

Active Management of a Patient with Endstage Pulmonary Emphysema Using Lung Volume Reduction Surgery and Intensive Rehabilitation

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Abstract

Introduction: Lung volume reduction surgery (LVRS) and pulmonary rehabilitation are newer options available in the treatment of advanced emphysema. We describe the progress of our first local patient to have undergone these 2 new treatment modalities. **Clinical Picture:** A 65-year-old man with advanced emphysema, limited by crippling dyspnoea despite maximal medical therapy with inhaled bronchodilator therapy, methylxanthines and supplementary oxygen. **Treatment:** Physical reconditioning with a 4-week inpatient pulmonary rehabilitation programme, followed by LVRS and a further 6-week outpatient pulmonary rehabilitation. **Outcome:** Dramatic improvements in spirometric indices, arterial blood gases, exercise capacity and overall functional status. Improvements maintained at one-year follow up period. **Conclusions:** LVRS with pulmonary rehabilitation is an exciting new treatment option in a select group of patients with advanced emphysema. Their role in improving overall performance status and quality of life should be considered in patients otherwise considered to have reached the limits of medical therapy.

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Key words: Dyspnoea, Palliation, Quality of life

Case Report

A 65-year-old Chinese man with a 30-pack year history of cigarette smoking was referred to our institution for further management of chronic obstructive pulmonary disease (COPD). Despite smoking cessation for 5 years, and pharmacological treatment with oral theophylline, inhaled ipratropium bromide and ventolin, his dyspnoea had progressed to the extent that he was unable to administer self-care. In addition, he required frequent hospitalisations for exacerbations, with one particularly severe exacerbation 2 years ago requiring mechanical ventilatory assistance.

Clinically the patient was barrel chested. He was cyanotic and tachypnoeic at rest and on room air. There was no clinical evidence of associated cor pulmonale. Chest auscultation revealed diminished breath sounds bilaterally.

His blood gases on intranasal oxygen 2 L/min showed pH 7.387, pCO₂ 47.8 mmHg, pO₂ 82.1 mmHg, O₂ saturation

95.7, standard bicarbonate (SBC) 26.8 mmol/L. Haemoglobin level was 12.5 g/dL. His chest X-ray (Fig. 1) and computerised tomography (CT) of the thorax (Fig. 2) confirmed emphysema involving both upper lobes, more severe on the right.

As a prelude to planned lung volume reduction surgery, the following tests were also done: transthoracic two-dimensional echocardiogram, cardiopulmonary exercise testing and 6-minute walk. The echocardiogram showed a normal left ventricular ejection fraction of 60%, but severe emphysema precluded accurate assessment of pulmonary pressures. An attempt at exercise testing was abandoned when the patient developed oxyhaemoglobin desaturation on unloaded pedalling on a cycle ergometer. He was unable to complete a 6-minute walk test.

The patient's condition was optimised as an inpatient and he underwent 4 weeks of inpatient pulmonary rehabilitation comprising both upper and lower extremity exercises and

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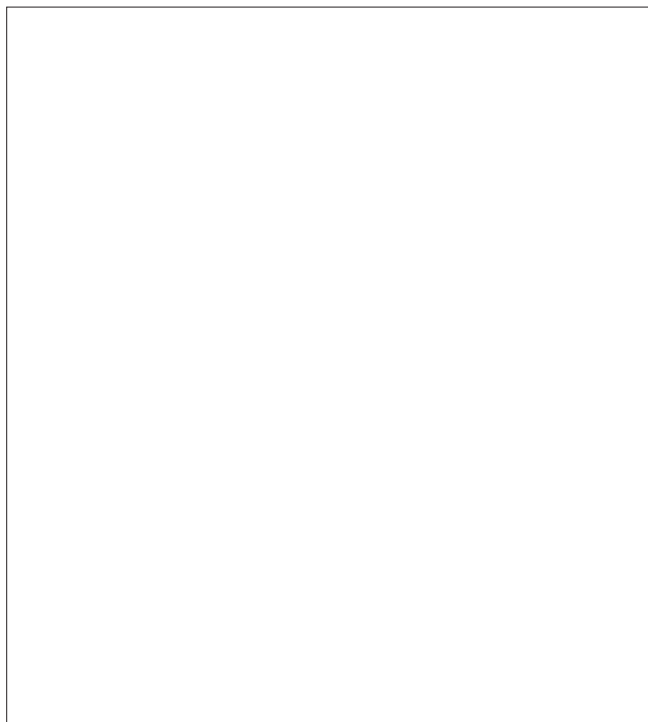


Fig. 1. Patient's preoperative chest X-ray.

