GENERAL THORACIC SURGERY

FUNCTIONAL AND OXIMETRIC ASSESSMENT OF PATIENTS AFTER LUNG REDUCTION SURGERY

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Objective: **The goal of this study was to clarify the issue of functional oxygen requirement by regimented exercise oximetry in patients undergoing lung reduction surgery.** ***Methods:*** **Thirty-seven patients underwent lung reduction surgery and were followed up for at least 3 months. Patients routinely completed a 6-week program of cardiopulmonary rehabilitation. Preoperative and postoperative spirometry, dyspnea scores, 6-minute walk distances, respiratory mechanics, and exercise oximetry were recorded.** ***Results:*** **After the operation, patients had a 37% increase in forced vital capacity and a 59% increase in forced expiratory volume in 1 second. Six-minute walk distance increased from 913 ± 310 feet before the lung reduction operation to 1202 ± 274 feet 6 months after the operation ($p < 0.001$). Maximal inspiratory and expiratory pressures were significantly increased in 16 patients after lung reduction surgery. Perceived dyspnea was significantly improved. Exercise pulse oximetry demonstrated that 83% of patients met American Thoracic Society criteria for supplemental oxygen use before lung reduction surgery. After the operation, 70% of patients continued to meet American Thoracic Society criteria for supplemental oxygen use. Notably, 10 patients with exertional desaturation while breathing room air discontinued supplemental oxygen use because of a reduction in dyspnea.** ***Conclusions:*** **These findings demonstrate significant subjective and functional improvements related to lung reduction surgery. Exercise-induced hypoxia was not reversed by lung reduction surgery. Discontinuance of supplemental oxygen use owing to reduction in dyspnea and improved physical performance may not be warranted in lieu of continued exertional desaturation. (J Thorac Cardiovasc Surg 1997;113:675-82)**

Lung reduction surgery (LRS) has recently been proposed as an effective palliative intervention for patients with advanced emphysema.[1-5] Reported benefits have included reduction in dyspnea, improvement in spirometric measurements, increased exercise capacity, and reduction in supplemental oxygen requirement. The mechanism whereby LRS reduces supplemental oxygen requirement in patients with emphysema remains hypothetical. In our experience, many patients discontinued supplemental oxygen use independent of physician recommendations because they felt less dyspneic. In reports relating to LRS, specific criteria for the discontinuance of supplemental oxygen have not been defined.[1, 2]

Accurately assessing a patient's need for supplemental oxygen remains important after LRS. Prolonged hypoxemia may result in pulmonary hypertension, cor pulmonale, and an increased risk of mortality.[6, 7] The benefit of supplemental oxygen in patients with hypoxemic chronic obstructive pulmo-

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nary disease (COPD) has been demonstrated in the Nocturnal Oxygen Therapy Trial[8] and in the report of the British Medical Research Council Working Party.[9] Both of these studies showed a clear survival benefit to the use of supplemental oxygen in patients who were hypoxic at rest. Premature discontinuance of supplemental oxygen in patients in whom desaturation continues after LRS may pose a risk to long-term survival after the procedure. We therefore undertook a detailed oximetric assessment of patients before and after LRS to accurately determine the effect of the procedure on the need for supplemental oxygen.

Patients and methods

From June 1994 to March 1996, 45 patients with advanced emphysema underwent LRS at the Medical College of Wisconsin Hospitals. The average age was 62 years (range 38 to 79 years). The group comprised 27 men and 18 women who were selected from 120 patients referred for LRS evaluation. Preoperative testing included measurement of arterial blood gases, spirometric values, lung volumes, and the diffusion capacity of the lung to carbon monoxide. Inspiratory and expiratory chest roentgenograms, computed axial tomographic scans, and quantitative ventilation-perfusion scintigraphy were used for radiologic evaluation. As a general rule, patients with marked hyperexpansion associated with heterogeneous emphysematous disease, a large residual volume, and a significant trapped gas volume were considered for the procedure. Patients with previous major thoracic surgery or those with a prominent component of bronchospasm, copious sputum production, or congestive heart failure were excluded. Patients routinely completed a 6-week program of preoperative cardiopulmonary rehabilitation that continued into the postoperative period. Poorly conditioned patients underwent a longer period of rehabilitation, and those who could not actively engage in the program were excluded from LRS consideration.

