Peer reviewed article

News on lung volume reduction surgery

Erich W. Russi, Walter Weder

Pulmonary Division and Division of Thoracic Surgery, Universitätsspital Zürich, Switzerland

Summary

Lung volume reduction surgery (LVRS) is an established therapeutic option for patients with advanced pulmonary emphysema after all conservative measures, including comprehensive pulmonary rehabilitation, have been exhausted. LVRS improves pulmonary function, shortness of breath, exercise capacity and hence quality of life in some 80% of cases for up to four years. Even patients with homogeneous types of pulmonary emphysema improve if those with extremely low FEV_1 and/or very low diffusion capacity are excluded. At experienced centres perioperative mortality is less than 2% in appropriately selected patients, and current results suggest that the five-year survival in COPD patients may even be improved by this palliative surgical intervention. In patients under 60 LVRS may serve as a bridging procedure to lung transplantation. Bronchoscopic creation of extra-anatomic bronchopulmonary passages – endoscopic LVRS – is a novel approach now under investigation.

Key words: pulmonary emphysema; lung volume reduction surgery

Several prospective case series [1–3] and, more recently, a few randomised, controlled prospective trials [4–8] have demonstrated that lung volume reduction surgery (LVRS) improves dyspnoea, lung function, exercise tolerance and quality of life [9] in patients with severe forms of emphysema. The degree of improvement is variable and may depend on different factors, including aetiology and morphological types of emphysema, preoperative lung function, and amount of resected emphysematous lung tissue.

Selection of patients

The selection of patients is based on sound pathophysiological concepts and contraindications which are known to increase the perioperative complication rate and mortality (table 1). Synopsis of all available data is important in this process. There is strong evidence that the two major symptoms, i.e. limited exercise capacity and shortness of breath during exercise, are mainly a consequence of pulmonary hyperinflation, a hallmark of severe pulmonary emphysema. Hence considerably increased thoracic gas volume remains the most important prerequisite for successful surgical volume reduction. It is not surprising that LVRS may have a high perioperative complication rate if performed in patients whose lung function, particularly gas exchange, is severely impaired due to an almost entirely destroyed lung, i.e. in those with so-called “vanishing” lung. This is unequivocally demonstrated by an interim analysis of the National Emphysema Treatment Trial (NETT) [10]. In 69 patients with a forced expiratory volume in one second (FEV_1) of no more than 20% of the predicted value and either homogeneous distribution of emphysema on computed tomography or a carbon monoxide diffusing capacity of no more than 20 % of the predicted value, 30-day mortality after surgery was 16% (95% confidence interval: 8.2–26.7%), as compared to nil mortality among 70 patients treated medically ($p <0.001$). Perioperative mortality is considerably lower (<5%) in centres with experience in selecting and operating on suitable patients [11]. In our 150 patients 30-day mortality was 2% [12].

Certain authors have stressed that patients with distinct regional differences in tissue destruction (i.e. with markedly heterogeneous emphysema) on computed tomography (CT), perfusion scintigram or both, profit most from LVRS because non-functional areas identified by imaging techniques are ideal targets for resection [1]. We have corroborated this concept in previous analy-

Supported by Grants: Swiss National Foundation 3200–063709.00 and Zurich Lung League.

Table 1

Patient selection for LVRS.

Indications	
Severe emphysema	hyperinflation: TLC >125% pred.
	RV/TLC: ≥0.65
	severe airflow obstruction: FEV_1 <35% pred.
Impaired exercise performance	12′ walking distance: <600 m
	VO_2 max: <12 ml/kg/min
Contraindications (absolute & relative)	
Vanishing lung	diffusing capacity <20% pred.
	HRCT: almost no lung tissue left
Lung function	FEV_1 <20% pred.
Pulmonary hypertension	PAP_{mean} ≥35 mm Hg
Hypercapnia	$PaCO_2$ >55 mm Hg
Extrapulmonary factors	coronary artery disease
	malignancy with life expectancy <2 y
	complicated osteoporosis
	emotional instability

ses but, more importantly, we have been able to demonstrate that even patients with a uniform pattern of emphysematous destruction (i.e. a homogeneous type) show significant and clinically relevant improvement after the operation [13]. Thus a homogeneous type of emphysema is not a contraindication for LVRS provided FEV_1 and diffusion capacity are not below 20% of the predicted values and if aspects of vanishing lung are absent from CT scan.

