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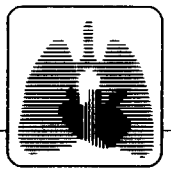

special report

Reduction Pneumoplasty for Giant Bullous Emphysema*

Implications for Surgical Treatment of Nonbullous Emphysema

Gordon L. Snider, MD, FCCP

A review of the literature on reduction pneumoplasty for giant bullous emphysema was undertaken to identify current criteria for this surgical treatment and in the hope of obtaining insights into evaluating reduction pneumoplasty for nonbullous emphysema. Twenty-two retrospective case series, published since 1950, were retrieved by a computer search of the literature and a search of the *Index Medicus* prior to 1966. Reduction pneumoplasty is most effective when bullae are larger than one third of a hemithorax with evidence of compression of adjacent lung tissue and an FEV_1 of less than 50% predicted; the presence of emphysema in nonbullous lung and the amount of compression are best judged by CT. The rationale for reduction pneumoplasty for nonbullous emphysema is supported by the similar early functional changes after reduction pneumoplasty for bullous and nonbullous—improvement of blood gas values and lung mechanics. A single study showing that decline of lung function after surgery for bullous emphysema was less in those who stopped smoking than in those who continued to smoke supports the need for preoperative and maintained smoking cessation in patients receiving reduction pneumoplasty. After 4 decades, the duration of improvement in lung function, whether worsening of emphysema occurs in remaining lung, and late morbidity and mortality after reduction pneumoplasty for bullous emphysema are not well defined. A registry with an unoperated-on comparison group could more rapidly accumulate such data after reduction pneumoplasty for nonbullous emphysema.

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Dco=diffusion of carbon monoxide; RV=residual volume; TLC=total lung capacity; VC=vital capacity

Key words: bullous emphysema; reduction pneumoplasty; review; surgical treatment; volume reduction surgery

A wave of excitement rippled through the pulmonary community in the spring of 1994 after Cooper and colleagues reported at national meetings that there was dramatic improvement in a small group of surgically treated patients with end-stage, nonbullous emphysema. The initial results of their observational study of volume reduction surgery in 20 patients with severe emphysema were subsequently published.[1] The operation, done through a median sternotomy, involved excision of 25 to 30% of the volume of each lung. The most affected portions were excised using a linear stapling device fitted with strips of bovine pericardium to minimize air leak through the staple holes.[2] There was no early or late mortality and a mean improvement in FEV_1 of 82% of baseline value (0.77 to 1.2 L). A mean rise of room air PaO_2 from 64 to 72 mm Hg and of 6-min walk distance from 1,000 to 1,600 feet was observed. There were improvements in dyspnea on effort, energy, physical mobility, and quality of life measures. It appeared that a treatment had become available that, like lung transplantation, would not just help to maintain patients with COPD but that could actually improve the function of the lungs and of the whole person.

Cooper et al[1] used the terms *pneumectomy* or *resection of air* and volume reduction surgery as terms to describe efforts to improve lung function in emphysema by resecting overdistended, poorly ventilated "functionless" lung. This review uses the term *reduction pneumoplasty* for the surgery because the word "reduction" reflects the resection and volume reduction of lung, and the word "pneumoplasty" (Greek *pneumon* lung+*plassein* to form) denotes the performance of plastic surgery on the lung. The term reduction pneumoplasty readily encompasses the different surgical techniques used to ameliorate the detrimental physiologic effects of emphysema.

Surgery has been used for more than 4 decades to

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improve lung function in patients with giant bullous emphysema. It therefore seemed reasonable to explore whether useful information for evaluating modern reduction pneumoplasty might come from a review of the published record of surgical treatment of giant bullous emphysema. Accordingly, this essay will briefly explore the pathophysiologic basis of reduction pneumoplasty for bullous emphysema. The recorded experience of surgery for giant bullous emphysema will then be critically reviewed. Current criteria for evaluation of patients with giant bullous emphysema for surgery will be summarized and the data will be evaluated to determine what the lessons are for reduction pneumoplasty for nonbullous emphysema.