Surgical procedure. The operation was performed via a median sternotomy in 44 patients and via a lateral thoracotomy in one patient. Bilateral lung reduction was achieved in 42 patients and a unilateral procedure was done in three. The most severely emphysematous regions of lung were resected with a linear stapling device reinforced with glutaraldehyde-preserved bovine pericardial strips. Early postoperative analgesia was maintained with epidural or intrathecal pharmacologic agents. A policy of early extubation and aggressive pulmonary toilet was maintained.

Preoperative and postoperative comparison was achieved by measurement of spirometric values including the forced vital capacity (FVC) and the forced expiratory volume in 1 second (FEV_1). Exercise capacity was measured by the 6-minute walk test. Change in dyspnea related to LRS was assessed by the transitional dyspnea index.[10] This index uses the following scale: minor improvement (+1), able to return to work at reduced pace or resumption of some customary activities with more vigor than previously because of improvement in shortness of breath; moderate improvement (+2), able to return to work at nearly usual pace or able to return to most activities with restriction only; major improvement (+3), able to return to work at former pace and able to return to full activities with only mild rest because of improvement in shortness of breath. Dyspnea was also assessed before and after LRS by the baseline dyspnea scale (0 to 4 scale, 4 = no impairment, 0 = very severe impairment) as described previously.[10] In a subgroup of 16 patients, maximal inspiratory and maximal expiratory pressures were measured before and 1 month after LRS by means of a "bugle" dynamometer (Applied Engineering, Inc., Rochester, Minn.) as described by Sobush and Dunning.[11] Exercise oximetry was performed on all patients before LRS and at 3 and 6 months after LRS on all available patients ($n = 37$, 3 months, $n = 25$, 6 months). Postoperative functional assessment was not obtained in five patients who ultimately died after the LRS either in the hospital or after discharge. In addition, three patients did not have formal functional assessment in the postoperative period. Two of these patients were well, with marked subjective improvement; the clinical condition of the third deteriorated as a result of pulmonary embolism 5 months after LRS. When possible, postoperative exercise oximetry was performed while breathing room air and then repeated with supplemental oxygen to more fully assess the effect of the procedure on exercise-induced oxyhemoglobin desaturation.

Six-minute walk evaluation. Resting heart rate and blood pressure were obtained before the 6-minute walk evaluation. Resting and continuous exercise pulse oximetry were performed with a Minolta Marquest Oxygen Saturation Monitor Pulsox-7 (Englewood, Colo.). The patients were instructed to cover as much distance as possible during the evaluation but were allowed to stop and rest on their own volition. All rest periods were recorded by the tester. The patients established the walking pace with the tester walking slightly behind carrying the oxygen canister. Oxyhemoglobin saturation (Sao_2), heart rate, blood pressure, distance walked, and the number of rest periods were recorded. Exertional desaturation was defined as a fall in Sao_2 to 88% or less. The perceived level of exertion was assessed with the use of the Borg Scale (6 to 20 scale).[12] Twenty-seven patients underwent 6-minute walk evaluation before and after preoperative cardiopulmonary rehabilitation and were again evaluated after LRS. The data presented in Table I are derived from these patients. Because of logistic factors, 10 additional patients underwent only prerehabilitation preoperative 6-minute walk testing. Their prerehabilitation and postoperative 6-minute walk distances were similar to those of the 27 cohorts with complete preoperative testing (891 ± 327 feet before rehabilitation, 1147 ± 310 feet 3 months after LRS, and 1169 ± 323 feet 6 months after LRS; p = not significant compared with the 27 cohorts for each time point; $p < 0.001$ comparing prerehabilitation to post-LRS distances, $n = 10$).

Data analysis. Preoperative and postoperative data for 6-minute walk variables were compared with the use of one-way repeated-measures analysis of variance. Continuous variables are reported as mean ± standard deviation.

Table I. *Performance data of 6-minute walk tests before and after LRS*

	Before rehab. (n = 27)	*Preop. (n = 27)*	*3 mo postop. (n = 27)*	*6 mo postop. (n = 23)*
Distance (feet)	838 ± 323	913 ± 310	1108 ± 269*	1202 ± 274*
Percent resting	67%	37%	11%*	4%*
Borg Score	13 ± 2	13 ± 2	12 ± 1*	11 ± 2*

Borg Scale 6 to 20, 20 = maximal exertion.
*p = 0.001 versus preop.

