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Research Article

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By contrast, in all patients who underwent bullectomy, there was a decrease in functional residual capacity but an increase in airway conductance, and an increase in the conductance/volume ratio. This change was sustained in patients who had had localized cysts removed. However, the measurements gradually reverted toward preoperative values in those patients who had generalized emphysema.

The increase in airway conductance after resection of blebs and bullae presumably was due to improved lung elastic pressure causing the airways to increase in diameter and conductance. In addition, some patients may have experienced relief of compression of neighboring airways.

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Effect of Removal of Bullae on Airway Conductance and Conductance Volume Ratios

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ABSTRACT Airway conductance is known to increase with an increase in the lung volume at which it is measured, owing to a change in transpulmonary pressure and lung tissue tension. We investigated the effect of surgical resection of lung tissue on functional residual capacity and airway conductance in patients with localized lung disease (i.e., carcinoma or tuberculosis) and in patients with lung cysts or bullous emphysema. In four out of five of the patients who had resection of one or more lobes of the lung to remove localized disease there was a reduction both in the airway conductance and in the functional residual capacity with relatively little change in the conductance volume ratio.

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The increase in airway conductance after resection of blebs and bullae presumably was due to improved lung elastic pressure causing the air-

ways to increase in diameter and conductance. In addition, some patients may have experienced relief of compression of neighboring airways.

INTRODUCTION

Airway conductance increases as the lung volume at which it is measured increases (1). Reduction in lung volume by chest strapping increases the lung elastic pressure for any given lung volume (2) with a resulting increase in airway conductance. Thus, the relationship between conductance and volume is a function of the lung elastic pressure rather than the lung volume itself (3). This increase of airway conductance has been found to be attributable partly to a greater bronchial transmural pressure, and partly to a greater tension exerted by the tissues pulling outward on the airway walls (4). In patients with emphysema, the functional residual capacity is increased, the airway conductance measured at functional residual capacity is reduced (2), and frequently the airway conductance approaches zero as the lung volume is reduced to residual volume.

The surgical resection of bullae has been reported to cause clinical improvement. The mechanism for this improvement has never been clearly established. The purpose of this investigation was to examine the effects of surgical removal of lung tissue on airway conductance. Two groups of patients were studied. In the first group, an anatomic segment of lung parenchyma, such as a lobe, was removed. In the second group, bullae were re-

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moved. The surrounding lung tissue was left undisturbed insofar as this was possible.

METHODS

A brief description of the patients who had large bullae (J. K., W. S., W. K., and F. J.) and diffuse pulmonary emphysema with small bullae (M. W., R. M., and J. G.) is given in the appendix.

Airway resistance (R_A), thoracic gas volume (TGV), and functional residual capacity (FRC) were measured with a body plethysmograph (5, 6). Airway conductance, the reciprocal of airway resistance, was calculated from the airway resistance values. The airway conductance was measured, first close to functional residual capacity, and next with the patient holding his chest in an inspiratory position, then in an expiratory position. The patients panted at a rate of approximately 2 cycle/sec with a small volume displacement. All subjects had measurements made just before surgery and 10–30 days after surgery. All emphysematous patients had intensive medical therapy before being admitted to this study. All subjects were judged to be free of bronchospasm or bronchial infection at the time of the studies. In none of these patients was there much change in airway conductance measured before and after inhalation of isoproterenol aerosol. All patients had preoperative measurements of airway resistance and thoracic gas volume on several occasions, and had additional pulmonary function studies, including vital capacity, maximum mid-expiratory flow rate, FEV₁, FEV₂, maximum breathing capacity, functional residual capacity, and arterial blood gas studies as part of their preoperative evaluation. The information obtained by the spirometric tests contributed to the present study mainly as an aid in establishing the diagnosis. The preoperative values are listed in Tables I and II. Table II also contains the postoperative values for all of the patients who underwent bullectomy (group II), except

for patient M. W. on whom the spirometry tests and blood gases were not performed postoperatively.