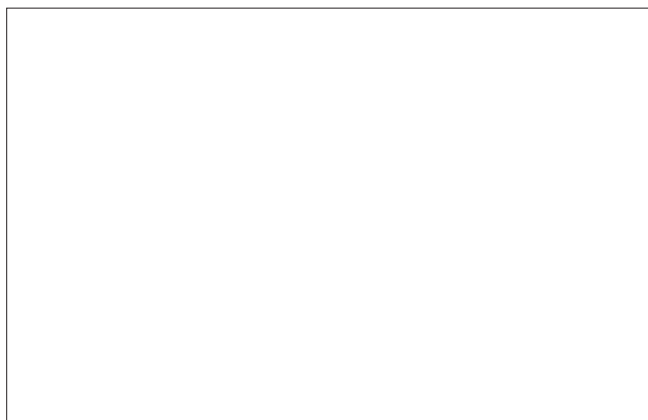


Fig. 2. Patient's preoperative CT thorax.

chest physiotherapy. At the end of the programme, his 6-minute walk test improved to 166 m though he still required supplementary oxygen of 2 L/min and stopped 3 times in between to rest. His blood gas on room air remained in type I respiratory failure.

Following the 4 weeks of inpatient pulmonary rehabilitation, he underwent lung volume reduction surgery (LVRS). Via a median sternotomy approach, 50% of his right upper lobe and 20% of the left upper lobe was resected and stapled.

Following surgery, the patient's 6-minute walk improved to 215.5 m. Significantly, he did not require the use of supplementary oxygen. Following his discharge on the

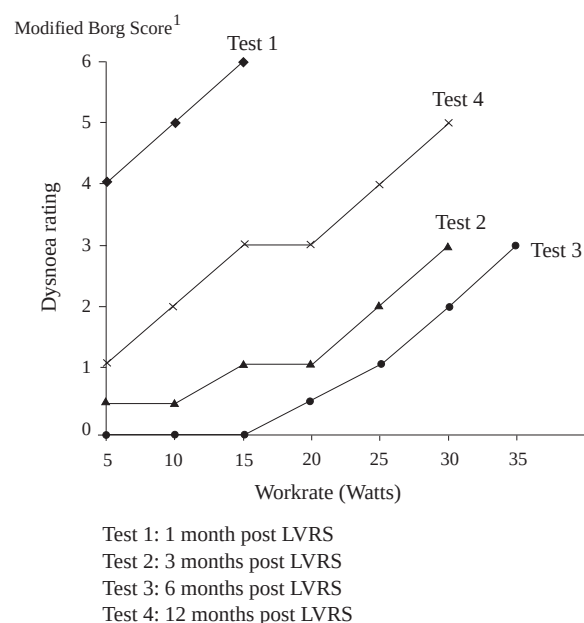


Fig. 3. Perceived dyspnoea ratings at various workloads on incremental cardiopulmonary exercise testing.

14th postoperative day, he commenced on a 6-week outpatient pulmonary rehabilitation programme starting from one month after LVRS. He was maintained on inhaled ipratropium bromide at two puff thrice daily and oral theophylline. Following the outpatient programme, his 6-minute walk improved further to 340 m. He remained free of exacerbations until the 11th month following surgery, when he was admitted for 2 days for haemoptysis from bronchitis. This was self-limited and he returned to his previous activity level after 2 weeks.

Cardiopulmonary exercise was conducted at interim periods of one month, three months, six months and twelve months following LVRS. This was performed on a cycle ergometer using an incremental workload protocol. Dyspnoea was assessed during exercise testing using the modified Borg scale, a 0 to 10 category ratio scale of perceived exertion.¹ Serial assessment of pulmonary function, exercise capacity and quality of life assessment is shown in Table I. His dyspnoea scores showed serial improvement during incremental exercise testing (Fig. 3). He maintained a longer duration of cycling at a constant workrate of 12 watts. Upon completion of pulmonary rehabilitation, he had gained 6 kg, and his body mass index increased from 18 to 20 kg/m². Most importantly, his quality of life scores showed a sustained improvement.