Table II. *Perioperative morbidity and mortality associated with LRS*

	No.	*%*
Hospital mortality	3	7
Total mortality	5	11
Respiratory failure/pneumonia	7	16
Prolonged air leak	24	53
Wound infection	1	2
Intestinal ischemia/ileus	4	9
Gastrointestinal bleeding	2	4
Pulmonary embolism	2	4
Pneumothorax	3	7

Table III. *Changes in spirometry related to LRS*

	Preop. (n = 37)	*3 mo (n = 33)*	*6 mo (n = 21)*
FVC	2.16 ± 0.76	2.66 ± 0.68* (37%)	2.66 ± 0.70* (33%)
FEV_1	0.68 ± 0.23	0.97 ± 0.38† (59%)	0.85 ± 0.35 (48%)

Average percent change from paired preoperative spirometric values is given in parentheses.
*p = 0.005 versus preop.
†p = 0.002 versus preop.

Preoperative and postoperative dyspnea scores were compared by means of the Wilcoxon signed rank test. Statistical analysis was performed with StatView statistical software (Jandel Corp., San Rafael, Calif.).

Results

Forty-five patients underwent LRS. There were three hospital mortalities, two caused by respiratory failure and one by multiple pulmonary emboli. Two patients died of causes related to LRS after hospital discharge; one died 2 months after transfer to a nursing facility as a result of recurrent respiratory failure and the other had the sudden onset of cardiopulmonary insufficiency 2 weeks after an uneventful hospitalization. The mean duration of hospitalization was 16 days. All surviving patients were discharged to their homes. Data regarding perioperative complications are presented in Table II. Respiratory insufficiency and prolonged air leak were the major sources of postoperative morbidity.

Thirty-seven patients underwent preoperative and postoperative assessment, including exercise oximetry, dyspnea scoring, and spirometry data. They constitute the basis of subsequent analyses. Spirometric assessment of these patients before and after LRS are presented in Table III. The data demonstrate significant improvement in FVC at 3 and 6 months after LRS. The FEV_1 increased from 26% ± 9% (percent predicted) before LRS to 40% ± 15% (p = 0.002) at 3 months, then decreased slightly to 35% ± 16% at 6 months after LRS.

Sixteen patients underwent respiratory pressure measurement immediately before and 1 month after LRS. Maximal inspiratory pressure increased from 73.1 ± 20.4 cm H_2O to 91.6 ± 22.0 cm H_2O ($p < 0.001$), and maximal expiratory pressure increased from 115.6 ± 35.7 cm H_2O to 139.4 ± 34.8 cm H_2O (p = 0.005).

There was less perceived dyspnea after LRS, reflected by an improved mean baseline dyspnea score from 0.83 before the operation to 2.4 ($p < 0.001$) at latest follow-up. The transitional dyspnea index indicated no significant change in dyspnea in three patients, mild (+1) improvement in 15 patients, moderate (+2) improvement in 11 patients, and major (+3) improvement in seven patients. One patient currently reports a mild (–1) worsening of dyspnea.

Exercise pulse oximetry was performed during 6-minute walk testing. In the postoperative period, patients performed more work during exercise assessment as their 6-minute walk distances were significantly longer than the preoperative values at both 3 and 6 months after LRS. Furthermore, postoperative 6-minute walks were performed with fewer stops and with a reduced level of perceived exertion (see Table I).

Before LRS, 18 patients used continuous supplemental oxygen, nine used supplemental oxygen with exertion or during sleep (or at both times), and 10 did not use supplemental oxygen. At latest follow-up 3 to 22 months after LRS, three patients use con-

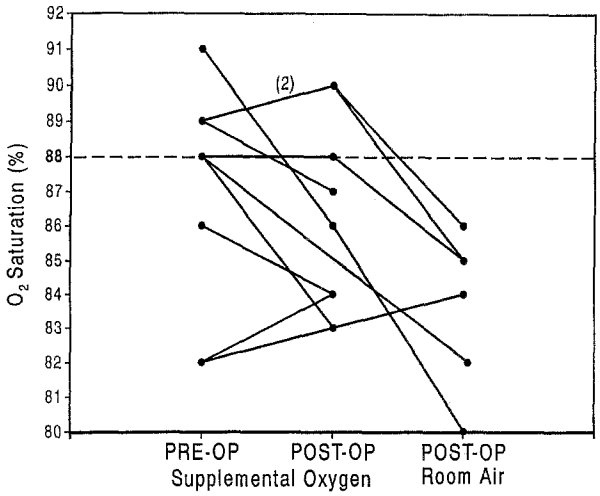

Fig. 1. Exercise oximetry results in 10 patients who had only exercise testing while breathing oxygen before the operation and then underwent postoperative exercise testing while breathing oxygen. Six of these patients were also tested while breathing room air after the operation. An Sao_2 of 88% is represented as a *dotted line,* the acceptable level qualifying for supplemental oxygen.

