Testing for Exercise Limitation in Obstructive Lung Disease

Marc Newton, Katherine A. Webb, Denis E. O'Donnell, Cristina Murariu, Heberto Ghezzo, Joseph Milic-Emili and Henry Gauthier

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Is Minimally Invasive Outpatient Pneumonectomy the Current Standard of Care for Lung Cancer?

To the Editor:

I read with some concern the article by Tovar in a recent issue of CHEST (November 1998).1 The author has demonstrated that a minimally invasive approach to pneumonectomy is feasible, but is it appropriate to advocate this approach for the definitive treatment of lung cancer? I submit that the answer is no—at least, not yet.

It would be important to know what percentage of all patients he treated for lung cancer required pneumonectomy and, of those patients, what proportion were candidates for this approach. In addition, what selection criteria did he use in determining who required a pneumonectomy and who was a candidate for the minimally invasive approach? Three tumors were staged as T2, but the basis for this designation (eg, size > 3 cm, or of smaller size but with visceral pleural invasion) was not elucidated.

How were the patients staged before pneumonectomy? If surgical staging occurred, did any patients receive induction therapy for N2 disease before pneumonectomy? Tovar states that each patient had a mediastinal lymphadenectomy at the same time as the pneumonectomy. How many lymph node stations were analyzed for each patient? What was the extent of disease in each of the lymph node stations? How does this compare with published results following traditional thoracotomy with lymphadenectomy?

Tovar states that the surgeon’s office communicated with the patient daily. How were patients followed for perioperative complications, such as atrial fibrillation and deep venous thrombosis? Were visiting nurses utilized? What analgesia was used and for how long was it required?

Tovar states that a 6- to 8-cm incision decreases the possibility of chest wall implantation of tumor, yet provides us with no long-term evidence to confirm this statement. I suspect that chest wall implantation with tumor would be likely (Tovar’s Fig 2) as long as the specimen is removed from the chest cavity without enclosing it in a protective plastic bag, as is now done when a video-assisted thoracic surgery approach is used.

I was disappointed that no mortality data (30 day, 1 or 2 year) were reported in his series of five patients, three of whom had stage IIIA non-small cell lung cancer.

This approach has not been proven in a randomized controlled trial to be a cancer operation equivalent to traditional thoracotomy. I would strongly caution the public and the medical profession against seeking out minimally invasive surgery for the definitive treatment of lung cancer at this time. A 4- to 5-day hospital stay with a curative resection should appeal more to an individual with lung cancer than a 1-day stay following an unproven minimally invasive procedure given the knowledge that recurrent or persistent disease is rarely cured by remedial surgery or adjuvant therapy. While thoracic surgeons everywhere continue to try to decrease the pain associated with thoracotomy and the length of stay following the procedure by utilizing smaller incisions, thoracic epidural analgesia, clinical pathways, and careful assessment of the impact that these changes have on the quality of the surgical procedure is mandatory.

Jemi Olak, MD, FCCP
Lutheran General Hospital
Park Ridge, IL

Correspondence to: Jemi Olak, MD, FCCP, Lutheran General Hospital, 1700 Luther Lane, Park Ridge, IL 60068; e-mail: jemi.olak@advocatemedical.com

REFERENCE


To the Editor:

“Surgery is not a province with fixed boundaries.”