Physiologic Basis of Reduction Pneumoplasty

Emphysema and Hyperinflation

Emphysema results in fixed airflow obstruction that is due to destruction of elastic tissue, with loss of elastic lung recoil and rupture of alveolar attachments to thin-walled bronchioles. As lung volume decreases during expiration, peripheral airway closure occurs at a much larger lung volume than in normal lungs. An increase in expiratory effort to overcome flow limitation results in dynamic compression of airways with resultant limitation of flow despite increased driving pressure. Hyperinflation of the lungs also occurs in emphysema because the outward pull of the chest wall is incompletely countered by the emphysematous lungs with diminished elastic recoil. Finally, central ventilatory drive increases, initially by increases in both respiratory frequency and tidal volume. The proportion of time spent in inspiration falls and because of the slowed expiration, hyperinflation worsens.

The most important muscle of inspiration is the diaphragm;[3] its fibers can shorten up to 40% between full inspiration and expiration and during quiet breathing the muscle accounts for most of the force needed to expand the lungs.[4] The marked hyperinflation of the lungs in expiration in severe emphysema results in marked flattening of the diaphragm, loss of its apposition to the chest wall, and shortening of its muscle fibers, thus putting them at a severe mechanical disadvantage for contraction during inspiration. Dyspnea limits exercise in COPD and appears to correlate better with measures of respiratory muscle function than with airflow obstruction.[5] Ventilation perfusion mismatch results in increased dead-space ventilation, initially with hypoxemia alone and later with hypercapnia and worsening hypoxemia.

Bullae

Bullae, defined as airspaces in the lung more than 1 cm in diameter,[6] were taken by Reid[7] to refer to the local elevation of pleura above the surface of the lung. She distinguished three types of bullae. Type 1 bullae project above the surface of the pleura like a mushroom, have a narrow neck and a sac that is empty except for a few tags or strands of tissue, and are composed of great overinflation of a small amount of lung. Type 2 bullae have a broad neck, are produced by relatively less overinflation of a shallow subpleural layer of lung, and the sac usually contains emphysematous lung. Type 3 bullae protrude only moderately above the surface, represent the least overinflation of a much deeper region of the lung, perhaps extending back to the hilum, have no well-defined neck, and contain emphysematous lung evenly throughout the bulla.

Radiographically, bullae are recognized as regions of hypertransradiancy with absent vascular markings surrounded by thin, circular, arcuate, or polygonal linear shadows. Giant bullae can sometimes be seen to compress adjacent lung tissue. Bullae are seen most frequently in smokers and, in concordance with the distribution of centrilobular emphysema, occur most often in the upper lung zones. A 2:1 right-sided preponderance is generally attributed to the larger size of the right lung.[7] Bullae are frequently seen with evidence of widespread emphysema.[8] CT is the method of choice for accurately delineating bullae and the state of adjacent lung parenchyma.[9,10]

Thurlbeck[11,12] points out that most bullae recognized radiologically are not so assessed morphologically. Those seen radiographically in the lower lung zones in the common form of emphysema or in association with alpha$_1$-protease inhibitor deficiency represent severe panacinar emphysema; the radiologic margins represent interlobular septae and are not readily identified pathologically as bullous margins. Radiologically determined upper zonal bullae with COPD usually represent centrilobular emphysematous spaces.[11] Commonly observed apical bullae in asymptomatic persons, which occasionally cause uncomplicated spontaneous pneumothorax, represent paraseptal emphysema.[12] Type 1 bullae of Reid[7] are due most commonly to paraseptal emphysema; type 2 and 3 bullae are due to centrilobular or panacinar emphysema.

Bullae and Lung Function

Bullae occupying one third to one half of one or both hemithoraces (giant bullae) may produce a dramatic chest radiograph, but such patients *may* have little abnormality of lung function.[13,14] More often, patients with giant bullae have airflow obstruction ranging from minimal to severe. The pathophysiologic basis for these observations is as follows. Bullae representing isolated bullous disease (paraseptal emphysema) without compression of adjacent tissue initially have well-preserved function. The bullae may enlarge progressively over time, trapping a progressively larger volume of air and

compressing adjacent lung tissue with loss of function. Bullae that are part of generalized emphysema are always associated with function loss.