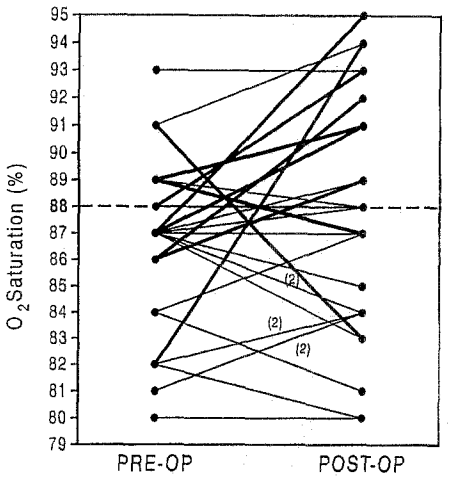

Fig. 2. Exercise oximetry results in 27 patients tested while breathing room air before and after LRS. Twenty-one patients had preoperative exertional desaturation, seven had Sao_2 increase above 88% after LRS, and two of six patients had Sao_2 decrease below 88% after LRS.

tinuous supplemental oxygen, 13 use supplemental oxygen with exertion and sleep, and 21 do not use supplemental oxygen. Notably, 10 patients with exertional desaturation while breathing room air do not use supplemental oxygen because of reduction in dyspnea with activity.

Ten patients who required continuous supplemental oxygen before LRS were tested on their prescribed level of supplemental oxygen both before and 3 months after the operation. The nadir level of Sao_2 recorded during exertion is represented in Fig. 1. Preoperatively, six of these patients had exertional desaturation while receiving supplemental oxygen. The four other patients are assumed to have had preoperative exertional desaturation on the basis of their preoperative oxygen requirement and marginal exertional Sao_2 (89% to 91%) while breathing supplemental oxygen. The remaining 27 patients were tested while breathing room air before and 3 months after LRS (Fig. 2). Twenty-one of these patients had preoperative exertional desaturation (≤88%). Thus a total of 31 patients met American Thoracic Society (ATS) criteria for supplemental oxygen use before LRS. Most significantly, the exercise Sao_2 increased above the 88% cutoff point after LRS in only seven of these 31 patients. Meanwhile, two patients not previously requiring oxygen demonstrated a reduction in exertional saturation such that they now qualified for supplemental oxygen use. In the total population there was no net change in exercise oximetry related to LRS (86% ± 3% before LRS, 86% ± 3% at 3 months, 86% ± 4% at 6 months).

Discussion

The findings of this series indicate significant improvements in pulmonary mechanics, exercise capacity, inspiratory and expiratory pressures, and perception of dyspnea after LRS. However, exercise-induced hypoxia persisted in most of these patients despite these gains. In accordance with these findings, a large proportion of our patients continue to use supplemental oxygen both during sleep and with exertion. Although 43% of patients continue to use supplemental oxygen, a total of 70% actually meet supplemental oxygen criteria by exercise testing. The observation that patients discontinued oxygen use of their own accord led us to investigate patient oxygen requirements more critically in the postoperative period. As we became aware that patients discontinued supplemental oxygen, perhaps inappropriately, we insisted that all available patients follow up at our facility for exercise oximetric evaluation and counseling. Nonetheless, 10 patients continue without supplemental oxygen use despite exertional saturation. On the other hand, we have noted that recovery from exertional hypoxemia appears to occur more quickly after

LRS. Future patients will have increased focus applied to recovery from exercise to assess postoperative physiologic changes that might be contributing to their overall sense of well-being despite exertional desaturation.

In this series the lung volume reduction procedure was performed in a manner similar to that described by Cooper and associates.[1] The most severely emphysematous regions of the lung were resected with the goal of reducing the lungs by 20% to 30%, and attempts were made to tailor the lung such that the finished product would contour well to the thoracic cavity. Patients characteristically had hyperinflation of the lung as measured by body plethysmographic techniques, a pseudorestrictive pattern of pulmonary function with decreased FVC and FEV_1, and heterogeneous yet diffuse emphysematous disease involving all lung zones.