O. H. Wagensteen

I thank Dr. Olak for her interest and comments regarding my recently published article (November 1998). Access to the chest cavity through the standard posterolateral thoracotomy consists of a large incision in which major thoracic muscles are divided, a rib resected, and one or two other ribs transected to improve visibility. The technique of opening the thorax and performing a pneumonectomy was a fairly straightforward exercise in applied anatomy, perfected by the end of the 19th century when less than 200 cases of lung cancer had been reported. This approach, by itself, has significant complications. In fact, patients who undergo exploration without parenchymal resection do not escape severe complications following an open-and-close procedure (Fig 1). Olak refers to the traditional thoracotomy as a “cancer operation” as though a paramedian incision to perform a radical gastrectomy is less of a cancer operation compared to one performed through a midline incision. There is no scientific evidence, to my knowledge, that a larger incision in itself produces more curative resections than a smaller one. The quality of the resection depends on the intracavitary technique used to resect the cancerous tissue and not the approach to access the chest cavity. It is therefore possible to perform an oncologically sound resection while minimizing collateral damage.
Like most surgeons trained before the advent of laparoscopic cholecystectomy, I was taught the standard posterolateral thoracotomy approach as the method of choice to access the chest cavity. The now anachronistic adage, “big surgeons make big incisions,” was omnipresent in operating suites throughout the country. To decrease morbidity and discomfort to patients in my practice, I made a gradual transition to the oblique muscle-sparing minithoracotomy. Initially, I stopped removing and transecting ribs, then I spared a muscle, and then I adopted the muscle-sparing approach. Throughout this process, I developed new skills that allowed me to perform exactly the same operation that I would have with a wide open thorax.

Olak would like to know what percentage of my patients were candidates for this approach and what criteria I used in determining who required pneumonectomy, as though the criteria should change. Since April 1993, without exception, every patient I have operated on for lung cancer has received an oblique muscle-sparing minithoracotomy. The criterion for lobectomy, bilobectomy, and pneumonectomy has never changed and the type of access has nothing to do with it. The criterion is universal and consists of the removal of the least amount of lung parenchyma necessary to completely clear all malignant tissue and lymphatic spread. There is no justification to jeopardize the quality of the resection in the name of a small incision. If the appropriate skill has not yet been developed, a more redundant approach should be used. As stated by Morgenstern, competence is at the heart of professional ethics and it is always challenged by new technology.

There are two kinds of intracavitary techniques for major lung resections: mass ligation vs individual ligation of hilar structures. The former was virtually abandoned in the early 1940s in favor of the latter. A stapling technique has emerged as a modern version of mass ligation in thoracoscopic procedures. The value of mediastinal lymphadenectomy vs nodal sampling remains controversial. I practice the isolation-ligation of the three hilar elements and find mass ligation to be an aberration. I also believe that systematic mediastinal lymphadenectomy is a sounder cancer operation.

Through a minithoracotomy, the specimen can be removed as easily as with a larger thoracotomy; that is shown in Figure 2 of the article. A nonrib-spreading thoracoscopic resection may lead to deposition of viable clumps of malignant cells during the extraction of the specimen and, therefore, a plastic bag in these cases seems appropriate.

The clinical pathway given in the article is implemented to prevent complications. If patients are active immediately after surgery, the possibility of deep vein thrombosis and many other common complications is nonexistent. Some of our patients did not require any analgesia, despite early activity, and others required only oral pain medication.

There were no deaths at 30 days, one death at 8 months due to distant recurrence, and one patient with metastatic disease from the appendix, who presented with brain metastasis 13 months after surgery and died 3 months later. The other three patients remain alive and cancer-free at 21, 23, and 24 months of follow-up, despite the advanced disease stage. None of the five patients has had local recurrence.

Olak would like me to present numbers, sizes, and percentages as though I could statistically prove that the access should be used. She forgets that the paper is about “can do,” not “should do,” as I clearly stated in the article.

Finally, I would like to close with the words of A. J. Walt: “If the objective of surgery is to achieve cure or palliation with as little discomfort to the patient as possible, conceptual transformations are inevitable. Genuine recognition of the tyranny of mind-sets that have inflexible and nonporous walls is essential to productive change. Paradigm-lepsis is dangerous but not rare.”

Eduardo A. Tovar, MD, FCCP
University of California
Orange, CA

Correspondence to: Eduardo A. Tovar, MD, FCCP, 100 E. Valencia Mesa Dr., Suite 301, Fullerton, CA 92835; e-mail: etovarmd@aol.com

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Pulmonary Embolism—Treatment vs Nontreatment

To the Editor:

We appreciated the excellent article by Miller and colleagues describing objective measurements of right ventricular (RV) function in hemodynamically stable patients with acute pulmonary embolism [PE] [March 1998]. Although there was demonstrable decrement in RV function (ejection fraction, end-systolic area), their findings reassure us that routine echocardiography is not necessary in these patients, as early quantification did not help in determining which patients were at risk for clinical deterioration, including recurrent PE.