Most bullae communicate poorly with the airways as demonstrated by ventilation scintigrams and by CTs taken in inspiration and expiration; they rarely ventilate sufficiently to contribute to dead-space ventilation.[15,16] The difference in functional residual capacity determined by body plethysmography and by gas dilution methods estimates the amount of trapped gas, which may be up to several liters.[17]

The mechanism of enlargement of bullae, maintenance of high intraluminal pressure, and compression of the adjacent lung is unclear. The giant bullae of paraseptal emphysema deflate readily at surgery and inflate with application of little pressure. The pressure-volume characteristics of excised bullae are those of a paper bag, which inflates to its maximum volume with a small inflation pressure, with subsequent rapid increase in pressure with little change in volume.[18] Measurement of intraluminal pressure by direct needle puncture showed that mean bulla pressure was always negative or below atmospheric pressure. Peak inspiratory pressure ranged from −5.5 to −19 cm H_2O and end-tidal pressure from +2.5 to +11 cm H_2O. During intermittent positive pressure breathing, bullous end-expiratory pressure was about 5 cm H_2O vs zero in the airway.[19] Morgan and colleagues[16] interpreted their finding of negative inspiratory intrabullous pressure as indicating that compression of adjacent tissue by a distended bulla could not be occurring. An alternative view is that the end-tidal intrabullous pressure is higher than end-tidal intra-alveolar pressure, which is atmospheric at end-expiration with an open glottis. This gradient between bulla and alveolar space provides an explanation for radiographically and intraoperatively observed compression of lung tissue immediately adjacent to a bulla. A valvular mechanism during expiration through a floppy airway is one explanation for distention of bullae *in vivo*. Preferential filling of bullae because of their very high compliance is another. Impairment of effective inspiration and dyspnea certainly also result from the large amount of space occupied in the thorax by the fixed-volume bulla with resultant loss of linkage between the chest wall and diaphragm and nonbullous lung tissue.[16,19,20]

Reduction Pneumoplasty for Bullous Emphysema

The presence of a giant, radiolucent airspace in the chest in a patient with compromised lung function has provided a fearsome challenge to pulmonary physicians and surgeons for more than 4 decades. On the one hand, if lung function is not improved by resectional surgery (a form of reduction pneumoplasty), the patient may die; on the other hand, the results of surgical treatment may be dramatic and long lasting.

Methods

Twenty-two articles dealing with resectional surgery for bullous emphysema were retrieved by a computer search of the literature and a search of the *Index Medicus* prior to 1966.[14,15,21-40] They are all case series, mostly retrospective, without control subjects. Only case series published since 1950 were included. Articles dealing *only* with therapy of bullae by pneumonostomy, surgery for bullous disease complicated by carcinoma, and series of surgery *primarily* for pneumothorax were excluded. The 22 reports include 476 patients, reflecting the relative infrequency of patients with bullous emphysema who were treated surgically. These studies are summarized in Table 1.

Results

Surgical Indications: Patients in the published series were selected by the authors mostly because of the presence of bullae compressing lung tissue and because they were dyspneic and had airflow limitation; a few patients were included because they had bullae and recurrent spontaneous pneumothorax. Only a small proportion of those treated surgically appear to have had Reid type 1 bullae; most had Reid type 2 and 3 bullae. The main determinants of the outcome of surgery were considered by the authors to be the size of the bulla, the presence of compressed adjacent lung tissue, and the impairment of function of the compressed tissue.

Early Postoperative Results: There was little improvement in function after surgery for bullae that occupied less than one third of the hemithorax, and when lung function was normal or minimally impaired.[23,28-30] Severe impairment of lung function, due mainly to the presence of generalized emphysema, with little evidence of lung compression, was also predictive of a poor result. Surgery in the face of a low FEV_1, hypercapnia, or cor pulmonale was fraught with danger unless compressed, relatively normal lung could be released by the operation.[17,40] Surgical treatment for severe impairment of lung function due to a giant bulla producing compression atelectasis of lung often produced the most dramatic of improvements; the descriptions suggest that these dramatic results were most often observed with the type 1 bullae of paraseptal emphysema.

