboratory has been found to be 0.112–0.317 1/cm H_2O (Guleria *et al.*, 1969). In 11 patients specific compliance, which is the ratio of lung compliance and FRC, was less than the lower limit of 0.038 cited by Marshall (1957). Inspiratory and expiratory pulmonary flow resistance were increased (i.e., >1·2 and >2·9 cm H_2O/l/sec) in 23 and 21 patients respectively. Mean C_L decreased further after aminophylline

TABLE III

REVERSIBILITY OF AIRWAY OBSTRUCTION

	C_L (l/cm H_2O) (mean ± S.E.)	Pulmonary Resistance (cm H_2O/l/sec) (mean ± S.E.)	
		Inspiratory	Expiratory
Resting	0·133 ± 0·014	3·68 ± 0·65	6·42 ± 1·50
After aminophylline:			
5 minutes	0·119 ± 0·013	3·21 ± 0·53[2]	5·84 ± 1·10
10 minutes	0·127 ± 0·014	2·80 ± 0·44[2]	5·49 ± 1·20[1]
15 minutes	0·119 ± 0·014	3·10 ± 0·57[2]	5·26 ± 1·00[1]

[1]$P < 0.05$; [2]$P < 0.001$.

but the changes were insignificant. The inspiratory resistance decreased very significantly ($P<0.001$) after aminophylline. The expiratory resistance also decreased though this change was less significant statistically ($P<0.05$).

Table IV gives the coefficients of correlation of various parameters of pulmonary function with the extent of disease as judged by the number of segments involved. This correlation was found to be the best with TL_{CO} ($P<0.001$). MMFR, MBC, and C_L also correlated highly significantly with the extent of disease ($P<0.01$). Vital capacity and FEV_1 showed the least significant correlation ($P<0.05$), while FRC and inspiratory and expiratory pulmonary resistance did not appear to be influenced by the extent of disease.

The results of blood gas analysis and fractionation of A–aD O_2 in seven patients are given in Table V. Arterial carbon dioxide tension was markedly increased in only one patient but all of them had significantly lowered oxygen tension. Total A–aD O_2 was widened in all (mean 36·6 S.E. $\pm$3.6 mmHg) and this resulted mainly from ventilation/perfusion ($\dot{V}/Q$) abnormalities which made up 22·5 mmHg of total A–aD O_2. True right-to-left shunts were also increased to 13·6% of the cardiac output and contributed 8·5 mmHg to the total A-aD O_2. All these patients had extensive bronchiectasis involving eight or more segments of the lung.

Table VI gives the pre and postoperative pulmonary function results for six patients who were treated surgically. All the bronchiectatic segments were removed in five, while in one patient (12) two abnormal segments were left behind because the disease was bilateral. Postoperative studies showed a worsening of function in a majority of patients. The most consistent changes were noticed in vital capacity, FRC, TL_{CO}, and C_L, each of which decreased in five of the six subjects.

TABLE IV

COEFFICIENTS OF CORRELATION BETWEEN PULMONARY FUNCTION AND EXTENT OF BRONCHIECTASIS IN 31 PATIENTS

Pulmonary Function	Coefficient of Correlation	P Value
TL_{CO}	−0·559	<0·001
MMFR	−0·554	<0·01
MBC	−0·549	<0·01
C_L	−0·449	<0·01
VC	−0·380	<0·05
FEV_1	−0·380	<0·05
Inspiratory resistance	+0·245	N.S.
Expiratory resistance	+0·241	N.S.
FRC	−0·017	N.S.

DISCUSSION

The results of pulmonary function tests obtained on the present series of patients are similar to those reported by earlier investigators. The patients were characterized by a reduced vital capacity with varying degrees of airway obstruction without any significant overinflation in most of them. The steady state pulmonary transfer factor was decreased. The dynamic compliance was reduced and the pulmonary flow resistance showed a variable increase. The airway obstruction was found to be partially reversible by a bronchodilator. Thus the pattern of pulmonary dysfunction differed from that seen in chronic obstructive lung disease only by the absence of any marked overinflation. The absence of significant overinflation in bronchiectasis was also reported by Kamener, Becklake, Goldman, and McGregor (1958), who found the mean FRC in 20 patients to be 110% predicted.