RESULTS

Group I: Resection of pulmonary carcinoma or granuloma. The first group consisted of five patients who were scheduled to have resection of all or a portion of one lung, and whose remaining lung tissue appeared normal on the chest roentgenogram. Four of the patients had carcinoma of the lung and one had a small tuberculous granuloma. This latter patient had a wedge resection, three others had lobectomies, and one had a pneumonectomy. In Table III are listed the pre- and postoperative values for airway conductance, thoracic gas volume, and conductance/volume ratios for the entire group. All had normal preoperative values for airway conductance except for H. McD. who had a low conductance and a slight elevation of functional residual capacity. The conductances are the mean conductances measured at the mean panting gas volume closest to functional residual capacity. Postoperatively in one patient who had a lobectomy there was essentially no change in airway conductance despite a decrease in lung volume, but in the other four patients there was a decrease in airway conductance with a decrease in lung volume (Table III). The preoperative and postoperative values for one such patient, who underwent a wedge resection, are shown in Fig. 1. The line was drawn by visual approximation. It appears that the pre- and postoperative values lie

TABLE I
Group I—Patients with Localized Lung Disease

Patient	V.C.	MMEFR	MMIFR	FEV ₁	FEV ₁ *	FEV ₂ *	MVV
	<i>liter</i>	<i>liter/min</i>	<i>liter/min</i>	<i>liter</i>	<i>%</i>	<i>%</i>	<i>liter/min</i>
J. B. Predicted	5.28	204–348		4.24	>70	>97	145
Preoperative	5.46	321	400	4.65	86	100	211
B. K. Predicted	3.22	162–246		2.77	>70	>97	83
Preoperative	2.59	165	178	2.20	87	100	61
H. McD. Predicted	4.38	120–240		3.38	>70	>97	99
Preoperative	3.50	39	175	2.10	58	77	90
A. B. Predicted	3.05	132–240		2.55	>70	>97	75
Preoperative	3.16	144	242	2.30	81	96	108
R. W. Predicted	4.02	180–336		3.39	>70	>97	115
Preoperative	2.26	142	135	1.94	86	100	59

B. K., H. McD., A. B., and R. W. had pulmonary carcinoma; J. B. had a small pulmonary granuloma (tuberculoma).

* Expressed as the percentage of the total forced expiratory volume.

TABLE II
Group II—Patients with Bullae

Patients		V.C.	MMEFR	MMIFR	FEV ₁	FEV ₁ *	FEV ₂ *	MVV	Blood gases (arterial)			
									O ₂ Sat	Po ₂	PcO ₂	pH
		liter	liter/min	liter/min	liter	%	%	liter/min	%	mm Hg	mm Hg	
Large bullae												
W. S.	Predicted	5.28	204–348		4.24	>70	>97	145				
	Preoperative	3.05	90	135	2.10	68	90	87	94	73	46	7.46
	Postoperative	2.95	175	250	2.45	83	98	75	94	73	47	7.46
J. K.	Predicted	4.82	180–336		3.84	>70	>97	127				
	Preoperative	5.08	181	434	3.56	72	95	167	—	—	—	—
	Postoperative	3.36	179	339	2.82	84	100	126	—	—	—	—
W. K.	Predicted	4.96	204–348		4.05	>70	>97	140				
	Preoperative	1.56	19	77	0.64	50	83	23	90.0	57	20	7.43
	Postoperative	2.29	59	144	1.42	72	100	48	94.0	76	33	7.38
F. J.	Predicted	4.96	204–348		4.05	>70	<97	140				
	Preoperative	3.10	36	174	1.53	63	75	61	88.8	—	33	7.44
	Postoperative 1	1.99	74	215	1.60	73	100	46	95.4	—	—	—
	Postoperative 2	2.69	156	253	2.35	84	99	131	94.3	—	43	7.42
Diffuse emphysema with "small" bullae												
M. W.	Predicted	3.18	132–240		2.62	>70	>97	77				
	Preoperative	2.36	13	110	0.54	31	56	18	94	61	37	7.49
	Postoperative	—	—	—	—	—	—	—	—	—	—	—
R. M.	Predicted	4.38	120–240		3.38	>70	>97	99				
	Preoperative	2.59	19	102	0.58	28	53	35	96	69	20	7.50
	Postoperative	3.15	26	126	0.82	30	54	24	94	62	43	7.52
J. G.	Predicted	4.68	150–282		3.66	>70	>97	114				
	Preoperative	2.80	19	130	0.65	27	54	24	94.8	76	38	7.39
	Postoperative	3.00	26	98	0.71	28	67	31	91.0	64	39	7.40

* Expressed as the percentage of the total forced expiratory volume.

approximately on the same line. The degree of reduction of airway conductance was consistent with the amount of decrease in thoracic gas volume.

Group II: Excision of localized lung cysts or emphysematous bullae. The second group consisted of seven patients who underwent bullectomy. Two of the patients had normal airway conductance before surgery, whereas five had low airway conductance. The results are summarized in Table IV. In all subjects there was a postoperative increase in airway conductance and, except for J. K., a decrease in lung volume. The increase in conductance and decrease in thoracic gas volume of the group were statistically significant. The last subject (F. J.) had no change in airway conductance or thoracic gas volume after the first operation, but following the second operation had a decrease in his thoracic gas volume and an increase in airway conductance. The results obtained on

one patient (J. G.) are plotted in Fig. 2. Preoperatively, the values lay to the right of the predicted line with the line of visual approximation intercepting the abscissa at a very high volume. Following operation there was a change in the slope of the line and also a shift of the volume intercept to the left.