The authors’ claim, however, that an objective decrement in RV function persists after 6 weeks “despite treatment” may be open to debate. Abnormal RV function is reported as persisting with a fairly high degree of standard deviation compared with that of the authors’ normal controls. Although they initially evaluated 64 patients, 3 of these died and another 35 could not be reevaluated, leaving only 26 patients who could be followed sequentially. Perhaps most important is the potential confounding variable of nontreatment. Although only 2 of the 26 patients who were followed up were not treated with anticoagulation, and are reported as not differing significantly from those who were, we wonder why they were included in the analysis at all.

Vena caval interruption alone should not be considered as treatment for PE. This stopgap measure merely helps protect patients from further hemodynamic compromise when their thromboembolic disease cannot be treated, patients from further hemodynamic compromise when their treatment for PE. This stopgap measure merely helps protect all.

To the Editor:

I appreciate the comments of Drs. Rascona and Gumbert on our article assessing right ventricular function in hemodynamically stable patients with pulmonary embolism. I agree that our analysis suffers from the shortcoming of relatively high drop-out rate, making strong conclusions about the extent and prevalence of persistent right ventricular abnormalities during treatment difficult, despite the presence of statistical significance. The inclusion or exclusion of the two patients treated with inferior vena cava interruption alone did not affect the statistical analysis of our results. To answer the last question, all patients who suffered adverse events were receiving anticoagulation with heparin alone.

Rachel L. Miller, MD

Columbia University College of Physicians and Surgeons
New York, NY

Correspondence to: Rachel L. Miller, MD, Department of Medicine, Columbia University College of Physicians and Surgeons, PH 8 Center, 630 W 168th St., New York, NY 10032; e-mail: rlm14@columbia.edu

Testing for Exercise Limitation in Obstructive Lung Disease

To the Editor:

In their recent article, Murariu and colleagues (October 1998) report that exercise performance in patients with obstructive lung disease is likely linked to the degree of resting lung hyperinflation as indirectly measured by inspiratory capacity (IC). Certainly, there is increasing evidence that dynamic lung hyperinflation during exercise (as assessed by serial IC measurements) contributes to the intensity and quality of dyspnea, and thus exercise limitation in COPD. Furthermore, increases in dynamic IC have been shown to correlate well with improvement in dyspnea and exercise endurance following pharmacologic and surgical volume reduction. The study by Murariu and colleagues is, however, the first to claim an association between the resting IC and exercise performance in patients with obstructive lung disease (COPD and asthma).

The authors report a correlation coefficient of 0.81 (p < 0.001) between maximal exercise power output (Wmax) and IC in 25 patients with mild COPD (FEV1, 0.68 ± 21) who had, on average, moderate exertional dyspnea (Borg 4 ± 1) and exercise curtailment Wmax (62 ± 29% predicted). There is, however, a significant con-
founding factor here that the authors did not correct for: the absolute $W_{\text{max}}$ and the IC would be expected to be linearly related, even in normal subjects. The predicted IC and $W_{\text{max}}$ are both functions of age, sex, and height. Simply stated, taller, younger male subjects would be expected to have high $W_{\text{max}}$ and IC, while shorter, older female subjects would have lower $W_{\text{max}}$ and IC. In fact, predicted $W_{\text{max}}$ is a linear equation of age, sex, and height. This explains the significant correlation between height and $W_{\text{max}}$ as reported by Murariu and colleagues (correlation coefficient, 0.61; $p < 0.005$).

To establish a causal relationship between reduced IC and diminished $W_{\text{max}}$, both IC and $W_{\text{max}}$ should be normalized and expressed as percent predicted. In their article, Murariu and coworkers provided us with the respiratory function and $W_{\text{max}}$ data (reported as percent predicted) for all study subjects. When viewed in this way, the relationship is not as strong (Fig 1). In fact, the correlation coefficient is only 0.294 ($p = 0.154$).