The patient population may have consisted of patients at higher risk as reflected by the relatively advanced age, low preoperative spirometric values, and short 6-minute walk distances compared with patients from earlier reports of bilateral LRS. In our first 15 patients, mean initial 6-minute walk distances were approximately 600 feet, considerably lower than in other experiences. Over time, the initial distances in patients undergoing LRS have increased, reflecting selection of more vigorous patients for LRS. Patients with target areas of more severe disease in either the upper or lower lobes were selected, although ease of operation and less complicated postoperative courses were noted for patients with upper lobe disease. This experience resulted in a trend favoring selection of patients with upper lobe disease during the second half of the series. Nonetheless, these changes in selection criteria were not associated with changes in exercise oximetry after LRS.

Improved ventilation/perfusion (V/Q) matching is the proposed mechanism whereby oxygen requirements are decreased with LRS. Improved oxygenation is thought to result from improved ventilation and perfusion of more normal areas of lung by the resection of space-occupying functionless regions of severe emphysema. Surgery for giant bullae that produce obvious atelectasis in normal adjacent lung has long been an accepted surgical intervention. In these instances, resection of bullae clearly improves V/Q matching. However, this series and that reported by Cooper's group[1] excluded such cases. All patients were believed to have diffuse emphysema with varying degrees of inhomogeneity. Computed tomographic scans in our patients usually demonstrated target areas with severe emphysema and larger bullae, as well as areas of lung with increased or "compressed" vascular markings in the remainder of the lungs. These latter areas are not normal; rather they demonstrate less severe emphysematous disease. In patients with this degree of residual lung disease, decompression evidently has not resulted in a change in V/Q matching sufficient to alter exertional desaturation. There is likely a spectrum of response that is dependent on the amount of parenchymal preservation in the remaining lung and the degree of decompression achieved by resection of target areas.

The fact that a greater proportion of our patients continued to require supplemental oxygen may reflect our strict adherence to ATS guidelines for its use. Previous reports have not given specific criteria by which supplemental oxygen was discontinued.[1, 2] Alternatively, the degree of residual lung disease may have been more severe in our patients such that improvements in V/Q matching were not by themselves sufficient to achieve independence from supplemental oxygen with exercise.

Our findings are supported by those of Sciurba and associates,[4] who found similar functional improvements in patients undergoing predominately unilateral LRS but noted no change in resting arterial oxygen tension. More compelling than an increase in oxygenation or lack thereof were biomechanical improvements achieved by patients after LRS. Sciurba and coworkers[4] documented increased elastic recoil of the lung, restoration of negative intrapleural pressure, and decreased right ventricular pressure after LRS. A decreased work of breathing resulting from reduction in airway resistance has also recently been demonstrated by Miller and coworkers[3] after bilateral LRS.

The salutary effects of supplemental oxygen in patients with hypoxemic COPD are well-founded. The British Medical Research Council study documented improved survival in patients with COPD receiving nocturnal oxygen versus those breathing only room air beginning 500 days after the initiation of therapy.[9] The Nocturnal Oxygen Therapy Trial,[8] studying patients with COPD who had a resting oxygen tension less than 55 mm Hg, compared patients receiving nocturnal oxygen with those receiving continuous oxygen therapy. There was a significant survival advantage to continuous oxygen use over nocturnal oxygen therapy. Patient demographic data for these two studies were similar. Combining the data

sets indicated that longer oxygen use was associated with progressively higher survivals.[13]

Chronic hypoxia in patients with COPD also produces well-defined adverse end-organ effects. Neuropsychiatric dysfunction characteristically involves disturbances in extracting ability, motor skills, and perceptual motor abilities. Before long-term oxygen therapy, neuropsychiatric deficits occurred three times more frequently in patients with hypoxemic COPD than in age-matched controls.[13, 14] Cardiac arrhythmias, particularly episodes of supraventricular tachycardia and premature ventricular complexes, have been documented during nocturnal desaturation ($Sao_2 < 80\%$).[15] Nocturnal desaturation also produces systemic hypertension and an increased heart rate, resulting in increased myocardial stress at a time of relative hypoxemia.[16] Such changes are readily reversible with nocturnal oxygen use. Finally, chronic hypoxia results in slowly progressive pulmonary hypertension in patients with COPD, which can be halted by supplemental oxygen use.[17]