Comparison between groups I and II. Data from the individual patients of groups I and II are plotted in Fig. 3. The individual preoperative (●) and postoperative (○) values for conductance, measured at a panting lung volume closest to functional residual capacity, are joined by lines. The decrease in conductance with the decrease in lung volume (solid line) can be clearly seen in four of the five patients of group I. The data from group II (broken lines) are distinctly different. All subjects had an increase in conductance and all but one had a decrease in lung volume.

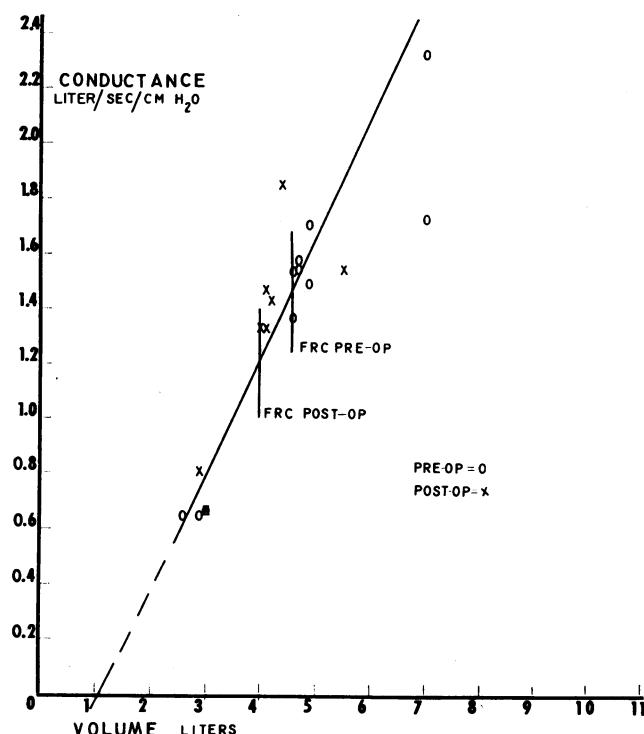


FIGURE 1 The conductance (G_A) (ordinate) is plotted against the thoracic gas volume (TGV) (abscissa) at which it was measured in a patient of group I (J. B.) who was to have a wedge resection of a tuberculous granuloma. The preoperative (O) and postoperative (X) values appear to fall along the same line (visual approximation) even though the functional residual capacity has decreased.

Differences within group II, the bullectomy group. Of the seven patients undergoing bullectomy, four (W. S., J. K., W. K., and F. J.) had large bullae occupying more than one-third of one hemithorax on the chest roentgenogram. The remaining three patients (M. W., R. M., and J. G.) had severe diffuse obstructive pulmonary emphysema with high airway resistance and no large bullae discernible on the chest roentgenogram. ^{131}I macroaggregated albumin lung scans

showed multiple areas of decreased perfusion which were found to represent small "bullous" areas at the time of surgery, when these areas were resected. All seven patients undergoing bullectomy had an increase in airway conductance after surgery. In the three patients with generalized emphysema, however, the values did not reach the predicted normal range, whereas in those patients with large, localized bullae, the values did reach normal. Long-term followup is

TABLE III
Group I—Patients with Localized Lung Disease (Pulmonary Carcinoma or Granuloma)

Patient	Operation	Preoperative values				Postoperative values			
		G_A^*	TGV	FRC†	G_A/V	G_A	TGV	FRC	G_A/V
		liter $\text{sec}^{-1} \text{cm H}_2\text{O}^{-1}$	liter	liter	$\text{sec}^{-1} \text{cm H}_2\text{O}^{-1}$	liter $\text{sec}^{-1} \text{cm H}_2\text{O}^{-1}$	liter	liter	$\text{sec}^{-1} \text{cm H}_2\text{O}^{-1}$
		Patient predicted				Patient predicted			
J. B.	Wedge resection	1.50 (0.60–1.63)	4.65	4.56	0.321	1.36 (0.53–1.42)	4.07	4.00	0.334
B. K.	Lobectomy	0.65 (0.29–0.77)	2.20		0.295	0.51 (0.22–0.60)	1.70	1.77	0.300
H. McD.	Lobectomy	0.66 (0.72–1.95)	5.57	5.20	0.118	0.57 (0.62–1.62)	4.47		0.127
A. B.	Lobectomy	0.35 (0.32–0.85)	2.43	2.67	0.144	0.37 (0.25–0.67)	1.93		0.192
R. W.	Pneumonectomy	1.10 (0.34–0.91)	2.60	2.49	0.428	0.57 (0.20–0.55)	1.56	1.62	0.365

* Mean airway conductance (G_A) was measured at a mean thoracic gas volume (TGV) at or close to functional residual capacity (FRC).