In summary, it is not likely that a particular patient’s exercise limitation would be strongly predicted by a single static pulmonary function test. That is not to say that lung hyperinflation, and particularly dynamic hyperinflation during exercise, is not important in dyspnea causation and exercise limitation in many patients with COPD. However, spirometric testing does not obviate the need for direct cardiopulmonary exercise testing, which remains the best and only way to accurately evaluate exercise capacity.

Marc Newton, MD
Katherine A. Webb, MSc
Denis E. O'Donnell, MD, FCCP
Queen’s University
Kingston, ON, Canada

Correspondence to: Marc Newton, MD, Division of Respiratory and Critical Care Medicine, Queen’s University, 102 Stuart St, Kingston, ON, Canada; e-mail: fmnewton@istar.ca

REFERENCES

To the Editor:

In normal subjects, there is a close association between maximal exercise power output ($W_{\text{max}}$) and vital capacity, the variation coefficient ($r^2$) amounting to 0.79. Nevertheless, in our patients with obstructive lung disease (OLD), the variation coefficient of $W_{\text{max}}$ to FVC was substantially weaker than that of inspiratory capacity (IC) (0.41 vs 0.66). Furthermore, in 21 patients with restrictive lung disease (RLD) who were included in our original manuscript, there was no significant correlation of $W_{\text{max}}$ to IC, though their exertional dyspnea and exercise curtailment were similar to those of the OLD patients. Thus, in OLD but not RLD patients, the resting IC is a better predictor of $W_{\text{max}}$ than either FVC or FEV1.

Although we argue that there is predictably a close association between $W_{\text{max}}$ and IC in OLD patients because of expiratory flow limitation, we do not state that “a particular patient’s exercise limitation can be strongly predicted by IC.” In fact, the $r^2$ between $W_{\text{max}}$ and IC was only 0.66, reflecting substantial scatter of the data.

Prediction of $W_{\text{max}}$ and IC is a nettlesome problem in old individuals, such as our OLD population. In fact, there are many predictive equations for $W_{\text{max}}$, each giving a different value. Furthermore, the predicted normal values of IC are obtained as the difference between predicted total lung capacity and FRC. Thus, assessment of the relationship of $W_{\text{max}}$ (% predicted) to IC (% predicted) may be problematic, particularly in small numbers of patients with moderate exercise limitation. In 34 OLD patients with a more marked decrease in $W_{\text{max}}$ (39% predicted on average), a significant correlation was found both between the absolute and the predicted values of $W_{\text{max}}$ and IC, with IC being the only significant contributor to $W_{\text{max}}$ (O. Díaz Patiño, MD, C. Lisboa, MD, J. Milic-Emili, MD; unpublished observations, July 1998).

In conclusion, we think that resting IC, the Cinderella of lung function testing, not only provides useful information about the effects of bronchodilators and surgical treatment on hyperinflation in OLD patients, but it also gives a useful estimate of a patient’s exercise capacity. If the actual $W_{\text{max}}$ in a given OLD patient is lower than that predicted by our regression equation, it is likely that the patient should benefit substantially by exercise rehabilitation because the exercise performance is probably mostly limited by mechanisms other than lung function impairment.

Cristina Murariu, MD
Heberto Ghezzo, PhD
Joseph Milic-Emili, MD
Henry Gauthier, MD
Meakins-Christie Laboratories
McGill University
Montreal, QC, Canada

Correspondence to: Joseph Milic-Emili, MD, Meakins Christie Laboratories, McGill University, 3626 St. Urbain Street, Montreal QC H2X 2P2, Canada; e-mail: milic@meakins.lan.mcgill.ca

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Figure 1. Relationship between IC (percent predicted) and $W_{\text{max}}$ (percent predicted) in 25 patients with obstructive airways disease, compiled from data provided by Murariu et al.1
Flutter Flap

To the Editor:

Homnick et al (October 1998) conclude that “...the flutter [device] appears to be safe, efficacious, and cost effective for [cystic fibrosis] inpatients capable of undertaking this type of therapy.” Unfortunately, data inconsistencies, omissions, and inadequate power preclude support of these conclusions.