Hypoxemia and hypercapnia appeared to be among the measurements most frequently improved by surgery. Increases in FEV_1, FEV_1/FVC, and diffusion of carbon monoxide (Dco) were generally modest; vital capacity (VC), residual volume (RV), and total lung capacity (TLC) and trapped gas generally decreased. Pulmonary hypertension and right ventricular hypertrophy were reversed if hypoxemia and hypercapnia

Table 1—*Summary of the Literature: Indications and Outcomes of Reduction Pneumoplasty for Bullous Emphysema

Reference	No. Male, No. Female, FU*	Indications	Surgery	Postoperative Mortality, No.	Comments
Waring, 1951[21]	2 M 3+ yr	Giant bullae, severe dyspnea	Unilateral, bilateral bullectomy	0	—Relief of dyspnea occurred in both patients —Pt 2, MVV improved from 24 to 83% predicted and VC from 27 to 82% predicted; resting SaO_2 unchanged
Pierce, 1962[22]	1 M 2 yr	Giant bullae bilaterally, severe dyspnea, cor pulmonale	Bilateral bullectomy	0	—Dyspnea and heart failure were relieved —Increase VC 1.12 to 4.36 L; —TLC 2.43 to 5.94 L; —FEV_1 0.64 to 3.15 L —$PaCO_2$ 67 to 40 mm Hg; SaO_2 53 to 94%
Woo-Ming, 1963[23]	48 M 5 F	Bullae ranging from <⅛ to >½ lung field	—Resections: bulla 32 lobes 21 segments 2 —Bilateral 2	7	—Results in 41 patients followed up >1 y: —Early results: 30 good, 6 fair, 5 poor —Late results: 14 good, 9 fair, 8 poor, 10 dead —The less lung resected the better the result —Among bullae >⅛, size had no effect —Resection bullae <⅛ did not help
Viola, 1966[25]	15 M 1-35 mo	Bullae: ¼ one lung to ½ both lungs	Bullectomy —Unilateral 12 —Bilateral 3	1	—9 pts, 0 to 1+ dyspnea; improvement occurred in: —VC 7, MVV 7, SaO_2 4, $PaCO_2$ 0 —6 pts, 2 to 3+ dyspnea; improvement occurred in: —VC 4, MVV 3, SaO_2 1, $PaCO_2$ 1 —Conclusion: compression is favorable, diffuse disease is unfavorable for outcome
Benfield, 1966[24]	19†	Bullous and generalized emphysema	—Resections: bulla 9 lobes 9 plication 2 bilateral 1	0	—Postoperative studies 15 patients at 1 mo to 7 yr: Improvement occurred in MVV, 5; FEV_1, 6 —5 had increased exercise tolerance not reflected in lung function studies
Foreman, 1968[26]	12 M 1 F 2-127 mo	—Enlarging, bullae >½ hemithorax —Dyspnea 2-4+ —History of pneumothorax 6	—Resection of bullae	0	—Improvement: symptoms 12; VC 8; MVV 8; FEV_1 10; PaO_2 mean preoperative 71, postoperative 85 mm Hg; $PaCO_2$ mean preoperative 42, postoperative 40 mm Hg —Conclusion: the only contraindication to surgery is the demonstration of abnormal, poorly perfused lung adjacent to bullae
Rogers, 1968[27]	6 M 1 F 8-88 mo	—Large bullae-4 —Diffuse emphysema with small bullae-3	—Bullectomy +plication 6 —Lobectomy 1 —Bilateral 2	1	—Function changes were compared with control group of 5 patients who had: wedge resection (1), lobectomy (3), and pneumonectomy (1) —In the bulla group, mean FEV_1 increased from 1.37 L preoperatively to 1.76 L postoperatively —In the control group, of 4 patients with lobectomy or pneumonectomy, there was reduction of FRC and G_A with no change in G_A/V —In the bulla group, FRC decreased but G_A and G_A/V increased in all; the changes were sustained in those with large bullae but reverted to preoperative values in the emphysema/small bulla group
Pride, 1969[29]	10 M 1 F 6-9 mo	Localized bullous emphysema with severe dyspnea	Removal and plication of bullae with several segmentectomies, lobectomies 2, and bilobectomy 1	1	—Increase in mean FEV_1 from 1.04 to 1.24 L (3 pts >0.3 L increase); increase PaO_2 of 8+ mm Hg in 8/10; fall $PaCO_2$ in 5/7 with preoperative value >45 mm Hg; no improvement in recoil pressure —Symptom improvement persisted only in 3 pts, those with increase of FEV_1 >0.31 or 10+ mm Hg increase in PaO_2