† FRC measured by body plethysmograph mean value of five consecutive measurements.

‡ Predicted value (3).

TABLE IV
Group II—Patients with Bullae

Patients	Preoperative values				Postoperative values			
	G _A *	TGV	FRC†	G _A /V	G _A *	TGV	FRC†	G _A /V
	liter sec ⁻¹ cm H ₂ O ⁻¹	liter	liter	sec ⁻¹ cm H ₂ O ⁻¹	liter sec ⁻¹ cm H ₂ O ⁻¹	liter	liter	sec ⁻¹ cm H ₂ O ⁻¹
	Patient predicted				Patient predicted			
Large bullae								
W. S.	0.75 (0.61–1.63)	4.20	4.52	0.215	0.96 (0.75–2.0)	3.30	3.15	0.291
J. K.	1.07 (0.57–1.54)	4.30	3.81	0.248	1.97 (0.58–1.56)	4.40	3.90	0.447
W. K.	0.29 (0.87–2.34)	6.73	6.85	0.043	0.57 (0.48–1.3)	3.73	3.72	0.152
F. J.‖								
1st operation	0.49 (0.57–1.54)	4.40	4.23	0.111	0.48 (0.57–1.54)	4.40	4.50	0.109
2nd operation					0.62 (0.41–1.09)	3.13	2.97	0.198
Diffuse emphysema with "small" bullae								
M. W.	0.14 (0.86–2.31)	6.60	6.87	0.021	0.22 (0.66–1.78)	5.10		0.043
R. M.	0.24 (1.05–2.84)	8.10	8.26	0.030	0.53 (0.68–1.70)	6.00	5.18	0.088
J. G.	0.33 (1.0–2.86)	7.91	8.08	0.041	0.54 (0.75–2.0)	5.71	5.80	0.094

* Mean airway conductance (G_A) measured at the stated mean thoracic gas volume (TGV) at or closest to functional residual capacity (FRC).

† FRC, mean of five consecutive measurements in the body plethysmograph.

‡ Predicted values (3).

‖ First operation was on the right lung and second operation on the left lung.

available in two of the three emphysematous patients, and in three of the four patients in the group with large bullae but without generalized emphysema. Table V summarizes the data on all five patients. In the two patients with generalized

emphysema, the conductance/volume ratios returned, over a period of months, toward their preoperative values. In the other patient, with emphysema (M. W.), we were unable to obtain measurements, but her clinical course was one of

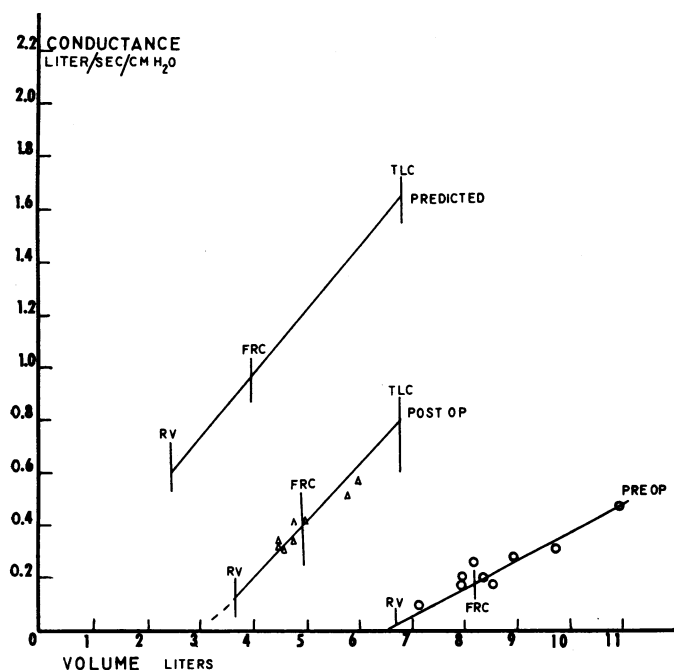


FIGURE 2 As in Fig. 1 the preoperative (○) and postoperative (△) conductances are plotted according to the TGV at which they were measured in a patient of group II, J. G., who had diffuse obstructive pulmonary emphysema with small bullae.