Fifteen subjects were admitted one time (15 hospitalizations) and 7 subjects were admitted more than one time (22 hospitalizations). This results in a total of 37 hospitalizations. Only 33 hospitalizations were included for analysis. Why were four (11%) of the hospitalizations excluded from analysis, and which groups were they excluded from?

Seven (32%) of their subjects provided 22 of the hospitalizations analyzed (59 to 67%, depending on whether the hospitalizations excluded from analysis come from the group of one-time admissions or multiple admissions). Did the subjects admitted multiple times alternate between the flutter device and manual chest physiotherapy (CPT), or did they receive the same treatment each time they were admitted? This weighting of data could have a significant impact on results.

Regardless of the power analysis used, it is clear that the sample size presented in this article falls far short of sufficient. Therefore, while this article is an interesting preliminary data acquisition abstract, it does not begin to support conclusions about relative efficacy, and should not be interpreted as justification to replace standard CPT with the flutter device in the hospital or at home.

Martin L. Bauer, MD, FCCP
Children’s Mercy Hospital
Kansas City, MO

Correspondence to: Martin L. Bauer, MD, FCCP, Section of Pulmonology, Children’s Mercy Hospital, 2401 Gillham Rd, Kansas City, MO 64108

REFERENCES

To the Editor:

We appreciate Dr. Bauer’s comments about our study comparing chest physiotherapy (CPT) and the flutter device in hospitalized patients with cystic fibrosis. We agree with Dr. Bauer’s observation that our study was underpowered. We clearly stated that this was a preliminary (pilot) study and our power analysis demonstrated that much larger subject numbers would be necessary to make definitive conclusions.

We disagree with the assertion that our study had “data inconsistencies” and “omissions.” Dr. Bauer misinterpreted our study design and data analysis when he stated that we excluded some hospitalizations. We had 33 hospitalizations, not 37, and none were excluded from analysis. Fifteen subjects had only one hospitalization. Seven subjects had two or more admissions, representing 18 hospitalizations, not 22.

The subgroup analysis included the 15 one-time admission subjects and the first admission of the 7 multiple-admission subjects, resulting in a total of 22 hospitalizations. Analysis of this subgroup compared to the 33-hospitalizations group demonstrated no differences.

Assignment to flutter device or CPT was alternated for each hospitalization, regardless of the previous therapy received by multiple-admission subjects. We did not analyze whether the seven subjects received the same therapy on subsequent admissions.

We observed and stated that “the flutter [device] appears to be safe, efficacious, and cost effective” in our study. We, of course, would not recommend that any airway clearance technique or device be routinely substituted for another until proven effective in clinical trials with adequate statistical power.

John H. Marks, MD, FCCP
Douglas N. Homnick, MD, FCCP
Michigan State University
Kalamazoo Center for Medical Studies
Kalamazoo, MI

Correspondence to: Douglas N. Homnick, MD, FCCP, Division of Pediatric Pulmonology, MSU, Kalamazoo Center for Medical Studies, 1000 Oakland Dr, Kalamazoo, MI 49008; e-mail: homnick@kcms.msu.edu

REFERENCE

Tuberculin Skin Testing

To the Editor:

We read with interest the article by Kendig et al (May 1998) and the subsequent correspondence by Dr. Scott (October 1998).

Interpreting the reaction to tuberculin protein administered by intradermal injection (Mantoux test) has several problems. There is variation in reading between health-care professionals and the general public, difficulty in ensuring correct placement, and uncertainty in the concept of sequentially greater concentrations.

In the United Kingdom, the multiple-puncture (Heaf) test is most commonly used. A standardized dose and depth of injection is provided when a fixed head apparatus is used. Selby and colleagues examined how well the general public read the Heaf test. Using a self-read card, 550 patients undergoing tuberculin skin testing were found to grade the reaction as accurately as a trained respiratory nurse. This contrasts sharply with the Mantoux test, where poor reproducibility has been found with medical and nonmedical health professionals, as well as with the general public.
Sarcoidosis Is a Significant Cause of Bullous Emphysema

To the Editor:

In a recent issue of CHEST (November 1998), Judson and Strange1 rationally discussed that sarcoidosis caused bullous emphysema through the three cases of pulmonary sarcoidosis. Although bullae may occur in subjects with otherwise healthy lungs, most bullae develop in smokers with centrilobular emphysema. Thus, cigarette smoking is considered to be an utmost risk factor in the development of bullous emphysema. However, the mechanism and cause of bullous emphysema have not fully been elucidated. Therefore, the current comprehensive report is important.