Pulmonary hemodynamic changes are also evident in patients with more mild degrees of hypoxemia. Levi-Valensi and coworkers[18] reported that patients with COPD who have mild hypoxia while awake have significantly higher mean pulmonary artery pressure if they spend at least 30% of their sleep time with an Sao_2 of less than 90%. The authors concluded that a causal relationship between nocturnal desaturation and permanent pulmonary hypertension is likely in patients with COPD. Fletcher and coworkers[19] studied patients with COPD and nocturnal desaturation without daytime hypoxemia and found that Sao_2 less than 90% for at least 5 minutes and less than 85% during rapid eye movement (REM) sleep was associated with pulmonary hypertension. These data are made more compelling by two separate studies, which document that exertional hypoxemia underestimates the degree of nocturnal hypoxemia in patients with COPD.[7, 18] Further, the Nocturnal Oxygen Therapy Trial revealed that a disproportionate number of patients died during sleep in the early morning hours[8, 16] Presumably, patients who have had LRS are subjected to the same tendencies toward nocturnal desaturation and its multisystem effects. Oximetric sleep studies should be obtained on patients who have had LRS before discontinuance of supplemental oxygen, particularly in patients with borderline exercise oximetry and in patients with underlying cardiac disease or elevated pulmonary artery pressures.

In summary, LRS offers dramatic symptomatic and functional improvement for patients with advanced emphysema. Although subjective improvement makes many patients eager to discard their oxygen, it is not clear that this is a safe practice inasmuch as many patients still have significant hypoxemia during exercise. The mechanisms by which lung volume reduction improves patients' sense of well-being may be more related to mechanical factors governing air flow and pleural dynamics than to a fundamental change in V/Q matching.

REFERENCES

1. Cooper JD, Trulock EP, Triantafillou AN, et al. Bilateral pneumectomy (volume reduction) for chronic obstructive pulmonary disease. J Thorac Cardiovasc Surg 1995;109:106-19.
2. Gaissert HA, Trulock EP, Cooper JD, Sundaresan RS, Patterson GA. Comparison of early functional results after volume reduction or lung transplantation for chronic obstructive pulmonary disease. J Thorac Cardiovasc Surg 1996;111: 296-307.
3. Miller DL, Dowling RD, McConnell JW, Skolnick JL. Effects of lung volume reduction surgery on lung and chest wall mechanics. Program of the Thirty-second Annual Meeting of The Society of Thoracic Surgeons, January 29-31, 1996:96-7.
4. Sciurba FC, Rogers RM, Keenan RJ, et al. Improvement in pulmonary function and elastic recoil after lung-reduction surgery for diffuse emphysema. N Engl J Med 1996;334: 1095-9.
5. Little AG, Swain JA, Nino JJ, et al. Reduction pneumonoplasty for emphysema: early results. Ann Surg 1995;222:365-74.
6. Summary of the Second Conference on Home Oxygen Therapy, Denver, February 28 and March 1, 1986. Problems in prescribing and supplying oxygen for Medicare patients. Am Rev Respir Dis 1986;134:340-1.
7. Mulloy E, McNicholas WT. Ventilation and gas exchange during sleep and exercise in severe COPD. Chest 1996;109: 387-94.
8. Nocturnal Oxygen Therapy Trial Group. Continuous or nocturnal oxygen therapy in hypoxemic chronic obstructive lung disease. Ann Intern Med 1980;93:391-8.
9. Report of the Medical Research Council Working Party. Long-term domiciliary therapy in chronic cor pulmonale complicating chronic bronchitis and emphysema. Lancet 1981;1:681-5.
10. Mahler DA, Weinberg DH, Well C., Feinstein AR. The measurement of dyspnea: contents, interobserver agreement, and physiologic correlates of two new clinical indexes. Chest 1984;85:751-8.
11. Sobush DC, Dunning M III. Assessing maximal static ventilatory muscle pressures using the "bugle" dynamometer. Phys Ther 1984;64:1689-90.
12. Borg GAV. Psychophysical bases of perceived exertion. Med Sci Sports Exerc 1982;14:377.
13. Weitzenblum E, Chaquat A, Oswald M, Kessler R. The scientific basis for long-term oxygen therapy in patients with chronic hypoxemia. In: O'Donohue WJ Jr, editor. Long-term oxygen therapy. New York: Marcel Dekker, 1995:25-51.