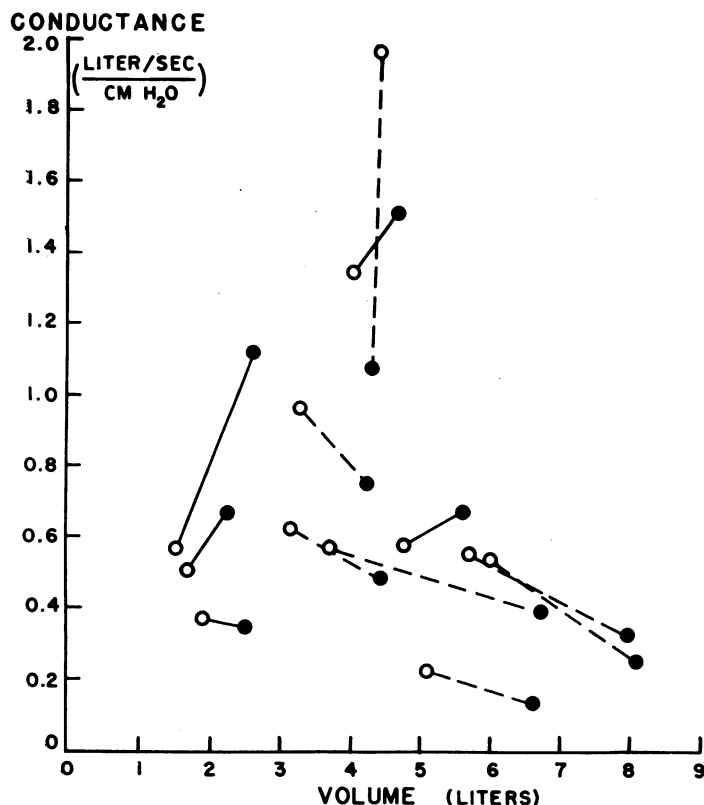


FIGURE 3 In this figure are plotted the pre-operative (●) and postoperative (○) conductances measured at or close to functional residual capacity in group I (solid line) and group II (dashed line). In group I four of the five patients have a decrease in conductance, whereas all of the group II (bullectomy) demonstrate an increase in conductance.

progressive respiratory failure. At least two of the subjects with large bullae maintained their postoperative improvement longer than did the patients with generalized emphysema. This difference is apparent in Fig. 4 wherein the conductance/volume ratios after bullectomy for two patients with large bullae [F. J. (×) and W. K. (●)] and one patient with generalized emphysema [R. M. (○)] are plotted.

DISCUSSION

Our data strongly suggest that airway conductance decreases after the removal of an anatomic segment of "relatively normal lung tissue" that includes a major airway (group I), but increases after the resection of bullae without the removal of a major airway (group II). This difference is present even though the thoracic gas volume becomes reduced in both instances.

Although all the patients who underwent bullectomy had an increase in their airway conductance, it would appear that in some, probably those with diffuse obstructive emphysema, the improve-

ment disappeared over the course of several months whereas in others, probably those without generalized emphysema, the increase of conductance was sustained.

The initial criteria for distinguishing between the patients in group II are not as yet definite. We used the pulmonary function studies, the chest X-ray, and ^{131}I -macroaggregated albumin lung scan as guides in that they seemed to indicate whether the bullae seen on the roentgenogram were associated with a diffuse process that we called generalized emphysema.

The decrease in airway conductance in group I might be explained as follows. Since the total airway conductance is the arithmetic sum of all airway conductances, then the removal of airways, as occurs with lobectomy or pneumonectomy, would reduce the total number of airways and therefore the total conductance. Furthermore, if the conductance/volume ratio within the resected lung were equal to the whole lung's conductance/volume ratio, then this latter would not change. The mathematical equivalent of the above state-

TABLE V
Group II—Conductance-Volume Ratios* after Bullectomy

Patient.....	Large bullae without diffuse emphysema			Diffuse emphysema§ with small bullae	
	W.S.	W. K.	F. J.†	R. M.	J. G.
Preoperative values				0.026	0.049
		0.043	0.110	0.032	0.057
	0.215	0.038	0.109	0.029	0.031
Bullectomy	—	—	—	—	—
Months after bullectomy 1	0.291	0.150		0.088	0.094
2				0.079	0.081
3				0.088	0.063
4					0.050
5	0.60				0.058
6				0.059	0.053
7				0.059	0.084
8				0.053	died
9				0.039	
10					
11		0.140	0.198	0.039	
12				0.046	
13					
14					
15				0.048	
16	0.223				
17				died	
18		0.110			
19		0.149	0.199		
—					
24		0.101			
25		0.106			
—					
34			0.171		
—					
88			0.202		

* R-units, sec⁻¹ cm H₂O⁻¹.