Discussion

Chronic obstructive pulmonary disease has been defined as a chronic irreversible and slowly progressive disorder characterised by airflow limitation confirmed by spirometry that does not change significantly, spontaneously or with

TABLE I: SERIAL VALUES OF PULMONARY FUNCTION AND EXERCISE CAPACITY AT PREOPERATIVE, 1, 3, 6 AND 12 MONTHS POSTOPERATIVE

Parameters	Predicted	Preoperative Pre inpatient rehab	1 month postoperative, pre outpatient rehab	3 months postoperative, post outpatient rehab	6 months postoperative	12 months postoperative
FEV ₁ (L)	2.25	0.480	0.960	1.16	0.88	0.81
FVC (L)	3.20	2.44	2.05	3.04	2.67	2.30
TLC (L)	5.13	6.15	5.83	6.81	7.77	6.42
PaCO ₂ (mmHg)	—	45.6	33.8	—	38.5	30.4
PaO ₂ (mmHg)	—	45.9	60	—	64.6	65
MIP (kPa)	5.78	—	2.25	1.72	5.52	4.77
MEP (kPa)	7.91	—	8.66	8.33	7.69	8.24
MVV (L)	30	—	26.62	48.7	45.2	31.93
VO ₂ max (mL/min)	1537	—	599	722	861	644
6-minute walk distance (m)	—	166	215.5*	340	—	318
Maximum workrate (W)	125.5	—	15	30	35	30
Duration of exercise at constant workrate:12 W (min)	—	—	3	20.1	20.5	30
QOL score	—	—	113	137	—	138

FEV₁: forced expiratory volume in one second; FVC: forced vital capacity; MEP: maximal expiratory pressures; MIP: maximal inspiratory pressure; QOL: quality of life (measured using the Chronic Respiratory Disease Questionnaire²³); RV: residual volume; TLC: total lung capacity;

VO₂ max: maximum oxygen uptake;

* 13 days following surgery

— test not performed

treatment over several months of observation.^{2,3} This simple definition underscores the heterogeneous patients that fall under this umbrella, ranging from emphysema, chronic obstructive bronchitis, and chronic bronchitis. These patients are similar only in their common complaint of dyspnoea and, in the vast majority, a prior history of heavy cigarette smoke exposure. Affected individuals fall along a continuum ranging from minor symptoms to crippling dyspnoea that impairs common daily activities and precludes self-care. Previously, the options for patients who have exhausted available medical avenues were limited to bullectomy in selected candidates, or lung transplantation. The only other option was palliative care.

The alleviation of dyspnoea is the primary goal when treating COPD. However, mechanisms involved in dyspnoea remain complex and poorly understood. The perception of dyspnoea is difficult to ascribe to any single cause and usually involves a complex interplay between peripheral receptors in the airways, lung and chest wall, central respiratory motor centre and cognitive function.⁴ Certainly, correction of hypoxaemia alone with supplementary oxygen does not necessarily lead to improvement in dyspnoea,^{5,6} as is the case with our patient. There is no sole physiologic parameter that correlates with the dyspnoea reported by patients with COPD. Dyspnoea in COPD patients is multifactorial, receiving contributions from increased ventilatory muscle resistive load secondary to airway narrowing, dynamic hyperinflation which compromises ventilatory muscle performance and

ventilation perfusion abnormalities resulting in arterial hypoxaemia.^{7,8} Consequent physical inactivity also leads to a deconditioned individual, producing accelerated blood lactate levels during exercise, which makes further demands on the ventilatory system.⁴

The foundation for LVRS was first laid 40 years ago by Dr Otto Bratigan.⁹ Unfortunately, his renegade surgical approach to emphysema was not widely endorsed due to a lack of objective data documenting benefits and unacceptable perioperative mortality. The technique was later revised and popularised in the 1990s by Cooper et al.¹⁰ By shaving off 20% to 30% of the most emphysematous areas of the lung in 20 patients, striking postoperative improvements were seen in FEV₁, dyspnoea scores, and quality of life. In their collective data on 150 patients who underwent bilateral LVRS via median sternotomy and staple approach, Cooper et al¹¹ documented mean increases in FEV₁ of 51% at 6 months. Over this similar period, corticosteroid reliance was reduced from 53% to 17%, and oxygen liberation allowed in 70% of the patients who had been previously oxygen dependent. The authors ascribed the improvements to relatively basic principles¹²: while the main function of the lungs was for gas exchange, adequate ventilation of the lungs depended on the bellows function of the thoracic-abdominal structures and diaphragm. Reversing thoracic hyperinflation was felt to improve ventilation by improving lung elastic recoil, improving respiratory muscle efficiency from diaphragm reconfiguration, and reducing the overall work of breathing.