TABLE V

ALVEOLAR-ARTERIAL OXYGEN TENSION GRADIENT

Serial No.	$\dot{V}_E$, BTPS (l/min)	$\dot{V}_A$, BTPS (l/min)	PAO_2 (mmHg)	PCO_2 (mmHg)	pH	PaO_2 (mmHg)	Alveolar-arterial Oxygen Tension Gradient				R→L Shunt (% CO)
							Diffusion Component (mmHg)	Distribution Component (mmHg)	True Shunt Component (mmHg)	Total (mmHg)	
1	7·08	3·10	90·9	56·5	7·44	46·5	4·0	38·2	2·3	44·5	7·9
2	8·17	3·83	105·9	38·5	7·44	56·5	10·9	22·8	15·7	49·4	17·0
8	5·36	2·00	85·4	45·5	7·40	50·5	2·0	24·9	8·0	34·9	17·4
10	7·41	2·64	81·5	45·0	7·42	52·0	5·5	19·0	5·0	29·5	12·8
11	7·41	2·69	89·6	45·5	7·44	45·0	5·6	36·0	3·0	44·6	8·8
12	7·38	2·61	99·3	45·0	7·42	72·0	6·0	9·3	12·0	27·3	14·0
14	5·83	2·45	92·1	41·0	7·46	66·0	5·6	7·0	13·5	26·1	17·1
Mean ±S.E.	6·95 0·37	2·76 0·22	92·1 3·1	45·3 2·2	7·43 0·02	55·5 3·9	5·7 1·0	22·5 4·9	8·5 2·0	36·6 3·6	13·6 1·5

V_E=minute ventilation; V_A=alveolar ventilation; P_{AO_2}=mean alveolar oxygen tension; P_{CO_2}=arterial CO_2 tension; Pa_{O_2}=arterial oxygen tension; CO=cardiac output

TABLE VI

PULMONARY FUNCTION AFTER SURGERY

Serial No.	No. of Segments Removed	Time of Study	VC[1]	FRC[1]	FEV_1 (%VC)	MMFR[1]	MBC[1]	TL_{CO}[1]	C_L (l/cm H_2O)	Pulm. Resistance (cm H_2O/l/sec) Insp.	Exp.
6	4	B	80	96	95	107	69	88	0·299	0·7	1·0
		A	50	90	97	88	53	54	0·079	2·0	3·0
11	8	B	40	91	69	14	17	65	0·056	4·5	6·5
		A	30	82	65	12	15	50	0·028	7·3	17·3
12	10	B	45	80	55	13	24	45	0·044	8·2	17·0
		A	33	94	51	9	17	49	0·034	19·3	28·8
13	2	B	58	59	92	89	50	67	0·135	1·5	2·5
		A	60	62	80	85	45	65	0·143	0	0·4
20	4	B	76	110	74	50	47	95	0·317	0·8	0·5
		A	70	102	75	52	50	88	0·153	0	0
24	6	B	75	101	53	18	46	54	0·161	3·0	4·3
		A	60	91	60	22	50	44	0·144	0·9	2·9
Mean	B	B	62·3	89·5	73·0	48·5	42·1	69·0	0·169	3·1	5·3
		A	50·5	86·8	71·3	44·7	38·3	58·3	0·097	4·9	8·7