Continued

Table 1—*Continued*

Reference	No. Male, No. Female, FU*	Indications	Surgery	Postoperative Mortality, No.	Comments
Boushy, 1969[28]	15† FU in 10 for 14-26 mo	Bulla: 1×20, 9×30, 3×40, 1×70, 1×90% hemithorax	Bullectomy	0	—Preoperative FEV_1 %predicted: >70=6, 50-70=5, <50=4 —Mean increase FEV_1=0.275 L —RV and TLC decreased —Changes were slight except when bullae occupied most of a hemithorax and even then all test results did not improve —Maximum intrathoracic pressure increased; elastic work of breathing increased and nonelastic work decreased
Wesley, 1972[31]	13 M 1 F 2-8 yr	—Bullous emphysema —Grade 2-4+ dyspnea —All but 2-compression	8-bullectomy 5-lobectomy 1-bilateral	1	—Improvement: marked 6, moderate 3, minimal 2 —Improvement of FEV_1 19-225% baseline —Improvement was stable over 2- 8-yr FU —Severe sputum production was an adverse risk factor
Pride, 1973[29]	7 M 1 F 6-9 mo	—Bullous emphysema —Dyspnea Grade 1-4+	Plication of bullae 5; pneumonostomy 3	0	—Most patients showed improvement in PaO_2 and reduction in plethysmographic FRC; increase in FEV_1 was modest and was greatest in those with best preoperative values; tests of regional function suggested that impairment was caused by bullae compressing lung
FitzGerald, 1974[30]	84 1-20 yr	—Dyspnea 57 —Pneumothorax 18 -Infection 6 -Hemorrhage 1	—Excision or plication 69 —Lobectomy 15	2	—Small bullae (<⅓ hemithorax) whether with normal or impaired function showed little improvement —Best results were in patients with giant bullae, moderate airflow obstruction, and bullectomy or plication rather than lobectomy —Worthwhile improvement occurred in some patients with cor pulmonale —Function was maintained for up to 20 yr after bullectomy and there was little tendency to recur
Potgeiter, 1981[32]	17 M 4 F 3-9 yr	—Large bullae compression of lung —Severe dyspnea, 10 —Cor pulmonale 4	Bullectomy, plication-15 Lobectomy 3 Bullectomy, decortication 3	2	—Improved symptomatically, 14; with improvement of FEV_1 and VC >10% in 9/13 —Four of 6 with hypercapnia survived and improved —Bronchogram helped show compression but had high complication rate —Bullectomy did better than lobectomy
Nakahara, 1983[14]	18 M 1 F 1-7 yr	—Bullae occupying more than ½ hemithorax —Dyspnea: absent or mild in 10 pt; moderate or severe in 9	Bullectomy, bilateral in 5	0	—Six of 9 patients with moderate or severe dyspnea improved and maintained improvement for 5-7 yr of FU; 2 improved for 1 yr and then suffered relapse —Mean FEV_1 was 60.6% predicted preoperatively vs 70.4% postoperatively; no change in VC%, PaO_2, or $PaCO_2$
Pearson, 1983[33]	12 M 5-10 yr	—Giant bullae —Dyspnea >grade 3	Lobectomy 7 Bullectomy 5	1	—FVC, mean 2.29 L, pre, 2.84 L 3-6 mo, 2.81 L, 5-10 yr; FEV_1, mean, 1.19 L, pre, 1.77 3-6 mo, 1.33 L, 5-10 yr; Mean dyspnea grade, 3.45 pre, 1.6 3-6 mo, 2.8 5-10 y —All not retired worked 5+ yr —Dyspnea slowly returned —Lobectomies fared as well as bullectomies
Wex, 1983[34]	25 M 2 F	—Multiple small and large bullae	—Plication bullae with	1	—Preoperative and postoperative function in 19 pts, 3-48 mo:

Continued

Table 1—*Continued*

Reference	No. Male, No. Female, FU*	Indications	Surgery	Postoperative Mortality, No.	Comments
	3-48 mos	—Compression adjacent lung was not required	dura (7) or pericardium (7) acrylic glued patch Bilateral 5 pt		—Improvement in 17/19; mean FEV_1, 0.8 to 1.3 L; FVC, 1.9 to 3.3 L; Raw cm/H_2O/L/s, 9.3 to 3.4; PaO_2, 59 to 72; $PaCO_2$ unchanged; mPAP rest, 21-18 mm Hg; exercise, 47-38 mm Hg; improved work capacity on ergometry 16/19 pts
Hughes, 1984[36]	11 M 4-20 yr	—Giant bullae	Lobectomy 8 Pneumonostomy 2 Bullectomy 1	—	—Postoperative FU of not <4 yr; 5 continued smoking, 6 stopped —All initial postoperative data were similar except $PaCO_2$, which was higher in ex-smokers —Mean annual decline for smokers vs ex-smokers: FEV_1 (post bd) 30.6 vs 44.0 (NS) Dco (mmol $kPa^{-1}min^{-1}$) −0.08 vs −0.18 ($p<0.025$); KCO (mmol $kPa^{-1}min^{-1}$) +0.01 vs-0.04 ($p<0.001$) —Continued smoking appears to adversely affect lung function after surgery for bullous disease
Vejlsted, 1985[35]	24 M 7 F 9 mo to 10 yr	Giant lung cysts or bullous emphysema with poor lung function and compression adjacent tissue	23 unilateral, 8 bilateral resections	7	—Of 21 patients with preoperative and postoperative studies: —MVV was improved in 18 —9 were asymptomatic, 8 were improved, 2 had impaired working capacity and 2 were disabled —$Xenon^{133}$ scintigraphy was considered helpful in about half the cases —Pulmonary angiography in 16 patients was considered of insufficient value to continue its use
Laros, 1986[37]	27 M 5-25 yr	Bullae occupying >50% hemithorax	—Giant bullae excised or sutured —Smaller bullae coagulated —Since 1975 staples used —Bilateral 10	0	—1957-1977, 142 pts considered for bullectomy and 65 were operated: —6 emphysematous lobes were resected and 3 patients were closed without resection; all of these lost function postoperative —29 with bullae <⅓ hemithorax underwent bullectomy; there was no improvement over 9 yr FU —Current: 27 with bullae >50% hemithorax with emphysema; —Dyspnea grade improved mean 27 preoperative to 10 early postoperative but worsened to preoperative in 5 yr —Mean FEV_1/FVC%: preoperative 42; 2 yr 48; last 39 —Mean VC%: preoperative 93; 2 yr 118; last 92 —7 died pulmonary insufficiency —No recurrence giant bullae but small bullae appeared in 13 pts
Connolly, 1989[38]	17 M 2 F 3-22 yr	—Giant bullae, upper lung fields —Definite evidence of compression adjacent lung	—Bullectomy with preservation of lung tissue —No lobe or segmental resections 13 bilateral	0	—All patients had preoperative and postoperative function studies but no detailed analysis is given —All patients had preoperative FEV_1/FVC <60% —Tomograms and angiograms provide the clearest evidence of compression lung tissue. —Even the most ill patients tolerate bullectomy