† The first value 0.110 is before bullectomy on the right, and the second (0.109) is after bullectomy on the right and before bullectomy on the left.

§ Bulla not discernible on the routine chest roentgenogram.

|| Patient died in respiratory insufficiency following bullectomy in the previous unoperated lung.

ment is as follows:

$$G_{AT} = G_{A_1} + G_{A_2} + G_{A_3} \quad (1)$$

$$V_T = V_1 + V_2 + V_3 \quad (2)$$

$$\frac{G_A}{V} = \frac{G_{A_1} + G_{A_2} + G_{A_3}}{V_1 + V_2 + V_3} \quad (3)$$

Where G_{AT} is the total airway conductance, and G_{A_1} , G_{A_2} , and G_{A_3} are the conductances of the airways of an individual segment of lung. V_T is the total volume of all lung segments with V_1 , V_2 , and V_3 equal to the volume of individual segments. With the removal of a segment or lobe then the

total conductance would decrease, since the conductances of the airways removed, for example, G_{A_3} would be subtracted from the total. However, the volume V_3 also would be removed and if the G_{A_3}/V_3 ratio were the same as the G_{AT}/V_{AT} then the latter (G_A/V) would be the same post-operatively. The increase in the conductance/volume ratio after bullectomy can be explained with analogous reasoning. With bullectomy the major airways to a lobe are not disturbed, i.e., the bulla is merely clamped and unroofed with suturing together of the relatively normal surrounding tis-

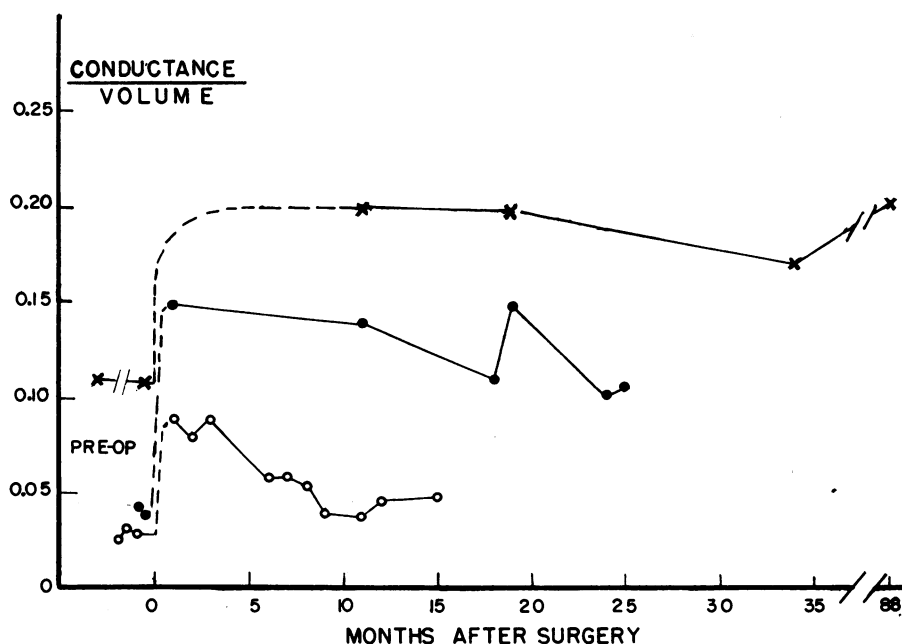


FIGURE 4 Conductance/volume (G_A/V) ratios are plotted for two patients who had large bullae resected (F. J. (X) and W. K. (●)) and one patient with diffuse obstructive emphysema who had small bullae resected (R. M. (○)). Preoperative values are plotted to the left of the zero (which indicate the time of bullectomy) on the abscissa. For the one patient (F. J.) (X), the first preoperative value is before bullectomy, on the right, and the second before bullectomy, on the left. Patients J. K. and W. K. are alive and doing well while R. M. died of respiratory insufficiency 16 months after surgery.

sue. If the conductance of the bullous area was extremely low preoperatively and the volume was disproportionately high, as would be expected, then postoperatively one would anticipate that the volume would be decreased proportionately more than the conductance, and therefore the total conductance/volume ratio would increase. This is what occurred in all the bullectomy patients in group II.