14. Heaton RK, Grant I, McSweeny AJ, Adams KM, Petty TL. Psychological effect of continuous and nocturnal oxygen therapy in hypoxemic chronic obstructive pulmonary disease. Arch Intern Med 1983;143:1941-7.
15. Tirlapur VG, Mir MA. Nocturnal hypoxaemia and associated electrocardiographic changes in patients with chronic obstructive airways disease. N Engl J Med 1982;306:125-30.
16. Shepard JW, Garrison MW, Grither DA, Evans R, Schweitzer PK. Relationship of ventricular ectopy to nocturnal oxygen desaturation in patients with chronic obstructive pulmonary disease. Am J Med 1985;78:28-34.
17. Weizenblum E, Sautegeau A, Ehrhart M, Mammosser M, Pelletier A. Long-term oxygen therapy can reverse the progression of pulmonary hypertension in patients with chronic obstructive pulmonary disease. Am Rev Respir Dis 1985;131: 493-8.
18. Levi-Valensi P, Weitzenblum E, Rida Z, et al. Sleep-related oxygen desaturation and daytime pulmonary haemodynamics in COPD patients. Eur Respir J 1992;5:301-7.
19. Fletcher EC, Luckett RA, Miller T, Costarangos-Galarza C, Kutka N, Fletcher JG. Pulmonary vascular hemodynamics in chronic lung disease patients with and without oxyhemoglobin desaturation during sleep. Chest 1989;95:757-64.

Discussion

Dr. Keith S. Naunheim *(St. Louis, Mo.).* Dr. Bousamra has pointed out that the physiologic benefit provided by lung reduction procedures, while substantial, is not absolute. The marked subjective improvement in dyspnea may mask a continuing and significant deficit in oxygenation, which should not be ignored.

There are some limitations in the manuscript which should be noted. The first problem is that conclusions are made on the basis of a relatively small sample size. However, this is a common and unavoidable situation during the start-up of any new technique and it will self-correct in time.

The second difficulty has to do with the timing of the functional testing performed. As noted in the manuscript, one third of the study population had exercise testing performed before pulmonary rehabilitation as opposed to after pulmonary rehabilitation, calling into question how much improvement resulted from the rehabilitation program itself as opposed to the surgical procedure. It is critical that standardized testing be performed after the rehabilitation and immediately before the operation so that the effect of the LRS itself can be assessed more accurately without the confounding effects of pulmonary rehabilitation. Finally, in the same vein, it is important that investigators reporting on lung volume reduction procedures do so in an organized and systematic fashion so that results can be compared both over time and from institution to institution.

Although Dr. Bousamra's reports have shown changes in FEV_1, FVC, 6-minute walk, and oxygen consumption, many parameters are unreported that might prove helpful in the future. These include spirometric indices such as maximum voluntary ventilation, volume measurements such as residual volume and total lung capacity, and simple gas exchange parameters such as resting oxygen and carbon dioxide tensions. For those of us who wish to participate in active investigation of lung volume reduction, it is critical that the reporting of these results be not only accurate but thorough. It is only through such efforts that real progress will be made. Despite these drawbacks, the manuscript stands as a confirmation of the beneficial effects of lung reduction procedures and it advances our understanding of the physiologic effects of such operations.

I have three areas of questions for the authors. The first deals with the surgical risk. Your 11% operative mortality is similar to that reported by others using a bilateral open approach, but it is still somewhat distressing. Did the deaths occur late in your clinical experience or early, during the so-called learning curve? Do you think that suboptimal patient selection played a role in these deaths? If so, what tips can you offer to avoid this pitfall? Do you think that alternative surgical strategy such as a thoracoscopic approach or a staged bilateral procedure might be advisable in elderly or high-risk patients?

The second area of questions deal with the parameters that were not reported in your manuscript. Did you measure lung volumes, shunt fractions, and room air blood gases? If so, did you see significant changes in these parameters in the postoperative period?

Finally, I have a question regarding functional improvement. Twenty percent of the patients in your series were not benefited by the operation either because they died or their functional and respiratory status did not significantly improve. Did you perform any risk analysis to try to identify predictors of success and failure? Do you have any experience using specialized techniques such as density mask imaging of computed tomographic scans or spectroscopy scanning to better assess patients in the preoperative period and to weed out those who are less likely to be benefited by the operation?

Dr. Bousamra. Thank you, Dr. Naunheim. To first address the question of exercise testing at initial evaluation and then preoperatively and then postoperatively: Eleven of our patients had only initial 6-minute walks. The mean distance for their 6-minute walks was 750 feet, the mean distance for the total group was approximately 850 feet, and the mean distance for those tested immediately before the operation, about 26 patients, was about 900 feet. Thus there was a 150-foot improvement comparing those two groups. Meanwhile, after LRS there was on average a 300-foot improvement in the 6-minute walk distance, from 850 feet to 1150 feet. Therefore I think that, although we do not have completely uniform data, we have demonstrated an effect of the LRS above and beyond the preoperative exercise training period.