Continued

Table 1—*Continued*

Reference	No. Male, No. Female, FU*	Indications	Surgery	Postoperative Mortality, No.	Comments
					—Results were often spectacular and persisted for some years, although deterioration eventually occurred
Ohta, 1992[39]	20 M	Bulla >⅓ hemithorax	Bullectomy —Unilateral 15 —Bilateral 5	0	—Group consisted of 15 patients who showed 1 grade improvement in dyspnea for more than 4 yr of follow-up; group 2 consisted of 5 patients who showed 1 grade improvement in dyspnea within the first year but were the same or worse than preoperative at 5 yr —Group 1: VC% 99 preoperative and 108 postoperative FEV_1 % 70 preoperative and 75% postoperative —Group 2: VC% 57 preoperative and 64% postoperative FEV_1 46% preoperative and 59% postoperative
Nickoladze, 1992[40]	38 M 8 F 5 yr	—Gr 1: 18 pt Bilateral bullae >⅓ hemithorax; all had history of pneumothorax —Gr 2: 16 pt small bullae <⅓ hemithorax; all had history of pneumothorax —Gr 3: 12 pt small bullae <⅓ hemithorax with lung fibrosis	—Gr 1: Bilateral bullectomy via median sternotomy and stapling —Gr 2:6 bullectomy via median sternotomy, 10 bullectomy —Gr 3: lobectomy with segmentectomy as indicated	0	—All had CT and plain radiographs preoperatively and at 5 yr; bulla proportion was determined planimetrically in CT images. —Gr 1: FEV_1 pre, 2.2, 5 yr, 2.6 L; VC, pre, 3.1, 5 yr, 3.4 L; TLC pre, 6.2, 5 yr, 5.3 L; TGV pre, 1,250 mL; Dco% pre, 74, 5 yr, 83. —Gr 2: FEV_1 pre, 2.8, 5 yr, 2.8 L; VC, pre, 3.9, 5 yr, 3.6 L; TLC pre, 6.5, 5 yr, 5.9 L; TGV pre, 307 mL; Dco% pre, 88, 5 yr, 71. —Gr 3: FEV_1 pre, 2.4, 5 yr, 1.9 L; VC, pre, 3.2, 5 yr, 2.0 L; TLC pre, 6.0, 5 yr, 5.7 L; TGV pre, 244 mL; Dco% pre, 60, 5 yr, 60 —No new bullae were seen in CT at 5 yr —Respiratory function improved significantly during long term follow-up after removal of bullae occupying >⅓ hemithorax, did not change if bullae were <⅓ hemithorax, and deteriorated when bullae were associated with fibrosis

*Reference=first author, year of publication (reference number); FU=duration of follow-up; Pt=patient; TGV=trapped gas volume; MVV=maximal voluntary ventilation; FRC=functional residual capacity; SaO_2=oxygen saturation; G_A=airway conductance; G_A/V=specific airway conductance; Raw=airway resistance; KCO=diffusion constant for carbon monoxide; mPAP=mean pulmonary artery pressure; bd=bronchodilator; pre=preoperatively.

†Gender not specified

improved.

A number of authors stressed the importance of resecting as little nonfunctioning lung as possible—performing bullectomy and plication of bullae rather than lobectomy or even segmentectomy.[24,32,34,37] In earlier series, bilateral operations were always consecutive, but in later series, simultaneous bilateral surgery via median sternotomy came into use.[40,41]

Tomography, pulmonary angiography, bronchography, bronchospirometry, and radionuclide ventilation and perfusion scans have all been used to attempt to predict postoperative results.[14,15,24-26,29-32,42] Of these, pulmonary angiography was for a long time considered the most useful for demonstrating compression of lung adjacent to bullae with a capillary blush in underlying lung tissue indicating absence of severe emphysema in compressed lung. CT is now the imaging technique of choice for preoperative evaluation.[9,16,40]

