Effects of lobectomy on lung function

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ABSTRACT The effect of lobectomy on lung function was investigated in 16 patients in whom comprehensive lung function tests were performed before and between six weeks and six months after lobectomy. The operation was well tolerated even by patients with quite severe airways obstruction. The effect of lobectomy on forced flow rates was in keeping with the change in lung volumes. There was no alteration in the distribution of ventilation and the diffusion capacity remained unchanged. The changes in the pressure volume curves were difficult to explain. All patients, even those with nearly “complete” hyperinflation of the remaining lung, had appreciable increases in maximal intrapleural pressure at full inspiration.

The effects of pneumonectomy and segmental lung resection on lung function have been extensively investigated. However, reports of the lung function effects of lobectomy in adults are relatively few and there are no reports of detailed mechanical lung function before and after lobectomy. We performed comprehensive lung function tests before and after lobectomy in 16 patients with varying degrees of airflow obstruction in order to document the changes in lung function.

Patients and methods

Sixteen patients presenting with small, peripheral lung lesions were selected for this study. The patients had screening tests consisting of a questionnaire, physical examination, chest radiograph, electrocardiograph, biochemical screen, and full blood count in order to exclude the presence of any disease other than chronic bronchitis or emphysema which may have resulted in lung function differences between the initial and repeat study—for example, asthma, renal failure, cardiac failure, collagen diseases, and so on. All patients were current or ex-smokers so that the presence of chronic bronchitis and emphysema in some or all was expected. All patients underwent fibreoptic bronchoscopy and in no case was an endobronchial lesion seen more proximally than the segmental level. All lesions were primary or secondary lung tumours. Patients with chronic bronchitis received physiotherapy so that at the time of the studies, their lung function was optimal.

Informed consent was obtained. The patients were studied one week before and between six weeks and six months after lobectomy. The age, sex, smoking history, grades of dyspnoea, time interval between lung function tests, lobe resected, and grade of emphysema are shown in table 1.

The lung function tests were performed by identical methods and equipment before and after lobectomy.

Lung function studies were performed in a sitting position. The forced expiratory volume in one second (FEV₁), vital capacity (VC), FEV₁/VC ratio, and maximal mid-expiratory flow rate (MMEFR) were performed with a nine litre Godart respirometer and corrected to body temperature and pressure, saturated with water vapour (BTPS). The lung volume subdivisions of residual volume (RV), functional residual capacity (FRC) and total lung capacity (TLC) were determined by the helium dilution method.¹

The maximal expiratory flow volume (MEFV) curves with air and an 80% helium–20% oxygen mixture (He–O₂) were performed with subjects seated in an Emerson volume displacement plethysmograph. Volume was measured with an attached water-filled wedge spirometer and flow was measured with a Fleisch pneumotachograph coupled to a Hewlett-Packard (HP) Model 270 pressure transducer. The curves were displayed

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on a storage oscilloscope and photographed. The patients performed MEFV manoeuvres while breathing air until reproducible results were obtained. The patients then took at least three VC inspirations of the He–O₂ mixture and produced further MEFV curves (the pneumotachograph calibration was corrected for the different gas mixture) until reproducible results were obtained. The values for VC while breathing air and He–O₂ were required to be within 5% of one another. The curves were then superimposed by tracing and matched at RV. The maximal flow at 50% VC (V₅₀) and the percentage increase in flow at 50% VC breathing the He–O₂ mixture (∆V₅₀) were then measured.

Total lung resistance (Rₐ) was determined by the method of Mead and Whittenberger, difference between mouth and oesophageal pressure being measured with an HP 267B differential pressure transducer. Balloon technique was as described by Milic-Emili et al.

Expiratory pressure–volume curves were produced by the method of volume interruption. Points obtained from four or five passive deflations from TLC to FRC were pooled and the curves of best fit drawn by eye. From the pressure–volume curve, lung compliance (Cₐ) was measured as the mean slope of the curve from FRC plus 700 mls and the maximal intrapleural pressure at full inspiration (Pmax) documented.

The single breath nitrogen test (SBNT) was performed by a standard method. The diffusing capacity for carbon monoxide (TLCOss) was measured by the single breath method. The KCO (TLCOss/alveolar volume) was calculated using the alveolar volume obtained during the single breath test. Steady state gas exchange was performed by the method of Filley et al and the steady state diffusing capacity (TLCOss) calculated.

Blood gas analysis was carried out using a radiometer PMS 3 Mark II system. Carbon monoxide concentration was measured using a Beckman infrared CO analyser and carbon dioxide with a Beckman LB-1 analyser.