B=before surgery; A=after surgery
[1]Per cent predicted

The presence of airway obstruction in bronchiectasis has been reported by Smith *et al.* (1954), Cherniack *et al.* (1959), and Cherniack and Carton (1966). The last of these investigators reported an increase in pulmonary flow resistance in a significant number of patients. Kamener *et al.* (1958) also found an increased pulmonary resistance in their four patients before surgery. The mechanism of airway obstruction in bronchiectasis has not been fully elucidated. It might be the result of bronchial plugging by secretions, mucosal oedema, bronchospasm, distortion, and kinking of bronchi or by excessive dynamic compression of the airways due to a greater pliability of the affected bronchi. Chronic bronchitis frequently complicates bronchiectasis. Thus bronchographic evidence of chronic bronchitis in the form of mucus gland filling was found to be present in six of the 20 patients with bronchiectasis studied by Fraser, Macklem, and Brown (1965). In a study of airway dynamics in bronchiectasis by a combined cinefluorographic and manometric technique, these investigators reached the conclusion that the mechanism of airway obstruction in bronchiectasis was similar to that seen in chronic obstructive lung disease. A highly significant decrease in the inspiratory resistance observed in the present study following the administration of aminophylline points to bronchospasm, particularly of the larger airways, as an important cause of airway obstruction.

The decrease in dynamic pulmonary compliance could be attributed to a non-uniform distribution of airway resistance as well as increased stiffness of the lungs due to patchy fibrosis and atelectasis. A smaller lung volume in some of the patients may have contributed to a decrease in dynamic compliance, but most of the patients having low dynamic compliance had decreased specific compliance as well. Cherniack and Carton (1966) observed a reduced pulmonary compliance in 13 of 31 patients. Kamener *et al.* (1958) also found decreased compliance in two of the four patients studied by them preoperatively.

The results of pulmonary transfer factor measurements have been variable in different series. Anderson *et al.* (1954) and Cherniack *et al.* (1959) determined TL_{O_2} by the method of Lilienthal, Riley, Proemmel, and Franke (1946) and obtained normal results in a majority of patients. Cherniack and Carton (1966) measured TL_{CO} by the single breath method in 42 patients and found it to be reduced below 70% predicted in only 12 of them. TL_{CO} measured by an end-tidal steady state technique was reduced in most of the patients studied by Bass *et al.* (1968). Our observations with a steady-state technique support the findings of Bass *et al.* (1968) that a significant decrease in pulmonary transfer factor is a frequent abnormality in bronchiectasis. Abnormalities of $\dot{V}/Q$ are frequently present in bronchiectasis. Since the steady-state method is particularly sensitive to $\dot{V}/Q$ disturbances, it is possible that this method for the measurement of TL_{CO} yields abnormal values more often than other methods.

Although significant correlations were found between the extent of disease and various measurements of function, correlation coefficients were much less than one. Thus only a minimal impairment of function was sometimes present in spite of extensive bilateral disease. Of all the variables tested, the TL_{CO} was found to correlate best with

the extent of involvement. Other correlations in descending order of significance were noted with MMFR, MBC, C_L, VC, and FEV_1. Pulmonary flow resistances were independent of the extent of disease. Cherniack and Carton (1966) found MBC, RV/TLC ratio, pulmonary flow resistance, and 7-minute N_2 index to correlate best with the extent of disease. Correlations with TL_{CO} and C_L were less significant.

The role of bronchospasm in producing airway obstruction is evident from the response to a bronchodilator. In addition to bronchospasm, airway obstruction may be caused by retained secretions, superadded infections, and other factors. It is not surprising that the degree of airway obstruction, as judged by pulmonary resistance, did not correlate with the anatomical extent of the disease.