Hypercapnia *per se* is not a contraindication for LVRS provided a reasonable amount of functional lung tissue is left (no vanishing lung) and the mechanical impairment is marked. Only a minority of patients with advanced emphysema have more than mild pulmonary hypertension [14]. It is therefore unnecessary to perform routine echocardiography or eventually right heart catheterisation in the preoperative evaluation. Approximately 15% of LVRS candidates have relevant ischaemic heart disease despite the fact that symptoms of myocardial ischaemia are lacking [15]. This is due to their low level of exercise capacity. Hence the decision whether coronary angiography is required preoperatively must be made on an individual basis. In this patient population no sensitive and specific method is applicable to noninvasive detection of relevant coronary ischaemia. In patients with *coronary artery stenoses*, percutaneous transluminal angioplasty may be performed preoperatively or coronary artery bypass grafting in the same surgical session.

Twenty-three of our LVRS patient population have alpha-1-antitrypsin deficiency. All except two were also heavy smokers. The natural history and morphology of these patients' lung disease is different from pure smoker's emphysema. We confirm other investigators' experience that in this patient population the degree and mean duration of functional improvement after LVRS is less than in patients with smokers' emphysema [16, 17]. However, we observed that individual patients experienced several years' benefit from the surgical intervention. Hence we do not consider alpha-1-antitrypsin deficiency *per se* as an absolute contraindication for LVRS [18].

Effects of LVRS

The beneficial effects of LVRS are summarised in table 2. The improvements in pulmonary mechanics have a marked impact on quality of life in these patients, who are severely impaired in their daily activities despite optimal medical treatment including pulmonary rehabilitation.

Almost all COPD patients with severe pulmonary emphysema have a history of heavy smoking. Some of them suffer from symptoms of chronic bronchitis and particularly from recurrent exacerbations. These problems are not improved by LVRS. 15% of our patients had hypoxaemia to a degree fulfilling the criteria for long-term oxygen therapy. After LVRS mean paO_2 improved slightly for up to one year due to a transient increase in alveolar ventilation as indicated by a lower $paCO_2$. However, improvement of gas exchange is not predictable in the individual patient. This is important to know when counselling the patient, who expects long-term oxygen therapy to be no longer necessary after LVRS.

Table 2

Changes after LVRS in patients with severe emphysema.

Shortness of breath (MRC): ▲▲ (1–2)
Breathing pattern: ▲
Cough and sputum production: ▶
Frequency of exacerbations: ▶
Health-related quality of life: ▲▲
Exercise tolerance: ▲
peak exercise (Watts): ▲ (15)
timed walking distance (m): ▲ (90)
Survival: ▶
Pulmonary function:
FEV_1 (% pred): ▲ (13)
IVC (% pred): ▲ (18)
RV: ▼
RV/TLC: ▼ (0.1)
DLCO: ▶
PaO_2: ▶
$PaCO_2$: ▶

Abbreviations: ▲: improved; ▶: unchanged; diminished: ▼; average changes at three months after LVRS are given in their respective units; MRC: Medical Research Council dyspnoea scale (0–4; from no shortness of breath to dyspnoea on minimal exercise; FEV_1: forced expiratory volume in one second; IVC: inspiratory vital capacity; RV: residual volume; TLC: total lung capacity; DLCO: carbon monoxide diffusing capacity; PaO_2: partial pressure of arterial oxygen; $PaCO_2$: partial pressure for carbon dioxide.

LVRS for other obstructive lung diseases

According to theoretical considerations [19] LVRS should improve lung function irrespective of the type of disease causing hyperinflation. The functional gain achieved by LVRS is greatest if the mismatch between the size of the lungs and of the chest wall is most pronounced, *i.e. hyperinflation of the lung is considerable*, and if the amount of resected lung volume is substantial. Hence patients with severe chronic and irreversible pulmonary hyperinflation of aetiology other than pulmonary emphysema may profit from LVRS as well. We have successfully performed LVRS in a 14-year-old boy with disabling airflow obstruction and hyperinflation secondary to postinfectious bronchiolitis unresponsive to medical therapy [20]. Within days of LVRS major improvements in symptoms and lung function had occurred and still persist after two and a half years. To our knowledge this is the first successful LVRS in a patient with obstructive lung disease not accompanied by pulmonary emphysema.