Although the above reasoning can account for the increase in the G_A/V ratio it does not account for the absolute increase in airway conductance. To explain this one must assume that preoperatively the bullae in some manner decreased the total conductance of the conducting airways, since postoperatively the total conductance increased. The algebraic expression of this situation would be as follows: $G_{AT} = G_{A1} + G_{A2} + G_{A3}$. If G_{A3} were removed, G_{AT} would increase postoperatively only if G_{A1} and G_{A2} or one of these increased postoperatively. Since G_{AT} would be increased, V_T would be decreased by the reduction in volume V_3 and the G_{AT}/V_T ratio would increase.

The following possible mechanisms whereby bullae might decrease airway conductance are suggested by the above. In the normal lung, the airways increase in diameter and length with increasing lung volume. This increase in size is a function of the lung elastic pressure (2), and requires either a uniform transmission of this pressure throughout the lung (7) or a local effect on tissues surrounding an airway (4). With the removal of bullae and the apposition of relatively normal tissue, the lung elastic pressure would be greater and might be better transmitted to all airways, leading to an increase in airway conductance with a change in the slope of the conductance-volume plot. Since bullae have been shown to have poor elastic properties, resembling those of a paper bag (8), they may not transmit the lung elastic pressure to surrounding airways, and therefore the airways in turn will not enlarge with increasing lung volume. This would then lead to a decreased slope of the conductance-volume plot, as has been observed in emphysema (2). Also, due to the large amount of air trapped in these bullae

(i.e., it cannot be expelled during expiration), the extrapolated intercept of the conductance-volume line on the volume axis would be at a very high volume. These mechanisms are consistent with the findings in the present study, although we do not have measurements of lung elastic pressure before and after surgery. We did not make this measurement because of the difficulty of doing repeated esophageal balloon studies on a patient.

If surgical resection of a part of the lung increases the lung elastic pressure at any given volume in a way comparable to the change after chest strapping (2, 3) then it might be expected that if there had been a generalized abnormality in lung elastic recoil preoperatively, postoperatively, airway conductance would be improved. But if the tissues exhibited stress relaxation, there would be a gradual return to the previous airway conductance and lung volume over a period of time. For example, bullae that were small before operation and that were able to withstand the lung elastic pressure before surgery might enlarge as a result of the new pressure-volume relationships. On the other hand, if the pulmonary parenchyma remaining after bullectomy were normal, then one might expect that the new pressure-volume relationships would lead to an increased conductance that would be maintained over a period of time, as occurred in the patients who had the resection of large bullae which did not appear to be part of a generalized process.

Other factors that have not as yet been evaluated may play a significant role in the increased conductance and the length of time it is maintained. The effect of a pleural reaction on bullae has not been adequately studied. It is conceivable that postoperative pleural reaction might fuse the visceral and parietal pleura which might limit the expansion of remaining bullae, since they would not be free to expand in all directions. In addition, if the pleural space became obliterated, this might alter the postural effects on vertical gradients of pleural pressure (9) and conceivably change the distribution of lung elastic pressure.

Bullae have been known to be associated with airway obstruction (10-14) but it has never been clearly demonstrated whether they cause or contribute to the obstruction, whether the obstruction produces the bullae, or whether there is a generalized disease that produces both the bullae and the

airway obstruction. Although the spirometric studies in most of our patients (Table II) demonstrated some improvement it is difficult to relate these results to the airways, since these studies are effort dependent. The body plethysmograph measures airway conductance and is not dependent on muscular effort. It is clear that bullectomy increases the airway conductance. Hence, one must conclude that bullae play a role in the decreased airway conductance seen before bullectomy. The predominant abnormality displayed by these patients was a mechanical one that was corrected by bullectomy, albeit in some cases only temporarily. Whether the bullae developed because of localized airway obstruction and then participated in the maintenance of generalized, as well as localized, obstruction, or whether they arose *de novo* and caused a generalized decrease in airway conductance is not clear. From our data, however, both possibilities must be considered and the question is at present unanswered.

On the basis of our findings, we see no reason to change the criteria for surgery outlined by Baldwin et al. (12) and further elaborated by Laurenzi, Turino, and Fishman (10). Bullectomy in patients with severe diffuse chronic obstructive emphysema cannot be supported by us on the basis of the present data. All such patients in our study returned toward their preoperative condition over a period of time. To establish whether a patient with large bullae also has diffuse obstructive emphysema is still a most difficult problem to which we have not found an easy answer.