Current Criteria for Selecting Patients With Bullous Emphysema for Reduction Pneumoplasty: From the foregoing review, it seems reasonable at this time to list the following as guidelines for performing reduction pneumoplasty in giant bullous emphysema. Study of serial chest radiographs is helpful in judging whether compression of normal lung by bullae is responsible for the patient's functional state or whether generalized emphysema is responsible. The presence of emphysema in nonbullous lung and the amount of compres-

sion can be best assessed by CT. Patients do best who have bullae larger than one third of a hemithorax and an FEV_1 less than half the predicted normal value.[20] Other physiologic parameters predictive of successful surgery include a large volume of sequestered gas (resection of which decreases lung volumes), a reasonably well-preserved diffusing capacity, and eucapnia.[39]

Late Postoperative Results: Postoperative mortality was not always given in published reports and varied greatly, from 0 to 22.5% with a weighted mean in 262 patients of 8.0%.[23,24,26,30-32,34,35] Long-term follow-up of clinical and physiologic data are given in relatively few articles and are difficult to interpret because of the variable way in which they are presented.[23,24,30,33,36,40] One third to one half of the patients appeared to maintain improvement for about 5 years.[23,24,30] Pulmonary function (FEV_1, VC, RV, and TLC [plethysmographic] and Dco) was better at 5 years than preoperatively in patients whose bullae occupied more than one third of a hemithorax. Late follow-up values were similar to preoperative values in patients whose bullae were less than one third of a hemithorax.[40] Nine of 12 patients reviewed 5 to 10 years after surgery[33] all reported a gradual return of dyspnea with a mean fall of FEV_1 of 82 mL/yr; 5 of the 9 still maintained some of their postoperative improvement. Among 11 patients operated on for bullous disease 4 to 20 years earlier, FEV_1 (prebronchodilator) Dco and diffusion constant for carbon monoxide declined more rapidly in 6 smokers than in 5 ex-smokers ($p<0.05$),[37] suggesting the great importance of smoking cessation after surgery. In general, resection of giant bullae does not seem to affect the size of other bullae.[30,40]

Lessons From Reduction Pneumoplasty in Bullous Emphysema

A number of lessons learned from surgery for giant bullous emphysema are applicable to reduction pneumoplasty for nonbullous emphysema. First, reduction pneumoplasty for bullous emphysema represents a special case of reduction of lung volume in severe emphysema; in both instances, the principle is to reduce lung volume by resecting the worst functioning lung tissue. As pointed out earlier in this essay, severely emphysematous tissue may appear radiographically but not pathologically bullous. If resection of an area of emphysema that appears bullous can restore impaired lung function, it follows that resection of similar emphysematous tissue that does not appear bullous radiographically may also ameliorate pulmonary dysfunction. The early functional changes after surgery for bullous disease are qualitatively similar to those in preliminary reports of reduction pneumoplasty for nonbullous emphysema—both blood gas values and lung mechanics are affected. Thus, the experience with surgery for bullous emphysema supports the rationale for reduction pneumoplasty for nonbullous emphysema.

Second, there is a single report in a few patients that preservation of postoperative lung function is much greater in those who stop smoking than in those who continue to smoke.[36] This study, coupled with the recent demonstration in the Lung Health Study of dramatically decreased decline of FEV_1 in smokers with moderate airflow limitation who stopped smoking as compared with those who continued to smoke,[43] emphasizes the need for smoking cessation before surgery and for maintaining smoking cessation after volume reduction surgery for emphysema.

Third, after more than 4 decades of reduction pneumoplasty for bullous emphysema, the duration of improved function, the effects of volume reduction on the lung remaining after surgery, and on long-term morbidity and mortality are not well defined. The criteria for selection of patients for reduction pneumoplasty are as yet unclear. It is important that this information be collected as rapidly as possible for reduction pneumoplasty for nonbullous emphysema. It has been argued elsewhere[44] that the best way of ascertaining this information is by establishing a reduction pneumoplasty registry (a prospective, cooperative, observational trial, with provisions for data auditing to confirm consecutive case reporting). A case-control group will be necessary to compare all-cause mortality, respiratory disease mortality, and morbidity with and without reduction pneumoplasty for emphysema. The group might be selected from existing databases or might comprise patients with comparable severity of airflow obstruction who refused surgery.

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