Long-term results

Several groups have reported beneficial long-term results after LVRS [1, 21]. 25 of our 150 patients have now reached four years post LVRS. In analysing their functional results one must consider a distinct positive bias due to deaths and patients lost to follow-up. Nevertheless, four years after LVRS the mean dyspnoea score is still better then preoperatively and improvement of various functional parameters also persists. To appreciate these results one must consider the natural history of COPD, i.e. emphysema, which is not influenced by any medical intervention besides smoking cessation and is attended by a constant decline in lung function. We recently analysed the evolution pattern of lung function, i.e. the time course of FEV_1 for up to four years after LVRS [22]. After reaching maximum value within 6 months postoperatively, the decline in FEV_1 was most rapid in the first year and slowed down in the succeeding years according to an exponential decay. We conclude that the long-term functional results of LVRS may be more favourable than could be expected from linear extrapolations of short-term observations.

Five-year survival in patients with emphysema of severity comparable to our population ranges around 50%. There are no controlled long-term prospective trials comparing the survival of medically treated patients with that in patients who underwent LVRS. Mean survival in our patients at five years is approximately 70%, which is comparable to observations by the group of Cooper et al. [23].

LVRS has the potential to defer lung transplantation and is therefore used in several centres as a procedure whereby the time of transplantation in patients under 60 can be delayed for several years.

Endobronchial (bronchoscopic) approaches to deflation of emphysematous lungs

The potentially favourable effect of LVRS on respiratory mechanics is unquestionable. However, since only relatively few patients are good candidates for this procedure and the benefit to be expected is often not in balance with the potential morbidity, various groups are currently investigating an endobronchial approach to deflation of emphysematous lungs by bronchoscopy.

Collateral ventilation, defined as the ability of gas to travel through non-bronchial pathways from one part of the lung to another, is extensively present in emphysema. However, due to the expiratory collapse of the small airways air is trapped and contributes markedly to pulmonary hyperinflation, which is believed to be the most important cause of dyspnoea. P. Macklen [24] has therefore suggested creating passageways through the chest wall allowing trapped gas to exit from emphysematous lung areas by bypassing the small obstructed airways. This concept was modified by Joel Cooper and colleagues, who proposed that the creation of direct stented passages from the large airways into the emphysematous pulmonary parenchyma would improve expiratory flow and respiratory mechanics (figure 1). Lausberg and colleagues [25] demonstrated in an ex vivo study in emphysematous human lungs that FEV_1 doubled when several stented passages were created. We are currently investigating this concept in emphysema patients who are not ideal candidates for LVRS.

Another group has proposed that the insertion of small one-way valves (Heimlich valves) in segmental bronchi would result in deflation of distal lung parenchyma. However, preliminary work has demonstrated no effect of this kind, probably because the resistance of the valves is higher than that through pathways for collateral ventilation. A different concept is being investigated by another group who propose that bronchial lavage of emphysematous lung tissue with antisurfactant and consequent sealing of the corresponding airway creates a collapse of this area. However, thus far no data have been forthcoming.

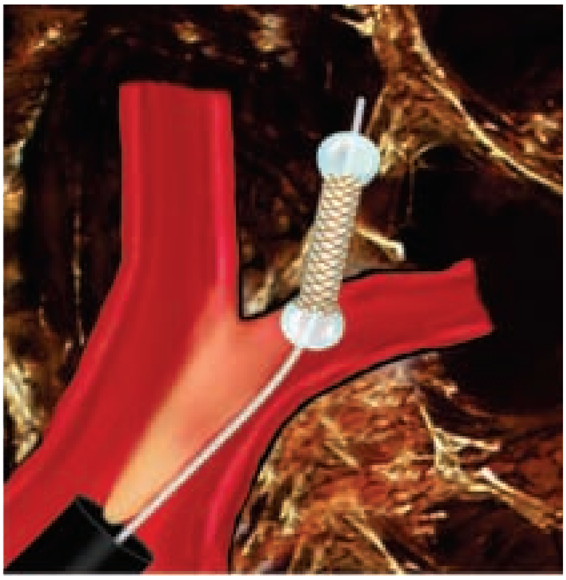


Figure 1

The transbronchial stent is placed over a guide wire and positioned in a newly created passage between a subsegmental bronchus and emphysematous lung tissue under flexible bronchoscopic control.