Selective resection of the more severely diseased lung would allow more synchronous lung functioning, and improve ventilation and perfusion matching. Subsequent reports were in general agreement with these principles.^{13,14}

More modest improvements in functional status have also been achieved after undergoing pulmonary rehabilitation. While not associated with significant changes in spirometric indices, pulmonary rehabilitation has been shown to improve dyspnoea, exercise endurance and quality of life,^{15,16} through either dyspnoea desensitisation,¹⁷ or and a true training effect.¹⁸ Our patient experienced dramatic postoperative improvements in dyspnoea and functional capacity, which is not unexpected, given his improved FEV₁ after LVRS. The later improvements seen in the respiratory muscle strength may be a reflection of the effects of ventilatory muscle training during pulmonary rehabilitation. Physical reconditioning with pulmonary rehabilitation also resulted in an improved maximal and submaximal exercise capacity on exercise testing. As demonstrated in this case, the roles of both LVRS and pulmonary rehabilitation are synergistic. The improvements in lung mechanics following LVRS, while dramatic, may begin to decline from as early as 3 to 6 months postoperatively.^{19,20} From these observations, it would be logical to capitalise on the dramatic short-term improvements in lung function and begin pulmonary rehabilitation soon following surgery. The improvement post-LVRS would allow for a more intensive exercise programme to optimise physical conditioning prior to the anticipated decline in lung function. The overall improvements in quality of life after LVRS and pulmonary rehabilitation appear to be more than a simple additive effect.²¹ Will we improve survival for this patient? From the early works on COPD mortality by Burrows,²² our patient, with FEV₁ of 480 mL preoperatively, is expected to have a median survival of 2 years. We may not have altered the course of his disease, but we did improve his quality of life.

It is too early to comment on the overall durability of these improvements achieved by LVRS. The benefits of LVRS on dyspnoea appear to be in excess of a year.^{13,18,19} Some of Bratigan's patients have reported sustained improvements up to 5 years postoperatively.⁹ Twelve months after LVRS, the benefits of the surgery and postoperative pulmonary rehabilitation appear to be maintained in this patient. FEV₁ seems to be on a downward trend following an initial peak at 3 months post LVRS. Nevertheless, his FEV₁ still remains approximately 68% above the preoperative value. Lung volumes are also starting to increase, consistent with hyperinflation. These negative points are matched by increases in maximal voluntary ventilation, which has increased from 26.6 L at 1 month post surgery to 45 L at 6 months post surgery. His maximal

inspiratory pressure (Pimax) has also improved from 2.25 kPa (39% of predicted) to 5.52 kPa (88% of predicted) at 6 months. These results are consistent with improved respiratory muscle strength. His submaximal exercise endurance continues to improve, being able to pedal against a constant workload for a longer duration. While his spirometric data and peak exercise capacity at one year show a marginal decline compared with 6 months before, it is noteworthy that his overall performance remains markedly improved from his baseline. In addition, some decline in his values at 1 year may be related to his recent exacerbation the month before. Most importantly, the improved quality of life was sustained up to one year in our patient.

LVRS must be viewed in its proper perspective as a palliative procedure and not as a cure for advanced emphysema. Further refinement is needed in defining the selection criteria for patients with LVRS. However, we feel that important features in candidacy include spirometric evidence of severe obstruction, heterogeneous emphysema, evidence of thoracic hyperinflation, symptomatic dyspnoea despite optimal medical therapy (including smoking cessation), motivation to complete a rehabilitation programme and absence of significant right heart disease.

In conclusion, treatment for dyspnoea in COPD has progressed from efforts centred primarily on pharmacological interventions. While exciting, the new treatment modalities of LVRS and pulmonary rehabilitation are not substitutes for primary and secondary prevention with smoking cessation. However, they do offer a potential for improvement in selected patients with COPD and intractable dyspnoea.

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