 Giant bullae can occur in patients with sarcoidosis and advanced stages of the disease, and appear to be bullous emphysema or vanishing lung syndrome.2–4 Although emphysema increases in frequency with age and is found most frequently in patients in the seventh decade, bullous emphysema is predominantly found in young patients. Further, the surgical procedures, including lung volume reduction surgery, are particularly beneficial for those who have bullous emphysema as well as bullous sarcoidosis.5–7 Sarcoidosis should be carefully considered in the differential diagnosis of bullous emphysema in young patients with chronic airflow limitation (CAL), in addition to the other hereditary disorders, including α1-antitrypsin deficiency, Fabry’s disease, Salla disease, cutis laxa, and Ehlers-Danlos syndrome.8 There is no doubt that sarcoidosis is a cause of bullous emphysema in young patients with CAL; however, the pathogenesis of large bullae in patients with sarcoidosis remains to be determined.

Interestingly, bullous emphysema can be found in dogs, cats, and pups. Hoover and colleagues9 suggested that congenital weakness of the bronchial cartilage and lung torsion might be involved in the development of bullous emphysema in the pup. This may, in part, support the authors’ hypothesis that endobronchial lesions of sarcoidosis in bronchi and bronchioles cause remodeling of the bronchial trees and airways. Further studies and reports are needed to clarify the pathology and etiology of bullous sarcoidosis.

Shinji Teramoto, MD, FCCP
Yasuyoshi Ouchi, MD
Tokyo University Hospital
Tokyo, Japan

Correspondence to: Shinji Teramoto, MD, FCCP, Department of Geriatric Medicine, Tokyo University Hospital, 7-3-1 Hongo Bunkyo-ku, Tokyo, Japan 113-8655

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Catheter Fragmentation of Pulmonary Emboli

To the Editor:

We read with interest Dr. Goldhaber’s editorial on catheter thrombectomy for treating severe pulmonary embolism in the November issue of CHEST. However, we do not share his enthusiasm. We do not believe that mechanical fragmentation of pulmonary emboli, as described in the careful and detailed report by Schmitz-Rode and colleagues in the same CHEST issue, has been shown to provide any clinical benefit to patients with severe pulmonary embolism. In that study, like virtually all published series on the same subject, most patients, if not all (8 of 10 patients in the report from Schmitz-Rode and colleagues) received medical thrombolysis together with mechanical fragmentation, so that at the very least, and as was acknowledged by the authors, the respective efficacy of each technique in the observed results cannot be assessed. Several investigators have demonstrated the dramatic and rapid (within 60 min) improvement of mean pulmonary artery pressure and cardiac index, resulting in a highly significant decrease in pulmonary vascular resistance after a simple bolus IV infusion of urokinase or alteplase in patients with severe acute pulmonary embolism.1–3

Even from a theoretical point of view, the fragmentation of a sphere of 1 cm³ into 1,000 spheres of 1 mm³ would transform a cross-sectional obstruction of 1 cm² into a cross-sectional obstruction of 10 cm², i.e., a proximal occlusion into a more distal but larger occlusion, with likely similar, if not worse, hemodynamic consequences. The supposed improvement in the efficacy of medical thrombolysis when applied to smaller clots remains to be demonstrated.

Thus, convincing arguments, either clinical or theoretical, to support the view that mechanical fragmentation of pulmonary emboli provides any clinical benefit over mere anticoagulant treatment and/or medical thrombolysis are lacking. In view of its cost and possible complications, including hemorrhagic complications of vascular access in patients who may receive thrombolytic agents, the addition of this aggressive technique should not be recommended, and catheter fragmentation of pulmonary emboli should not “be integrated into our armamentarium to treat acute pulmonary embolism” until careful clinical trials have clearly demonstrated its clinical efficacy.