APPENDIX

Brief clinical summaries of the patients in group II

J. K. was a 46 yr old white male with a 40-pack-yr¹ history of cigarette smoking, who had a large bulla (11 × 16 × 16 cm) noted on a tuberculosis control survey chest roentgenogram. His only previous chest roentgenogram had been made while he was in the Armed Services some 20 yr ago. Physical findings were restricted to a pectus excavatum of a moderate degree. He denied any pulmonary symptomatology. A right upper lobectomy was performed.

W. S. was a 35 yr old Negro male who had a 3 month history of dyspnea on exertion and a nonproductive cough. A chest roentgenogram taken 2 months be-

¹ 1-pack-yr, the consumption by a person of 20 cigarettes/day for 1 yr.

fore surgery revealed a large bulla in the right upper lobe and "fibrosis" in the right lower lobe. Microscopic examination of the resected bulla and surrounding tissue revealed "sarcoid-like" lesions, in addition to the bullae. The patient also had a reduced single breath DL_{CO} (14.7 ml/min per mm Hg. Predicted: 26.7 ± 8).

W. K. was a 36 yr old Negro male with a 10-pack-yr history of smoking cigarettes who noted gradually progressing dyspnea over the 5 yr before admission. The dyspnea progressed more rapidly in the yr before admission so that at the time of surgery the patient was dyspneic at rest and severely dyspneic on mild exercise (such as walking to the bathroom). A chest roentgenogram revealed bilateral large apical bullae that occupied over 50% of each hemithorax; in addition, there was a small mass noted in the right mid-lung field. The patient underwent a wedge resection of the mass, which proved to be an adenocarcinoma, and excision and plication of bullae in the right upper and lower lung.

F. J. was a 33 yr old Negro male with a history of over 20-pack-yr of smoking cigarettes who complained of dyspnea and chest pain, both of which had become progressively more severe over a 2 yr period. He had a chronic cough with occasional paroxysms of coughing that caused an exacerbation of his chest pain. His cough was productive of thick, viscid, mucoid material. 4 yr before admission the patient had had bronchopneumonia. A chest roentgenogram revealed advanced bullous emphysema most marked in both lower lung fields. The patient had plication of bullae in the left lower lobe, and lingula and apical posterior segment of the upper lobe. After this procedure, the patient experienced little relief of his symptoms, so that 8 months later, plication and resection of bullae in the right lung were carried out. The majority of bullae were found in the lower lobe, but several small bullae were noted in the middle and upper lobe and also were resected.

M. W. was a 50 yr old white housewife with a 21-pack-yr history of cigarette smoking. The patient stated that for "many years she had had frequent colds which tended to persist" and they usually were associated with sputum production. 4 yr before surgery, she noticed the onset of exertional dyspnea that progressed to dyspnea at rest by the time of surgery. She received little relief from any medication. Her chest roentgenogram showed bilateral pulmonary emphysema with areas of increased radiolucency at both bases. Plication and resection of bullae in the right lung, predominantly in the lower lobe, were performed.

R. M. was a 64 yr old white male with a history of 40-pack-yr of cigarette smoking. He was told 5 yr before surgery that he had emphysema, and at that time he stopped smoking. He had a morning cough that was productive of minimal amounts of sputum and occasionally the cough occurred in paroxysms causing him to feel extremely weak. Over the 5 yr since the onset of his symptoms, the patient had progressive dyspnea so that at the time of the present admission he was able to walk only 5 to 10 steps without dyspnea. He noticed the most

severe dyspnea when he arose from a sitting position and this gradually lessened as he remained standing erect. A chest roentgenogram showed hyperaerated lung fields bilaterally with flattened diaphragms and widened intercostal spaces. The increased radiolucency was more marked in the upper lobes with some distortion of the vascular markings suggesting the presence of bullae, although no definite bullae could be clearly outlined in the roentgenogram. Plication and resection of bullae were carried out on the right upper and lower lobes.

J. G. was a 59 yr old white male with a 45-pack-yr history of cigarette smoking. The patient had first noticed dyspnea on exertion 9 yr before surgery. The patient's dyspnea increased gradually and insidiously over the subsequent 8-9-yr period so that at the time of admission he could walk only 15 yd without stopping and had to rest 3-4 min after ascending one flight of stairs. During this time, the patient also had a chronic cough productive of a teaspoon of white, clear sputum in the morning. The chest roentgenogram showed flattened diaphragms, with a marked degree of hyperaeration and increased radiolucency in both apices. Plication and resection of bullae in the right lung were performed. After an initial improvement in his symptoms, the patient returned toward his preoperative status. 6 months after the initial surgery on the right lung, the patient had a plication of bullae in the left lung. He died on the 15th postoperative day. His death was attributed to pneumonia and respiratory insufficiency.

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