Significant arterial hypoxaemia was constantly present in our patients with extensive bronchiectasis. Fractionation of the total A-aD O_2 showed that it was widened predominantly due to $\dot{V}/Q$ abnormalities. Right-to-left shunts were increased but contributed to a lesser extent to hypoxaemia. Perfusion of unventilated alveoli could be partly responsible for the intrapulmonary right-to-left shunt observed in the present series of patients, but in a group of 10 patients with severe chronic obstructive lung disease who had more pronounced $\dot{V}/Q$ abnormalities, the magnitude of true right-to-left shunt was found to be much smaller (Gupta, 1970). It is possible, therefore, that new vascular communications in the bronchial wall and granulation tissue might shunt blood from the pulmonary arterioles to pulmonary veins. The presence of bronchial artery to pulmonary artery shunts in bronchiectasis has been demonstrated by Liebow, Hales, and Lindskog (1949) and Roosenburg and Deenstra (1954). Weibel (1959), on the basis of morphological studies, concluded that there was little possibility of pulmonary arterial blod being shunted to the pulmonary veins without passing through the alveolar capillary network. von Hayek (1960), on the other hand, has presented evidence in favour of the existence of such pulmonary arteriovenous anastomoses chiefly in the bronchial walls. These communications may, indeed, become prominent in bronchiectasis and thus account for the increased right-to-left shunt. True right-to-left shunts in bronchiectasis, of the magnitude reported in the present series, have not previously been reported. Anderson *et al.* (1954) and Cherniack *et al.* (1959) measured total venous admixture but did not investigate separately the contribution of true right-to-left shunts. The recognition of right-to-left shunts in bronchiectasis may be of practical importance since these shunts are likely to diminish following resection of the affected segments with an increase in arterial oxygen tension. On the other hand, virtual shunts due to $\dot{V}/Q$ abnormalities are unlikely to improve since pulmonary resistance does not show any decrease postoperatively (Kamener *et al.*, 1958). Smith *et al.* (1954) found that total venous admixture decreased after surgery. This decrease probably resulted from a reduction in true right-to-left shunts rather than from an improvement in $\dot{V}/Q$ abnormalities.

The slight deterioration which we observed postoperatively in all variables of pulmonary function, including TL_{CO} and pulmonary mechanics, is consistent with the observations of Kamener *et al.* (1958) and Smith *et al.* (1954). The former investigators reported an improvement in pulmonary functions after a lapse of more than five months from the time of surgery. Unfortunately, few data are available on the postoperative blood gas tensions in their patients. The effect of pulmonary resection on right-to-left shunts in bronchiectasis would be a suitable subject for further investigation.

REFERENCES

Anderson, L. L., Bell, J. C., and Blount, S. G., Jr. (1954). An evaluation of factors affecting the alveolar-arterial oxygen tension gradient in chronic pulmonary disease. *Amer. Rev. Tuberc.*, **69**, 71.

Ayres, S. M., Criscitiello, A., and Grabovsky, E. (1964). Components of alveolar-arterial O_2 difference in normal man. *J. appl. Physiol.*, **19**, 43.

Bass, H., Henderson, J. A. M., Heckscher, T., Oriol, A., and Anthonisen, N. R. (1968). Regional structure and function in bronchiectasis. *Amer. Rev. resp. Dis.*, **97**, 598.

Bates, D. V., Boucot, N. G., and Dormer, A. E. (1955). The pulmonary diffusing capacity in normal subjects. *J. Physiol. (Lond.)*, **129**, 237.

—— Woolf, C. R., and Paul, G. I. (1962). Chronic bronchitis: a report on the first two stages of the co-ordinated study of chronic bronchitis in the Department of Veterans Affairs, Canada. *Med. Serv. J. Can.*, **18**, 211.

Bawa, Y. S., and Guleria, J. S. (1957). A chest clinic survey for 1952–1954 with special emphasis on bronchiectasis. *J. Ass. Phycns India*, **5**, 144.

Bhargava, R. K., and Woolf, C. R. (1967). Changes in diffusing capacity after bronchography. *Amer. Rev. resp. Dis.*, **96**, 827.

Cherniack, N. S., and Carton, R. W. (1966). Factors associated with respiratory insufficiency in bronchiectasis. *Amer. J. Med.*, **41**, 562.

—— Vosti, K. L., Saxton, G. A., Lepper, M. H., and Dowling, H. F. (1959). Pulmonary function tests in fifty patients with bronchiectasis. *J. Lab. clin. Med.*, **53**, 693.