Philippe Girard, MD, FCCP
Institut Mutualiste Montsouris
Paris, France
Gérald Simonneau, MD
Hôpital Antoine Béclère
Clamart, France

Correspondence to: Philippe Girard, MD, FCCP, Département Thoracique, Institut Mutualiste Montsouris, Choisy 6, Place de Port au Prince, 75013 Paris, France; e-mail: pgirard@imm.fr

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Prevention of Air Leaks After Lung Surgery

To the Editor:

We read with interest the paper from Abollhoda and colleagues (June 1998). The authors must be commended for reporting on a common and potentially serious complication. Air leakage after major pulmonary resections is a well-known problem which occurs more frequently when interlobar fissures are incomplete or absent, and if the pulmonary resection is performed in older patients with emphysema. A number of reports in the literature corroborate the hypothesis that the ideal treatment begins with prevention; in fact, when the fissures are incomplete, meticulous attention should be given to anatomic planes of interlobar dissection and staplers should be used. However, notwithstanding these measures, air leaks may still occur, compromise lung reexpansion, prolong hospitalization, and lead to the onset of other complications.

The resurgence of interest in lung volume reduction surgery in patients with COPD has been accompanied by a recognition of the importance of preventing postoperative air leaks. Various materials have been used to buttress the staple line and the use of this reinforcement technique is generally accepted as an effective means of prevention in this subset of patients. Patients receiving pulmonary resections generally present other problems; however, in our opinion, incomplete fissures should be approached using all the skills learned from lung volume reduction surgery.

Abollhoda and colleagues stated that this group of patients does not present with the typical pulmonary pathologic changes found in end-stage emphysema and, thus, the routine use of such staple-reinforcing techniques is neither cost-effective nor justified. There is indeed evidence in the literature that these measures can improve the postoperative course in this group of patients. We would like to draw attention to some experimental and clinical studies recently published. Robertson and colleagues have demonstrated in a canine model that the use of reinforcing materials to buttress the staple line can help in preventing postoperative air leaks in normal lungs. In a prospective clinical randomized study, we have compared different techniques to complete interlobar fissures during pulmonary lobectomies. The use of GIA staplers (Ethicon, Inc; Somerville, NJ) and pericardial sleeves was compared to that of TA-55 staplers and the “old-fashioned” silk, crile, and cautery. Staplers and bovine pericardium sleeves significantly reduce the duration of postoperative air leaks and hospital stay. No complications were associated with the use of this technique. Only a small number of patients could be enrolled in our prospective study, but the results were statistically significant in favor of the use of bovine pericardium buttressing.

We would also like to stress the importance of creating a pleural tent after upper lobectomy and lung volume reduction surgery. Creation of the pleural tent is also technically feasible using thoracoscopy, this dynamic and reversible tailoring of the postresectional pleural space may contribute to improving the outcome of patients receiving a partial or complete resection of the upper lobes.

One can also inject air within the peritoneal cavity (pneumoperitoneum) to elevate the diaphragm and reduce postresectional pleural spaces, pulling the residual lung towards the chest wall.
This technique has recently gained new acceptance and has been described after standard pulmonary resections and lung volume reduction surgery.\textsuperscript{5,6}

In conclusion, the surgical procedure and the measures adopted to prevent, reduce, and treat postoperative air leaks must be tailored patient by patient. The use of staplers and buttressing to create interlobar fissures must be encouraged. Additional measures, like the creation of a pleural tent and pneumoperitoneum, should be taken into consideration.

Federico Venuta, MD
Erino A. Rendina, MD
Tiziano De Giacomo, MD
Giorgio F. Coloni, MD, FCCP
University of Rome
Rome, Italy

Correspondence to: Federico Venuta, MD, Cattedra di Chirurgia Toracica, Policlinico Umberto I, University of Rome “La Sapienza,” V.le del Policlinico, 00100 Rome, Italy; e-mail: Fevenuta@tin.it

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Erratum
Testing for Exercise Limitation in Obstructive Lung Disease
Marc Newton, Katherine A. Webb, Denis E. O'Donnell, Cristina Murariu, Heberto Ghezzo, Joseph Milic-Emili and Henry Gauthier

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