Not all the patients had all the tests. In a few instances, informed consent for oesophageal intubation could not be obtained. Some patients presented before the introduction in this laboratory of MEFV curves using the He–O₂ mixture. Because of technical difficulties, diffusion studies could not be performed in some cases.

After resection, the lobes were inflated with 10% formalin at a constant pressure of 25 cmH₂O for a period of 48 hours. The lobes were then cut into parasaggital slices of 1-0 cm thickness. These were numbered in order from the lateral pleural surface. Slide 3 in each case was impregnated with barium sulphate and used for assessment of emphysema by comparison with standard emphysema reference pictures which graded the degree of emphysema arbitrarily from zero to 100.

Results

The results of the lung function studies are shown in tables 2–4. The operation was well tolerated by all the patients and the dyspnoea grade changed in only two patients (table 1).
### Table 2  Results of lung function tests before and after lobectomy—spirometry, lung volumes, pressure–volume curves

<table>
<thead>
<tr>
<th>Patient</th>
<th>FEV₁(l)</th>
<th>VC(l)</th>
<th>FRC(l)</th>
<th>RV(l)</th>
<th>TLC(l)</th>
<th>CV(l)cmH₂O</th>
<th>Pmax(cmH₂O)</th>
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Mean 2:5 2:2† 4:1 3:7† 4:2 3:7† 2:7 2:3† 7:1 6:2; 0:42 0:31* 23 36‡

SD 0:7 0:6 0:7 0:7 1:0 0:8 0:8 0:7 1:1 0:9 0:16 0:10 8 9

FEV₁ = forced expiratory volume in one second
VC = vital capacity
FRC = functional residual capacity
RV = residual volume
Cᵥ = lung compliance
Pmax = maximum intrapleural pressure at full inflation

*p < 0.05; †p < 0.01; ‡p < 0.001 compared to pre-lobectomy values, paired t test.

### Table 3  Results of lung function tests before and after lobectomy—lung resistance, maximal expiratory flow-volume curve and single breath nitrogen test

<table>
<thead>
<tr>
<th>Patient</th>
<th>RL(cmH₂O/l/s)</th>
<th>V̅ₐₒ(l/s)</th>
<th>ΔV̅ₐₒ(%)</th>
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<th>CV%VC</th>
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Mean 3:5 3:8NS 1:9 1:5‡ 17 35‡ 2:8 2:7NS 27 27NS

SD 1:4 1:5 0:8 0:7 11 15 1:4 1:4 9 7

RL = lung resistance
V̅ₐₒ = maximal flow at 50% vital capacity (VC)
ΔV̅ₐₒ = percentage increase in flow at 50% VC breathing a helium-oxygen mixture
%N₂/l = slope of phase 3 of the single breath nitrogen test
CV%VC = closing volume as percentage of VC
NS = not significant

For statistical notes see table 2.
Table 4 Results of diffusion studies on the 16 patients before and after lobectomy

<table>
<thead>
<tr>
<th>Patient</th>
<th>Pao₂</th>
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Pao₂ arterial oxygen tension in mmHg
FCO fractional uptake for carbon monoxide (CO) in percentage
TLCOₓSB = diffusing capacity for CO, steady state method, in ml/min/mmHg
KCO = TLCOₓSB/TLCOSBKCO, KCO, or fractional uptake for carbon monoxide (FCO). Although there was a greater reduction in TLCOSB, this was also not significant. The mean Pao₂ remained at 75 mmHg pre- and post-lobectomy.

Discussion

There are several potential factors which might affect the results of the repeated lung function studies apart from the loss of lung tissue itself. If insufficient time has elapsed for loss of pain, this interferes with forced manoeuvres. In this study, the time interval after lobectomy was not important in the findings since it has been shown that the residual effects of thoracotomy are no longer present six weeks after surgery. Many of the patients either stopped or reduced their cigarette smoking after surgery. It is possible that some improvement in lung function, particularly small airway function, resulted from this.
Finally, as pointed out by Frank et al\textsuperscript{12} and McIlroy and Bates,\textsuperscript{13} the measurement of intrapulmonary pressure may not accurately reflect pleural pressure changes in patients after pneumonectomy, though this is less likely to be a problem after lobectomy.

This study showed that lobectomy was very well tolerated by patients even with moderately severe lung disease. Only two patients admitted to worsening respiratory symptoms after surgery. These were patients 10 and 16 who changed their dyspnoea grade from 0 to 1, and from 1 to 2, respectively. Patient 5 (FEV\textsubscript{1}/VC\%=30\%) and patient 11 (FEV\textsubscript{1}/VC\%=41\%) were the patients with the most severe airways obstruction and were considered borderline risks before operation. However, neither showed deterioration of lung function after operation.