Regarding surgical risk, all of the deaths in this series were truly related to the operation, and all deaths were within 5 months of the operation. I think a learning curve has been involved; four of the deaths were within the first half of our series. We found that patients who had uniform disease, patients who had a diffusion capacity of the lung to carbon monoxide of less than 20% of predicted, especially if they also had carbon dioxide retention, were at an increased risk of mortality. We currently avoid those patients.

Regarding a staged procedure, we do not have enough experience with the unilateral approach to comment from experience.

Regarding measurement of lung volumes, we did not have complete data in that regard on our 37 patients, but I do have information on approximately 20 patients. The diffusion capacity to carbon monoxide in our patient group was relatively stable; it went from 29% to 35% of predicted during the postoperative period. The trapped gas volume decreased from a median value of 1.7 to 1.0 L. The residual volume went from 210% of predicted to 136% of predicted. The FRC was reduced from 5.1 to 3.9 L, and the total lung capacity was not significantly changed but did decrease from 6.5 to 5.6 L. All of these results are consistent with previously reported series of bilateral LRS.

Regarding predictors of patients with improved functional results, we evaluated our patients whose saturation improved from below to above 88% with exercise. All of those patients did have apical disease. We have had some spectacular results in patients with lower lobe disease, although those patients had a marked gradient of disease, their lower lobes being very severely affected and their upper lobes being relatively normal. I think that this disease process is a spectrum of severity in which the amount of diseased lung resected should be balanced against the amount of functioning lung that remains. Perhaps our oximetric results reflect that the underlying lung in our patients was not of sufficient quality to improve the oximetry in patients, at least during exertion. On the other hand, we also did note that the majority of our patients were able to be weaned from continuous supplemental oxygen. It was only with exertion that they continued to require supplemental oxygen.

Dr. Joel D. Cooper *(St. Louis, Mo.).* I was puzzled by your results compared with our own series. The FEV_1 started off in exactly the same place, but the improvement that you reported was a little less than half what we have observed in our patients. The 6-minute walk results that you started off with were exactly where we started off, but you ended up where we ended up after the period of preoperative rehabilitation and before, not where we ended up after the operation. Thus I was puzzled by the fact you did not achieve similar results. All of our results are obtained under controlled, measured circumstances such as you described, using oximetric studies. Of course, one must also remember that if you enable the patient to walk faster both by the period of training and by the effects of the operation, inevitably the minute consumption of oxygen has risen because they are less dyspneic and are able to achieve a higher level of physical activity. Postoperatively, 70% of our patients were able to discontinue the use of continuous supplemental oxygen, and more than 50% stopped needing supplemental oxygen during maximum exertion, even though that level of maximum exertion was now much, much greater than it was before.

Can you explain these differences in the results between our series? The operation has two components. One is a volume reduction effect on respiratory mechanics—that affects dyspnea, affects performance, affects mechanics of breathing. A second is a redistribution effect, that is, improving ventilation to the better parts of the lung. That is what I believe yields the improved oxygenation. In different patients there are different degrees of each of these components. Of course, they are not necessarily related. I would be interested to know how uniform the disease was in your patients. You indicated the residual volume was 210% to start with. That was markedly below our lower limit.

Dr. Bousamra. With regard to FEV_1 measurements, we demonstrated a 59% improvement at 3 months-identical to your data at 6 months. Our 6-month data were less complete. The improvement in FVC, I believe, is similar between our two series.

With regard to the 6-minute walk distances, I am confident that our physiotherapists trained the patients well; it is what they do for a living. The fact that the patients continue to improve after the operation, as well as the magnitude of this improvement, is evidence that our rehabilitation program is effective in our subgroup of patients. Part of the difference may be that we operated on 45 of 120 patients, and I believe you mentioned that you probably operate on 20% of the patients who are referred to you. That is, we were less selective.

The residual volumes reflect that we have been "less selective" than you have been. We operated on four patients who truly had diffuse disease, and two of those patients died, one patient had a modest benefit from the procedure, and the other one had a moderate improvement in dyspnea as a result of the procedure. I agree with you that improvements in oxygenation may be due to relative improvements in V/Q mismatch. In our patients, I postulate that the remaining lung is less diseased but still dysfunctional from underlying emphysema; thus the improvement in oxygenation is less than in your series.

Monitoring exercise oximetry is an important issue for all of us as we follow our patients after lung volume reduction, because patients in whom desaturation occurs during exertion also experience desaturation at night. Nocturnal desaturation may last for longer periods, not for just the few moments that occur during exertion. There are important issues at stake, for instance, the development of pulmonary hypertension, cor pulmonale, and the possibility of reduced life-expectancy.