Correspondence:
Prof. Dr. Erich W. Russi
Pulmonary Division
University Hospital of Zurich
CH-8091 Zurich
E-Mail: erich.russi@dim.usz.ch

References

1 Cooper JD, Patterson GA, Sundaresan RS, Trulock EP, Yusen RD, Pohl MS, et al. Results of 150 consecutive bilateral lung volume reduction procedures in patients with severe emphysema. J Thorac Cardiovasc Surg 1996;112:1319–30.
2 Bingisser R, Zollinger A, Hauser M, Bloch KE, Russi EW, Weder W. Bilateral volume reduction surgery for diffuse pulmonary emphysema by video-assisted thoracoscopy. J Thorac Cardiovasc Surg 1996;112:875–82.
3 Brenner M, McKenna RJJ, Gelb AF, Fischel RJ, Wilson AF. Rate of FEV_1 change following lung volume reduction surgery. Chest 1998;113:652–9.
4 Geddes D, Davies M, Koyama H, Hansell D, Pastorino U, Pepper J, et al. Effect of lung-volume-reduction surgery in patients with severe emphysema. N Engl J Med 2000;343:239–45.
5 Pompeo E, Marino M, Nofroni I, Matteucci G, Mineo TC. Reduction pneumoplasty versus respiratory rehabilitation in severe emphysema: a randomized study. Pulmonary Emphysema Research Group. Ann Thorac Surg 2000;70:948–53.
6 Criner GJ, Cordova FC, Furukawa S, Kuzma AM, Travaline JM, Leyenson V, et al. Prospective Randomized Trial Comparing Bilateral Lung Volume Reduction Surgery to Pulmonary Rehabilitation in Severe Chronic Obstructive Pulmonary Disease. Am J Respir Crit Care Med 1999;160:2018–27.
7 Lödfahl, Hillerdal G, Ström K. Preliminary results up to 12 months. Am J Respir Crit Care Med 2000;161:[A585].
8 Wilkens H, Demertzis S, Konig J, Leitnaker CK, Schafers HJ, Sybrecht GW. Lung volume reduction surgery versus conservative treatment in severe emphysema. Eur Respir J 2000;16: 1043–9.

9 Hamacher J, Buchi S, Georgescu CL, Stammberger U, Thurnheer R, Bloch KE, et al. Improved quality of life after lung volume reduction surgery. Eur Respir J 2002;19:54–60.
10 NETT trialists. Patients at high risk of death after lung-volume-reduction surgery. N Engl J Med 2001;345:1075–83.
11 Young J, Fry SA, Hyde C. Lung volume reduction surgery (LVRS) for chronic obstructive pulmonary disease (COPD) with underlying severe emphysema. Thorax 1999;54:779–89.
12 Tutic M, Bloch K, Weder W, Russi EW. Long term results after bilateral lung volume reduction surgery. Am J Respir Crit Care Med 2002;165[8]:A196.
13 Weder W, Thurnheer R, Stammberger U, Burge M, Russi EW, Bloch KE. Radiologic emphysema morphology is associated with outcome after surgical lung volume reduction. Ann Thorac Surg 1997;64:313–9.
14 Thurnheer R, Bingisser R, Stammberger U, Muntwyler J, Zollinger A, Bloch KE, et al. Effect of lung volume reduction surgery on pulmonary hemodynamics in severe pulmonary emphysema. Eur J Cardiothorac Surg 1998;13:253–8.
15 Thurnheer R, Muntwyler J, Stammberger U, Bloch KE, Zollinger A, Weder W, et al. Coronary artery disease in patients undergoing lung volume reduction surgery for emphysema. Chest 1997;112:122–8.
16 Cassina PC, Teschler H, Konietzko N, Theegarten D, Stamatis G. Two-year results after lung volume reduction surgery in alpha-1-antitrypsin deficiency versus smoker's emphysema. Eur Respir J 1998;12:1028–32.
17 Gelb AF, McKenna RJ, Brenner M, Fischel R, Zamel N. Lung function after bilateral lower lobe lung volume reduction surgery for alpha-1-antitrypsin emphysema. Eur Respir J 1999; 14(4):928–933.
18 Tutic M, Bloch K, Lardinois D, Brack T, Russi EW, Weder W. Long term result after lung volume reduction surgery in patienst with alpha-1-antitrypsin deficiency. Society of Thoracic Surgeons 2003. (Abstract submitted).
19 Fessler HE, Permutt S. Lung volume reduction surgery and airflow limitation. Am J Respir Crit Care Med 1998;157(3 Pt 1): 715–22.
20 Bloch KE, Weder W, Boehler A, Zalunardo MP, Russi EW. Successful lung volume reduction surgery in a child with severe airflow obstruction and hyperinflation due to constrictive bronchiolitis. Chest 2002;122:747–50.
21 Gelb AF, McKenna RJ, Jr, Brenner M, Epstein JD, Zamel N. Lung function 5 yr after lung volume reduction surgery for emphysema. Am J Respir Crit Care Med 2001;163:1562–6.
22 Bloch KE, Georgescu CL, Russi EW, Weder W. Gain and subsequent loss of lung function after lung volume reduction surgery in severe emphysema with different morphology. J Thorac Cardiovasc Surg 2002;123:845–54.
23 Meyers BF, Yusen RD, Lefrak SS, Cooper JD. Improved long-term survival seen after lung volume reduction surgery compared to continued medical therapy for emphysema. Ann Thorac Surg 2001;71:2081.
24 Macklem PT. Collateral ventilation. N Engl J Med 78;298: 49–50.
25 Lausberg HF, Chino K, Patterson GA, Meyers BF, Toeniskoetter PD, Cooper JD. Bronchial fenestration improves expiratory flow in emphysematous human lungs. Ann Thorac Surg 2002 (in press).