The changes in lung volumes were of the same order of magnitude as have previously been reported for segmental resection\textsuperscript{14} and lobectomy.\textsuperscript{15} The mean changes in flow rates can be explained entirely by changes in lung volume. However, it is of interest that five patients showed decreased RV after surgery. This could not be related to the degree of hyperinflation of the remaining lung, upper or lower lobectomy, or the presence or absence of emphysema. It may be related to smoking cessation or surgical removal of diseased airways.

The pressure-volume curve reflects the overall elastic properties of the lung. There are several mechanisms by which the pressure-volume curve may be changed after lobectomy. Firstly, disease may not be equally distributed throughout the lung—for example, in centrilobular emphysema—and removal of an upper or lower lobe may influence the resultant overall elastic properties. Secondly, the pressure-volume curve may be affected by the degree of hyperinflation of the remaining lung. If no hyperinflation occurs, the post-lobectomy values for TLC and C\textsubscript{L} are expected to decrease and Pmax will increase. The C\textsubscript{L} measurement of the remaining lung depends on the position of FRC, governed by the balance of lung and chest wall elastic recoil, on the post-lobectomy pressure-volume curve. Thirdly, a variable amount of pleural and extrapleural fibrosis may result from the surgery.

The patients studied by McIlroy and Bates\textsuperscript{13} after pneumonectomy generally did not increase TLC to predicted normal levels and showed reduced C\textsubscript{L} and increased Pmax. However, one of their patients did reach a normal TLC (although preoperative data were not available). This patient also showed reduced C\textsubscript{L} and a high Pmax. None of the patients studied by Frank et al\textsuperscript{12} after segmental resection reached their preoperative TLC, and the reduction in C\textsubscript{L} observed was correlated with the number of segments resected.

The patients in the present study demonstrated a significant decrease in C\textsubscript{L} although, when this was corrected for change in FRC (specific compliance), no difference was apparent. An increase in Pmax was observed in all the patients and the magnitude of the increase did not correlate with the change in TLC. It is unlikely that all the patients with small changes in TLC increased their inspiratory effort on retesting. It appears that even small reductions in lung volume can give rise to quite large increases in Pmax, although a relationship between reduction in lung volume and increase in Pmax would be expected. Therefore, although the direction of change in Pmax was expected, the lack of relationship to changes in lung volume may be related to the complex interaction of the factors previously discussed.

The lack of changes of the slopes of phase 3 of the SBNT after surgery indicated little disturbance to the distribution of ventilation by the procedure and this is in line with the findings of Miller et al\textsuperscript{14} and Anthonisen et al\textsuperscript{15} after pneumonectomy.

It is of interest that all but one of the patients increased the \(\Delta V_{50}\) and the only patient who failed to do so was the patient with the largest decline in lung volumes after lobectomy. Perhaps hyperinflation of the lungs produced some fall in peripheral resistance. Klingele and Staub\textsuperscript{17} showed in cats' lungs that bronchiolar diameter increased with lung inflation, so that the relationship between central and peripheral resistance may be altered. Reduction or cessation of smoking and clearance of mucus may also have been factors in the increase in \(\Delta V_{50}\).

There was no change in the mean CV\%/VC before and after surgery but there was considerable individual variation and the results do not parallel those of the \(\Delta V_{50}\). The changes in CV\%/VC were not related to whether upper or lower lobectomy had been carried out or to the presence or absence of emphysema.

It had been shown previously by Dietiker et al\textsuperscript{18} that the TL\textsubscript{CO} falls immediately after pneumonectomy and lobectomy and that it rises again with time up to about 20 weeks after the operation, although still falling short of the preoperative value. These authors found that, after pneumonectomy, the loss of TL\textsubscript{CO} was commensurate with the reduction in lung volume whereas after lobectomy, the loss of TL\textsubscript{CO} was greater than ex-
plained by the reduction in lung volume. An even
greater reduction in TLCO was seen after seg-
mental resection. This explained this on the basis
of operative interference to the blood supply of
the remaining lung. Gimeno et al. on the other
hand, found that TLCO 20 years after pneumon-
ectomy was relatively greater than expected for
the loss of lung volume. In the present series there
was no significant change in any of the diffusion
measurements. This probably means that when
the residual lungs hyperinflate the increase in their
alveolar capillary membrane area, in addition to
the increased blood flow, is able to compensate
for the loss of about 25% of the lung tissue. How-
ever, more even distribution of ventilation and
perfusion after the removal of a diseased area of
lung is another possible explanation.

This study has shown that lobectomy is very
well tolerated even by patients with severe degrees
of airway obstruction and that remarkably little
change in lung function results from this pro-
cedure. Most of the changes in lung function
after lobectomy can be explained simply on the
basis of changes in lung volume.

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partment of Veterans’ Affairs, Australia.

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