Christoforidis, A. J., Nelson, S. W., and Tomashefski, J. F. (1962). Effects of bronchography on pulmonary function. *Amer. Rev. resp. Dis.*, **85**, 127.

Fraser, R. G., Macklem, P. T., and Brown, W. G. (1965). Airway dynamics in bronchiectasis. A combined cinefluorographic-manometric study. *Amer. J. Roentgenol.*, **93**, 821.

Goldman, H. I., and Becklake, M. R. (1959). Respiratory function tests: normal values at median altitudes and the prediction of normal results. *Amer. Rev. Tuberc.*, **79**, 457.

Guleria, J. S., and Chitkara, R. K. (1966). Natural history of bronchopulmonary suppuration with emphasis on symptomatology. *Indian J. Chest. Dis.*, **8**, 54.

—— Pande, J. N., Markose, M. M., Gupta, R. G., and Jain, B. P. (1971). Pulmonary function in chronic severe anaemia. *Clin. Sci.*, **40**, 317.

—— Sharma, M. P., Pande, J. N., and Ramchandran, K. (1970). Pulmonary diffusing capacity in normal Indian subjects. *Indian J. Physiol. Pharmacol.*, **14**, 245.

—— Talwar, J. R., Malhotra, O. P., and Pande, J. N. (1969). Effect of breathing cold air on pulmonary mechanics in normal man. *J. appl. Physiol.*, **27**, 320.

Gupta, R. G. (1970). Quantitative fractionation of alveolar-arterial oxygen tension difference in normal man and in patients with bronchiectasis and chronic obstructive lung disease. Thesis for M.D., All India Institute of Medical Sciences, New Delhi.

Hayek, H. von (1960). *The Human Lung*, translated by V. E. Krahl. Hafner, New York.

Kamener, R., Becklake, M. R., Goldman, H., and McGregor, M. (1958). Respiratory function following segmental resection of the lung for bronchiectasis. *Amer. Rev. Tuberc.*, **77**, 209.

Liebow, A. A., Hales, M. R., and Lindskog, G. E. (1949). Enlargement of the bronchial arteries in bronchiectasis. *Amer. J. Path.*, **25**, 211.

Lilienthal, J. L., Jr., Riley, R. L., Proemmel, D. D., and Franke, R. E. (1946). An experimental analysis in man of the oxygen pressure gradient from alveolar air to arterial blood during rest and exercise at sea level and at altitude. *Amer. J. Physiol.*, **147**, 199.

Marshall, R. (1957). The physical properties of lungs in relation to the subdivisions of lung volume. *Clin. Sci.*, **16**, 507.

Mead, J., and Whittenberger, J. L. (1953). Physical properties of human lungs measured during spontaneous respiration. *J. appl. Physiol.*, **5**, 779.

Pande, J. N., Jain, B. P., and Guleria, J. S. (1970). Reversibility of airway obstruction in chronic obstructive lung disease. *Amer. Rev. resp. Dis.*, **101**, 371.

Roosenburg, J. G., and Deenstra, H. (1954). Bronchial-pulmonary vascular shunts in chronic pulmonary affections. *Dis. Chest*, **26**, 664.

Smith, G. A., Siebens, A. A., and Storey, C. F. (1954). Preoperative and postoperative cardiopulmonary function studies in patients with bronchiectasis. *Amer. Rev. Tuberc.*, **69**, 869.

Snedecor, G. W. (1956). *Statistical Methods*, 5th ed. Iowa State University Press. Ames, Iowa.

Weibel, E. (1959). Die Blutgefässanastomsen in der menschlichen Lunge. *Z. Zellforsch.*, **50**, 653.

Williams, M. H., Jr. (1954). Pulmonary function in bronchiectasis. Walter Reed Centre Army School, Army Medical Science Graduate School, Washington, D.C.