Swiss Medical Weekly

Official journal of
the Swiss Society of Infectious disease
the Swiss Society of Internal Medicine
the Swiss Respiratory Society

The many reasons why you should choose SMW to publish your research

What Swiss Medical Weekly has to offer:

- SMW's impact factor has been steadily rising, to the current 1.537
- Open access to the publication via the Internet, therefore wide audience and impact
- Rapid listing in Medline
- LinkOut-button from PubMed with link to the full text website http://www.smw.ch (direct link from each SMW record in PubMed)

- No-nonsense submission – you submit a single copy of your manuscript by e-mail attachment
- Peer review based on a broad spectrum of international academic referees
- Assistance of our professional statistician for every article with statistical analyses

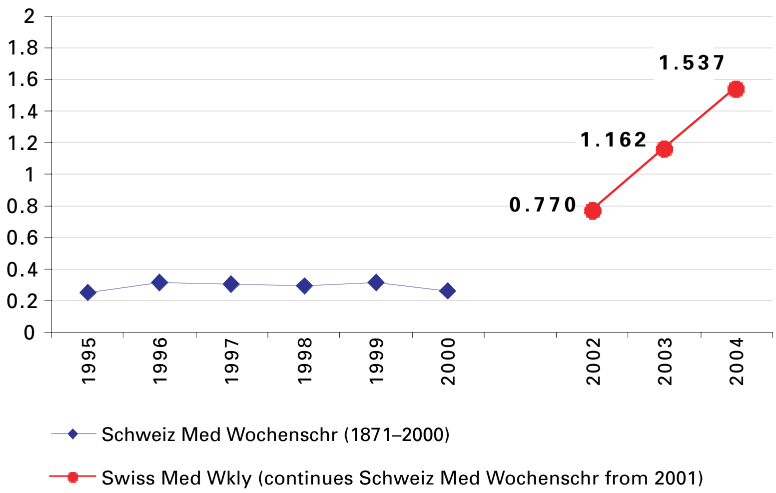
- Fast peer review, by e-mail exchange with the referees
- Prompt decisions based on weekly conferences of the Editorial Board
- Prompt notification on the status of your manuscript by e-mail
- Professional English copy editing
- No page charges and attractive colour offprints at no extra cost

We evaluate manuscripts of broad clinical interest from all specialities, including experimental medicine and clinical investigation.

We look forward to receiving your paper!

Guidelines for authors:
http://www.smw.ch/set_authors.html

Impact factor Swiss Medical Weekly



EMH
FMH Schwabe
Editores Medicorum Helveticorum

All manuscripts should be sent in electronic form, to:

EMH Swiss Medical Publishers Ltd.
SMW Editorial Secretariat
Farnsburgerstrasse 8
CH-4132 Muttenz

Manuscripts:	submission@smw.ch
Letters to the editor:	letters@smw.ch
Editorial Board:	red@smw.ch
Internet:	http://www.smw.ch